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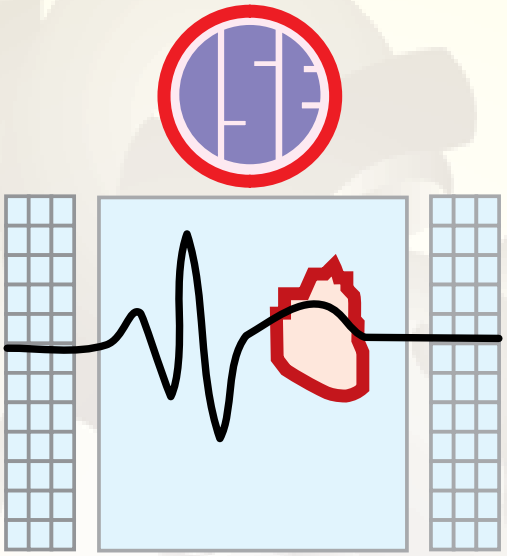

Indian Journal of ELECTROCARDIOLOGY

EDITORS
Dr. Yash Lokhandwala
Dr. Ulhas Pandurangi

Dr. Tailor who is 80 years old, our youngest candidate to give exam.

ECG exam in progress on 11th January, 2009 at Ahmedabad

C O N T E N T S

SECRETARIAT
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Editorial

Dear Friends,

As we release this issue of the IJE, we are at the threshold of ISECON 2009, Ahmedabad. Ajay Naik and his colleagues have prepared an exciting academic programme. I am sure this will be a very good learning experience.

The current issue of the IJE carries a wide range of interesting articles. Dr. Jaishankar and colleague have written a comprehensive review on right bundle branch block. The anatomy, vascularity, physiology and applied aspects of the right bundle are succinctly covered in this article.

Dr. S.B.Gupta has written an article on stress testing for the diagnosis, evaluation and prognostication of arrhythmias. This focused article carries several aspects which are not well recognized and therefore provide a good reference.

Gussak and co-workers have covered a common clinical problem, that of early repolarization. This article has been re-printed with permission from the web-based Indian Pacing and Electrophysiology journal.

As always, the ECG Quiz is one of the highlights of the IJE. This time around we bring some of the ECGs that were discussed at the last RAC in Ranchi, 2008. RAC 2008 at Ranchi was a resounding success with excellent participation from the faculty and the audience. Dr Deepak Gupta, Dr Subir Pal and colleagues deserve a pat on the back for this event.

Last but not the least are two interesting case reports from Chennai and Mumbai.

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

We acknowledge the untiring efforts of Dr Gopi Krishna Panicker in editing this issue.

Yash Lokhandwala
Editor

Ulhas Pandurangi
Editor

From Vice President's Desk

Dear Members,

Indian Society of Electrocardiology is known to be for its academic programs.

Dr Rabin Chakraborty and his team organized ISECON-2008 at Kolkata from 22nd to 24th February 2008. It was indeed a great scientific feast.

Dr Deepak Gupta and Dr Subir Pal and their team members organized Ranchi Arrhythmias Course on 27th and 28th September 2008 - A real ECG based program.

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

a. Second seminar on "Global Approaches to *Cardiac* Safety" at Mumbai in collaboration with Quintiles on 8th and 9th March 2008

b. "ECG Learning Course" for postgraduate students at Bangalore on 24th and 25th May 2008, at Silvassa on 23rd and 24th August 2008, at Mumbai on 3rd and 4th January 2009 and at Ahmedabad on 11th January 2009. About 70-80 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading

c. Satellite Symposia were organized at Mumbai on 18th May 2008, at Thane on 22nd June 2008 and on 6th July 2008 at Lucknow and were appreciated by one and all.

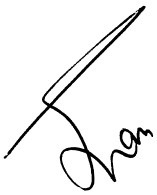
d. A unique "Training the Trainers" was organized at Mumbai on 29th June 2008 to streamline the "ECG Learning Course". Most of the cardiologists in the field of Electrocardiophysiology attended the seminar.

All the members are eagerly awaiting for ISECON 2009, to be held at Ahmedabad from 20th to 22nd February 2009 under the dynamic leadership of Dr Ajay Naik and the team.

I look forward to see you all at ISECON-2009, which will be a real treat.

My sincere thanks to Dr Yash Lokhandwala, Dr Ulhas Pandurangi and the Editorial Team for bringing out the ISE Journal - 2009,1st Volume.

Long Live Indian Society of Electrocardiology



Dr S B Gupta
Vice President
Indian Society of Electrocardiology

Right Bundle Branch Block

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The right bundle branch, which is a part of the specialized cardiac conduction system, is a continuation of the Bundle of His. It is a discrete, distinct structure in contrast to the left bundle branch and is given off distal to the insertion of medial leaflet of tricuspid valve on the pars membranacea or membranous part of the ventricular septum. Its length is 40-55 mm and its diameter averages 1 to 2mm (Figure1).

It passes along the lower septal band just distal to the muscle of Lancisi and reaches the moderator band. Thereafter it proceeds to the anterolateral papillary muscle of the right ventricle and divides into three segments- one supplying the anterolateral papillary muscle, one to parietal wall of the right ventricle and the other to distal septal surface of the right ventricle. The various divisions fan out to the right ventricular free wall and septal musculature. Because of this anatomic arrangement, catheter manipulation along the right side of the septum or operative procedure involving the septum or which produces a transverse or vertical right ventriculotomy scar that interrupts the radiations of the right bundle branch can result in transient right bundle branch block.[1]

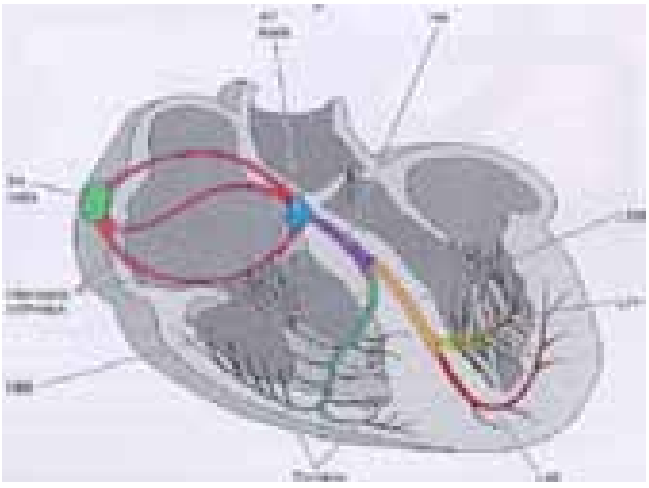

Figure 1 : (RBB-Right bundle branch, LBB-Left bundle branch, LAF-Left anterior fascicle, LPF-Left posterior fascicle)

The beginning of the right bundle branch and its third part are subendocardial and usually the second part has an intramyocardial course. Unusually if the AV bundle is located on the left ventricular aspect, then the whole right bundle branch may have an intramyocardial course.[2]

Histopathology: On microscopy the cells of first part of right bundle branch are of the same size as AV bundle cells. The cells of the second part are about the size of surrounding myocardial fibres and only the third part consists of typical Purkinje cells. These are larger than the myocardial cells and characterized by cross-striations and intercalated discs.

On electron microscopy there are few myofibrils and mitochondria with an irregular arrangement and an absent transverse tubular system. The connections are well developed with desmosomes and gap junctions. The subendocardial Purkinje cells become transitional as they penetrate the myocardium and become regular myocardial cells.[2]

Blood supply: The right bundle branch has a dual blood supply. The AV node artery, a branch of the right coronary artery supplies the first segment of the right bundle branch. The second and third segments are supplied by perforating septal branches of the left anterior descending coronary artery. Knowledge of the vascular supply is critical to understand the mechanism of right bundle branch block in the setting of ischaemic heart disease.

Electrophysiology of right bundle branch: Activation of right ventricle occurs initially in the lower third of the inter-ventricular septum and spreads through peripheral branches to cover the apex, the inter-ventricular septum and the right ventricular free wall. The basal portion near the tricuspid valve ring has delayed activation. This is due to slow conduction through the ventricular muscle because of absence of specialised conduction fibres in that region.[3] The conduction velocity (2 m/s) of the right bundle branch is slightly faster than that of the left bundle branch with a longer refractory period. Hence aberrant conduction along the right bundle branch is commoner during SVT.

Right bundle branch block (**RBBB**): It is a defect in the cardiac conduction system.During a right bundle branch block, the right ventricle is not directly activated by impulses travelling through the right bundle branch. The left ventricle however, is still normally activated by the left bundle branch. The impulses travel transeptally and thereafter activate the right ventricle .

The initial part of the QRS complex remains unchanged despite absent right septal depolarization (Figure 3). This is because the initial septal activation which occurs from the left bundle branch is left to right and anterior. The latter part of the QRS complex represents an unopposed slow conduction across the interventricular septum to the right ventricle in the anterior and superior or inferior direction. [4]

ECG diagnosis

The criteria to diagnose a complete right bundle branch block on the electrocardiogram:

- The heart rhythm must be supraventricular in origin
- The QRS duration must be ≥ 120 ms
- There should be a terminal R wave in lead V1 (e.g., R, rR′, rsR′, rSR′ or qR)
- There should be a slurred S wave in leads I and V6.

The T wave should be deflected opposite the terminal deflection of the QRS complex (Figure 2).

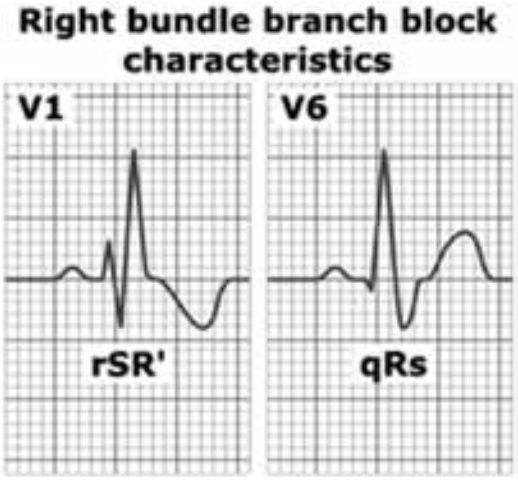

Figure 2 : ECG characteristics of a typical RBBB showing wide QRS complexes with a terminal R wave in lead V1 and slurred S wave in lead V6

Other ECG features to be considered in right bundle branch block include the following:

- For complete right bundle branch block, the duration of the QRS complex is prolonged for the patient's age. Maximum QRS durations are 70 ms for newborns less than 6 days, 80 ms for patients aged 1 week to 7 years, and 90 ms for patients aged 7-15 years.
- An rSR′ or rR′ pattern, with the initial r wave less than the R′ or r′, may be seen in leads V1-V3R .The initial R wave represents septal activation, the S wave represents left ventricular activation, and the R′ represents activation of the right ventricle from the septum and left ventricle.
- The S wave, which represents left ventricular activation, is wide in leads I and V6
- The QRS axis is usually normal, but right or left axis deviation may be present.
- The T wave is almost always inverted in lead V1 and may be inverted in V2. In the other precordial leads and in the limb leads, the T wave is directed opposite to the terminal portion of the QRS complex (Figure 3a & 3b).

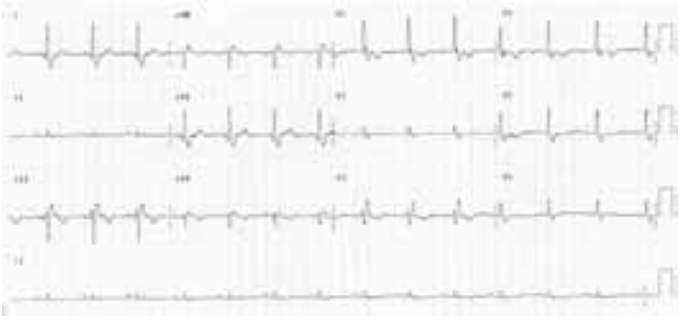

Figure 3a

Incomplete RBBB: The characteristic pattern in lead V1 is

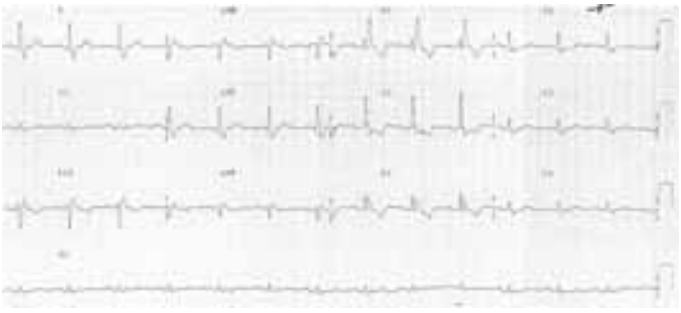

Figure 3b

rSr′ with an inverted T wave and a QRS duration of <0.12sec. Usually it is not involved with any pathologic process. The r′ deflection represents the normal terminal depolarization of the crista supraventricularis, proximal septum and the base of heart.

