C O N T E N T S

SECRETARIAT
S. B. GUPTA
HON. GENERAL SECRETARY

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

Editorial

Dear Colleagues,

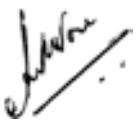
Greetings for 2008 to the members of the IJE ! We present to you an interesting and informative compilation this time.

Sudden death in India has hardly received any attention when it comes to scientific data. We do know however that there is an increasing incidence of cardiovascular disease in India. In 2020, 50% of the deaths are estimated to be due to coronary artery disease! This staggering figure would also mean a large number of sudden deaths. In this context, the article by Dr. Panicker and colleagues is an eye-opening compilation of currently available data and projected estimates from India/Asia, compared to the developed West.

ECG artifacts have numerous sources and may go unrecognized. Dr. Nathani and colleague have put together a comprehensive article with their own experiences in this regard. They have also suggested ways to avoid these pitfalls.

Two interesting case reports are followed by an ECG Quiz, as always. Some of these ECGs were discussed at the last ISECON 2007 in Hyderabad. This conference was a grand success, for which kudos are due to Dr. Narasimhan and his dedicated team.

Yash Lokhandwala
Editor



Amit Vora
Editor

From Hon. Secretary's Desk

Dear Members,

Indian Society of Electrocardiology has become a name for academics. I am thankful to all of you for the suggestions and the support.

Dr C Narasimhan and their team organized ISECON-2007 at Hyderabad on 24th and 25th February 2007. It was indeed a great scientific feast. Many Mid-term and ISECONs have been organized. For the first time, from the organizers of different ISECONs, Dr C Narasimhan contributed Rs. 1.5 Lacs to Indian Society of Electrocardiology from the savings of ISECON 2007. ISE is highly thankful and we wish the same will be followed by the other organizers too. The amount such received will be utilized for the academic activities of ISE, sponsorship to candidates for higher learning and in developing guidelines and registry data of ISE.

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

a. A seminar on "Global Approaches to Cardiac Safety" at Mumbai in collaboration with Quintiles on 25th and 26th November 2006
b. Euro-India Satellite Symposium on "Atrial Fibrillation" at Goa on 16th February 2007.
c. "ECG Learning Course" for postgraduate students at Mumbai on 21st and 22nd July 2007. About 100 students participated and successful candidates were awarded the Certificate of Competence for ECG reading
d. A meeting on "Heart Failure" at Mumbai on 16th December 2007

However, we missed the Mid-Term CME this year.

Every one is eagerly waiting for ISECON 2008, to be held at Kolkata from 22nd to 24th February 2008 under the dynamic leadership of Dr Rabin Chakraborty.

2nd Seminar on "Global Approaches to Cardiac Safety" at Mumbai is planned in collaboration with Quintiles on 8th and 9th March 2008.

ISECON-2009 will be at Ahmedabad from 20th to 22nd February 2009.

Dr Ajay Naik has taken the lead in organizing the event.

I look forward to see you all at ISECON-2008, 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 – 2008.

Long Live Indian Society of Electrocardiology

Dr S B Gupta
Hon. Secretary, Indian Society of Electrocardiology

Review Article

Sudden Death: India/Asia and the West

Gopi Krishna Panicker[#], Shantanu Deshpande[#], Yash Lokhandwala[*]
[#]Quintiles ECG Services; [*]Arrhythmia Associates

Introduction

Sudden death is defined as death, in which the time and mode of death is unexpected, which occurs in an individual with or without pre-existing cardiac diseases and which occurs within 1 hour of the onset of the heralding symptoms.[1] This definition is a modified combination of the various definitions of sudden death, which differ according to clinical, scientific and medico-legal requirements. For example, in the operational definition of the forensic pathologist, death may be considered "sudden" if the subject was seen alive and functioning normally in the preceding 24 hours. Similarly, the advances in life support system interventions have also resulted in delaying the biological death beyond 1 hour or in survival of the patient and is often referred to as 'aborted sudden death', although it contradicts the definition of death as an irreversible event. [2]

Sudden cardiac death (SCD) is defined as death due to cardiac causes, in which the time and mode of death is unexpected, which occurs in an individual with or without pre-existing cardiac diseases and which occurs within 1 hour of the onset of the heralding symptoms.[1]

Epidemiology of SCD – Difficulties in estimating incidence

Estimating the overall incidence of SCD is difficult, partly due to the varying definitions of SCD used in epidemiological studies. Moreover, the etiological categorization of SCD, on the basis of clinical circumstances, is an estimate considering that 40% of sudden deaths are un-witnessed at the time of the event.[3]

The reported incidence of SCD in the United States vary depending upon the source of information, with emergency rescue data giving an estimate of 200 thousand sudden deaths per year and retrospective analysis of vital statistic mortality data estimating more than 450 thousand sudden deaths per year. [4, 5] In a study of Gillum, on the data for deaths occurring out of hospital and in the emergency room from 1980 to 1985 in 40 states in USA, the incidence of SCD paralleled the prevalence of coronary artery disease (CAD). [4] Consequently, it is difficult to estimate, with fair approximation, the overall incidence of SCD, due to the significant difference in CAD prevalence in different countries.

SCD accounts for 300,000 to 350,000 deaths annually in the United States, which corresponds to about 50% of cardiac deaths in the United States and other developed countries. [6,7] The incidence of SCD ranges from 36 to 128 per 100,000 inhabitants per year in different studies. [8] These figures suggest an overall incidence between one and two deaths per 1000 persons among the general population. [9]

Risk Factors for SCD

Assessing the risk factors of SCD is of importance in identifying populations at risk and considering strategies for prevention of SCD. The important risk factors for SCD are:

Cardiac Disease Profile

Population sub-groups with cardiovascular diseases, understandably, show a significantly greater risk of SCD, as compared to the normal population. The major cardiovascular diseases are as follows:

- Coronary artery disease – the single largest causative risk factor
- Cardiomyopathies
  - Idiopathic dilated cardiomyopathy – heart failure
  - Hypertrophic cardiomyopathy
  - Arrhythmogenic right ventricular dysplasia
- Electrophysiologic abnormalities
  - Long QT syndrome
  - Brugada syndrome
  - Sudden Infant death syndrome (SIDS)
  - Pre-excitation syndromes
  - Conduction system abnormalities
- Valvular heart disease
  - Aortic stenosis
- Congenital cardiac abnormalities

Age and gender

There are two ages of peak incidence of sudden death: between birth and 6 months of age (SIDS) and between 45 and 75 years of age. The Gillum study showed that the incidence of SCD increases with age as the prevalence of CAD increases with age. [4]. Thus In the study, the cause of death varied for the 35 to 64 years and ≥ 65 years age group. Acute CAD, unspecified cardiovascular disease, cardiomyopathy and arrhythmias were more common in the younger age group. Chronic CAD and heart failure, in contrast, were more common in the older age group. In a study of vital statistics mortality data in United States, CAD was listed as the underlying cause of 62.2% of the death certificates, with successive age groups showing increased SCD rates. [8]

The annual incidence of SCD is three to four times higher in men than in women, with approximately 75% of SCD in men. The reason for this difference is surmised by the gender difference in the incidence of CAD and the protection from atherosclerosis in women before menopause.[8]

Time-dependent risk factors

Risk of SCD has been analyzed in the context of both biological and clinical chronology. Epidemiological analyses of SCD risk among populations have identified three patterns: diurnal, weekly, and seasonal. General patterns of heightened risk during the morning hours, on Mondays, and during the winter months have been observed.[11]

Clinically, the risk of SCD is not linear as a function of time after changes in cardiovascular status. Survival curves after major cardiovascular events, which identify risk for both sudden and total cardiac death, usually demonstrate the most rapid rate of attrition during the first 6 to 18 months after the adverse cardiac event. This time dependence of risk emphasizes the importance of an intervention during the early period after an adverse cardiovascular event for maximum efficacy. The addition of time as a dimension for measuring risk may increase the resolution within subgroups.

Life-style and Psychosocial Risk Factors

The present day urban lifestyle with its contributing psychosocial factors has been implicated in increased risk of SCD. In a study of 2320 men who survived MI, social isolation and high-level psychological stress were associated with an increased risk of SCD. These factors were also directly associated with low educational levels. [12] Study of SCDs among women showed an increased risk for women who were not married, who had fewer or no children and who had greater educational discrepancy with their spouses. Type A personality has also been associated with an increased incidence of CAD and its manifestations, including SCD.[13]

Other risk factors of SCD include a history of psychiatric treatment, greater alcohol consumption, obesity and cigarette smoking. Cigarette smoking and obesity were amongst the few risk factors in the Framingham study which showed the proportion of sudden deaths due to CAD increased in association with the risk factor for SCD. The Framingham study also demonstrated that cigarette smokers have a twofold to threefold increase in SCD risk in each decade of life between 30 and 59 years.[14]

Epidemiological observations show a relationship between low levels of physical activity and increased CAD risk. In a case-controlled crossover study, a strong association between vigorous exercise and the onset of MI, particularly in persons who are habitually sedentary was observed.[15] It was suggested that the acute bouts of exercises increase the sympathetic activity and decrease the vagal activity, leading to an acute increase in susceptibility to ventricular fibrillation.[16] In contrast, regular vigorous exertion increase the vasovagal tone, resulting in increased cardiac electrical stability and protection against ventricular fibrillation.[17] However, there is a dearth of studies on the effect of physical activity in various clinical settings.

Scenario in Asia

In Asia, the levels of coverage of vital registration and the reliability of the cause of death as stated on the death certificate is often low, especially in the rural areas. Consequently, the estimation of SCD incidence has to be done from surrogate endpoints. Therefore, the calculation is often done from the epidemiological estimates of cardiovascular disease profiles in the population.

By these estimates, the rates of SCD are considerably lower, paralleling the rates of CAD in developing countries. However, 'newly industrialized countries' in Asia like India, Malaysia, Thailand, in recent years, have shown an increase in the rates of CAD and a corresponding increase in SCD incidence.

This trend is similar to the increase in prevalence of the major cardiovascular diseases in the South Asian countries like India, Pakistan and China.[18,19] (See Tables 1, 2 and 3)

However, an estimation of SCD in the Indian subcontinent is skewed, with observations from studies indicating a rural-urban divide in the prevalence of CAD. In a study by Bahl et al in 2001, the prevalence of CAD in urban areas ranged from 7.6% to 12% as compared to rural areas with a range of 3.1% to 7.4%.[20] These findings related to the rural-urban gradient in CAD may be attributed to the lower risk factors in rural areas – plausibly due to the socio-economic patterns.

Since autopsies are difficult to perform on a large scale, the exact cause of sudden death is often unclear. However, one can be definite about the commonest etiology (MI), if there is ECG documentation of the same. In the absence of this, classical symptoms of rest angina with sweating immediately

preceding death would indicate a high probability of the same diagnosis. But often, the exact cause of sudden death goes down as "unknown". Death certificates world over have been found to be very deficient when making scientific analyses of the cause of death in general, leave alone sudden death. An interesting concept of a "verbal autopsy" (Tables 4 and 5) was undertaken on a large scale in South India.[21] Lay volunteers, who were graduates, were given brief training before they undertook a survey among family members of those who had died. This revealed a significant decrease in the category of "unspecified" medical causes as compared to the death certificate data. Such verbal autopsies have been scientifically validated. Such data is very meaningful for estimating the magnitude of SCD, since we know that SCD incidence parallels the incidence and mortality from CAD.

The prevalence of CAD in the urban areas in this study were already found to be comparable to the recent update on CAD prevalence in USA.[22] The Global Burden of Disease Study has also estimated that by the year 2020, India will have more individuals with CAD than any other region. [10] Nevertheless, this aspect must be judged in the context that the information on SCD incidence is usually obtained from secondary and tertiary care hospitals in various parts of the country. Therefore, extrapolation of the conclusions drawn from the available data to rural regions may not be entirely valid because of the ethnic, economic and cultural diversity, and the differing levels of literacy and awareness among the population, access to healthcare and standards of healthcare.

An evaluation of the data, compiled by Dr. Ashish Nabar and Dr. Rahul Gupta (November, 2007), of KEM Hospital in Mumbai for a period of 3 months showed a similarity in the trend of SCD in urban India and the West (See Table 6).

In the study by Bahl et al, acute coronary syndromes were observed to be a significant cause of SCD, which if managed systematically can significantly reduce the incidence of SCD. [20] This includes improving the time from symptom onset to presentation at hospital and in-hospital care including management and drug treatment.

The time from symptom onset to presentation at hospital is also typically longer among patients in India than in the West. The mortality of MI showed that 60% of deaths occurred out of hospital, mostly sudden.[4] Thus hospital based treatment of MI, including high-tech primary angioplasty, would prevent only a

Table 1 : Estimated prevalence of major cardiovascular diseases in the South Asian region (India & China), 2004*

Country	Heart Failure	Atrial Fibrillation	Angina	Peripheral artery disease
China				
Prevalence (000s)	18,703	4,546	42,862	68,839
Prevalence rate (%)	1.40%	0.40%	3.30%	5.30%
India				
Prevalence (000s)	15,763	4,260	38,982	60,176
Prevalence rate (%)	1.50%	0.40%	3.70%	5.70%

*Data from: The Asian Cardiovascular Market Outlook to 2010, Business Insights, 2006. By Revati Nehru and James Fox-Tucker[18]

Table 2 : Estimated incidence of myocardial infarction (MI) in the South Asian region, 2004*

Country	Incidence (000s)	Incidence (%)	Share in 2004 (%)
China	4,156	0.30%	45.20%
India	3,728	0.40%	40.50%

*Data from: The Asian Cardiovascular Market Outlook to 2010, Business Insights, 2006. By Revati Nehru and James Fox-Tucker[18]

Table 3 : Distribution of Acute Myocardial Infarction Cases in south Asia*

	Overall			Male			Female		
	No. of Cases	Age, Mean (SD), y	No. (%) of Cases < 40 y	No. of Cases	Age, Mean (SD), y	No.(%) of Cases < 40 y	No. of Cases	Age, Mean (SD), y	No.(%) of cases < 40 y
Worldwide	12460	58.1 (12.2)	751 (6.0)	9458	56.3 (12.0)	683 (7.2)	3002	63.7 (11.4)	68 (2.3)
South Asia	1732	53.0 (11.4)	154 (8.9)	1480	53.0 (11.2)	143 (9.7)	252	58.6 (11.6)	11 (4.4)
India	470	53.0 (11.4)	55 (11.7)	411	51.0 (10.4)	52 (12.7)	59	57.3 (11.6)	7 (11.9)
Pakistan	637	53.3 (11.1)	57 (8.9)	543	52.4 (10.9)	54 (9.9)	94	58.3 (10.9)	0

*Data from Joshi P, et al [19]

Table 4 : Cause of death classified by Vital Statistics Department and based on Verbal Autopsy of 48 000 adult deaths (aged ≥ 25) in Chennai (urban), south India: 1995–97*

Causes of death (ICD9 codes)	Cause of death in VSD		Cause of death based on Verbal Autopsy	
	M (%)	F (%)	M (%)	F (%)
Vascular disease (390–415, 418–459)	8319 (30)	5168 (25)	11056 (41)	7435 (37)
Respiratory tuberculosis (TB) (011, 012, 018)	1399 (5)	372 (2)	2231 (8)	575 (3)
Other respiratory diseases (416, 417, 460–519)	1088 (4)	596 (3)	1597 (6)	855 (4)
Neoplasm (140–239)	1163 (4)	1002 (5)	2344 (9)	1999 (10)
Infection except respiratory & TB (rest of 1–139,279.8 [HIV], 320-6, 590, 680-6)	584 (2)	303 (2)	1034 (4)	618 (3)
Unspecified medical causes (780-9, 797-9)	12291 (44)	115 11 (56)	4367 (16)	5889 (29)
Other specified medical causes	1899 (7)	1045 (5)	4414 (16)	2804 (14)
No cause given in VSD (hence probably medical)	983 (4)	634 (3)	Nil	Nil
Total deaths – medical	27 726	20 631	27 043	20 175
Re-assigned by VA to external causes	*Excluded from the study		683	456
Total deaths (medical causes+external causes)	27 726	20 631	27 726	20 631

*Data from Gajalakshmi V and Peto R [21]

Table 5 : Cause of death classified by Vital Statistics Department and based on Verbal Autopsy of 48 000 adult deaths (aged ≥ 25) in Chennai (rural), south India: 1995–97*

Causes of death (ICD9 codes)	Cause of death in VSD		Cause of death based on Verbal Autopsy	
	M (%)	F (%)	M (%)	F (%)
Vascular disease (390–415, 418–459)	3351 (20.3)	1614 (14.4)	3928 (24.6)	2404 (22.0)
Respiratory tuberculosis (TB) (011, 012, 018)	1659 (10.1)	686 (6.1)	1841 (11.5)	671 (6.1)
Other respiratory diseases (416, 417, 460–519)	717 (4.4)	471 (4.2)	1044 (6.5)	728 (6.6)
Neoplasm (140–239)	415 (2.5)	594 (5.3)	488 (3.1)	664 (6.1)
Infection except respiratory & TB (rest of 1–139,279.8 [HIV], 320-6, 590, 680-6)	1818 (11.0)	1584 (14.1)	1954 (12.2)	1411 (12.9)
Unspecified medical causes (780-9, 797-9)	5829 (35.4)	4565 (40.7)	4173 (26.1)	2737 (25.0)
Other specified medical causes	2237 (13.6)	1346 (12.0)	2570 (16.1)	2334 (21.3)
No cause given in VSD (hence probably medical)	451 (2.7)	343 (3.1)	Nil	Nil
Total deaths – medical	16 477	11 203	15 998	10 949
Re-assigned by VA to external causes	2817	1291	3296	1545
Total deaths (medical causes+external causes)	19 294	12 494	19 294	12 494

*Data from Gajalakshmi V and Peto R [21]

small proportion of MI deaths. The time from symptom onset to emergency department arrival for patients with acute ST elevation myocardial infarction (STEMI) ranges between 110 and 140 minutes in North America, while in India, it is 180–330 minutes.[22-25] This delay in presentation is due to several factors such as lack of symptom awareness, longer distances travelled to reach hospital and problems of transportation. Only 5.4% of patients are brought to hospital in an ambulance, with the large majority using public transport (buses) and hired vehicles. Interestingly, consultation with the family doctor, local practitioner or local primary health centre has been found to be an important cause of delay. Thus, the true incidence in general population remains to be determined.