It may also depend upon the position of the heart relative to the exploring electrode. The incidence of incomplete RBBB increases as the precordial electrode is moved to the right. This was found in 4% of normal persons in lead V3R and in 6% in lead V4R.[5]

Tapia and Proudfit [6] have suggested the following criteria for normality:

a. Amplitude of initial R wave<0.8mV
b. Amplitude of r′ <0.6mV
c. R/S ratio of <1.0

In their study most of the normal subjects met these criteria.

Pathological incomplete RBBB is due to slowed conduction or delayed activation of the right ventricular conduction system.[4]

a. In acute corpulmonale as a result of massive pulmonary embolism the incomplete RBBB is caused by acute dilatation of the right ventricle causing a delay in conduction

b. Peri-ischaemic block may occur in the setting of inferior wall infarction with involvement of the right ventricle due to a slowed conduction in the ischaemic ventricle.

c. Incomplete RBBB is the most common pattern of aberrant intraventricular conduction to occur in supraventricular arrhythmias. This is because the right bundle branch is the site of longest action potential and the longest refractory period in the conduction system.

d. In right ventricular hypertrophy of any cause incomplete RBBB is caused by a slowed conduction in the hypertrophied ventricle.

e. Frequently seen following coronary bypass surgery and cardiac transplantation; the mechanism is not well understood.

ECG findings of RBBB and histopathologic changes of bundle branch have shown very good correlation. Lev et al in a pathological study of bundle branches in patients of coronary artery disease and RBBB found significant but incomplete lesions in all[7]. They suggested that presence of complete RBBB on ECG does not imply complete disruption of anatomic continuity of the bundle branch.

RBBB is a well known complication after right ventriculotomy. In a study by Krongrad et al there was no correlation between length of incision and QRS duration.[1] However it was seen with an incision of 1cm or less at a specific site in the distal portion of the right bundle branch. Electrophysiological studies in postoperative patients have identified that RBBB occurs at three levels-proximal, distal and the distal ramifications of the right bundle branch[8].

Right ventricular hypertrophy in isolation can cause RBBB pattern and there is a higher incidence of RBBB in those living at high altitudes[9]. In a study by Gatzoulis et al the effect of right ventricular volume overload in postoperative Tetralogy of Fallot patients with RBBB was analysed. They observed an increase in QRS duration with chronic right ventricular volume overload. This suggested that the QRS duration is affected by the degree of right ventricular distension[10].

RBBB in structurally normal hearts:

The prevalence of RBBB increases with age. In one prospective study of 855 men followed for 30 years, the prevalence was 0.8 percent in subjects at age 50 and 11.3 percent by age 80. There was no significant association with risk factors for or the presence of ischemic heart disease, myocardial infarction, or cardiovascular deaths, suggesting that RBBB is usually a marker of a slowly progressive degenerative disease that also affects the myocardium.

RBBB may occur in an otherwise normal heart as illustrated by the following observations:

- In a study of 237,000 airmen under age 30; there were 394 cases of complete RBBB, representing a prevalence of 0.2 percent.
- In a series 7392 middle-aged men without myocardial infarction or stroke, RBBB was present in 50 (0.7 percent).[11]

The prognosis for patients with isolated right bundle branch block is excellent because the course of right bundle branch block is generally benign

Electrophysiology of right bundle branch block

Three levels of right bundle branch block have been identified in electro physiologic studies.

a. Proximal, or central right bundle branch block occurs when a conduction block is present just distal to the bundle of His.

b. Another type of right bundle branch block occurs when the impulse is interrupted between the proximal and distal aspects of the right bundle branch.

c. Distal right bundle branch block is observed when distal ramifications of the right bundle are disrupted.

All the three levels of right bundle branch block are commoner after surgical intervention involving the right ventricle. Regardless of the type of right bundle branch block, the ECG patterns remain similar.

Natural history-In general when right bundle branch block occurs in isolation with a structurally normal heart it may not be of any concern. Also surgically induced right bundle branch block is not clinically significant and usually has a benign course. Rarely it may progress to complete heart block. Sudden death is a concern, particularly if the right bundle branch block is accompanied by additional evidence of injury to the His-Purkinje system (eg, left anterior hemiblock, first-degree AV block).

Patients who have undergone repair for Tetralogy of Fallot and who have an right bundle branch block pattern with a markedly prolonged QRS duration (>180 ms) may be at increased risk for important ventricular arrhythmias and sudden death. Patients with right bundle branch block from other causes may have diverse natural histories depending on the underlying disease. The outcome may be benign in some forms of familial right bundle branch block, or sudden death may result if the right bundle branch block pattern on ECG is due to Brugada syndrome.

References

1. Krongrad E, Hefler SE, Bowman FO Jr et al. Further observations on the etiology of right bundle branch block pattern following right ventriculotomy: Circulation 50:1105-1113,1974

2. Silver, Gotlieb, Schoen et al. Cardiovascular pathology 3rd edition:612,2001

3. Guiliani ER, Gersh B, Mc Goon M, Hayes DL, Schaff HV et al. Mayo Clinic Practice of Cardiology 3rd edition : 732.

4. Surwicz B, Knilans TK. Chou's electrocardiography in clinical practice.6th edition 95-107,2008.

5. Andersen HR, Nielsen D, Hansen LG. The normal right chest electrocardiogram. J Electrocardiol20:27,1987.

6. Tapia FA, Proudfit WL: Secondary R waves in right precordial leads in normal persons and in patients with cardiac disease.Circulation 21:28,1960.

7. Lev M, Unger PM, Lesser ME et al. Pathology of the conduction system in acquired heart disease: complete right bundle branch block. Am Heart J61:593,1961.

8. Horowitz LN, Alexander JA, Edmunds LH. Postoperative right bundle branch block: identification of three levels of block. Circulation 62:319,1980.

9. Laham J. Les Blocs de Branche en Clinique. Paris, Malsone Editeur, 1985.

10. Gatzoulis MA, Till JA, S omervilleJ et al. Mechano-electrical interaction in tetralogy of Fallot:QRS prolongationrelates to right ventricular size and predicts malignant ventricular arrhythmias and sudden death. Circulation92:231,1995

11. Eriksson P, Hansson PO, Eriksson H, Dellborg M. Bundle-branch block in a general male population: the study of men born 1913. Circulation 1998;98:2494

Stress Testing in Arrhythmias – Salient Aspects

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Stress ECG Testing is the cornerstone in evaluating patients with chest pain and high-risk asymptomatic people for the evidence of ischaemia. However, Stress ECG Testing can also be performed for the evaluation of arrhythmias.

The ACC/AHA have recommended following guidelines for evaluation of arrhythmias by Stress ECG Testing[1] :

Class I

- Identification of appropriate settings in patients with rate-adaptive pacemakers
- Evaluation of congenital complete heart block in patients considering increased physical activity or participation in competitive sports

Class II a

- Evaluation of patients with known or suspected exercise induced arrhythmias
- Evaluation of medical, surgical, or ablative therapy in patients with exercise induced arrhythmias (including atrial fibrillation)

Evaluation of Ventricular Arrhythmias

- If underlying ischaemia is suspected, Stress ECG Testing may uncover this
- Serious arrhythmias are uncommon in unselected population undergoing exercise testing

Udall et al[2] reported a follow up study in 6500 patients who underwent exercise testing:

- 1327 patients exhibited VPBs during exercise
- Subsequent annual coronary events
  - Neither ST changes nor VPBs 1.7%
  - VPBs alone 6.4%
  - VPBs and ST changes both 11.4%
- Prognostic Value
  - 12 month mortality is 3 times greater in persons exhibiting exercise-induced ectopy
  - Mortality with complex ectopy exceeds that for those with simple ectopy

Paris Prospective Study I[3] reported :

- EIVAs (Exercise induced Ventricular Activation) – Frequent PVCs - > 10% of all the beats during any 30 second recording, or a run of > or = 2 consecutive PVCs during exercise or recovery
- 6101 males, free of cardiovascular disease
- Follow up – 23 years
- EIVAs were seen only in 2.3% of the above population
- Cardiovascular mortality – 2.6 times higher in patients with frequent VPBs
- Independent of age, cardiovascular risk factors and ST segment depression

Veterans Affairs Medical Centers[4] reported :

- EIVAs – Frequent PVCs - > 10% of all the beats during any 30 second recording, or a run of > or = 3 consecutive PVCs during exercise or recovery
- 6213 consecutive males – mean follow up 6 +/- 4 years
- 1256 patients died (20%) died during follow up

Partington S et al[5] reported the prevalence and prognostic value of exercise-induced ventricular arrhythmias as below **:**

- EIVAs – 503 patients (8%)
- Prevalence of EIVAs increased in older patients, with cardiopulmonary disease, resting PVCs and ischaemia during exercise

Tamakoshi K et al[6] (2002) reported :

- 25075 consecutive patients – 14037 men and 11038 women
- Mean age 53.3 +/- 8.8 years
- Non-sustained VT – 8 or more consecutive ventricular ectopic beats at > 100 beats/min.
- Twenty patients (0.08%) had exercise-induced VT.

VPBs after Exercise

If VPBs appear during exercise or during recovery, does it have any significance?

- After cessation of exercise, parasympathetic tone should be reactivated quickly and should suppress catecholaminergic

ventricular ectopy

- If VPBs persist after exercise – marker of attenuated vagal reactivation with prognostic significance

Frolkis JP et al[7] reported that VPBs occuring during recovery have more significance :

- 30000 patients
- Follow-up period – 5.3 years
- 1862 patients died
- VPBs during recovery after exercise – increased risk of all-cause mortality (hazard ratio 2.4)

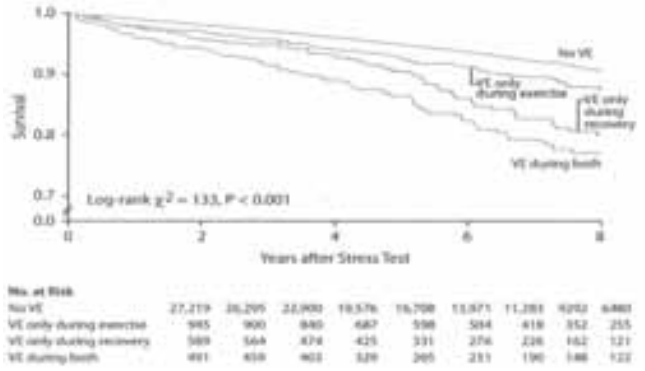


Figure 1 : Prognostic impact of ventricular ectopy (VE) during and after exercise. Adapted from Frolkis JP et al N Engl J Med (2003)

O'Neill JO et al[8] reported the prognostic value of VPBS in presence of LV dysfunction :

- 2123 patients with LVEF ≤ 35%
- Severe VPBs (triplets, NSVTs and VTs) were noted in 7% of the study group
- 530 deaths in 2.9 years, significantly more in patients with complex VPBs (3 year mortality 37% versus 22%, hazard ratio 1.76)

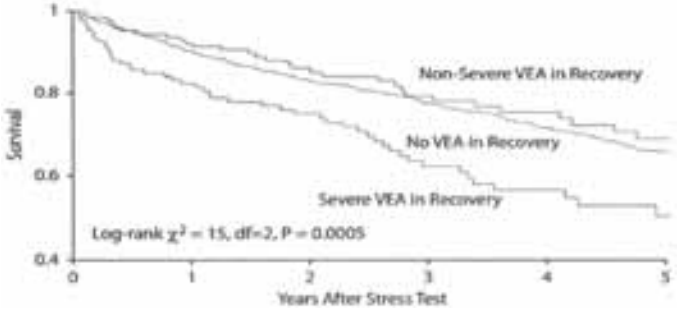


Figure 2 : Prognostic impact of ventricular ectopy activity (VEA) after exercise. Adapted from O'Neill JO et al J Am Coll Cardiol (2004)

Role of Exercise ECG Testing in Supraventricular Arrhythmias

WPW Syndrome :

- Exercise testing – to evaluate the risk of developing rapid ventricular response during atrial arrhythmias
- Abrupt loss of pre-excitation during exercise – longer antegrade refractory period in the accessory pathway than in the AV node – unlikely to develop rapid ventricular response

Atrial fibrillation :

- Effective rate control at rest – does not necessarily signify adequate rate control during exercise
- Titration of additional drugs for this purpose may be felicitated by exercise testing

Sinus Node Dysfunction

- Exercise testing may distinguish resting bradycardia with a normal exercise heart rate response from sinus node dysfunction with resting bradycardia
- However, normal test result dose not negate the possibility of sinus node dysfunction

Role of Stress ECG Testing in patients for Cardiac Pacemakers

- Exercise testing – inappropriate in most patients with a permanent pacemaker
- Adaptive rate pacemakers – exercise response may be used to fine tune these devices
- Evaluation of quality of atrial sensing – best expressed by the percentage of synchronized atrioventricular events
- Evaluation of evolution of P-wave amplitude during exercise