In-hospital care is determined by the type of hospital that the patient attends. In prospectively collected data from 14 hospitals in three southern Indian states, George et al found that government hospitals were least likely to follow guidelines for

Table 6: SCD incidence – Hospital data*

Evaluation Period of 3 months King Edward Memorial Hospital, Mumbai, India	
Total no. of Admissions	21,126
Total no. of Deaths	1194
Total no. of Sudden Non-cardiac deaths	40 (3.4% of total deaths)
Total no. of Cardiac deaths	126
Total no. of Sudden cardiac deaths	55 (4.6 % of total deaths)
Percentage of SCD	
SCD/Total admissions	0.26%
SCD/ Total Deaths	4.6%
SCD/Total Sudden deaths	45.5%
SCD/Total cardiac deaths	43.7%
SCDs confirmed on Autopsy	27.3%

*Data from Dr Ashish Nabar and Dr Rahul Gupta (electronic communication, November 2007)

the treatment of acute STEMI compared with private hospitals or those run by voluntary organisations.[26] Patients treated at hospitals affiliated to medical colleges were more likely to receive fibrinolytic treatment and β blockers than those admitted to non-teaching hospitals. [26]

Conclusion

Thus, the scenario in Asia as regards the epidemiology and management of SCD is one of striking heterogeneity. There is a need for correctly estimating the incidence and risk factors of SCD in the various regions. There is a need for the creation of a central database which must be collated from various regional registries. Guidelines incorporating evidence-based, cost-effective treatments should be formulated taking into consideration the resource-constraints of the region and widely disseminated for implementation.

Incidence of SCD in India appears to be significantly greater in the West, due to the increasing burden of CAD. The urban areas of Asia are already showing a trend similar to the West with CAD being the commonest underlying cause of SCD.

References

1. Torp-Pedersen C, Kober L, Elming H, Burchart H: Classification of sudden and arrhythmic death. *Pacing Clin Electrophysiol* 1997; 20:245.
2. Eldar M, Sauve MJ, Scheinman MM. Electrophysiologic testing and follow-up of patients with aborted sudden death. *J Am Coll Cardiol* 1987;10:291–298.
3. de Vreede-Swagemaker JJ, Gorgels AP, Dubois-Arbouw WI, et al. Out-of-hospital cardiac arrest in the 1990's: a population based study in the Maastricht area on incidence, characteristics and survival. *J Am Coll Cardiol* 1997; 30:1500–1505.
4. Zheng ZJJ, Croft JB, Giles WH, et al. Sudden Cardiac Death in the United States, 1989 to 1998. *Circulation* 2001;104:2158– 2163.
5. Cobb LA, Fahrenburch CE, Olsufka M, et al. Changing incidence of out of hospital ventricular fibrillation. 1980–2000. *JAMA* 2002; 288:3008–3013.
6. Gillum RF. Sudden coronary death in the United States: 1980–1985. *Circulation* 1989; 79:756–765.
7. DiMarco JP, Haines DE. Sudden cardiac death. *Curr Probl Cardiol* 1990;15:183–232.
8. Rasekh A, Razavi M, Massumi A: *Sudden cardiac death.* In: Willerson JT, Cohn JN, Wellens HJJ, Holmes DR (Jr), ed. *Cardiovascular Medicine*,3rd ed. London, UK: Springer-Verlag; 2007:2039-2083.
9. Myerburg RJ, Castellanos A: *Cardiac Arrest and Sudden Cardiac Death.* In: Libby P, Bonow RO, Mann DL, Zipes DP, ed. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.* 8th ed. Philadelphia, USA: Saunders Elsevier; 2007.
10. Murray JL, Lopez AD. *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability From Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020*. Boston, MA: The Harvard School of Public Health, 1996.
11. Arntz HR, Willich SN, Schreiber C, et al: Diurnal, weekly and seasonal variation of sudden death. Population-based analysis of 24,061 consecutive cases. *Eur Heart J* 2000; 21:315.
12. Ruberman W, Weinblatt E, Goldberg JD, Chaudhary BS. Psychosocial influences on mortality after myocardial infarction. *N Engl J Med* 1984;311:552–559.
13. Weinblatt E, Ruberman W, Goldberg JD, et al. Relation of education to sudden death after myocardial infarction. *N Engl J Med* 1978;299:60–65
14. Hallstrom AP, Cobb LA, Ray R: Smoking as a risk factor for recurrence of sudden cardiac arrest. *N Engl J Med* 1986; 314:271.
15. Albert CM, Mittleman M, Chac CU, et al. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med* 2000;343:1355–1361.
16. Peronnet F, Cleroux J, Perrault H, et al. Plasma norepinephrine response to exercise before and after training in humans. *J Appl Physiol* 1981;51:812–815.
17. Hull SS Jr, Vanoli E, Adamson PB, et al. Exercise training confers anticipating protection from sudden death during acute myocardial ischemia. *Circulation* 1994; 89:548–552.
18. Nehru R, Fox-Tucker J. The Asian Cardiovascular Market Outlook to 2010. Business Insights Ltd. 2006. Available at: http://bizwiz.biomarketgroup.com/bw/Archives/Files/TOC_RBHC0151.pdf. Accessed January 25, 2008.
19. Joshi P, Islam S, Pais P, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. *JAMA*.

2007 17; 297(3):286-94.

20. Bahl VK, Prabhakaran D, Karthikeyan G. Coronary artery disease in Indians. *Indian Heart J* 2001;53:707–13

21. Gajalakshmi V, Peto R. Verbal autopsy of 80,000 adult deaths in Tamilnadu, South India. *BMC Public Health* 2004, 4**:**47. Available at: http://www.biomedcentral.com/1471-2458/4/47. Accessed January 25, 2008.

22. American Heart Association. *Heart Disease and Stroke Statistics — 2007 Update*. Dallas, Texas: American Heart Association; 2007

23. George E, Savitha D, Pais P. Pre-hospital issues in acute myocardial infarction. J Assoc Physicians India 2001;49:320–3.

24. Rajagopalan RE, Chandrasekaran S, Pai M, et al. Pre-hospital delay in acute myocardial infarction in an urban Indian hospital: a prospective study. Natl Med J India 2001;14:8–12.

25. Malhotra S, Gupta M, Chandra KK, et al. Prehospital delay in patients hospitalized with acute myocardial infarction in the emergency unit of a north Indian tertiary care hospital. Indian Heart J 2003;55:349–53.

26. George E, Hunsberger, Savitha D, et al. Treatment of acute myocardial infarction: Does the type of hospital make a difference? Indian Heart J 1999;51:161–6.

Review Article

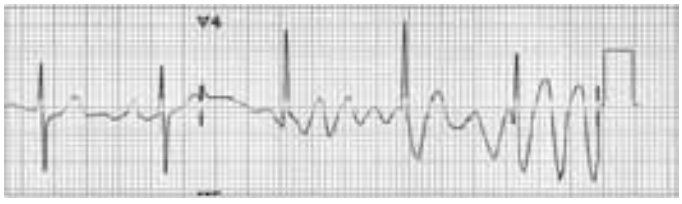
ECG Artefacts: Recognition and Prevention

Pratap J Nathani, Sathish R.
Dept of Cardiology, KEM Hospital, Mumbai, India

The surface ECG is a graphic representation of the sequence of myocardial depolarization and repolarization. The object of ECG monitoring is to observe and assess the heart's electrical activity. The ECG contains important diagnostic information routinely used to guide clinical decision making. Obviously ECG diagnoses are valid only if ECG is recorded correctly i.e. electrodes are placed in correct anatomic locations, lead wires are attached to the appropriate electrode, and the recording is of good technical quality. Cardiac monitoring is mandatory and of value in as well as for diagnosis in ambulatory setting. Advancements in signal processing have produced instrumentation with the potential of providing high fidelity ECG recordings. Despite these improvements in instrumentation, monitoring systems continue to be plagued by problems related to poor signals. Wandering baselines, small complexes, fuzzy tracings, and frequent electrode replacement make patient assessment difficult and induce false heart rate alarms. Improvement in electrode preparation techniques and a better understanding of the sources of artefact can enhance equipment performance, result in improved ECG and patient assessment, more effective utilization of nursing time, and reduced operating costs. This article discusses the physiologic and non-physiologic sources of ECG monitoring artefact. We briefly review commonly encountered errors in clinical electrocardiography, related to inaccurate lead placement, lead wire reversals, and noisy ECG signals. In addition, recommendations are made for preventing these pitfalls.

The word 'artefact' is similar to artificial in the sense that it is often used to indicate something that is not natural (i.e. man-made). An ECG artefact is used to indicate something that is not "heart-made." ECG artefacts result from but are not limited to electrical interference by other equipment, electrical noise from elsewhere in the body, poor electrode contact, and ECG machine malfunction. Artefacts are extremely common, and knowledge of them is necessary to prevent misinterpretation of a heart's rhythm.

Figure 1 : Artefact mimicking arrhythmia



The phenomenon of ECG artefact was recognized and categorized shortly after ECG monitoring became available.[1-10].Artefacts are divided according to Krasnow and Bloomfield's[8] original classification into 2 general categories, pseudoarrhythmias and nonarrhythmias. (Arrhythmias can simulate ventricular tachycardia, supraventricular tachycardia, Mobitz type II atrioventricular block, and sinus arrest.)

Clinical Implications of ECG Artefacts

Arrhythmias (Fig 1) are unique among transient pathologic states because, even in the absence of symptoms, they prompt intensive investigations and treatments that have long-term repercussions. Several therapies may be prescribed for patients in whom ECG artefacts were misinterpreted as arrhythmias as was seen in the study done by Knight and colleagues[11] who reported on the clinical implications of the misdiagnosis of artefact as ventricular tachycardia in 12 patients. Clinical consequences of the misdiagnoses in these patients included unnecessary cardiac catheterization in three patients, unnecessary medical therapies including intravenous antiarrhythmic agents in nine patients, precordial thumps in two patients, implantation of a permanent pacemaker in one patient, and insertion of an implantable cardioverter-defibrillator in one patient. These misinterpretations resulted resulted in higher hospital costs because of unnecessary testing, and patients were transferred to a higher level of intensive care or treated longer than necessary in the hospital. For example an artefactual recording of atrial flutter or atrial fibrillation may occur in a patient who has tremors due to Parkinson's disease, which if not correctly recognized may result in initiation of unnecessary treatments.

Sources of ECG Artefact

The electrical activity of the heart is sensed by monitoring electrodes placed on the skin surface. Since electrical signal is small, other artefactual signals of similar frequency and often larger amplitude reach the skin surface and mix with the ECG signals.[12,13] Artefactual signals arise from several internal and external sources. Sources of artefact are: (1) signals from other

Figure 2 : Pacing artefact (Ventricular Pacing)

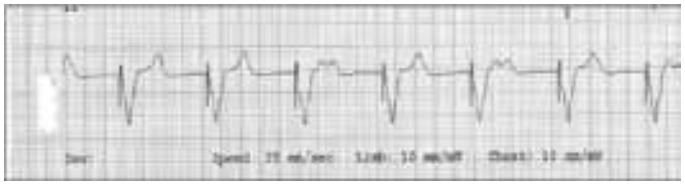
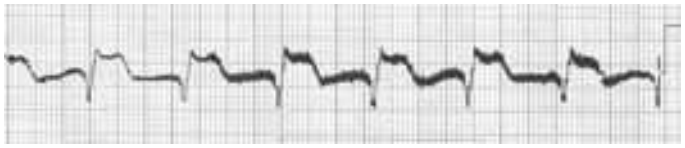


Figure 3 : 50 Hz artefact



muscles (electromyographic signals) and (2) signals produced in the epidermis. (3) 60 Hz. electrical interference signals, (4) offset signals produced by the electrode itself, (5) signals produced by the interaction of body fluids and the electrode gel, and (6) lead wire and patient cable problems, and (7) inaccurate lead placement, lead wire reversals and artificial pacemaker spikes. Some of these sources of artefact are discussed here under.

Pacing Artefacts

These appear as regular spikes preceding either the P' wave or the QRS' complex depending on whether the atrium or the ventricle is paced respectively. Further, the spikes are small when pacing is bipolar whereas the spikes are large during unipolar pacing (fig 2).

Electromyographic signals

All muscle activity produces electrical signals. Signals from muscles other than the heart are called EMG signals and appear as narrow, rapid spikes associated with muscle movement.

50 Hz artefact

This is produced by the electrical current from the mains source. The artefact rate, if measured, is 50/second. Hence in this ECG, there are 10 artefacts for every 200 ms interval. Most ECG machines have a 50 Hz notch filter to eliminate this source of artefact. Yet it sometimes manifests, especially in the ICU setting, when there are various electrical appliances hooked on to the patient.

Limb lead wire reversals

There are several limb lead wire reversals that produce abnormal ECG findings. One of these errors, in connecting the ECG cable, reversal of the right leg (RL) and left leg (LL) electrodes, does not alter the ECG because potentials recorded from the right and left legs are practically the same. Some of the arm-leg reversals may show variations depending on the ECG machine used.

Figure 4 : Right arm-Left arm reversal

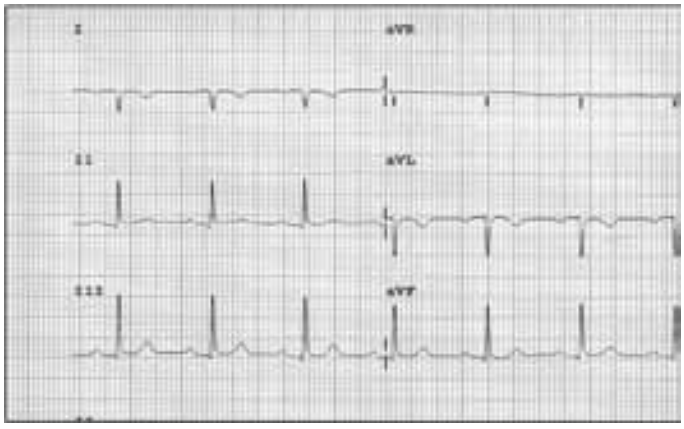
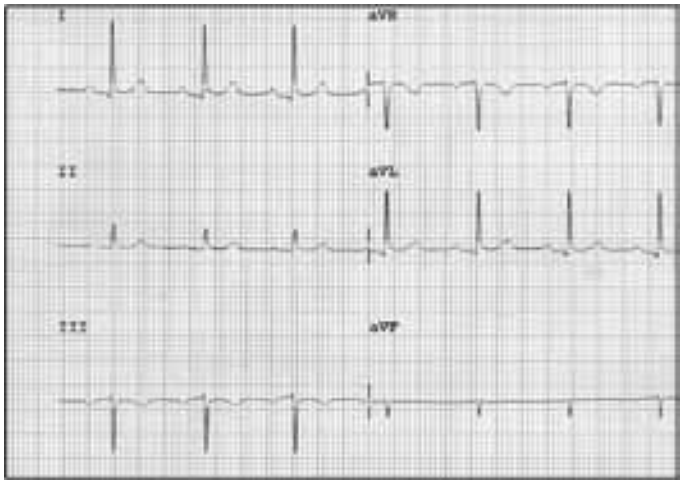


Figure 5 : Left arm-Left leg reversal



Right Arm–Left Arm Reversal

The most common error when attaching an ECG cable to a patient is right arm (RA)–left arm (LA) lead wire reversal, which means that lead I looks upside down (inverted P, QRS, and T waves)(fig 4). If the P wave is upright and only the QRS is negative, the problem is not lead wire reversal but an abnormal QRS axis, most likely resulting from some pathology causing extreme right axis deviation. If, however, the P wave is inverted also, there are two possible causes: (1) RA-LA lead wire reversal, or (2) dextrocardia. These can be differentiated by examining the precordial leads. If they have normal R-wave progression then it is due to lead reversal and abnormal R-wave progression with a negative QRS in V6 suggests dextrocardia.