Stress ECG Testing in patients with Implantable Cardiac Defibrillators

- May provoke arrhythmias or ICD discharge
- Test to be terminated 10 bpm below the detection interval of the device
- Slower programmed rates – reprogrammed to faster heart rate or temporarily deactivated
- Care should be taken to avoid unnecessary shocks

Role of Stress ECG Testing in patients with LBBB :

- Exercise-induced ST depression usually with LBBB and has no association with ischaemia
- Even upto 10 mm ST depression can occur in healthy nor-

mal subjects

- There is no level of ST-segment depression that confers diagnostic significance in LBBB
- However, Stress ECG Testing can be performed to judge the effort tolerance, other vital parameters and patient's symptomatology

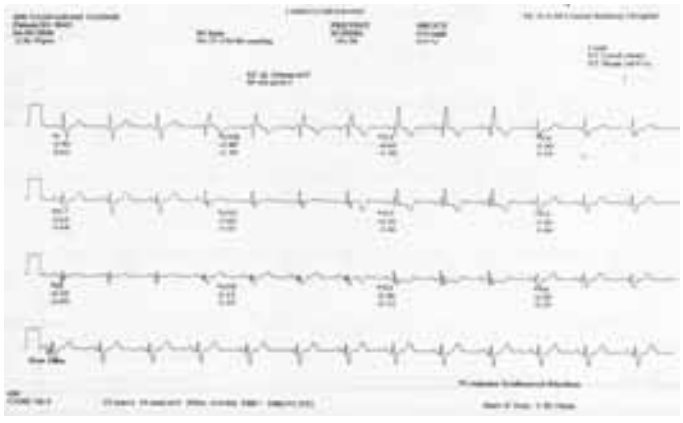
Exercise induced LBBB :

Incidence :

- Transient LAHB – 0.3 %
- Transient LBBB – 0.4 %

Candell Riera J et al reported :

- Exercise-induced LBBB does not always denote the presence of underlying CAD
- 9318 consecutive stress tests
- 20 patients – Exercised-induced LBBB
- Mean follow up 6.9 years
- 8 normal coronaries (A)
- 12 had CAD (B)
- Group A
  - 7 patients had chest pain coinciding with the first beat of LBBB
  - No deaths
  - Permanent LBBB appeared in follow up
- Group B
  - 3 deaths, 2 Acute MI
- One patient in each group developed AV Block requiring PPM



Figigure 3A : Stress ECG Testing in a patient of RBBB (Pre-Test ECG)

Conclusions

- Prognosis of patients with painful LBBB and normal coronaries is good
- Development of permanent LBBB is frequent
- AV Block, although rare, may occur.

Vasey et al reported :

- 2584 patients – Rate dependent LBBB – 28 patients (1.1%)
- 7 out of 10 patients developed classical angina pectoris – found to have CAD
- 12 out of 13 patients – atypical chest pain – normal coronaries
- All 10 patients who developed LBBB at a heart rate of 125 beats or higher – free of CAD
- 9 out of 18 patients who developed LBBB at a heart rate of < 125 bpm had CAD

Grady TA et al[9] concluded :

- Exercise induced LBBB was associated with a three times higher risk of death and major cardiac events
- Exercise induced LBBB – in association with or without structural heart disease (SHD)
- Pooled mortality:
  - 2.7 % per year – SHD
  - 0.2 % per year – No SHD

Role of Stress Testing in the presence of RBBB :

- Exercise-induced ST depression usually occurs with RBBB in anterior chest leads (V1 – V3) and is not associated with ischaemia

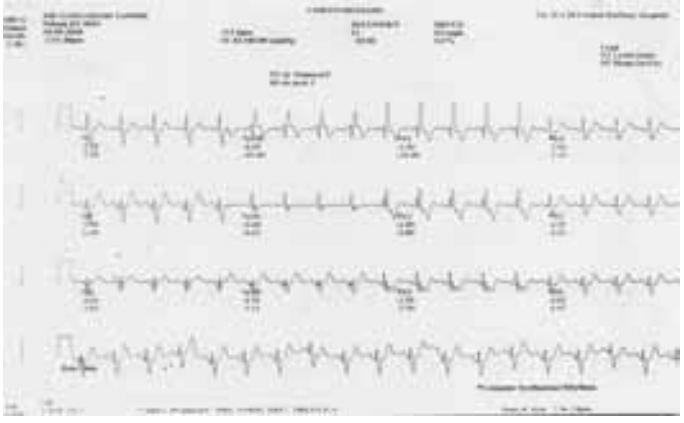


Figure 3 (B) : Stress ECG Testing in the above patient of RBBB (ECG at the peak-exercise), showing false positive changes seen in leads $V_1 – V_3$

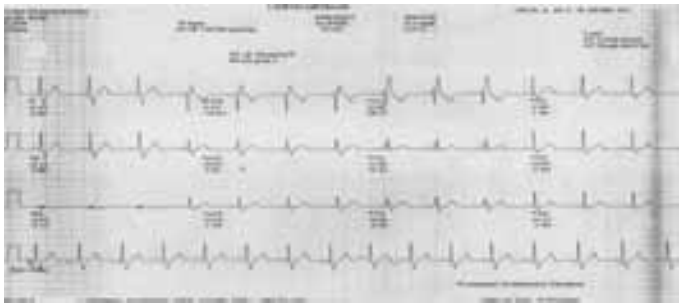


Figure 4 (A) : Stress ECG Testing in a patient of RBBB (Pre-Test ECG)

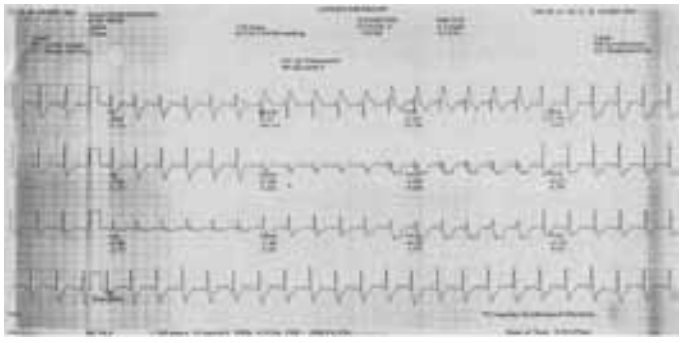


Figure 4 (B) : Stress ECG Testing in the above patient of RBBB (ECG at the peak-exercise), showing true positive changes seen in inferior leads and leads $V_4 - V_6$

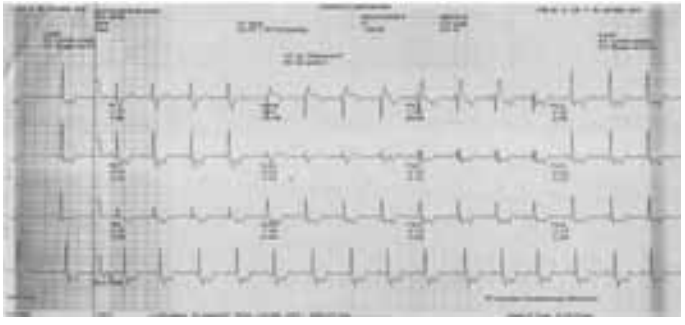


Figure 4 (C) : Stress ECG Testing in the above patient of RBBB (ECG during recovery), showing true positive changes seen in inferior leads and leads $V_4 - V_6$

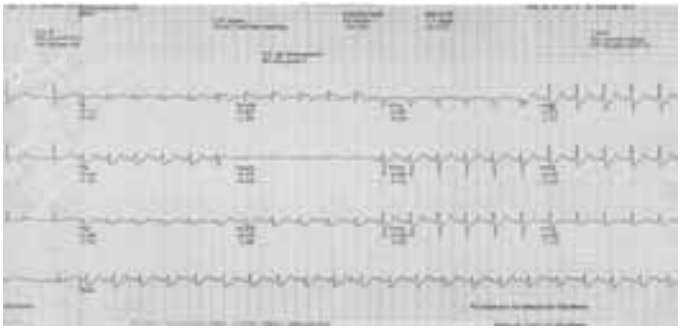


Figure 5 (A) : Stress ECG Testing in a patient with base line normal ECG

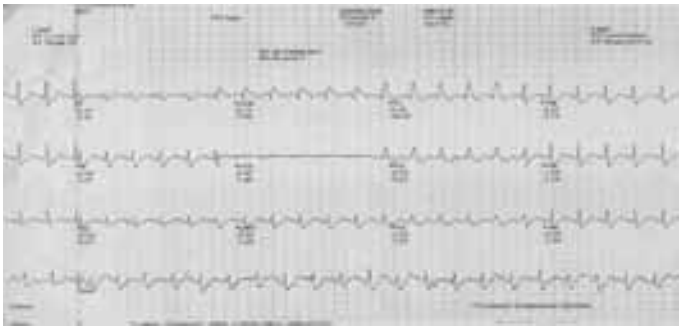


Figure 5 (B) : Stress ECG Testing in the above patient – developed exercise-induced RBBB

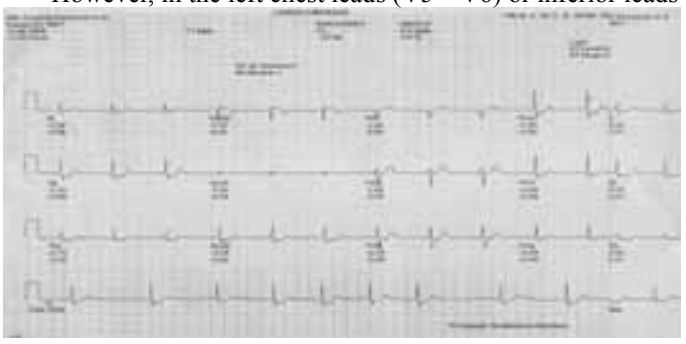


Figure 5 (C) : Stress ECG Testing in the above patient who developed exercise-induced RBBB, returned back to normal during recovery

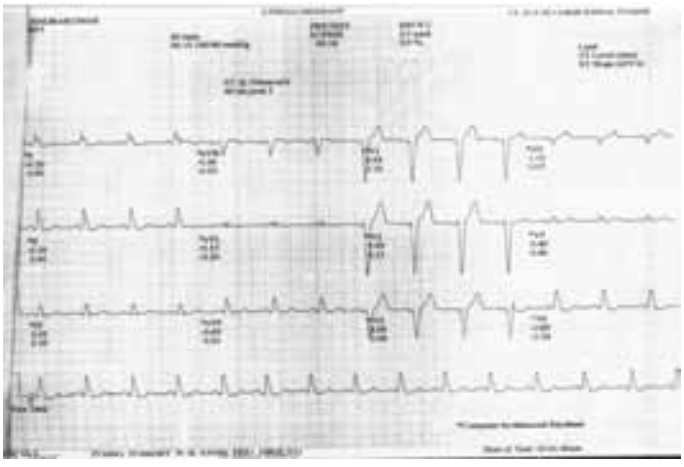


Figure 6 (A) : Stress ECG Testing in a patient of LBBB (Pre-Test ECG)

- However, in the left chest leads (V5 – V6) or inferior leads (II and aVF), test characteristics are similar to those of a normal resting ECG.
- The presence of RBBB does not appear to reduce the sensitivity, specificity or predictive value of the stress test for the diagnosis of ischaemia

Exercise induced RBBB :

- 0.1%

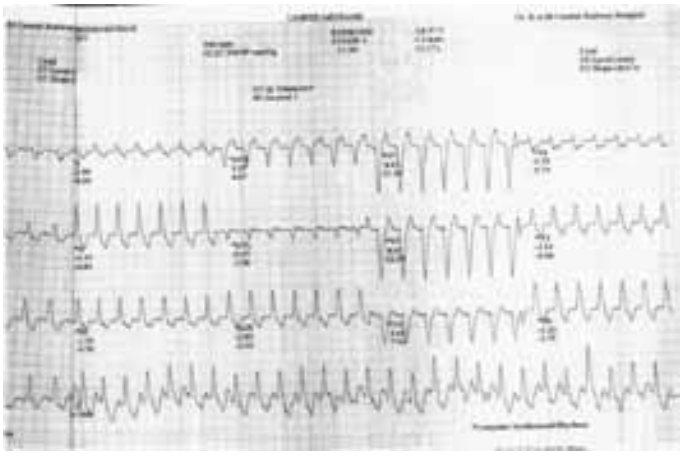


Figure 6 (B) : Stress ECG Testing in the above patient of LBBB (ECG at the peak-exercise), showing false positive changes

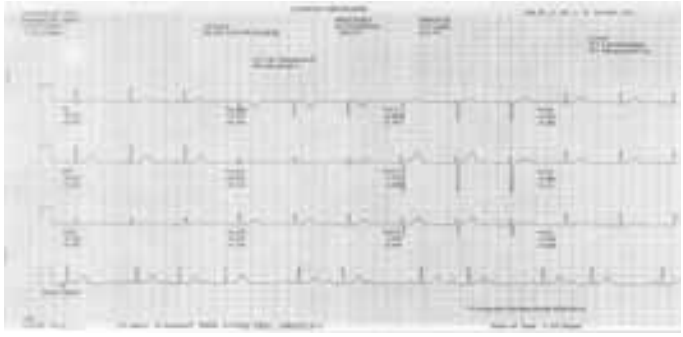


Figure 7 (A) : Stress ECG Testing in a patient of AV Block Mobitz Type I (Pre-Test)

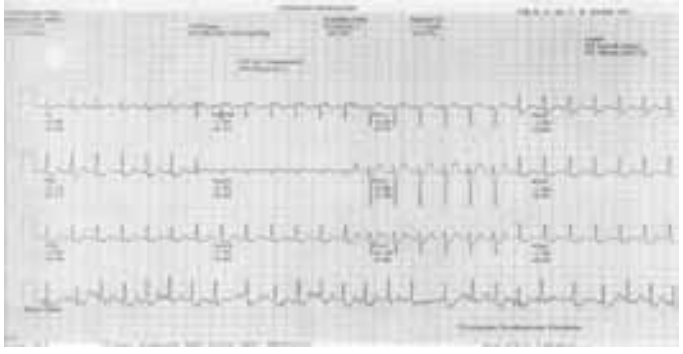


Figure 7 (B) : Stress ECG Testing in a patient of AV Block Mobitz Type I (During Exercise) showing improvement in conduction

- Significance – not known
- Mostly benign rate-related RBBB

Complications arising during Stress ECG Testing

Sometimes complications may arise during such testing. One should be prepared in the exercise room to resuscitate the patient with fully equipped facilities of defibrillator, endotracheal

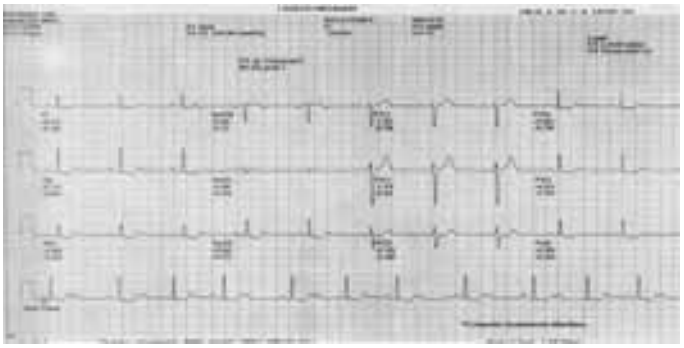


Figure 7 (C) : Stress ECG Testing in a patient of AV Block Mobitz Type I (During Recovery) – Reappearance of block

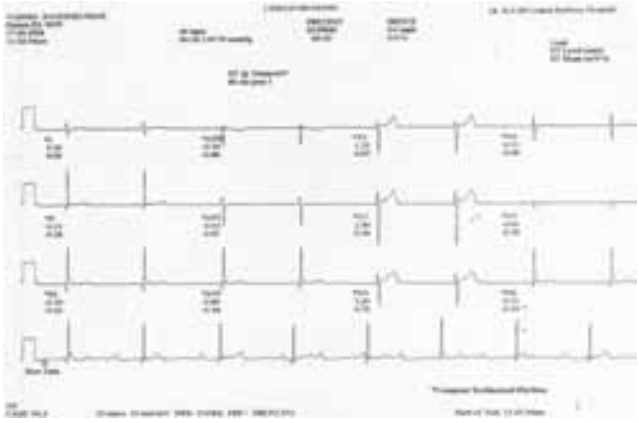


Figure 8 (A) : Stress ECG Testing in a patient of CHB with narrow QRS escape rhythm (Pre-Test)

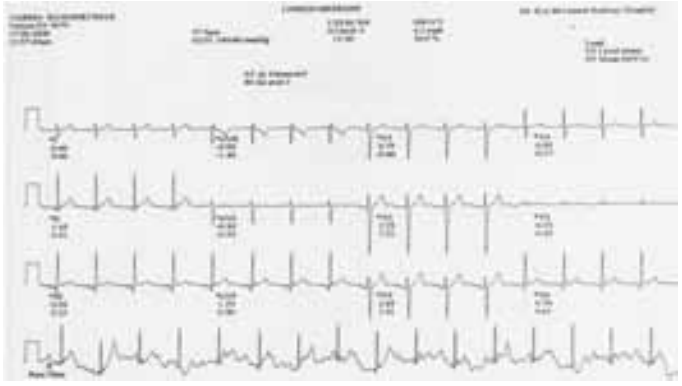


Figure 8 (B) : Good chronotropic response

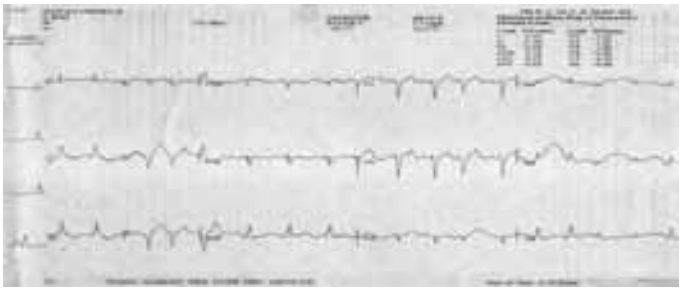


Figure 9 (A) : Stress ECG testing in a patient with old CABG evaluated for ventricular arrhythmias (Pre-Test ECG)

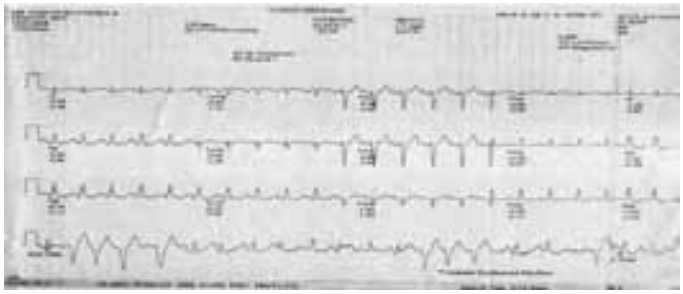


Figure 9 (B) : Developed frequent PVCs

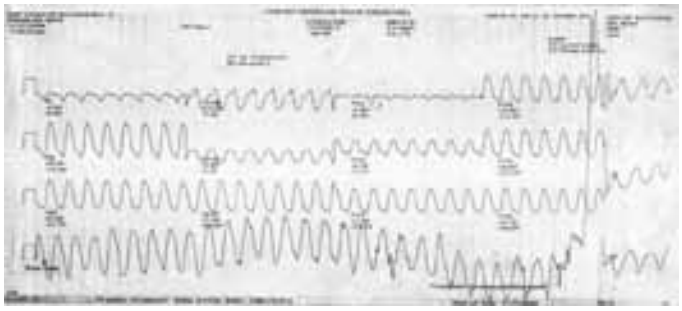


Figure 9 (C) : Later, developed monomorphic VT

intubation and respiratory support with necessary life- saving injections.

The patient collapsed, was successfully resuscitated and later underwent an ICD implantation.

Conclusion

Stress Testing in Arrhythmias, if done judiciously can provide a good deal of diagnostic and prognostic information and is a quiet safe diagnostic tool

References

1. Gibbons RJ, Balady GJ, Bricker JT et. al. ACC/AHA 2002 Guideline Update for Exercise Testing : Summary Article. Circulation 2002; 106:1883-1892.
2. Udall JA, Ellestad MH. Predictive implications of ventricular premature contractions associated with treadmill stress testing. Circulation 1977; 56:985-989.
3. Jouven X, Zureik M, Desnos M et. al. Long-term outcome in asymptomatic men with exercise induced premature ventricular depolarizations. N Engl J Med 2000; 343:826-833.
4. Beckerman J, Mathur A, Stahr S et. al. Exercise-induced ventricular arrhythmias and cardiovascular death. Ann Noninvas Electrophysiol 2005; 10:47-52.
5. Partington S, Myers J, Cho S et. al. Prevalence and prognostic value of exercise-induced ventricular arrhythmias. Am Heart J 2003; 145:139-146.
6. Tamakoshi K, Fukuda E, Tajima A et. al. Prevalence and clinical background of exercise-induced ventricular tachycardia during exercise testing. J Cardiol 2002; 39:205-212.
7. Frolkis JP, Pothier CE, Blackstone EH, Lauer MS. Frequent ventricular ectopy after exercise as a predictor of death. N Eng J Med. 2003; 348:781-790.
8. O'Neill JO, Young JB, Pothier CE, Lauer MS. Severe frequent ventricular ectopy after exercise as a predictor of death in patients with heart failure. J Am Coll Cardiol 2004; 44:820-826.
9. Grady TA, Chiu AC, Snader CE, et. al. Prognostic significance of exercise-induced left bundle branch block. JAMA 1998; 279:153-156.

ECG Phenomena of the Early Ventricular Repolarization in the 21 Century

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Key Words: Early Ventricular Repolarization : Syndromes.

Introduction and Terminology

Clinical interest in electrocardiogrpahic (ECG) phenomena of early ventricular repolarization (EVR) has been rekindled recently mainly because of their clinically established association with fatal cardiac arrhythmias, particularly in otherwise healthy individuals with no (or minimal) structural diseases of the heart. ECG phenomena of EVR have often been misdiagnosed, misinterpreted, or undermined. this happened mainly because of prevailing opinion of the "beningn" or misleading" nature of various EVR phenomena. For instance, early repolarization changes consistent with Brugada syndrome have been interpreted "innocent" and overlooked for decades until 1992.[1] Another example - so - called "early repolarization syndrome" (ERS) that universally and unequivocally has been regarded as "normal", normal variant", beningn early replarization" ntil 2000.[2] In 2008, seminal article by Haïssaguerre et al.[3] accompanied by editorial comments by Wellens[4] and letter to the editor by Nam et al.[5] brought clinical attention to an incr3eased prevalence of the ERS among patients with a history of idiopathic ventricular fibrillation (VF).

The ECG phenomena of EVR include (but not are limited to):

- J-deflections
- "Slurring or notching" of the QRS complex (with or with-out J-deflections)
- J-waves (with or without ST-segment elevation)

The clinical validity of ECG manifestations of EVR as makers of sudden cardiac death (SCD) and their utility in risk stratification are not well defined. In addition, it is common for such ECG phenomena to vary significantly depending upon a variety of intrinsic cardiac and extracardiac factors. The main focus of this review is to characterize the *ECG patterns of the EVR* with a special focus on the ERS and its arrhythmogenic potential.

Ventricular repolarization begins when ventricular depolarization ends. In the normal heart, the evolutin of depolarization into repolarization is relatively short process, and the magnitufde of the overalp between the latest depolarization and the earliest repolarization does not exceed 10 msec.[6] The duration of this temporal overlap between the end of depolarization and the beginning of repolarization is greatly influenced by various physiological and pathological, cardiac and extracardiac conditions that affect either: (a) propagation of the excitation wave through venricular wall or (b) recovery of its excitability, or (c) both. Among intrinsic cardiac factors are:

- An intrinsic configuration of the early phase of the actions potentials in different ventricular layers (endomyocardium, midmyocardium and epicardium)
- A modification o the time-course of repolarization across the ventricular wall (transventricular vector gradient).

In a normal ECG, the transition of ventricular depolarization into ventricular repolarization corresponds onthe surface ECG to the *J-point.* The J-point defined as the point at which there is abrupt transition from the QRS complex to the ST-segment. Deviation of the J-point from the isoelectric line leads to the presence of a *J-deflection,* which is a common ECG feature of ERS, but also seen in acute myocardial ischemia, hypercalemia, and various intraventricular conduction disturbances. A J-deflection inscribed on either the downsloping limb of the QRS complex or S wave is commonly regarded as a *QRS-notching,* whereas a smooth and prolonged ransition from the QRS segment to the ST segment - *QRS-slurring.* Increased amplitude and duration of the J-deflection that takes the shape of a 'dome or a hump' is usually described as a *J-wave*.[6]

Noteworthy to emphasize that a clear distinction between the delayed conduction and the EVR cannot always be made on the basis of an ECG alone. Nevertheless, distinguishing the two is very important since commonly EVR presents greater arrhythmia risk than a delayed intraventricular conduction, especially in otherwise healthy individuals. To limited extent, depolarization and repolarization processes can be differentiated based by their differneces with respect to the heart rate, different cardiac drugs, and neurotransmitters (see below). The signal-averaged ECG could be helpful diagnostic tool but of limited clinical value in many cases,[7] and the search for new ECG tools and makers to differentiate these two processes is accelerating.

"Slurring or Notching" of the QRS Complex

In 1998, Garg and his associates[8] reported a case of "familial sudden cardiac death associated with a terminal QRS-abnormality on surface 12-lead electrocardiogram". the

abnormal low-amplitude deflections in the downsloping limp of the QRS complex in leads II, III, aVF and I, aVF and I, aVL and V6 were coincident with the late potential on the signal-averaged ECG. This deflection appeared to be more prominent after procainamide or beta-blockers and normalized after quinidine. Moreover, sustained polymorphic ventricular tachycardia, which degenerated to VF was easily inducible during programmed stimulation from the right ventricular apex, despite administration of procainamide or atenolol. The clinical significance of these ECG findings is not fully understood at present and further investigation is warranted.