Left Arm–Left Leg Reversal

LA-LL reversal means that lead I on the ECG is actually lead II, lead II is actually lead I, and lead III is upside down because the positive and negative poles are reversed. In addition, leads aVL and aVF are reversed (fig 5). This type of reversal may be difficult to identify because it may not appear out of the ordinary except for left axis deviation, which is common in

Figure 6 : Right arm-Left leg reversal

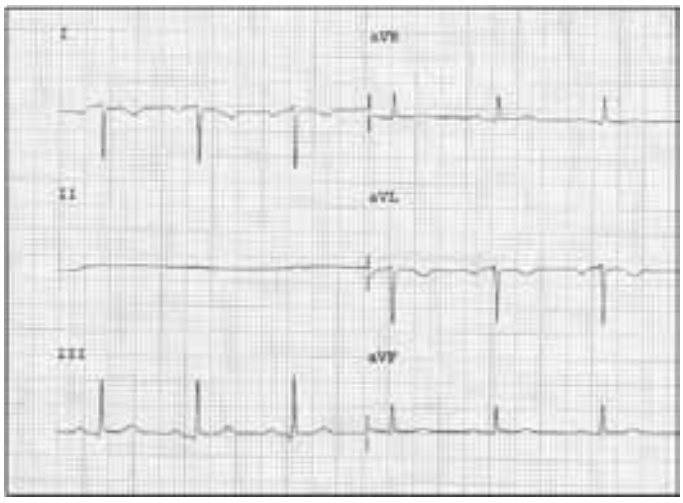


Figure 7 : Left arm-Right leg reversal

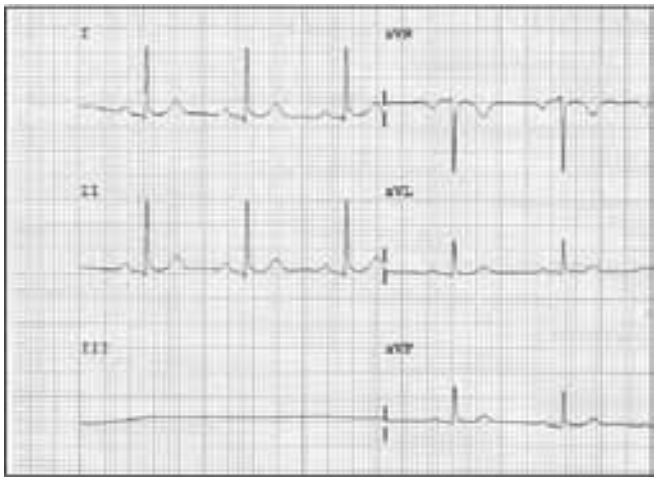
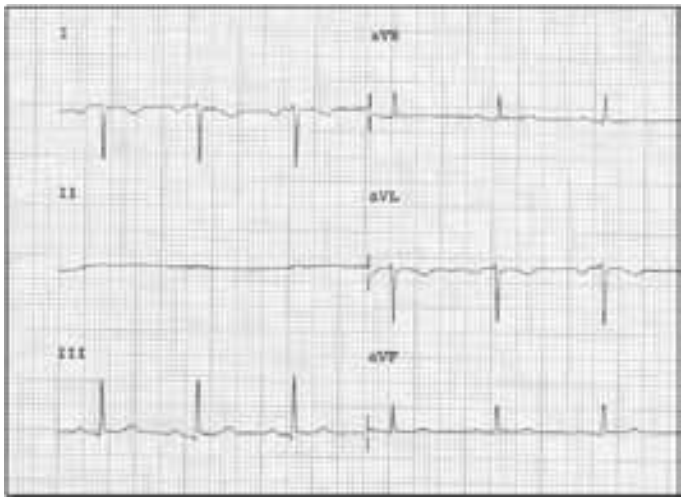


Figure 8 : Right arm-Right leg reversal



hospitalized patients. One clue is the appearance of a P wave in "lead I" that is larger in amplitude than in "lead II." The "lead I" P wave actually represents the P wave of the true lead II, which typically has the largest P-wave amplitude of any limb lead.

Figure 9 : Noisy signal artefact

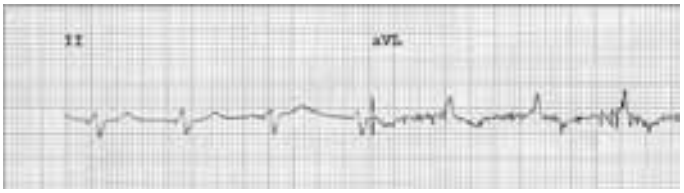
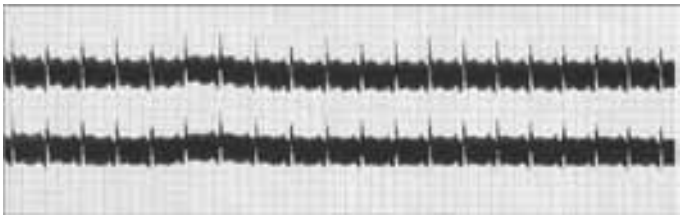


Figure 10 : Filter artefact



Right Arm–Left Leg Reversal

RA-LL reversal causes lead I to be the inverse of lead II, lead II to be the inverse of lead I, and lead II is upside down because the positive and negative poles are reversed. In addition, aVR and aVF are reversed. This situation produces highly abnormal-looking limb leads, with leads I, II, III, and aVF being negative and aVR being upright(fig 6). During sinus rhythm, it is highly unlikely to have a QRS axis in the bizarre quadrant of -90° to ± 180°, which is typical of this type of lead reversal.

Reversals Involving the Right Leg And Left or Right Arm

In reversals involving one of the arm lead wires switched with the RL lead wire, one lead [either lead III in the case of LA-RL reversal(fig 7) or lead II in the case of RA-RL reversal (fig 8)] records a zero potential difference between the legs, resulting in a flat line (termed a "far-field" signal).

Excessively noisy signals

Hospitals are filled with equipment that can be a source of electrical artefact during the recording of an ECG. Moreover, ECGs recorded in the emergency department or other immediate care setting where patients may be restless or confused may be plagued with a noisy signal (fig 9).

Inappropriate filter settings

Lowering the high-frequency filter from 100 to 40 Hz will eliminate noise caused by 50-cycle interference and other artefact (fig 10). However clinically important high-frequency signals (eg, pacemaker stimuli or notches in the QRS complex) will be damped/eliminated.

In patients who have severe pain, tremor, or some other cause of an unacceptably noisy ECG signal, the filter switch on the ECG machine can be used after all attempts to eliminate the interference have failed. The change in filter setting should be documented on the final ECG.

Figure 11 : Artefact mimicking atrial flutter

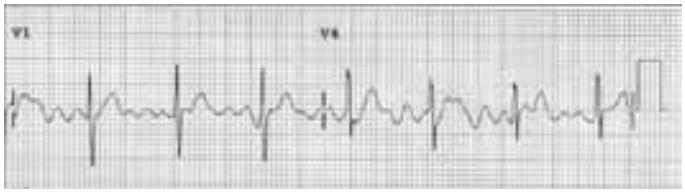
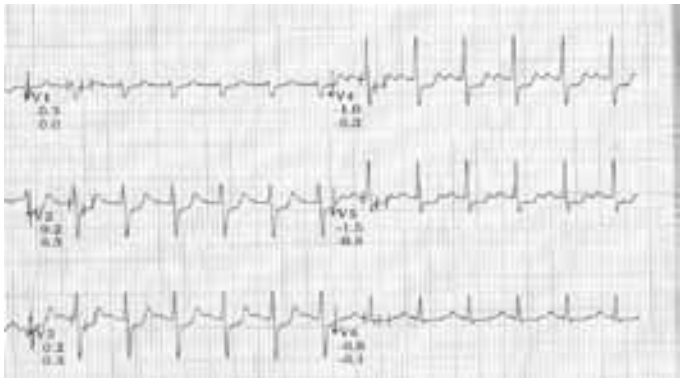


Figure 12 : Linked Median ECG showing ST depression



Excessive body movement during ECG acquisition can mimic various arrhythmias especially atrial flutter(fig 11) as depicted below. Changes in R wave amplitude are commonlycan be prod, as well as varying intercostal spaces when comparing serial ECGs in acute coronary syndrome and high V_1 / V_2 electrode placement.

During stress test, ECG taken in the "Linked Median" mode may show ST depression (fig 12), while the unaveraged ECG not showing any ST changes (fig 13). This may result in the test being reported as positive and patient subjected to unnecessary cardiac catheterization.

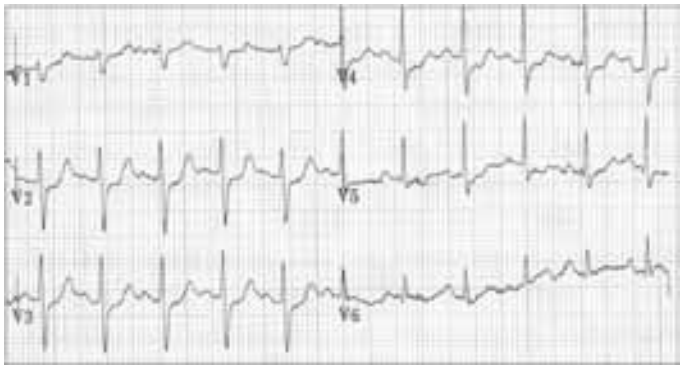
Interventions to Reduce ECG Artefact

To reduce artefact and a good monitoring tracing, the ECG signal must be as large as possible and the artefact signals as small as possible. Proper preparation of the site is required (these are more relevant for stress test, Holter and Event Recorder).

An ideal skin preparation consists of four steps:

1. Removal of part of the stratum corneum to allow for electrical signals to travel to the electrode. This can be achieved by vigorous rubbing with gauze pad or sandpaper which helps in removing dead and non conductive cells
2. Scratching the stratum granulosum to reduce motion potentials generated in this layer. Fine scratches produced by the sandpaper or grit allows the conductive gel to penetrate the stratum granulosum and "short circuit" the epidermal potentials responsible for motion artefact.
3. Defatting the skin to permit the adhesive base on the electrode to grip the skin by rubbing the skin with an alcohol/ acetone gauze pad.

Figure 13 : Unaveraged ECG without significant ST depression



4. Assuring the presence of conductive gel.

After obtaining good skin contact, it is necessary to confirm proper attachment of the electrodes to corresponding limbs to prevent artefacts.

The electrical grounding should be good when the ECG machine is connected to the mains power source. Battery-operated machines are free from this source of artefact.

For myocardial infraction patients where comparison of serial ECGs is necessary, the precordial electrode positions should be marked by non-fading ink. This will maintain uniformity. This simple measure is rarely undertaken, even in tertiary hospital ICUs.

Stress test machines that only have Linked Median traces should be avoided. There should be an option to print the raw, unaveraged 12 lead ECG at any time during the test. Moreover, this raw ECG should be of good quality.

Conclusion

Skills in correctly identifying ECG artefacts are mandatory for those who record ECGs and for physicians who interpret them. Artefacts are more common than appreciated. Clues are usually present, but you see only what you look for and recognize only what you know. The value of the ECG depends upon the accuracy with which it is obtained. Currently, many different types of health personnel record ECGs, including physician office workers, minimally trained hospital aides, ECG technicians, nurses from a variety of hospital units, and house staff. Thus, a potential exists for inconsistent recording methods that are especially problematic when accurate diagnosis depends on comparison of serial ECG tracings. It is important to have a hospital/clinic policy for recording of the ECGs that includes appropriate filter settings, diagrams of accurate lead placement, and most commonly encountered errors. Training of personnel should include a demonstration to identify difficulties identifying anatomic landmarks and to clear up any other inconsistent techniques or misconceptions. Finally, a quality improvement program should monitor the incidence of common errors such as RA-LA lead wire reversal and provide retraining when indicated.

References

1. Dyke DBS, Rich PB, Morady F. Wide complex tachycardia in a critically ill patient: what is the rhythm? J Cardiovasc Electrophysiol 1997;8:1327-1328

2. Kleinman B, Shah K, Belusko R, Blakeman B. Electrocardiographic artifact caused by extracorporeal roller pump. J Clin Monit 1990;6:258-259.

3. Schiller EC, Heerdt P, Roberts J. Life-threatening ECG artifact dur-ing extracorporeal shock wave lithotripsy. Anesthesiology 1988;68:477-478.

4. Paulsen AW, Pritchard DG. ECG artifact produced by crystalloid administration through blood/fluid warming sets. Anesthesiology 1988;69:803-804.

5. Kantor HI, Bowman A, Abbott PD Jr. Misdiagnosis of fetal life from an artifact in the electrocardiogram. Am J Obstet Gynecol 1966;94:287-289.

6. Lampert BA, Sundstrom FD. ECG artifact simulating supraventricular tachycardia during automated percutaneous lumbar discectomy. Anesth Analg 1988;67:1096-1098.

7. Falk RH, Knowlton AA. Atypical ventricular tachycardia or motion artifact? Am J Cardiol 1987;59:924-924.

8. Krasnow AZ, Bloomfield DK. Artifacts in portable electrocardiographic monitoring. Am Heart J 1976;91: 349-357.

9. Murdock DK, Moran JF, Stafford M, King L, Lowb HS, Scanlon PJ. Pacemaker malfunction: fact or artifact? Heart Lung 1986;15: 150-154.

10. Lin SL, Wang SP, Kong CW, Chang MS. Artifact simulating ventricular and atrial arrhythmia. Jpn Heart J 1991;32:847-851.

11. Knight etal. Clinical Consequences of Electrocardiographic Artifact Mimicking Ventricular tachycardia. NEJM 1999; 17, 341: 1270-74

12. Patterson, R.E. The Characteristics of Some Commercial ECG Electrodes. *Journal of Electrocardiology*, 1978;11(1): 23-26,.

13. Odman, S., and Oberg, P. Movement Induced Potentials in Surface Electrodes. *Medical Engineering and Computing*, 1982 20: 159-166.

Case Report

Tachycardiomyopathy:
A Diagnosis not to be missed

Ajay Kumar Sinha
Cardiac Care, HMRI & Magadh Hospital, Patna

Introduction

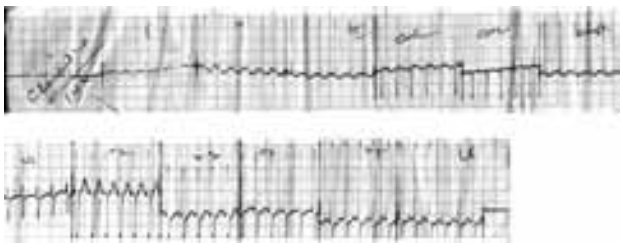
Heart failure is a common disease with major social and economic repercussion for health system. All potentially curable and reversible cause of heart failure should be investigated and treated if possible.

Dilated cardiomyopathy due to incessant tachycardia is a well-known entity especially in infants and children. This is often confused with myocarditis leading to dilated cardiomyopathy. This is potentially reversible form of cardiac failure[1].

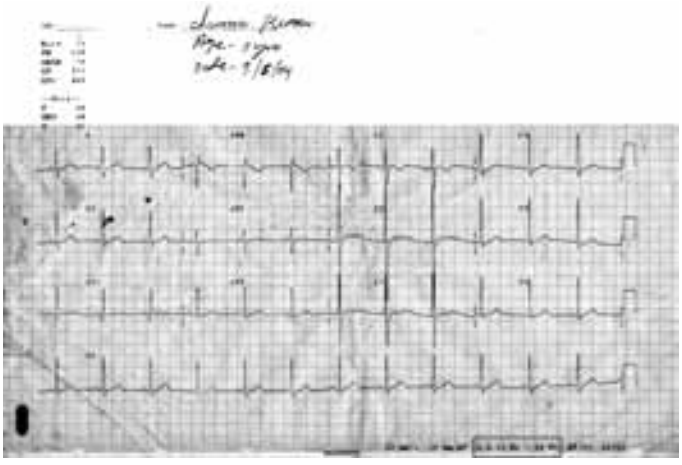
Present case of persistent tachycardia and left ventricular dysfunction was treated with amiodarone, ace-inhibitor and β - blocker with complete recovery within 4 months.