J-Waves

Different names have been used at different time for the J-wave. They include "camel hump sign", "hathook junction", "K wave", "H wave", "late delta wave", "current of injury", "J point wave", "hypothermic wave", "hypothermic hump", "Osborn wave", J waves can be classified as :

- Hypothermic
- Non-hypothermic
- Idiopathic

Hypothermic J-waves

Clinical as well as experimental data linking hypothermic J-waves and cardiac arrhythmias remain sparse and somewhat conradictory; the occurence of ventricular tachyarrhythmias associated with hypothermic J-wave varies from 0 to almost 100%.[9] Sodium channel blockers, such as procainamide and lidocaine, are inefective, indeed proarrhythmic in both the prevention and treatment of the malignant ventricular tachyarrhythmias in hypothermic patients during their rewarming.[9]

Non-hypothermic J-Waves

ECG changes resembling those in hypothermia-induced J-waves have been observed in a various clinical and experimental settings with normal body temperature, such as acute myocardial ischemia, acute pulmonary thromboembolism, right ventricular infarction, electrolyte or metabolic disorders, pulmonary or inflammatory diseases, or abnormalities of cenral or peripheral nervous system, intoxication by heterocyclic antidepresssant or cocaine, and many other abnormal conditions.[10] Among those clinical situations, prominent J-waves most frequently are observed in acute myocardial ischemia and hypercalemia, and their arrhythmogenic potentials in most clinical settings are chiefly dependent upon the underlying disease.

Idiopathic J-Waves

In the absence of any structural cardiac abnormalities or extracardiac disease, changes of EVR can be classified as primary or "idiopathic". Several forms of idopathic appearance of a J-wave in human with or without accompanying ST-segment elevation have been described. Among them, the most investigated is so-called ECG marker of Brugada syndrome. Idiopathic J-wave followed by downsloping ST-segment elevation with inverted T-waves in the right chest leads is an ECG hall mark of the "typical" Brugada syndrome.

However, in many clinical cases of symptomatic Brugada syndrome (atrial and/or ventricular tachyarrhythmias or SCD), the pattern of ST-T abnormalities and/or their ECG "localization" is different from that in the "typical" Brugada Syndrome. In such cases, the terms "atypical" or "J-wave - like" are appropriate. Clinically noteworthy that J-wave-like ECG abnormalities in leads other that the right precordial (e.g. inferior or lateral) ("atypical" Brugada syndrome) have been also described in otherwise healthy individuals prone to paroxysmal ventricular tachycardia/fibrillation.

Early Repolarization Syndrome

The term "early repolarization syndrome" was introduced nearly half a century ago and has traditionally been regarded as idiopathic and benign or "innocent"[11] or "misleading"[12] ECG pattern of EVR until 2000.[2] The ERS has been ascribed a number of names, including "early repolarization", "early ventricular repolarization", "benign early repolarization", "benign J wave", "nonspecific changes of ventricular repolarization", "repolarization variant", "normal variant RS-T segment elevation", and "juvenile or unconventional ST-T pattern" and cetera to describe the "characteristic" ECG pattern of J-deflection followed by horizontal ST-segment elevation in the mid-precordial leads.[2]

To avoid further confusion and inconsistencies with terminology, we prefer to use the term "early repolarization syndrome", acknowledging at the same that this term is not the best one to use in the cases of asymptomatic forms of EVR. By its own definition, any clinical syndrome is a combination of signs and symptoms that occur together and characterize a particular abnormality. The term "syndrome" is best reserved for a description of clinical manifestation of the disease. In this context, the "true ERS" is best defined as an arrhythmogenic entity that is characterized by (a) ECG marker of ERS that is associated with (b) arrhythmogenic complications, including SCD, and/or family history of SCD in otherwise healthy individual.

The prevalence of ERS varies between 1%[13] and 2%.[14] It is more commonly seen in young individuals (27.5%),[15] especially those predisposed to vagotonia, and shows a clear *mate preponderance* (77%).[13] The syndrome is also often observed in:

- Athletes[16]
- Cocaine users[17]
- Obstructive hypertrophic cardiomyopathy[18]
- Defects and/or hypertrophy of the interventricular sep-

tum.[18]

The electrocardiographic manifestation of ERS is often dependent on heart rate. normalizing during exercise or with rapid pacing, as well as with advancing age.[19] Familial occurence of the syndrome has been observed.[20,21] Althoughearly studies were interpreted to suggest that the syndrome is more prevalent in the Black population. More-recent studies challenge this notion.[22]

In 2000, based on preclinical experimental work from Dr. Charles Antzelevitch, it has been suggested that: (a) ERS should not be considered as normal or benign ECG abnormality *a priori,* unless otherwise proven , as generally thought and (b) under certain conditions known to predispose to ST-segment elevation, patients with ERS may be at greater arrhythmogenic risk.[2]

The classical ECG pattern of ERS consists of : (a) prominent notch or slur on the downsloping portion of the QRS complex, or J-deflection, (b) followed by diffuse upward ST segment concavity concordant with the QRS complex, and (c) positive T wave in the same lead. Additional ECG feature include:

- *Localization* of the ECG pattern of ERS in scalar ECG. Mid-to-lateral precordial leads V_2-$V_{4\ (5)}$ hve been recognized as showing the most prominent repolarization changes consistent with ERS. Notewrothy, similar changes might appear in other leads but to a lesser extent
- Reciprocal ST segment depression in aVR
- "Waxing and waning" of the ST-T segment over time
- Often, ERS is associated with shorter-than-normal QT interval duration, even adjusted to the heart rate.

Nevertheless, in many clinical instances, it is still very difficult to distinguish subjects with ERS from those with the Brugada syndrome or various intraventricular conduction blocks, or Short QT syndrome based solely on a resting ECG. Furthermore, the ECG alterations in response to changes in heart rate, drugs effects, and autonomic tone observed in ERS are very similar to those observed (a) under hypothermic conditions and (b) the Brugada syndrome (**Table 1**)

Many clinical features of ERS are similar to that of Brugada syndrome, including (a) predominance in young otherwise healthy males, (b) predisposition to familial occurence, (c) in many individuals, a transient normalization of ECG manifestation, and (d) ERS similar responses to drugs and autonomic modulation.

Two major features permit differentiation of the ECG signatures of ERS and the Brugada syndrome: (a) pattern and (b) leads specificity. The elevated ST-segment in ERS is usually localized in leads V_2-$V_{4\ (5)}$ and has an upward concavity with positive T-wave polarity accompanied by a notched (or slurred) J-deflection. In contrast, the ECG of Brugada patients generally displays a prominent J-point elevation (J-wave), followed by a downsloping ST segments and negative T-wave in the right precordial leads V_1-V_3 ("typical") or other leads ("atypical").

It is often difficult to distinguish subjects with ERS from those with the Brugada syndrome, especially when the latter also display intraventricular conduction slowing or block. In 1993. Aizawa and his colleagues[23] discribed several patients with idiopathic ventricular fibrillation in whom they found "bradycardia-dependent *intraventricular block".* The common ECG features of these patients included: (a) incomplete right bundle branch block, (b) prominent "notch" on downsloping limb of the QRS complex in leads V_3-V_5, II, III and aVF, (c) elevated ST segment with positive T waves leads V_2-V_3, and (d) rate (deceleration) - dependent accentuation of the prominence of the "notch".

The dynamicity of ECG changes in ERS and Brugada syndrome is also confounding. When the clinical history is malignant, an invasive electrophysiological study examining the inducibility of ventricular tachycardia/fibrillation may be useful in assessing arrhythmogenic risk.

Interestingly, ECG changes consistent with the Brugada syndrome have also been referred to as a benign repolarization variant or "Ideiken phenomenon" for more than three decades and some patients having such ECG abnormalities have been described as "unwitting victims of electrocardiography".[2]

No doubt, the intrinsic capability of the standard 12-lead ECG are significantly limited in the differential diagnosis of the ERS, since only a small portion of the available diagnostic information can be obtained from conventional scalar 12-lead ECG tracings. The search for new additional and more advanced and sophisticated computer-assisted methodologies and ECG markers that can help to differentiate ST-segment elevations in ERS from other ECG phenomena of EVR (e.g. Brugada syndrome, acute ischemia) have intensified in most recent

Table 1 : Autonomic and pharmacologic modulation of the J-wave magnitude under hypothermic conditions, in the early repolarization (ERS) and Brugada syndrome (BrS).

	Sodium blockers	Isopro-terenol	β-blockers	Exercise	Nitroglycerine
Hypothermic JWave	↑	↓	↑	↓	**not determined**
ERS	↑	↓	↑	↓	**no effect**
BrS	↑	↓	↑	↓	**no effect**

years. The "three-dimensional (3D) approach" is expected to be valuable in EVR evaluation. Some of those 3D-ECG markers (e.g. ST-T and QRS-T angles) are already in their "clinical validation" stages of development (**Figure 1**).

Possible Cellular and Ionic Mechanisms

Concordant with the QRS complex. ST segment elevation is most commonly recognized as a sign of acute myocardial damage, often associated with the development of cardiac arrhythmias. The electrophysiological nature of the ST-segment elevation in ERS, whether idiopathic or due to so-called "current of injury", has been investigated by means of a direct-current magnetocardiogram, which, in contrast to conventional 12-lead ECG, is capable of determining a TQ interval shifts. The results showed clearly that ST-segment shifts in subjects with ERS are unrelated to ischemic injury.[24]

One might also explain the principal difference between ERS and Brugada syndrome based on an abbreviation of the action potential due to loss of its "done" resulting in the development of a very significant transmural as well as epicardial dispersion of repolarization and refractoriness, setting the stage for both phase 2 and circus movement reentry culminating in much more severe arrhythmogenic manifestations of the Brugada syndrome if compared with ERS.[2]

Some believe that parietal (myocardial)conduction defects (delayed conduction) could contribute to both the ECG manifestation and the arrhythmogenic potentials of the different ECG phenomena of EVR.

Figure 1 : Acute ischemia induces wide angle between QRS and T vector loops, but the angle remains narrow in ST elevation associated with early repolarization. In acute myocardial ischemia, the QRS and T loops become widely divergent and the spatial angle between them is greater than 75^0 in a significant majority of cases. In contrast, the angle is unaffected in early repolarization (from http://www.newcardio.com)

Modulation by Drugs, Rate, and Neurotransmitters

The changes in the magnitude of the EVR abnormalities in ERS and Brugada syndrome, like a hypothermic J wave, display qualitatively similar responses to a variety of drugs as well as to changes in rate and autonomic tone (**Table 1**)[2]:

- *Slowing of heart rate* exaggerates J-waves and ST-segment elevation, while increase in heart rate during exercise or following isoproterenol reduces or even eleiminates these ECG abnormalities
- *Sodium channel blockers* (known to unmask the Brugada syndrome) increase ST-segment elevation in ERS subjects and hypothermic J-waves
- *Sympathetic stimulation and ß-adrenergic* agonists normalize the ST-segment in both syndromes, whereas β-adrenergic blockers augment ST-segment elevation in both, while propranolol increases its magnitude and its toxicity may even induce classical pattern of ERS.

The Role of Nervous System

Clinical and experimental studies point to high spinal cord injury as a cause of ERS-like changes in the ECG. High cervical spinal chord injury can lead to significant deterioration or even complete disruption of the cardiac sympathetic activity, leaving parasympathetic activity unopposed.[25] parasympathetic activation has an opposite effect in both syndromes, causing ST segment elevation due to depression or loss of the action potential plateau (see below). ECG patterns "wax ans wane" in both syndromes, possibly due to variations of autonomic activity.[2]

Arrhythmogenic Potential

In vast majority of cases, ERS *is not a malignant* EVR syndrome per se, *unless otherwise proven.* However, the similarities between the Brugada syndrome and ERS in response to rate neuromodulation and pharmacologic agents strogly suggest a parallelism of mechanisms.

When ERS has long been considered to be benign, in experimental models, the ECG signature of ERS can be ***converted*** to that of the Brugada syndrome.[2, 26, 27] This raises the possibility that ERS *may not be as bening (unless otherwise proven)* as generally believed, and that under certain conditions known to predispose to ST-segment elevation, ERS subjects may be at increased risk. In considering these possibilities, it is instructive to remember that (a) the Brugada syndrome was regarded as bening for more than three decades, and (b) that one syndrome can be readily converted to the other in experimental models involving the wedge preparation.

One hypothesis presumes that depression of the epicardial, but not endocardial, action potential plateu creates a transmural gradient that manifests on the scalar ECG as a complex ST-segment elevation with a positive T-wave. As such the cellular substrate is not arrhythmogenic, but in case of further increase in net repolarizing current as a result of complete loss of the epicardial action potential dome and the attendant development of a large transmural dispersion of repolarization could become arrhythmogenic. This proposed mechanism also explain the

highly arrhythmogenic character of the Brugada syndrome and apparently benign exceptions.

Alternative hypothesis is based on assumption that the delayed (parietal) intraventricular conduction delay in the proximity of interventricular septum (mid-precordial leads could contribute to the ECG phenomenon of ERS and its potential link to electrical instability.

It is important to emphasize that these hypotheses remain to be more rigorously tested and that the distinct nosologic entity that is referred as the ERS needs to be more fully delineated within the framework of what we have learned about the Brugada syndrome in recent years. A careful clinical history and invasive electrophysiological studies may be required to determine whether or not the early ventricular repolarization abnormalities in a given patient are benign or malignant.

Summary, Clinical Implementation, and Future Directions

The available clinical data suggest that the different ECG phenomena of the EVR syndromes often share similar ECG presentations, mechanisms with wide range of their arrhythmogenic potentials. ECg similarities between different ECG phenomena of EVR raise certain concern for their misdiagnosis and clinical relevance.

In our opinion :

1. ERS, as a diagnosis, should not be regarded as either benign or malignant *a priori* unless otherwise proven.
2. Clinical judgment based on clinical presentation ("arrhythmogenic" anamnesis), family history or syncope/SCD, potential use of cardio-active drugs, including psychotropic medications is the most essential in the risk stratification of the ERS subject. Special attention should be devoted to the family history of primary EVR abnormalities and/or SCD.
3. Additional diagnostic work-up, such as tilt-test, signal-averaging ECG, and electrophysiological studies with or without drug testing, should be considered on the case-by-case basis upon physician's discretion. Genetic screening of ERS subjects at risk, once availabe, will be one of the most important diagnostic tools to identify and/or confirm the diagnosis of primary electrical abnormalities/diseases of the EVR.
4. It is important to keep in mind that ERS subjects might be predisposed to the drug-induced ventricular arrhythmias.
5. Standard 12-lead ECG is very valuable diagnostic tool to identify ERS subjects, yet its clinical utility in the risk stratification is of limited intrinsic value. New ECG technologies, methodologies, and markers should be developed and clinically validated to help to identify and differentiate ERS from other forms of EVR as well as to stratify this risk.

References

1. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome. A multicenter report. J Am Coll Cardiol. 1992;20:1391-6.
2. Gussak I. Antzelevitch C. Early Repolarization Syndrome: Clinical Characteristics and Possible Cellular and Ionic Mechanisms. J Electrocardiol 2000;33:299-309.
3. Haissaguerre M. Derval N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. N. Engl J Med 2008;358:2016-23.
4. Wellens HJ. Early repolarization revisited. N Engl J Med. 2008;358:2063-5.
5. Nam GB, Kim YH, Antzelevitch C. Augmentation of J waves and electrical storms in patients with early repolarization. N. Engl J Med 2008;358:2078-9
6. Gussak I. Bjerregaard P, Egan TM, Chaitman BR. ECG phenomenon called J wave. J Electrocardiol. 1995;25:49-58
7. Gussak I, Bjerregaard P, Hammill SC, Clinical Diagnosis and Risk Stratification in Patients with Brugada Syndrome. J Am Coll Cardiol. 2001;37:1635-8
8. Garg A, Finneran W, Feld GK, Familial sudden cardiac death associated with a terminal QRS abnormality on surface 12-lead electrocardiogram in the index case. J Cardiovasc Electrophysiol 1998;9:642-7.
9. Gussak I, Bjerregaard P, Egan TM, Chaitman BR. ECG phenomenon called J wave. J Electrocariol 1995;28:49-58
10. Gussak I., Bjerregaard P., Antzelevitch C, Towbin J, Chaitman BR. The Brugada syndrome: clinical, electrophysiological and genetic aspects. J Am Coll Cardiol 1999;35:5-15
11. Martinez-Lopez JI. ECG of the month. Innocent abnormality. Early repolarization pattern. J La State Med Soc 1991;143;7-9
12. Netter FH. Misleading electrocardiographic findings. In: Heart. The CIBA collection of medical illustrations. V. 5., Section II - Plate 32. Ed: Yonkman FF. Ciba Pharmaceutical Company, USA, 1987.
13. Mehta MC, Jain AC. Early repolarization on scalar electrocardiogram. Am J Med Sci 1995;309:305
14. Mehta M, Jain AC, Mehta A. Early Repolarization. Clin Cardiol 1999;22:59
15. Lazzoli JK, Annarumma M. de Araujo CG. Criteria for electrocardiographic diagnosis of vagotonia. Is there a consensus in the opinion of specialists? Arq Bras Cardiol 1994;63:377
16. Bjornstad H, Storstein L. Meen HD, Hals O. Electrocardiographic findings according to level of fitness and sport activity. Cardiology 1993;83:268
17. Hollander JE, Lozano M. Fairweather P, Goldstein E. gennis P, Brogan GX, Cooling D, Thode HC, Gallagher EJ. "Abnormal" electrocardio-

grams in patients with cocaine-associated chest pain are due to “normal” variants. J Emerg Med 1994;12:199

18. Vorob’ev LP, Gribkova IN, Petrusenko NM, Trofimenko NB. Te clinico-electrocardiographic classification of the early ventricular repolarization syndrome. Ter Arkh 1992;64:93.

19. Huston TP, Puffer JC, Rodney WM. The athletic heart syndrome. N. Engl J Med 1985;313:24

20. Gritsenko ET. Several aspects of early ventricular repolarization syndrome. Kardiologiia 1990;30:81.

21. Beliaeva LM, Rostovtsev VN, Novik II. The role of genetic factors in determining the ECG indicators, Kardiologiia 1991;31:54

22. Vitelli LL, Crow RS, Shahar E, Hutchinson RG, Rautaharju PM, Folsom AR. Electrocardiographic findings in a healthy biracial popylation. Atherosclerosis Risk in Communities (ARIC) Study Investigators. Am J Cardiol 1998;81:453

23. Aizawa Y. Tamura M. Chinushi m, Naitoh N, Uchiyama H, Kusano Y, Hosono H, Shibata A. Idiopathic ventricular fibrillation and bradycardia-dependent intraventricular block. Am Heart J 1993;126:1473

24. Mirvis DM, Evaluation of normal variations in S-T segment pattern by body surface mapping: S-T segment elevation in absence of heart disease. Am J Cardiol 1982;50:122

25. Lehman KG, Shandling AH, Yusi AU, Froulicher VF. Altered ventricular repolarization in central sympathetic dysfunction associated with spinal cord injury. Am J Cariol 1989;63:1:1498.

26. Yan GX, Antzelevitch C. Cellular basis for the Brugada Syndrome and other mechanisms of arrhythmogenesis associated with ST segment elevation. Circulation 1999;100:1660-6

27. Antzelevitch C, Dumaine R. Electrical heterogeneity in the heart: Physiological, pharmacological and clinical implications. In: Page E, Fozzard HA, Solaro RJ, eds. Handbook of Physiology. The Heart, New York: Oxford University Press, 2002:654-92.

ECG Quiz

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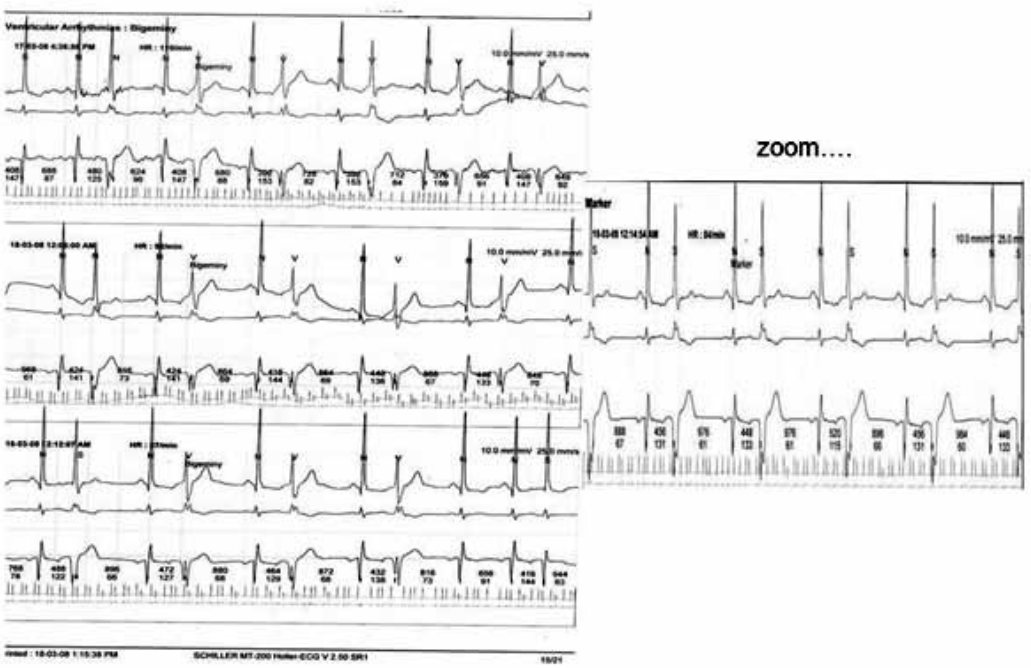
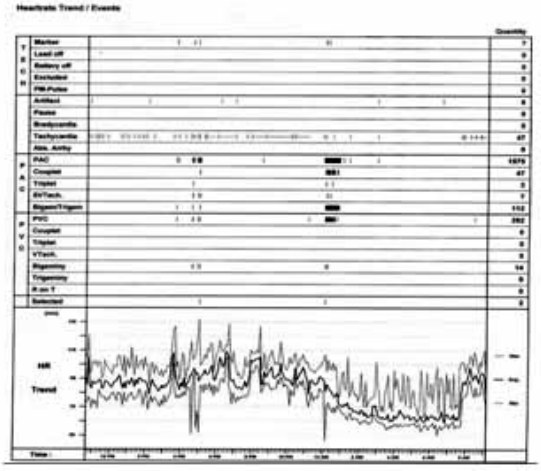
The answers and explanations are on the reverse side of the page.

ECG - 1

Holter recording of a middle-aged lady with frequent palpitations





1. The tracing shows

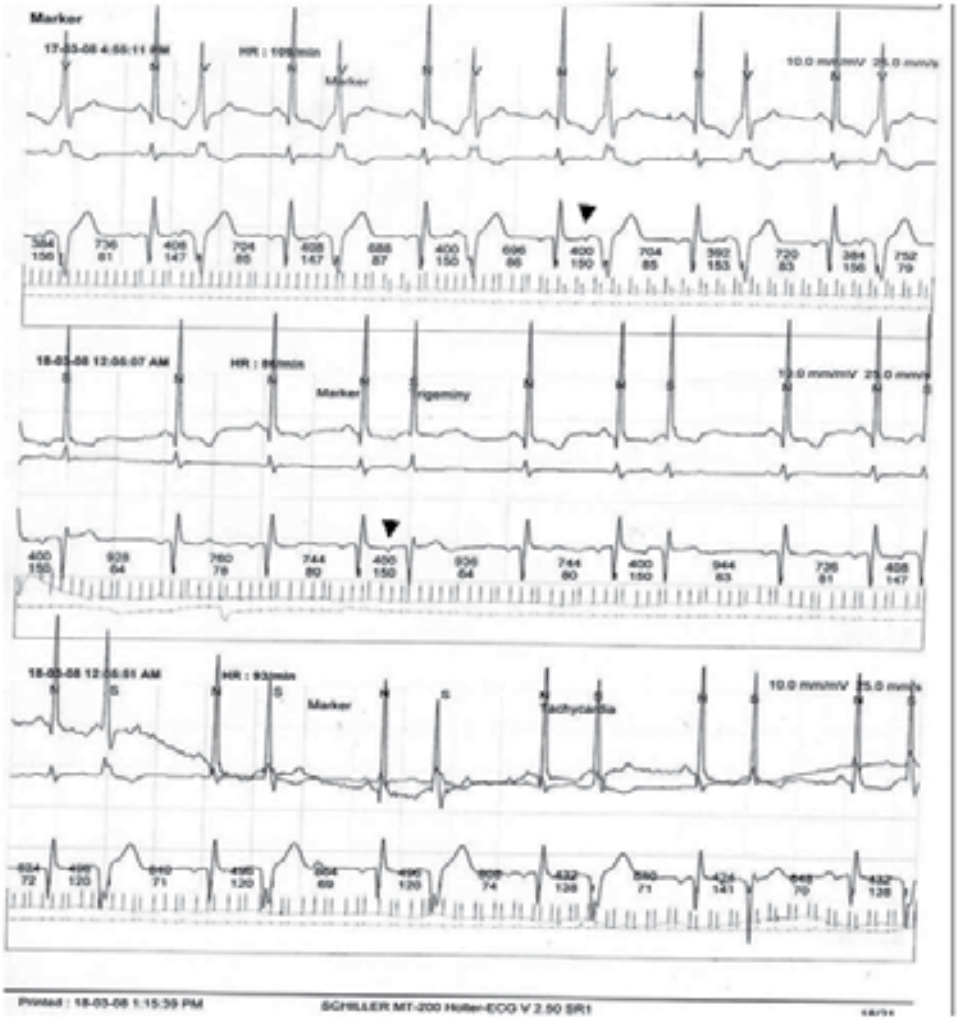
a. Premature atrial complexes (PACs)
b. Premature ventricular complexes (PVCs)
c. PACs and PVCs