Case Report

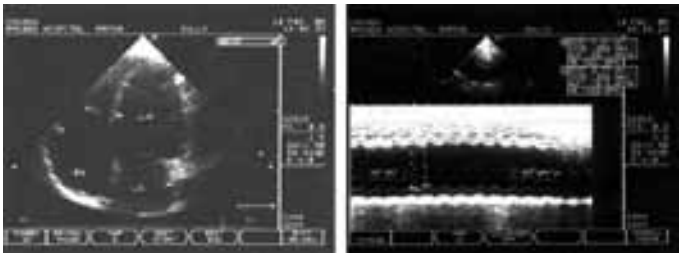
A 11 year old boy presented with symptoms of breathlessness, orthopnea and swelling of body of 6 months duration. On examination he had regular tachycardia rate being more than 150 per minute. BP was 80/60 mmHg with elevated JVP and bilateral pedal edema. Cardiovascular examination revealed cardiomegaly with normal 1st and 2nd heart sounds. ECG showed narrow complex regular tachycardia at the rate of 150 per minute. Echocardiogram showed dilated LV with global hypokinesia and severe systolic dysfunction (LVEF – 0-20).Biochemical parameters and haemogram were normal Parents refused admission due to poverty. He was put on digoxin, amiodarone, small dose of ace-inhibitor and diuretic. In follow-up patient improved and his echo-parameters showed improvements. He was then in sinus rhythm with rate around 100 per minute. Small dose of metoprolol was added. On third visit after nearly 4 months he was asymptomatic with normal echo parameters and LVEF of 60%. He was maintained on amiodarone and ace-inhibitor.



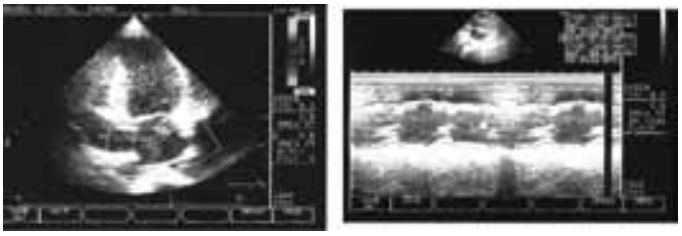
Narrow complex regular tachycardia before treatment



Sinus rhythm after 4 months



Echocardiogram before treatment



Echocardiogram after treatment

Discussion

Dilated cardiomyopathy due to persistent tachycardia is a reversible condition which should not be missed[2]. Causes can be incessant atrial tachycardia, AV nodal reentrant tachycardia, or atrial fibrillation[3]. Ventricular tachycardia is also known

to cause this condition. Radio frequency ablation has given permanent cure not only for arrhythmia but also reversal of LV systolic dysfunction. Present case was treated with drugs with good results, which is better than radio-frequency ablation in infants and children[4].

References

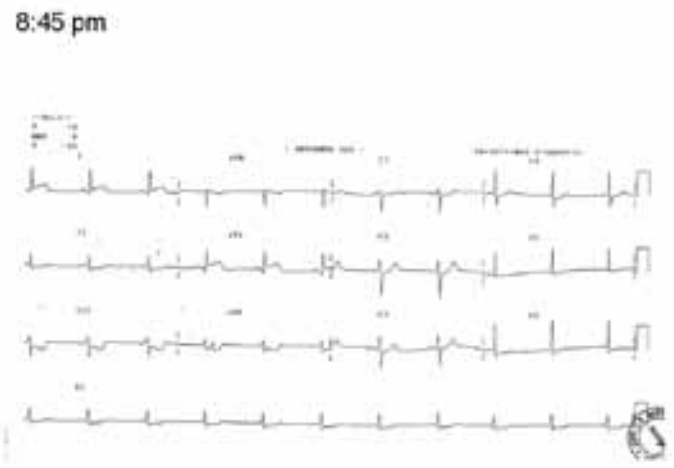
1. Packer DL, et al.Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. Am J Cardiol. 1986 Mar 1;57(8):563-70.
2. Walker NL, Cobbe SM, Birnie DH. Tachycardiomyopathy: a diagnosis not to be missed. *Heart* 2004;90 (2):e7
3. Calò L, et al. Tachycardia-induced cardiomyopathy: mechanisms of heart failure and clinical implications. J Cardiovasc Med (Hagerstown). 2007 Mar;8(3):138-43.
4. Juneja R, Shah S, Naik N, Kothari SS, Saxena A, Talwar KK. Management of cardiomyopathy resulting from incessant supraventricular tachycardia in infants and children. Indian Heart J. 2002 Mar-Apr;54(2):176-80.

Case Report

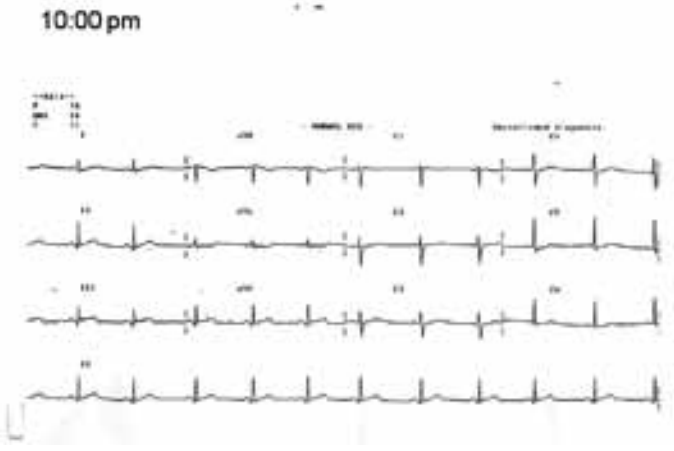
The Flitting, Fleeting ST Elevation

Bharati K.
BPT Hospital, Wadala, Mumbai

A 50 year old man presented with rest angina. The ECG showed ST elevation in leads 1 and aVL, with ST depression in the inferior leads. This suggested a high lateral M.I.



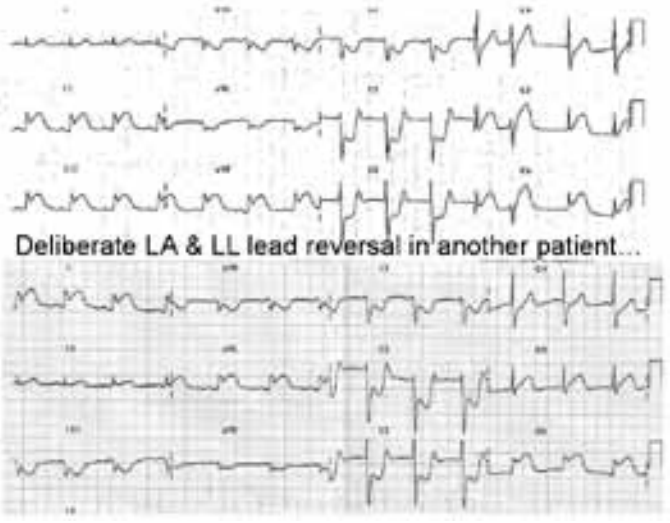
The ECG after 75 minutes showed that the ST elevation had now shifted to the inferior leads, while leads 1 and aVL now showed ST depression! How does one explain this?



Explanation

This bizarre change does not have any logical explanation. Therefore one must consider a recording error. The commonest lead interchange is right arm-left arm. However, this is not the case here. The QRS/ST in Lead I in the first ECG is similar to lead II in the second ECG. Also, lead II in the first ECG is similar to lead I in the second ECG. This suggests interchange of left arm-left leg leads in one of the 2 ECGs. This is confirmed by the interchange of the P waves also. Typically, lead II shows a taller P wave than lead I. The best way out of this quandary would be to repeat another ECG, to know which of the 2 above ECGs is the correct one. In this patient, it turned out the 2nd ECG was the correct one.

Another deliberate left arm-left leg interchange is shown below:



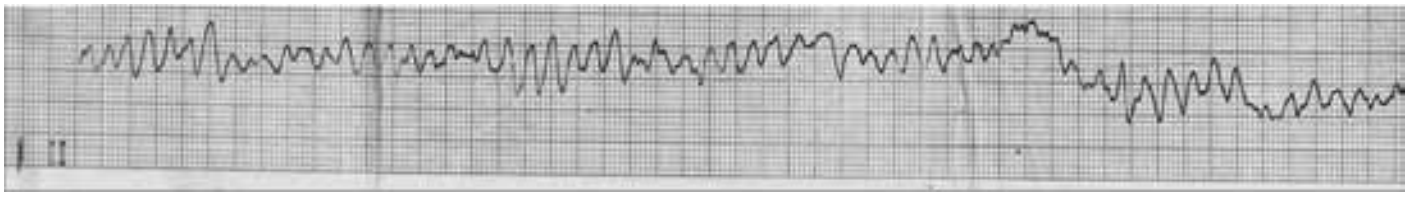
ECG Quiz

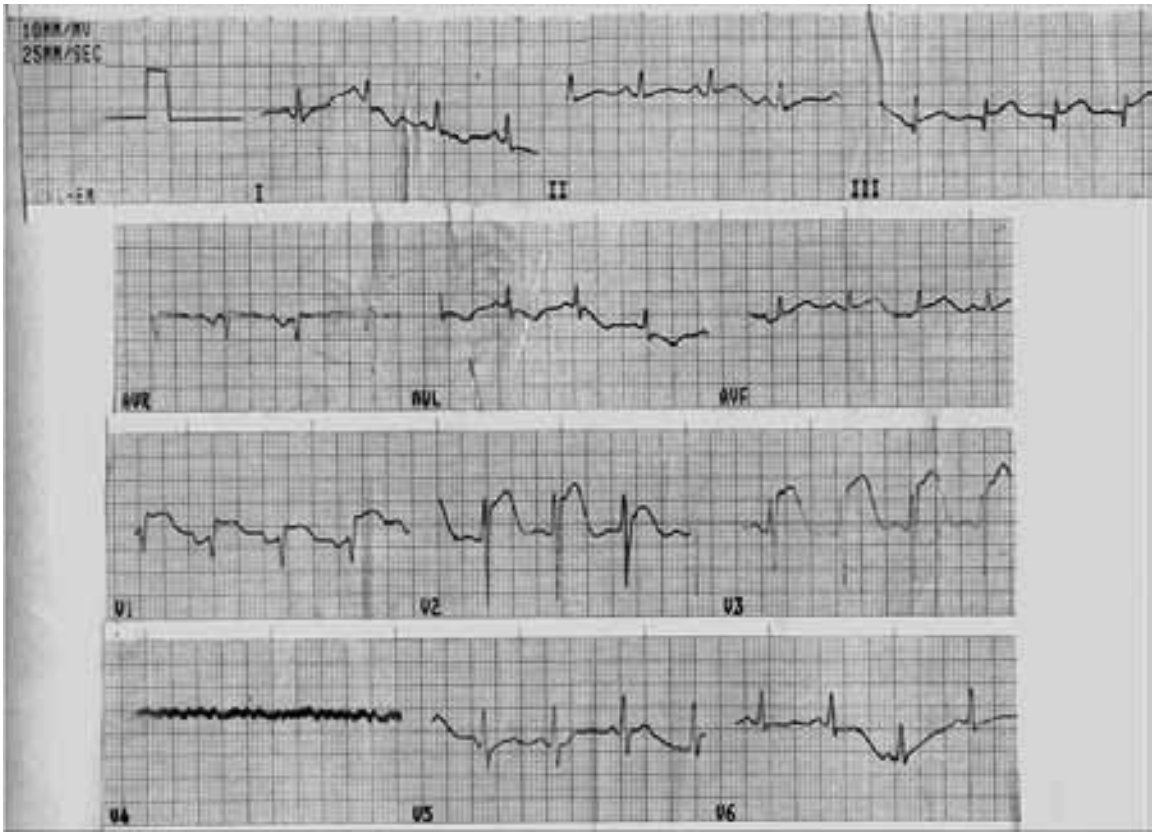
COMPILED BY
Ghanshyam Kane[#], Gopi Krishna Panicker[#], Yash Lokhandwala[*]

[#] Quintiles ECG Services
[*] Arrhythmia Associates

ECG - 1a

A 50 yr old man,
brought to the emergency room after sudden collapse on the road.





1a. The culprit coronary artery is:

a. Left anterior descending (LAD)
b. Left main
c. Right (RCA)
d. Circumflex

For correct answer see overleaf

ECG - 1a

The correct answer is 'c' – RCA

The 12-lead ECG shows ventricular fibrillation with ST elevation from V1 to V3, maximal in V3. There is also ST elevation in lead II, III and aVF. ST elevation in inferior leads can be seen with occlusion of any of the 3 major coronary arteries.

With LAD occlusion, the ST elevation in the inferior leads would be seen when the occlusion is in the mid-portion of the LAD. However, the marked ST elevation in V1, as seen in this patient, would not be seen in a mid-LAD occlusion.

In a critical left main stenosis, classically, there would be marked ST depression in the inferior and lateral leads.

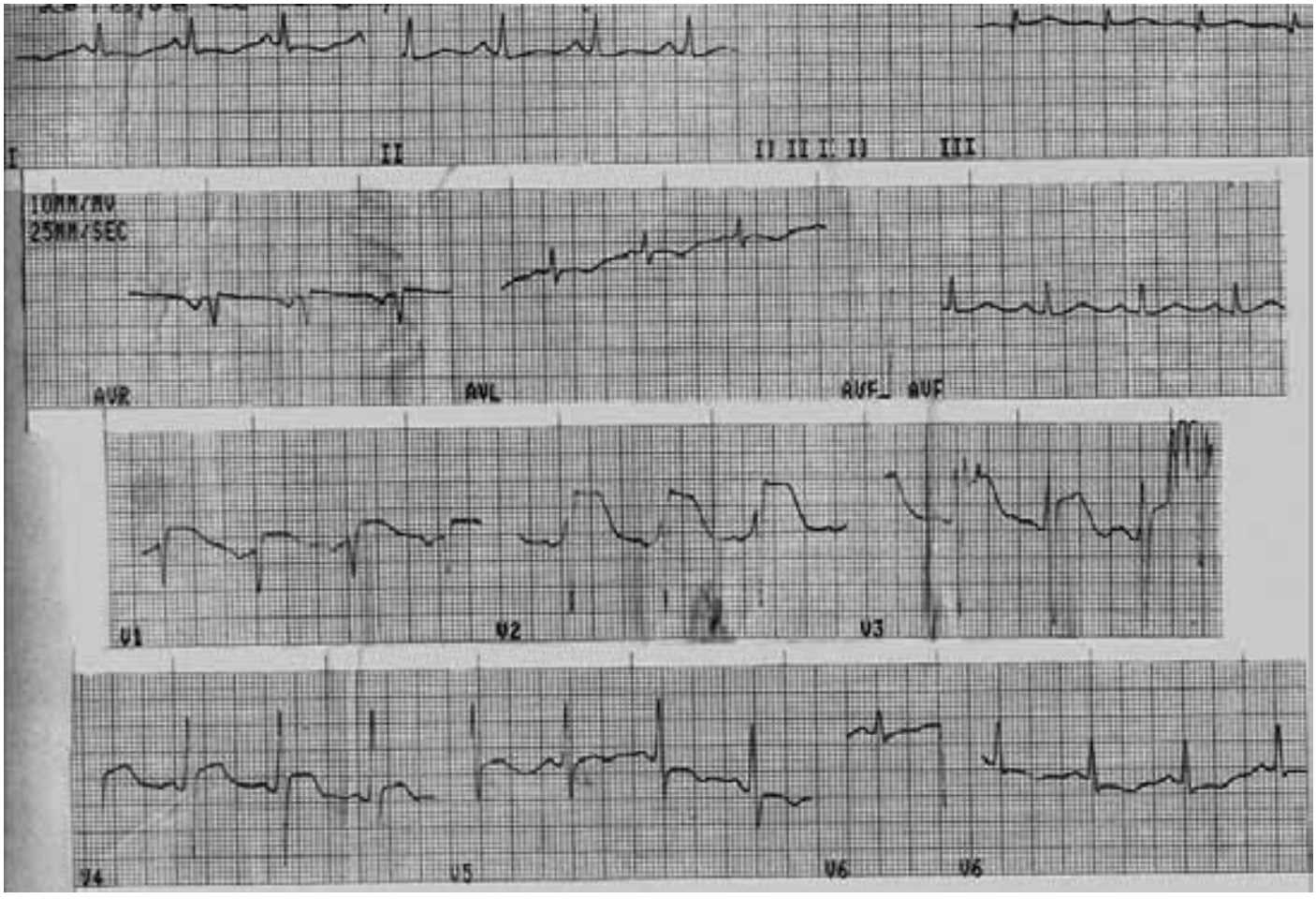
In a circumflex occlusion, there would be ST depression in leads V1 to V3.

In a RCA occlusion, usually, there would be ST depression in V1 to V3. However, occlusion of a major anterior right ventricular branch could give a pseudo-impression of antero-septal MI.

In summary, this ECG, though not a classical for any of the four options, comes closest to choice 'c'.

ECG - 1b

Surprisingly, the angiogram was reported showing no significant stenosis. The first reaction was that this was either a coronary artery spasm (and therefore, normal) or the occluded artery had recanalized. Unfortunately, even after angiography the ECG (below) showed the same ST elevation as before, making the explanations unlikely.