For correct answer see overleaf

ECG - 1

The correct answer is 'a' – Premature atrial complexes (PACs)

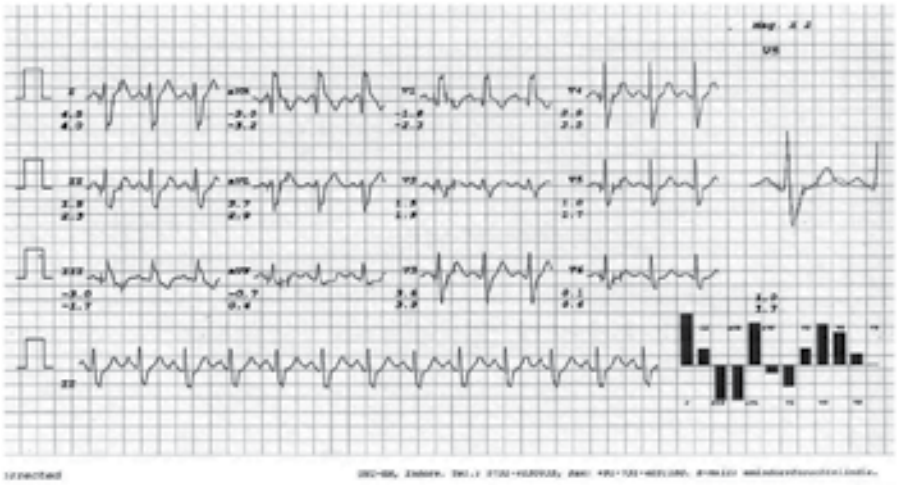
The tracing shows frequent premature complexes, often in a bigeminal rhythm. Some of them show narrow QRS complexes and others show wide QRS complexes. Narrow QRS complexes clearly indicate supraventricular ectopics. One can clearly make out abnormal premature P waves within the T wave of the previous complex (arrowheads). The wide QRS complexes are also preceded by similar abnormal P waves within the preceding T waves as indicated by arrows. It is not uncommon for very early PACs to find one of the bundle branches refractory and therefore produce a wide QRS complex. Such wide QRS complexes are commonly mistaken for PVCs on a Holter tracing.



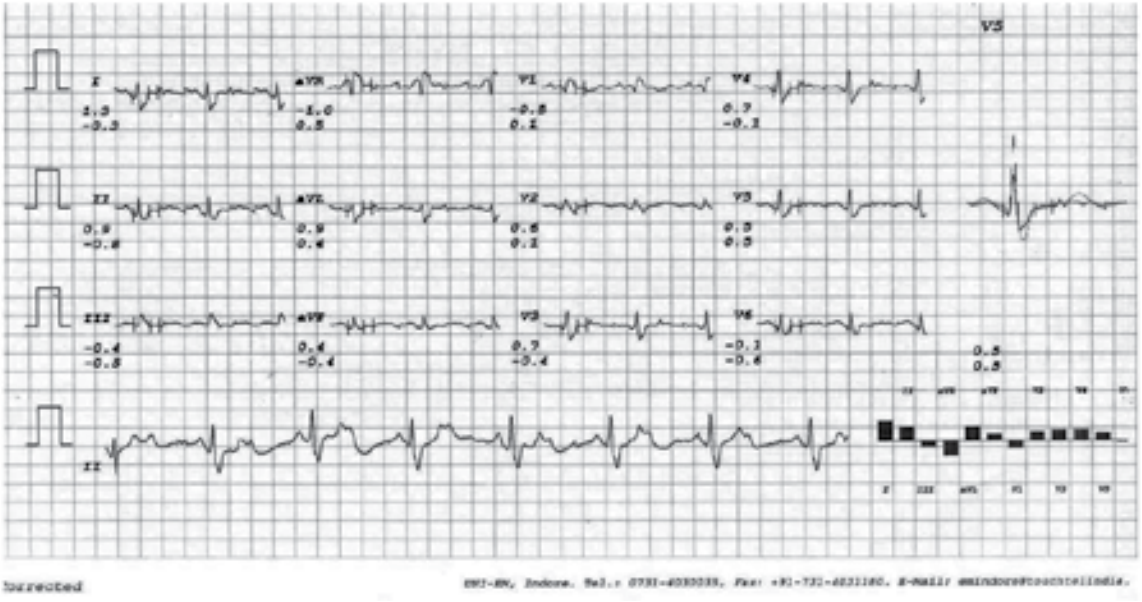
Interestingly, the computer-generated Heartrate Trends/Events graph shows PACs and "PVCs" at ***identical*** times.

ECG - 2

A 66 yrs-old-man, intermittent exertional fatigue. The baseline ECG showed sinus rhythm with RBBB.



During treadmill test….



2. The cause of fatigue is

a. Ischaemia
b. Sinus nodal dysfunction (SND)
c. AV nodal disease
d. Infra-nodal disease

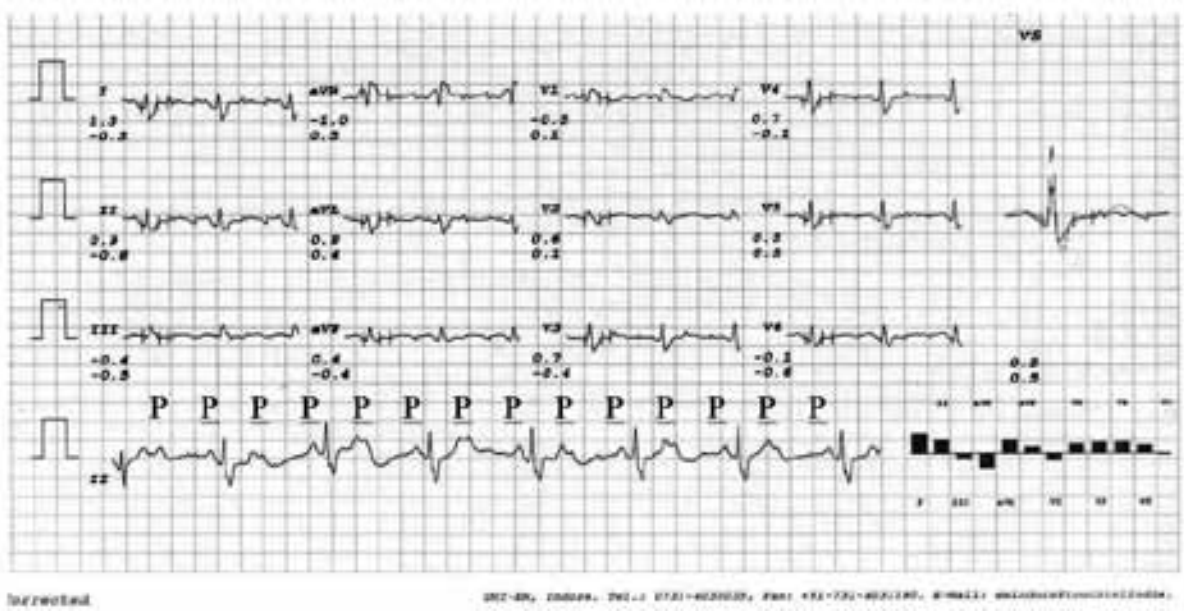
For correct answer see overleaf

ECG - 2

The correct answer is 'd' – Infranodal disease

The ECG in stage 1 shows sinus rhythm with 1:1 AV conduction. There is a right axis deviation (RAD) and a complete RBBB. The PR interval is normal.

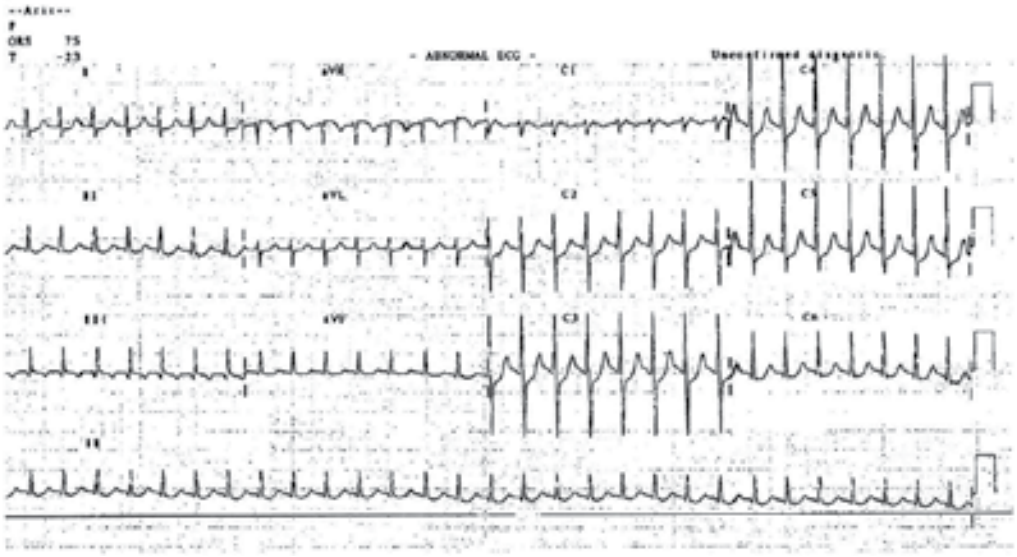
During the recovery phase one sees a sudden slowing of the ventricular rate. On careful observation there is 2:1 AV block. The sinus rate is 140 bpm while the ventricular rate is half of that. The PR interval of the conducted P wave is normal. Ischaemia does not present as isolated AV block. Since there is adequate sinus acceleration, the sinus node function is normal. AV nodal conduction improves with exercise. Hence AV block that appears on exercise is indicative of infranodal disease, typically in the bundle branches.



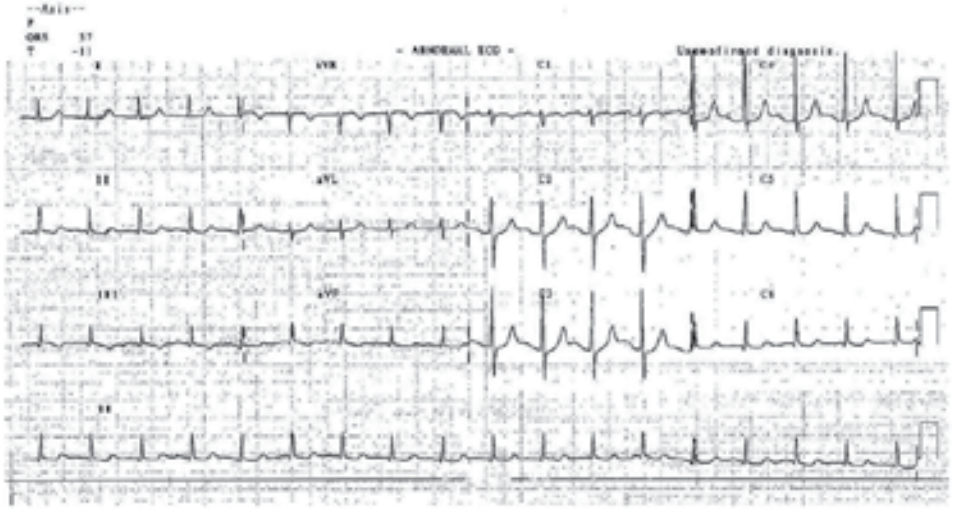
An additional support to choice (d) is the baseline RBBB. Also the PR interval is normal. Hence, this is a case of infranodal block and the subject needs a pacemaker.

ECG - 3

A 45 yrs-old patient with recurrent, paroxysmal palpitations…



Later…



3. The upper trace shows:

a. Sinus tachycardia
b. Atrial tachycardia
c. Atrioventricular nodal re-entrant tachycardia (AVNRT)
d. Atrioventricular re-entrant tachycardia (AVRT)

For correct answer see overleaf

ECG - 3

The correct answer is 'c' – Atrioventricular nodal re-entrant tachycardia

The ECG shows a regular narrow QRS tachycardia of 170 bpm. No P waves are clearly discernible. The QRS complexes are nearly normal. No ST depression and T waves are normal.

Since no P waves are seen with such a rapid rate, sinus tachycardia is not tenable.

Atrial tachycardia would typically show normal or abnormal P waves at least in some leads.