1b. What is, therefore, the diagnosis?

a. Acute myocardial infarction (MI)
b. Brugada Syndrome
c. Myocarditis

For correct answer see overleaf

ECG - 1b

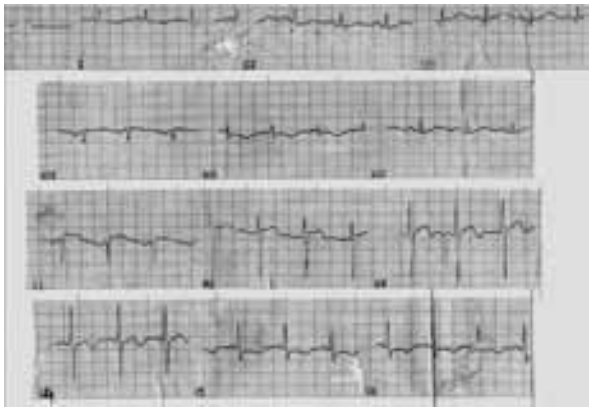
The correct answer is 'a' - Acute MI

Brugada syndrome along with ST elevation in lead V1, will also show a RBBB-like pattern, which is not seen in this ECG.

Myocarditis is more common in children and adolescents. It can give multiple ECG patterns including ST elevation. However, the regional distribution of the ST elevation as seen in this ECG would be unusual in myocarditis.

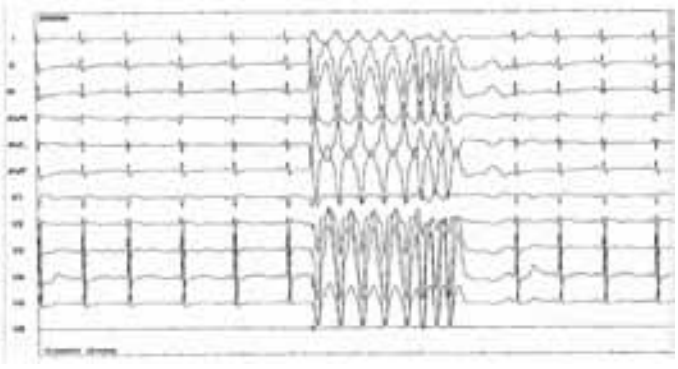
Despite, a reportedly normal angiogram, therefore, it would be correct to stick with the diagnosis of acute MI. At times, flush occlusion of an artery from a branch of its origin can be missed, if there are no collaterals to that branch.

The ECG, on the next day, shows the evolution of an infarct pattern as expected.

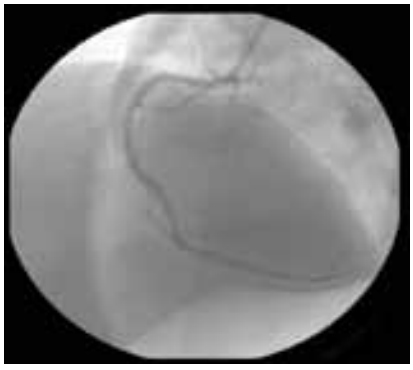


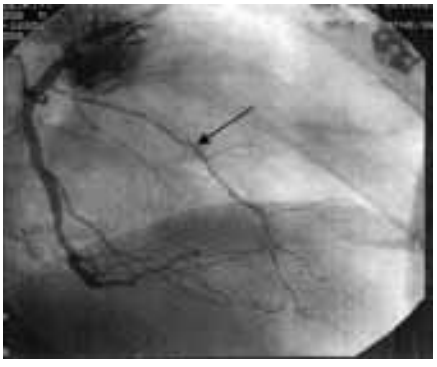
Initial

The EP study, after a week, shows no VT is inducible, despite delivering multiple ventricular extrastimuli.



After 1 week

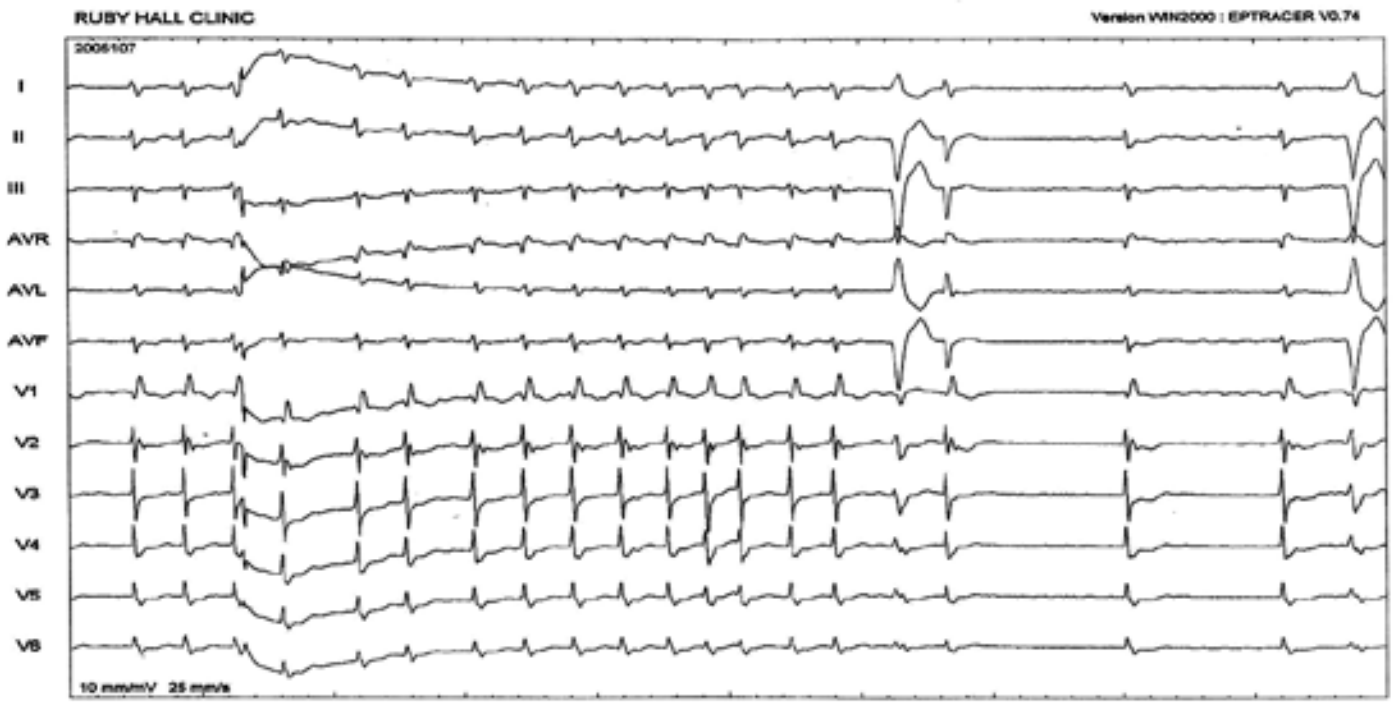




After 1 week, the RCA angiogram shows a long RV branch (arrow), with severe stenosis at its origin. This branch was totally absent in the initial angiogram, since it was occluded from the origin itself. Since this branch runs across the anterior surface of RV, it produced ST elevations, which were most remarkable in V1 – V3. The parent RCA being spared, the inferior leads did not show much ST elevation. It is important to bear in mind (as illustrated by this example) that even a small MI can produce ventricular fibrillation.

ECG - 2

A 79 yr old lady, during treatment for chronic atrial fibrillation (AF) with persistent rapid ventricular rate and increasing dyspnea and refractory to drugs and DC cardioversion…



2. The treatment is:

a. I.V. verapamil
b. I.V. adenosine
c. I.V. amiodarone
d. None of the above

For correct answer see overleaf

ECG - 2

The correct answer is 'd' – None of the above

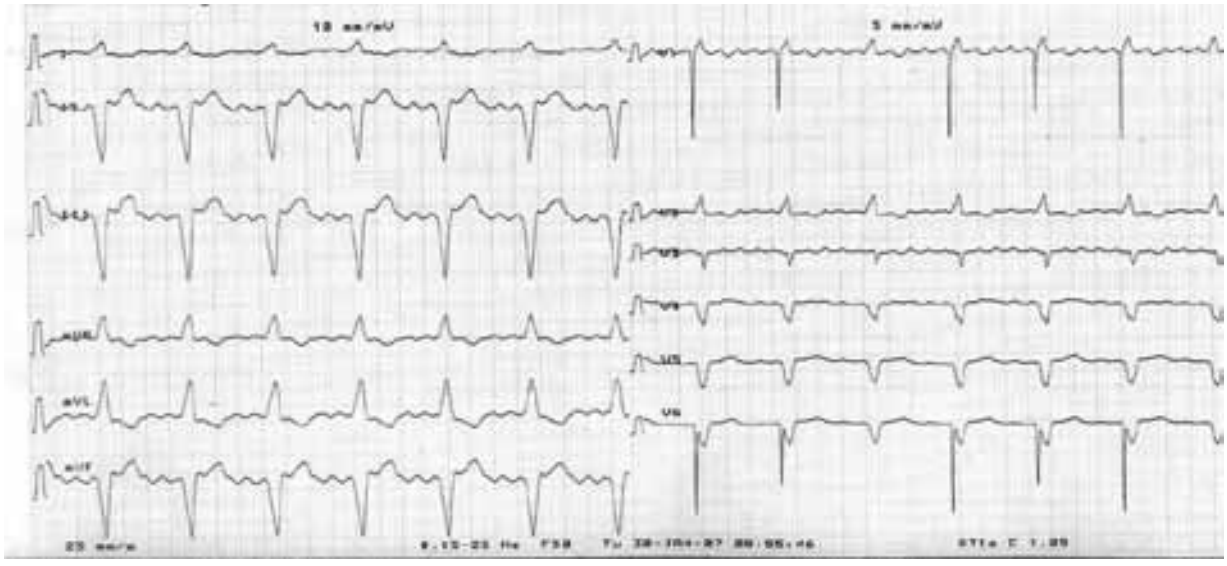
The strip shows a sudden reduction of the ventricular rate towards the end of the tracing. Even during this, there is atrial fibrillation. Since the patient is already refractory to drugs, it is highly unlikely that the treatment options a, b or c would have produced this effect. Especially in the case of verapamil and amiodarone, the effect is not abrupt as seen in this tracing. The artefact seen after the third QRS complex was produced by the onset of RF (Radiofrequency) energy.

In patients of AF with fast VR who are resistant to DC cardioversion and multiple drugs there are only 2 options

- Attempt RF ablation of AF
- Ablate the AV node so that the ventricles are protected from the high atrial rate

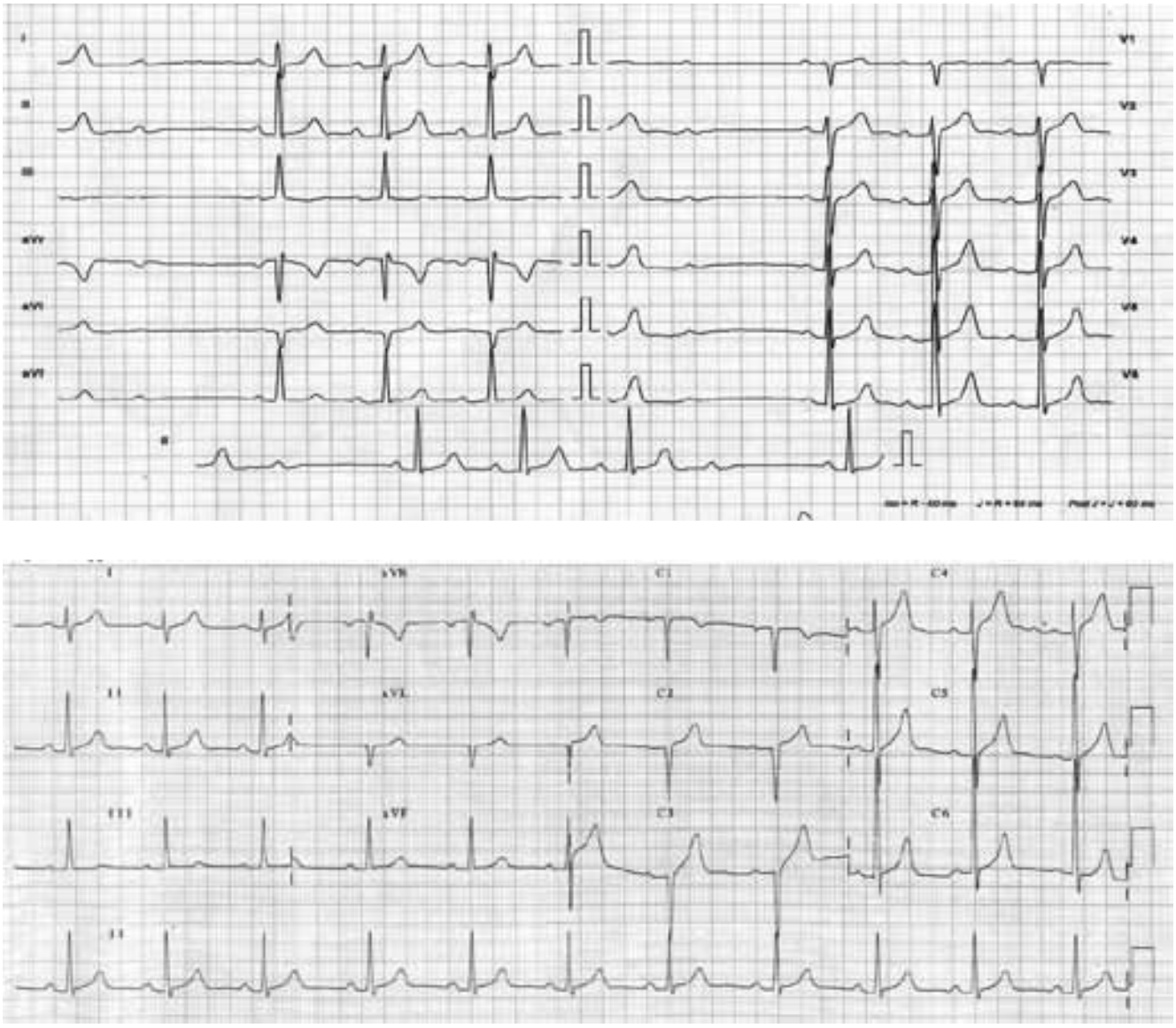
The patient also then needs implantation of a VVIR pacemaker. Some patients with chronic resistant AF with persistent fast ventricular rate require repeated hospital admissions and may develop tachycardiomyopathy. Such patients benefit with AV nodal ablation and pacemaker implantation.

After pacemaker



ECG - 3

A 22 yr old male; asymptomatic nationally competitive tennis player



3. This ECG shows:

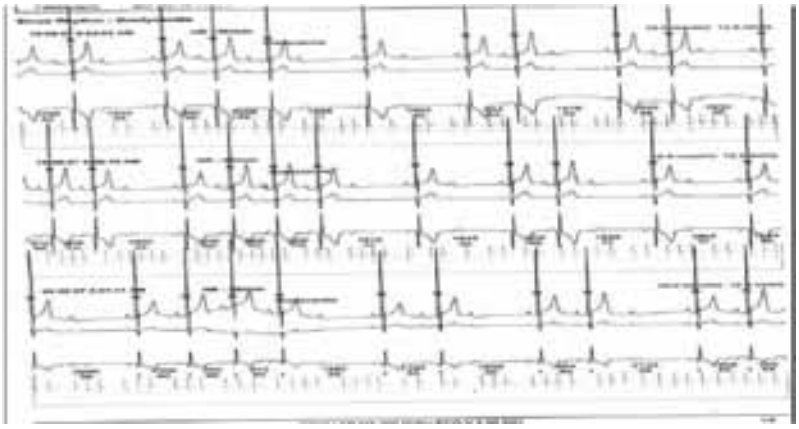
a. Mobitz type 1 AV block (Wenckebach)
b. Mobitz type 2 AV block
c. Intermittent AV block
d. SA exit block

For correct answer see overleaf

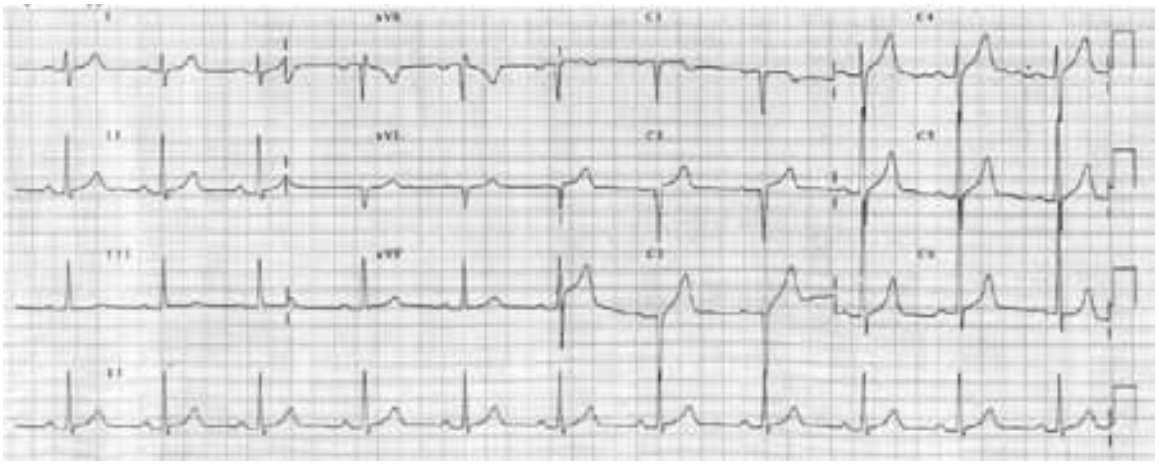
ECG - 3

The correct answer is 'a' – Mobitz type 1 AV block (Wenckebach)

There is clearly progressive PR prolongation followed by a blocked P wave. The Holter strips shown below did not show in any higher grades of AV block. Repeatedly, AV Wenckebach was seen.



At other times during the Holter there was 1:1 AV conduction even upto a heart rate of over 101 bpm.

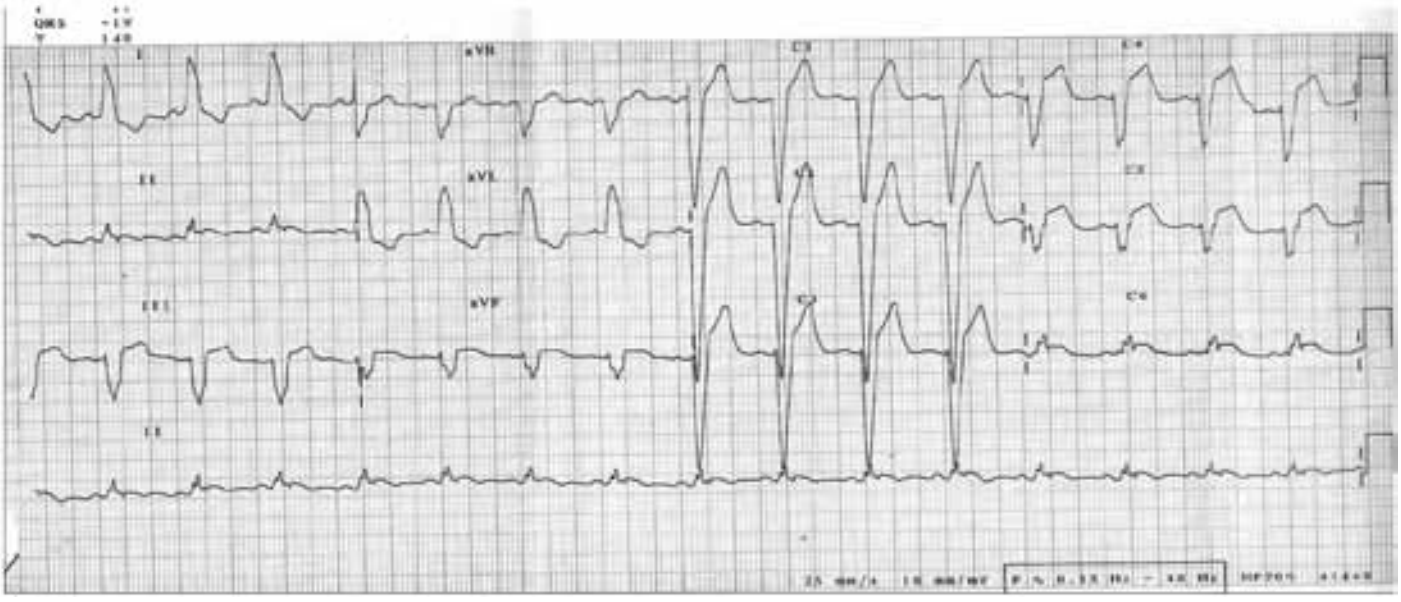


During stress test, he walked for 15 minutes on standard Bruce protocol without symptoms; the ventricular rate increased to 130 bpm.

All the above information put together suggests a 'vagally mediated' AV nodal Wenckebach. In this athlete it is physiological, asymptomatic and not requiring any treatment. He was allowed to continue his playing career with precautions of avoiding dehydration and fasting.

ECG - 4

A 62 yr old lady with diabetes,
hypertension, Creatinine 3.9; comes with heaviness in chest…



4. This ECG shows:

a. LBBB
b. Acute MI
c. Both A and B
d. None of the above

For correct answer see overleaf

ECG - 4

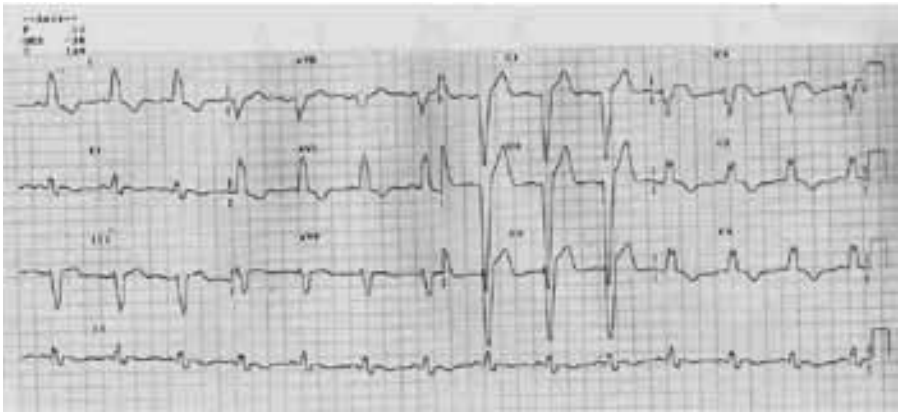
The correct answer is 'c' – LBBB and Acute MI

The ECG shows wide QRS complex of 140 ms, which confirms the LBBB pattern in lead V1 (C1). The QRS axis is -300 and the PR interval is normal.

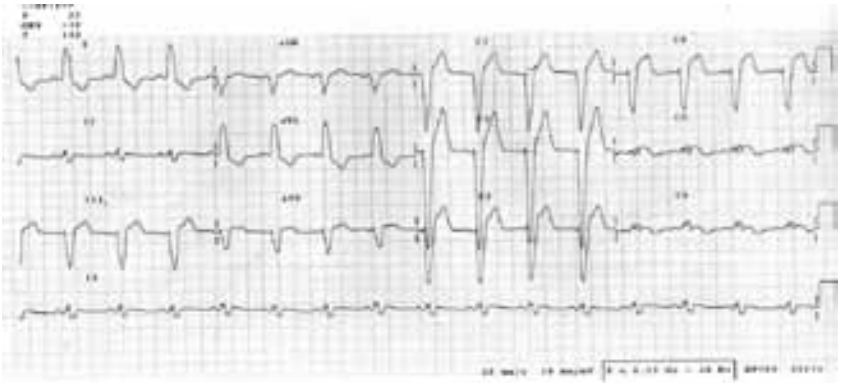
In addition to this, there is ST elevation in V6.

In the presence of LBBB, secondary ST-T changes are the rule. These ST-T changes are in a direction opposite to the QRS complex. However, in this ECG, despite the R wave in V6, there is concordant ST elevation. This clinches the diagnosis of acute MI. Therefore, in this instance, one can diagnose LBBB with AMI, especially with the associated clinical profile.

This patient was give IV heparin along with other medication. After the chest heaviness settled, the ECG the next day (shown below) shows resolution of the ST segment in lead V5 (C5) and V6 (C6), along with the appearance of R wave in lead V5 (C5). This evolution suggests reperfusion.

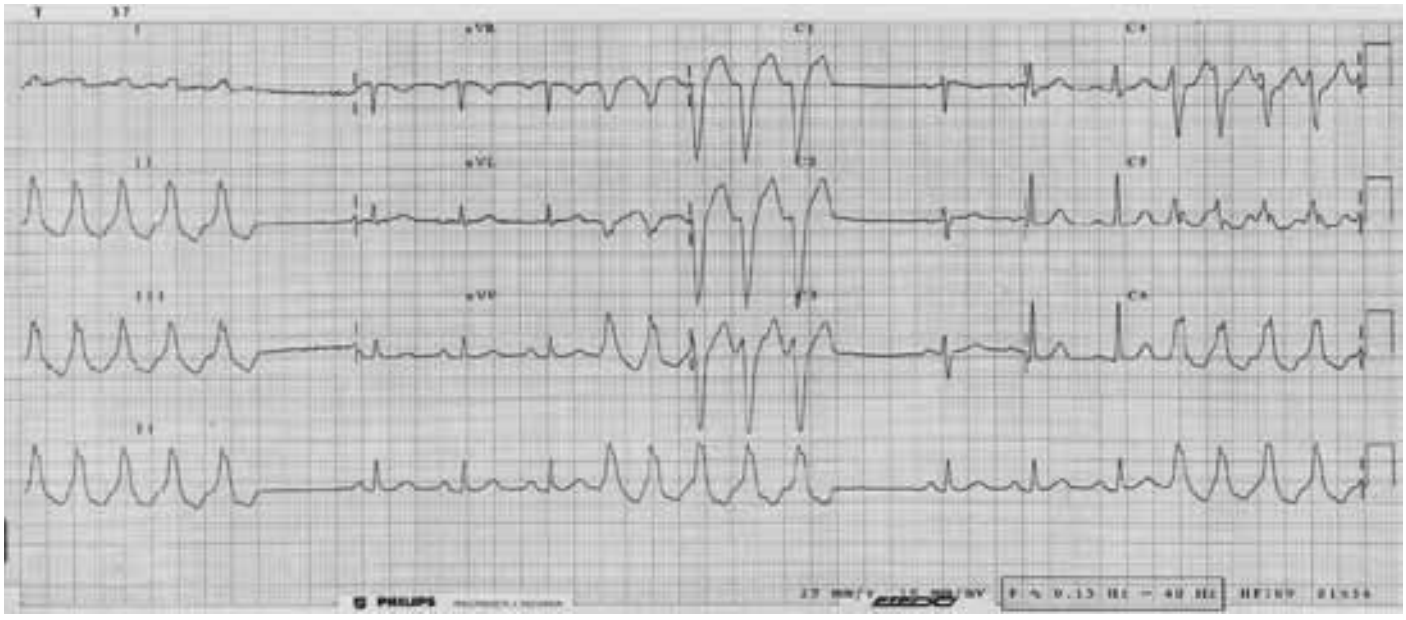


The next day, the patient again had chest discomfort. The ECG now seen below shows reappearance of ST elevation in V5 and V6, suggesting re-infarction.



ECG - 5

53 yr old lady; occasional transient rapid palpitations with uneasiness since 5 years; increased frequency since 2 days….



5. This ECG shows:

a. SVT with aberrancy
b. Ventricular tachycardia
c. A Fib with fast ventricular rate
d. None of the above

For correct answer see overleaf

ECG - 5

The correct answer is 'b'- Ventricular tachycardia

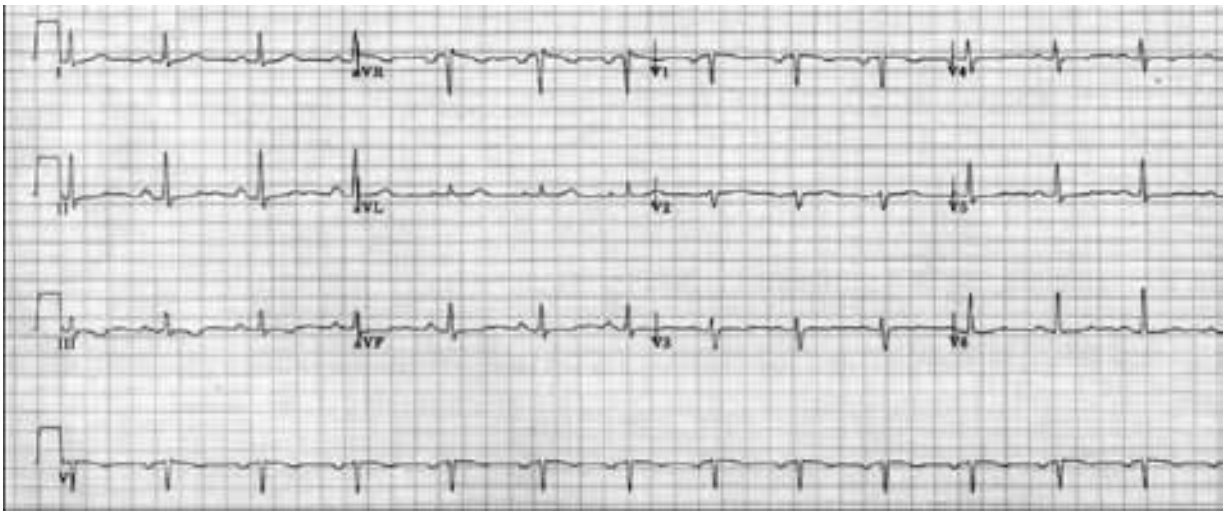
The ECG shows recurrent non-sustained monomorphic regular wide QRS tachycardia at the rate of 170 bpm. The QRS morphology during these episodes is LBBB-like with a QRS axis of 80o. The sinus rhythm complexes appear normal.

If SVT produces LBBB, the axis moves leftward compared to before. Moreover, short bursts of SVT as seen here would be unlikely to always show LBBB.

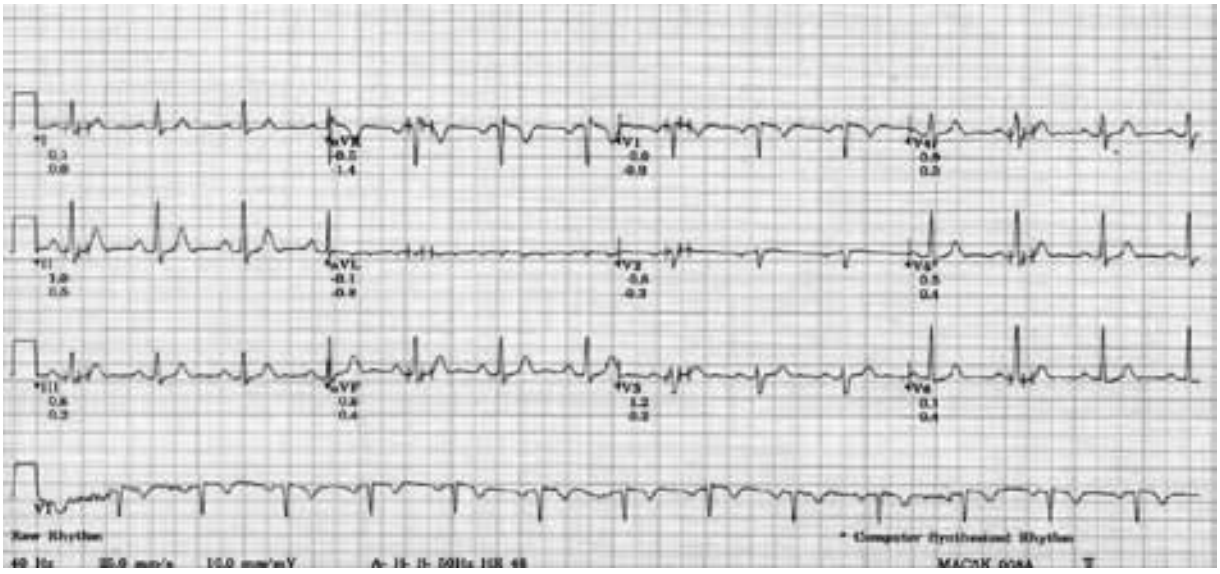
This pattern is typical of idiopathic RVOT (Right ventricular outflow tract) tachycardia. This type of VT is rarely dangerous, though it can cause recurrent symptoms, including syncope. This VT is curable by RF ablation.

ECG - 6

A 53 yr old lady with effort dyspnea. At start of stress test….



3 minutes of exercise. Tiredness. The profile suggests:



6. This ECG shows:

a. Ischemia
b. AV nodal block
c. Infranodal block
d. None of the above

For correct answer see overleaf

ECG - 6

The correct answer is 'c' – Infranodal block

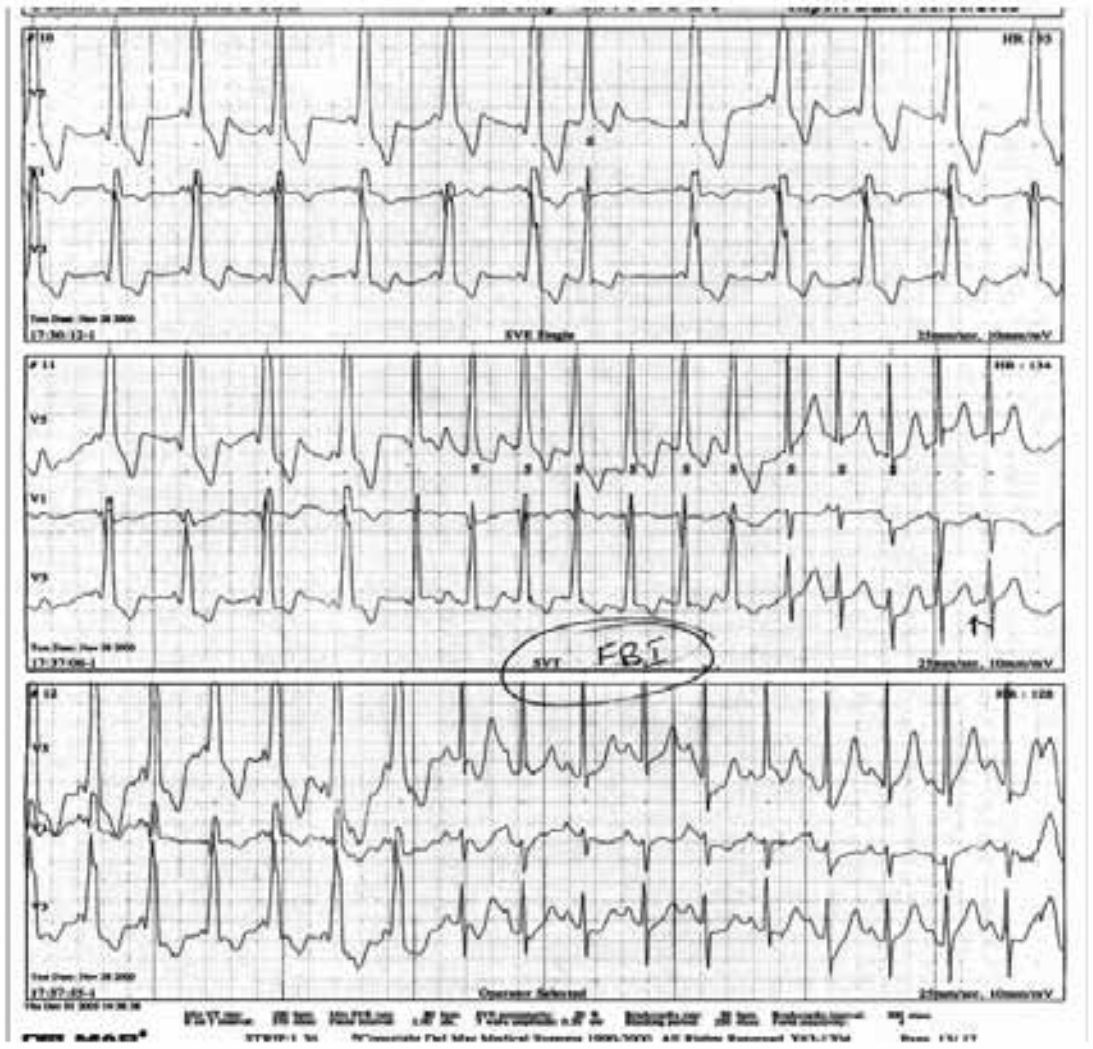
Careful observation of ECG during exercise shows that there is deformation of the T wave which has become pointed in lead II suggesting the presence of 2:1 AV block. This is confirmed by pointed T wave lying exactly between the midpoint of the 2 surrounding p waves.

Ischemia is unlikely as there are no ST segment changes. The T wave changes have occurred because of superimposed P waves. In any case, ischemia does not produce isolated AV block on exercise.

Any AV block which comes or worsens on exercise, has to be 'Infranodal block' as AV nodal conduction improves with exercise. This patient therefore needs a pacemaker.

ECG - 7

A 43 year old man with recurrent paroxysmal palpitations



7. This ECG shows:

a. WPW syndrome
b. Atrial fibrillation
c. Sinus tachycardia
d. All of the above

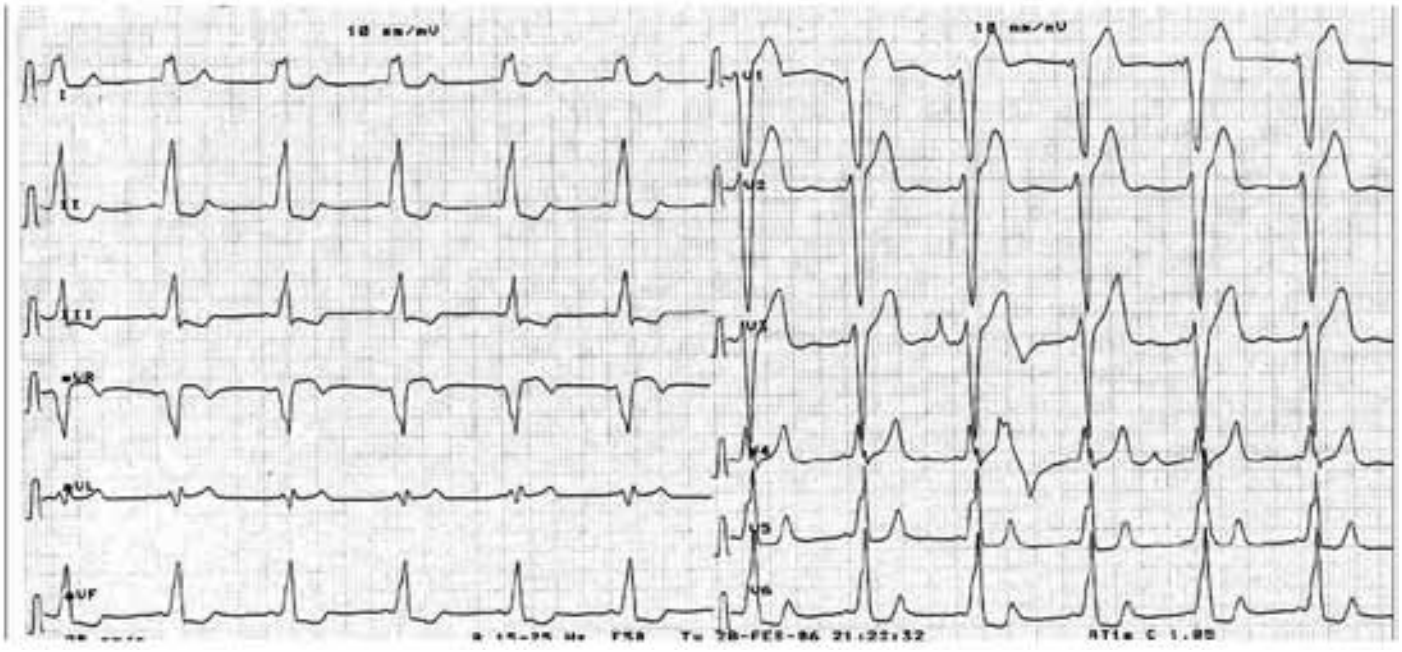
For correct answer see overleaf

ECG - 7

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

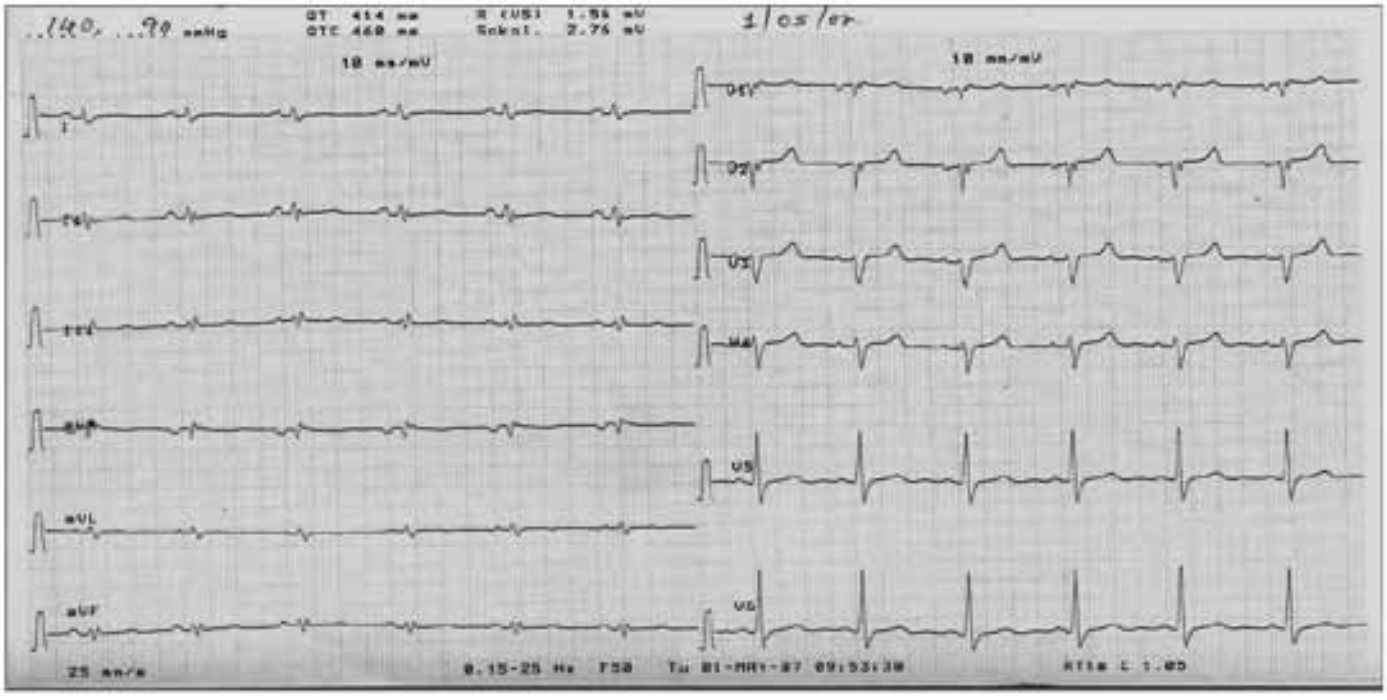
The top and middle panel shows fast, broad and irregular rhythm which suggests atrial fibrillation with fast ventricular rate in a patient with WPW syndrome. In the right half of the bottom panel, one can see sinus tachycardia, normal PR and narrow QRS. This suggests that the patient has an intermittent preexcitation syndrome.

The ECG in sinus rhythm, of same patient, clearly shows the WPW pattern



ECG - 8

49 year old man, recent unconsciousness after an alcohol bout. This was followed by total recovery within a few minutes. Several years ago, the patient had undergone an ICD (implantable cardioverter-defibrillator) implantation for ventricular tachycardia.



8. The likely diagnosis:

a. Hypoglycemia
b. Subdural hematoma
c. Tachyarrhythmia
d. Vagal episode

For correct answer see overleaf

ECG - 8

The correct answer is 'c' - tachyarrhythmia

The ECG shows low voltage complexes in the limb lead. Leads V1 and V2 show notching towards the end of the QRS complexes. There is poor progression of R wave up to lead V4. These abnormalities in the ECGs are not diagnostic of any heart disease. However, they do suggest a pathology of the right ventricle since the leads V1 and V2 lie over the RV.

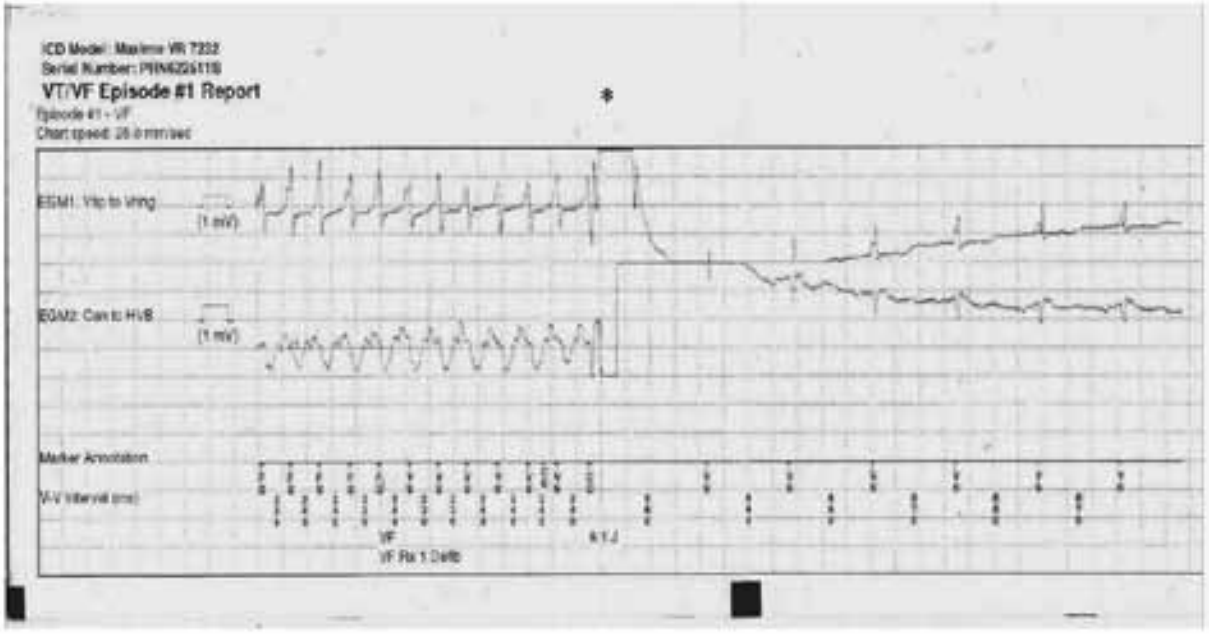
Hypoglycemia does not produce abrupt loss of consciousness. There would be a prodrome of uneasiness and sweating.

There was no vagal prodrome/association such as pain, nausea or sweating.

Subdural hematoma would be unlikely to occur abruptly. Also, there was no history of injury. And spontaneous total recovery would be unusual.

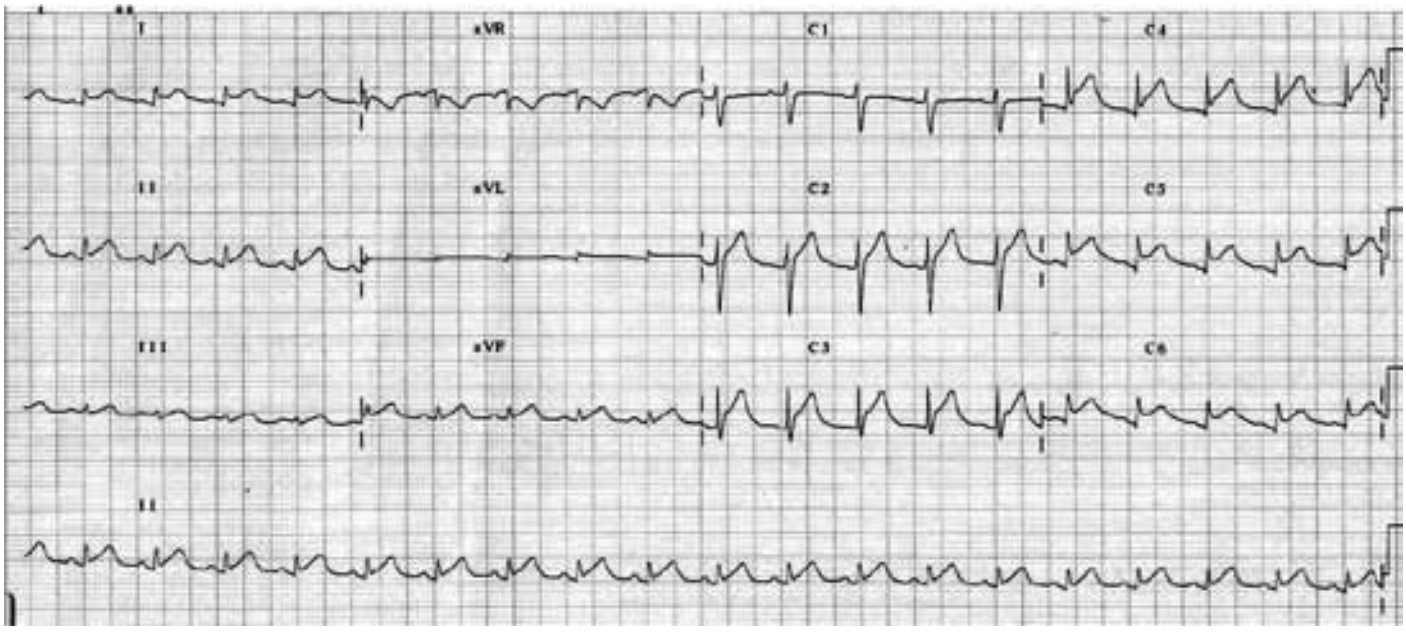
Since the patient had received an ICD for VT several years ago, one should strongly suspect that the recurrence of VT was the causes of recent unconsciousness.

Interrogation of the ICD (shown below) reveals that the patient had developed a rapid VT at the rate of 260 bpm, which was terminated by a shock (*). Since the ICD was already several years old, it took time to charge and deliver the shock. This delay resulted in the loss of consciousness. His underlying diagnosis was ARVC (Arrhythmogenic right ventricular cardiomyopathy).



ECG - 9

A 45 year old man with severe chest pain



9. This ECG suggests:

a. Proximal LAD occlusion
b. RCA occlusion
c. Left main critical stenosis
d. Acute Pericarditis

For correct answer see overleaf

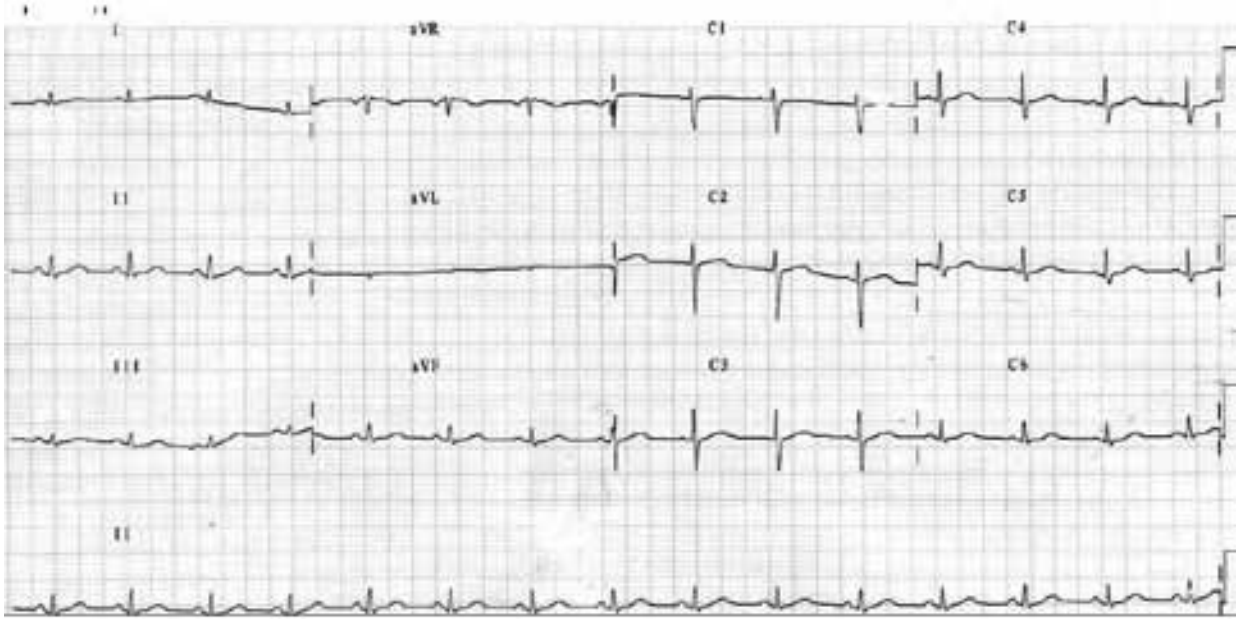
ECG - 9

The correct answer is'd' – Acute pericarditis

Except leads aVR and V1, all leads show ST elevation. Also, there is ST depression in lead aVR. Close scrutiny shows the ST elevation to be "concave upward", best seen in lead V5; however, this is only a supportive criterion for the diagnosis.

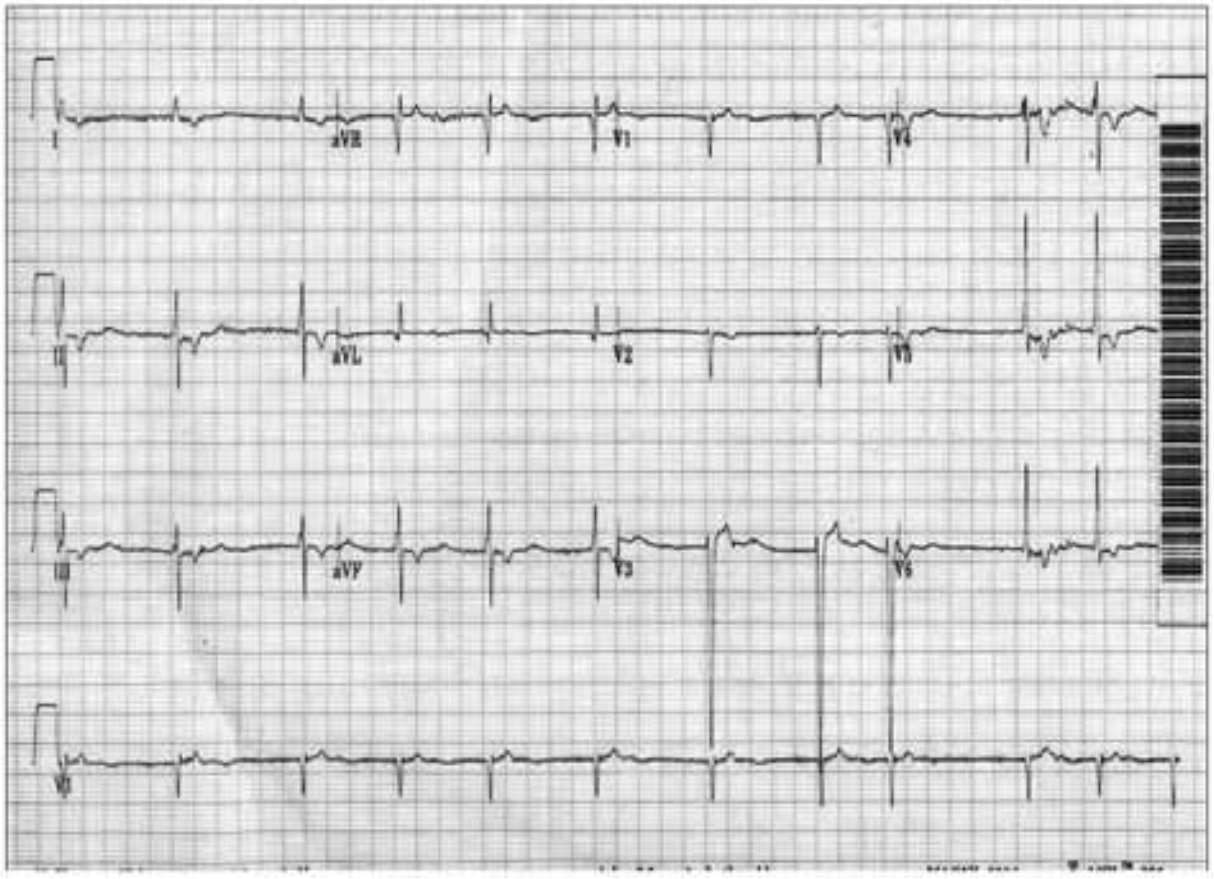
Acute pericarditis causes inflammation of the epicardium resulting in repolarization abnormalities of both atria and ventricles. In ventricles, it results in ST elevation which is seen in most of the leads and in atria as repolarization occurs during the PR segment. The current of injury causes PR segment elevation in aVR and depression in other leads. In acute pericarditis, PR and ST segment change directions. In conclusion, aVR may show PR elevation with ST segment depression where as other leads show PR depression with ST elevation.

The ECG after 3 days (below) showed complete normalization.



ECG - 10

A 35 year old man with dyspnea on exertion.



10. This ECG suggests:

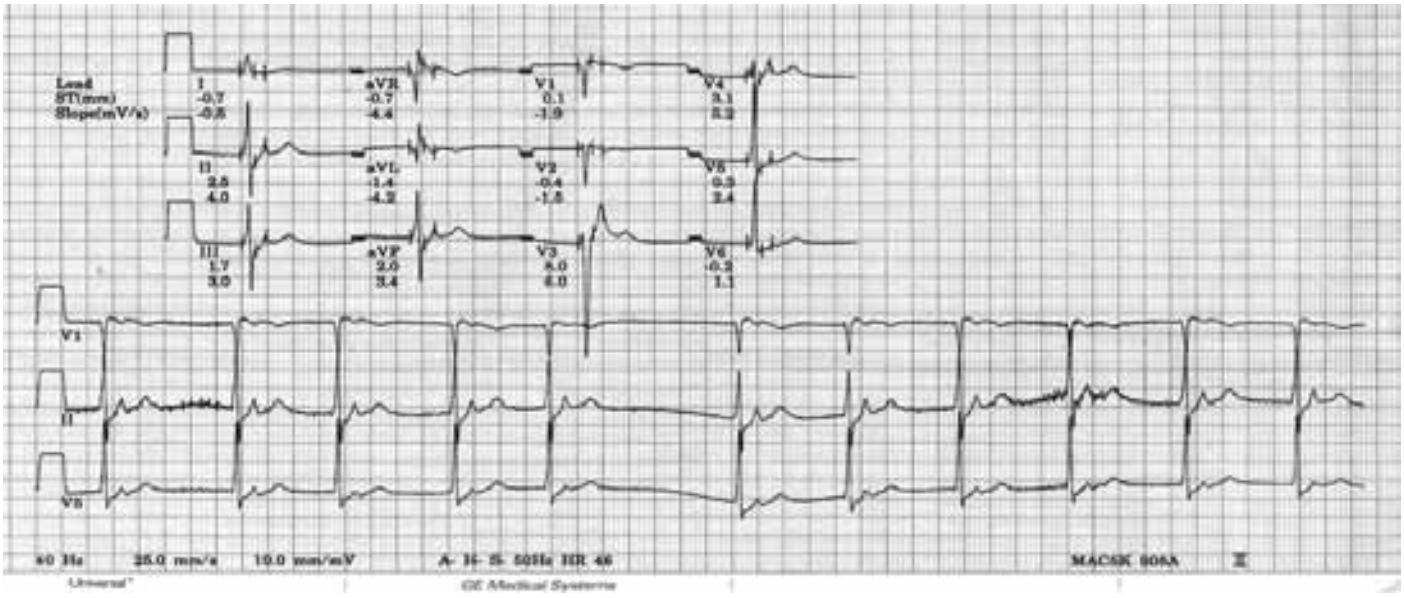
a. Junctional rhythm
b. Complete AV Block
c. Atrial Fibrillation
d. None of the above

For correct answer see overleaf

ECG - 10

The correct answer is 'c' – Atrial fibrillation

The ECG shows an irregular ventricular rate. The QT interval is extremely short. Thus, the inverted T wave in leads II, III and aVF are easily mistaken as retrograde P waves of junctional rhythm. In fact, this is an example of a rare anomaly known as short QT syndrome. The ECG after exercise (seen below) show that the T waves have changed polarity and have become positive in lead II, followed by a prominent U wave. Short QT syndrome can be familial and is associated with atrial fibrillation. It can also result in sudden cardiac death.



INDIAN SOCIETY OF ELECTROCARDIOLOGY

APPLICATION FORM FOR LIFE MEMBERSHIP/FELLOWSHIP

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

Dear Sir,

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

My particulars are as follows :

Name in full (Surname first) _____

Qualifications _____

University (Post-Graduation obtained) _____

Year of obtaining first Post-Graduation _____

Mailing Address _____

_____

Tel. No. Hospital _____ Clinic _____ Residence _____

Fax _____ E-Mail _____

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

No. _____ Dated _____ of _____

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

Thanking you,

Yours sincerely,

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

Name _____

Address _____

Signature _____

FOR OFFICE USE ONLY

Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No.

_____

Hon. Secretary, ISE

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

***Life Members :**

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

****Fellowship:**

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

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

Indian Society of
ELECTROCARDIOLOGY

ISECON 2009
Ahmedabad

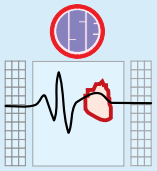
The Final Frontiers: AF & HF

First Announcement:

First ISECON in Ahmedabad

February 20-22, 2009

Invitation



Dear Colleague

Rapid, bold strides in the field of Electrocardiology and Electrophysiology have tamed many types of Cardiac Arrhythmias. Advances in Implantable Devices have reduced the morbidity and mortality in many life-threatening cardiac diseases. However, two diseases still defy the clinician: Atrial Fibrillation and Heart Failure.

Regionalization of health care is an inexorable and welcome process - the vast population of our country cannot be subserved by limited urban centers. Over the past few years, Ahmedabad has evolved as an important health care destination. Ahmedabad has been given the opportunity of hosting the ISECON 2009, to be held on February 20th - 22nd.

The theme for ISECON 2009 is **"The Final Frontiers: AF & HF".** We have many international and national luminaries in the faculty, who promise delectable fare during the conference. We will be delighted to welcome you to our city and enjoy our hospitality in the land of the Mahatma.

Core Committee
Dr. Alok Ranjan
Dr. Anup Gupta
Dr. Bhavesh Thakkar
Dr. Jayesh Rawal
Dr. Vinit Sankhla

Dr. Ajay Naik
Organizing Secretary

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

Welcome to Ahmedabad

Gandhi Ashram

Gandhiji's first ashram was at Kochrab near Paldi of the present day Ahmedabad. It was a bungalow of his barrister friend Jivanlal Desai. The present Ashram is on the banks of the river Sabarmati. Gandhiji first stayed in the Vanatshala - a place where handlooms were installed-but later on moved to `Hridaykunj', so named by Kakasaheb Kalelker as it was the pulse of the Ashram. This Spartan accomodation was to witness Gandhiji's evolution from Mohandas to Mahatma, who rose to be the Father of the Nation.

Akshardham Temple

A unique edifice celebrating the teachings of Swaminarayan faith is an amazing example of great craftsmanship and sandstone carvings. This architecturally diaphanous masterpiece is a symbol of humanity and has glorified the Indian Culture. The temple houses idol of Hindu Lord Swaminarayan, the founder of Swaminarayan Faith.

Adalaj Stepwell

The stepwell is a masterpiece in water architecture. It is situated 17 kms north of Ahmedabad. Adalaj Vav is richly carved, every pillar and wall surface covered with leaves and flowers, birds and fishes and friezes of ornamental designs. The five storeyed Octagonal shaft - wonderful sculptures of a king seated on a stool beneath a parasol with two bearers, gods, goddesses, dancing maidens, musicians, birds, animals, leaves one spellbound. From the steps of the well one can literary fee, the presence of the royal ladies peeping outside the intricalely carved jharokhas.

Bhadra Fort

Bhadra Fort is one of the majestic buildings in the city of Ahmedabad. The fort was constructed by Sultan Ahmed Shah in the year 1411 AD. It houses a number of palatial buildings, along with the sprawling Nagina Baugh. Bhadra fort covers an astounding area of 43 acres and its walls once encircled the entire city. The fort even houses a temple dedicated to the Hindu goddess, Bhadra Kali, which is the main reason why it was named as Bhadra Fort.

ISECON 2009 REGISTRATION FORM

ISECON 2009, Ahmedabad

February 20-22, 2009

❑ Dr. ❑ Mr. ❑ Mrs. ❑ Ms.

Last Name First Middle Name

Degree Speciality

Address

City State Pin Code

Email

Delegate Fees

	31-Mar-08	31-Dec-08	31-Jan-09
Delegate (Member)	2500/-	3000/-	4000/-
Delegate (Non Member)	3500/-	4000/-	5000/-
Foreign Delegate (US $)	100	100	100
Postgraduate Trainee Delegate (Proof from the HOD is required)	1500/-	2000/-	2500/-
Spouse/Accompanying person	2000/-	2000/-	2000/-
Technicians and Nurses	750/-	1000/-	1500/-

- Delegate Registration limited to 300 only (First come first serve basis)
- Demand Draft or Cheque to made in favour of "ISECON - 2009"

For Registration:

Conference Secretariat: **ISECON-2009**
The Heart Care Clinic
201, Balleshwar Avenue, Opp. Rajpath Club, Bodakdev,
S. G. Highway, Ahmedabad - 380015, Gujarat, INDIA.
Phone : + 91-79-26873101, + 91-79-26873325, + 91-79-26873224
Fax : + 91-79-26872195
Mobile : +91 98250 66664, +91 98250 66668

Global Approaches to Cardiac Safety

8th - 9th March, 2008
Hyatt Regency, Near Sahar International Airport, Mumbai

FOR MORE DETAILS VISIT

www.iseindia.org

In patients uncontrolled on monotherapy,
achieve desired target BP goals with 24-hour BP control

℞ Tazloc*H

Telmisartan 40 mg + Hydrochlorothiazide 12.5 mg

Stringent control for all 24 hours

As compared to losartan + HCTZ[1]

- Greater reductions in BP over the entire 24-hour dosing period[1].
- Allows more no. of patients achieve 24-hour BP goals.

Also available

Tazloc*

Telmisartan 20/40 mg Tablets

ARB that never gives up

Ref : 1. Am J Cardiol 2002; 89 (Suppl) : 27A-33A 2. Clin Ther. 2005 Nov; 27(11) : 1795-805