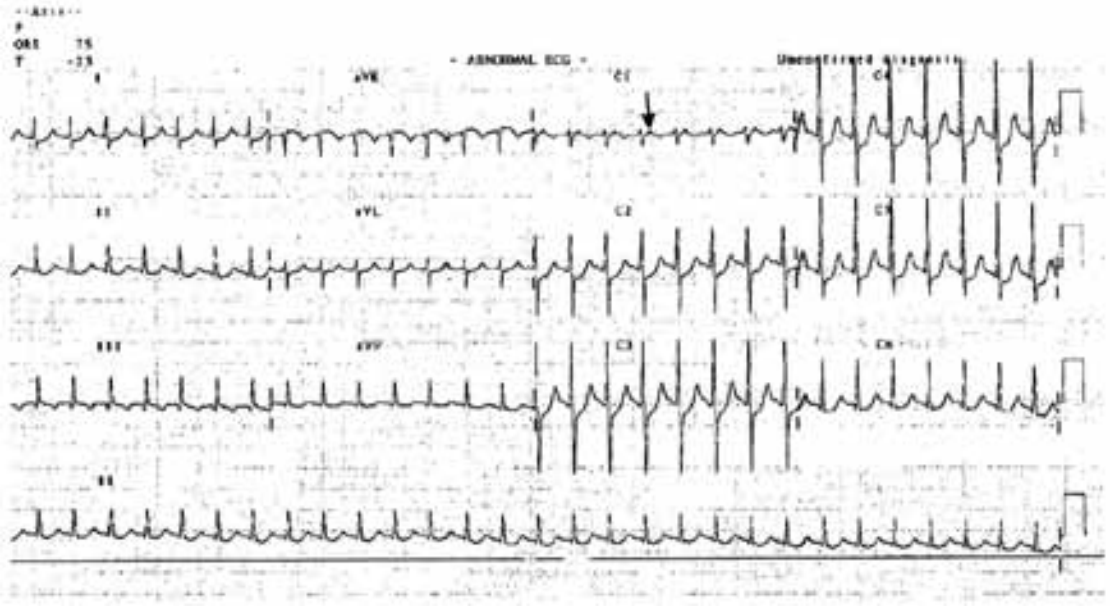
AVRT (involving a retrograde accessory pathway) typically shows ***inverted*** P waves, which follows soon after the QRS complex.Even if these retrograde P waves are not clearly visible in AVRT they would certainly cause marked ST segment depression especially in some of the inferior leads.This is not seen in this ECG.

If we compare this ECG to that in sinus rhythm, we can clearly see some differences in the QRS complexes:

-During tachycardia there is a distinct R' in lead V1 (arrow).This is absent in sinus rhythm.

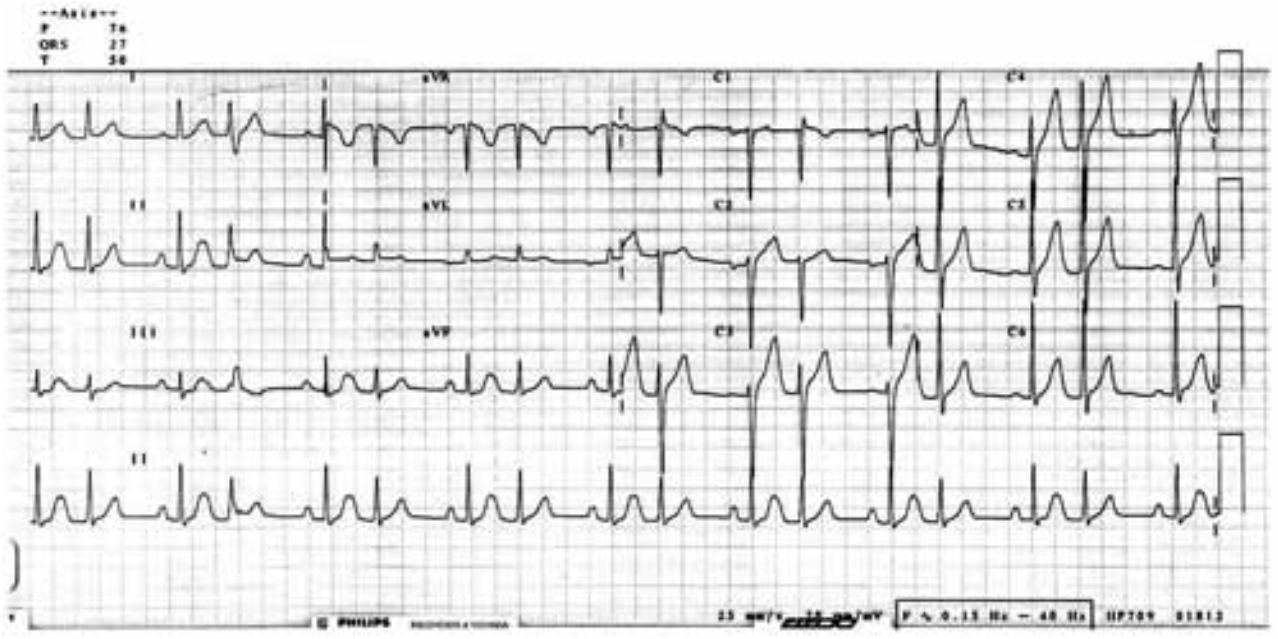
- Also seen during tachycardia are small S waves in leads II and V6. These are absent in sinus rhythm

The R' and these pseudo S waves are classical signs for AVNRT. In AVNRT, the atria and ventricles being activated ***simultaneously*** result in the P wave being submerged in the QRS complex.The R' in V1 and the small S in the inferolateral lead are thus tell-tale signs of atrial activation.



ECG - 4

A 42 yrs-old smoker with uneasiness & chest discomfort…



4. The ECG shows:

a. AV block
b. Atrial bigeminy
c. Acute coronary syndrome
d. Sinus node dysfunction

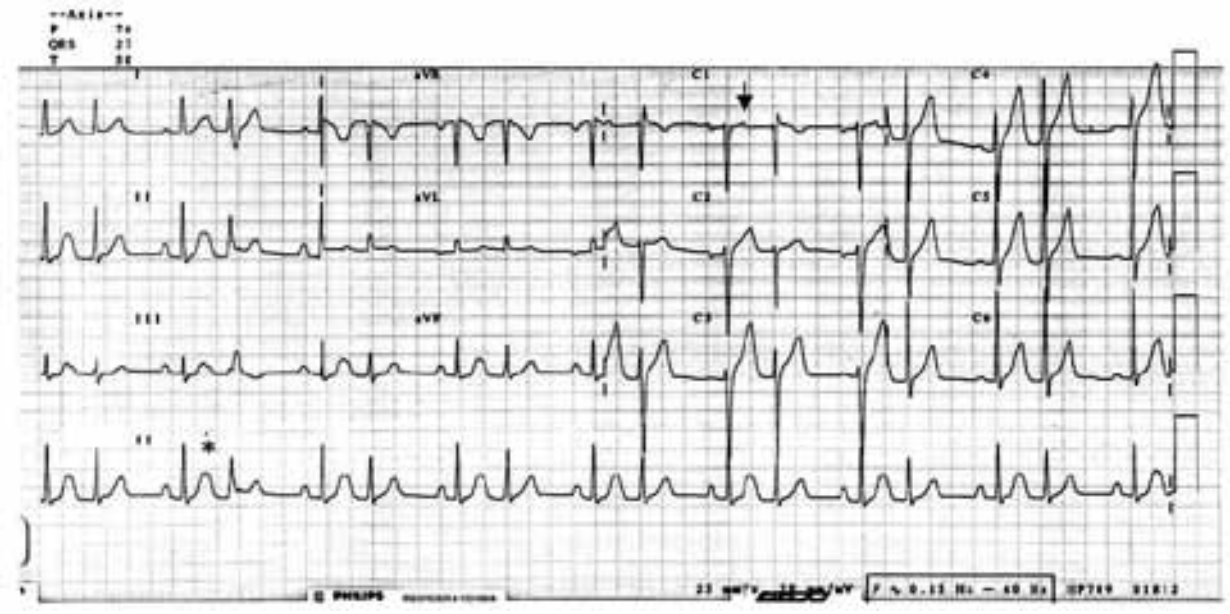
For correct answer see overleaf

ECG - 4

The correct answer is 'b' – Atrial bigeminy

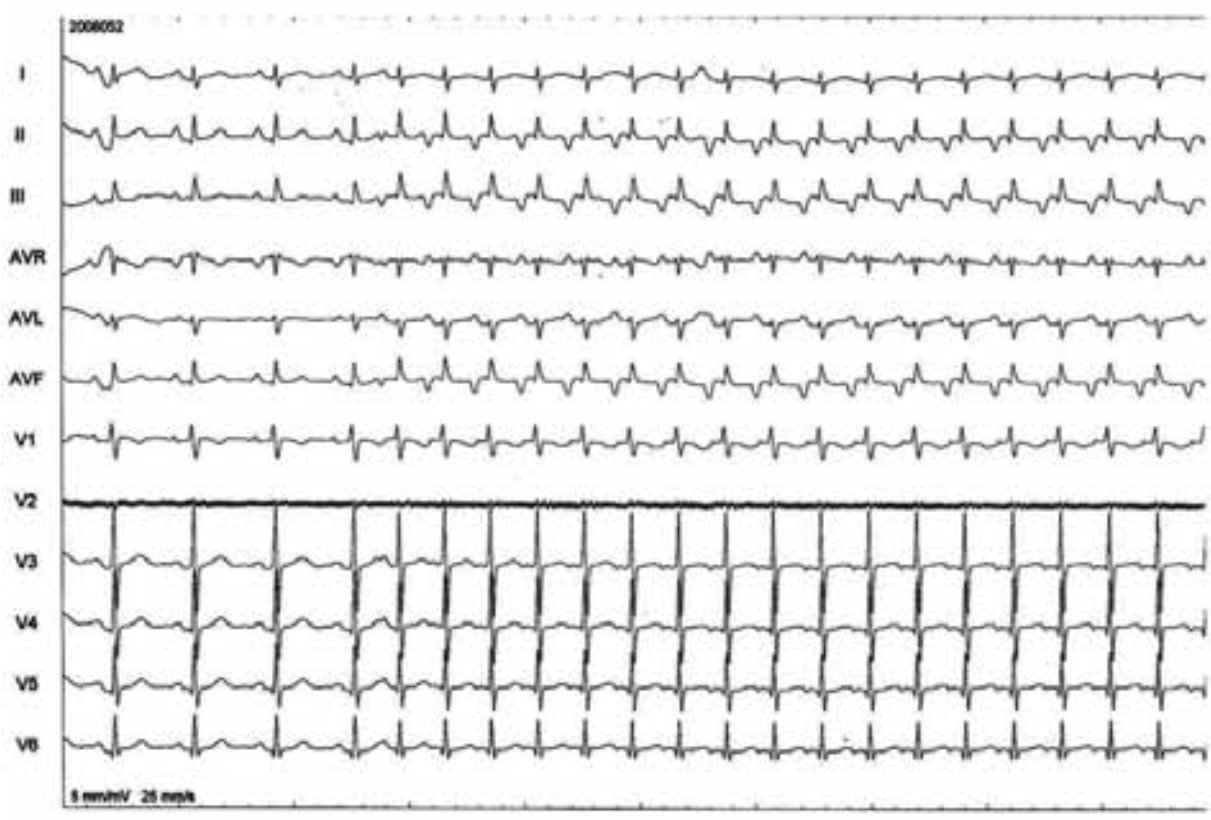
The long lead II clearly shows paired QRS complexes, which then would be described as a bigeminal rhythm. A clear P wave is seen before the first of these pairs of QRS complexes. The T wave shows alternating amplitude and width. The first T wave of the bigeminal pair (*), is broader and taller. This clearly suggests a P wave sitting on a T wave. If one looks at lead V1 (C1), one can clearly make out these premature ectopic P waves (arrow). Since the P wave is very premature, it finds the AV conduction partly refractory and conducts with a prolonged PR interval best seen in V1.

Therefore, it is clear that there are alternating unifocal atrial ectopics producing an atrial bigeminy pattern.



ECG - 5

A 15 yrs-old patient presenting with recurrent palpitations.



5. The mechanism of tachycardia is:

a. Atrial tachycardia
b. Atypical (Fast-slow) AVNRT
c. Atypical AVRT (slowly conducting accessory pathway – Coumel type)
d. Any of the above

For correct answer see overleaf

ECG - 5

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

The ECG shows a sudden initiation of a narrow QRS tachycardia @160 bpm. Lead V2 is disconnected.

The tachycardia shows inverted P waves in the inferior leads which precede the QRS complex (RP longer than PR).

The tachycardia starts with a premature P wave which is different in morphology from P waves during tachycardia. From the P wave morphology one can say that the atrial activation starts in the low atria and proceeds superiorly. So a low atrial tachycardia arising typically from near the coronary sinus ostium or even from low left atrium is one possibility. The positive P waves in aVR and aVL suggests an atrial activation near the inter-atrial septum spreading to both right and left shoulders at near-equivalent vectors.

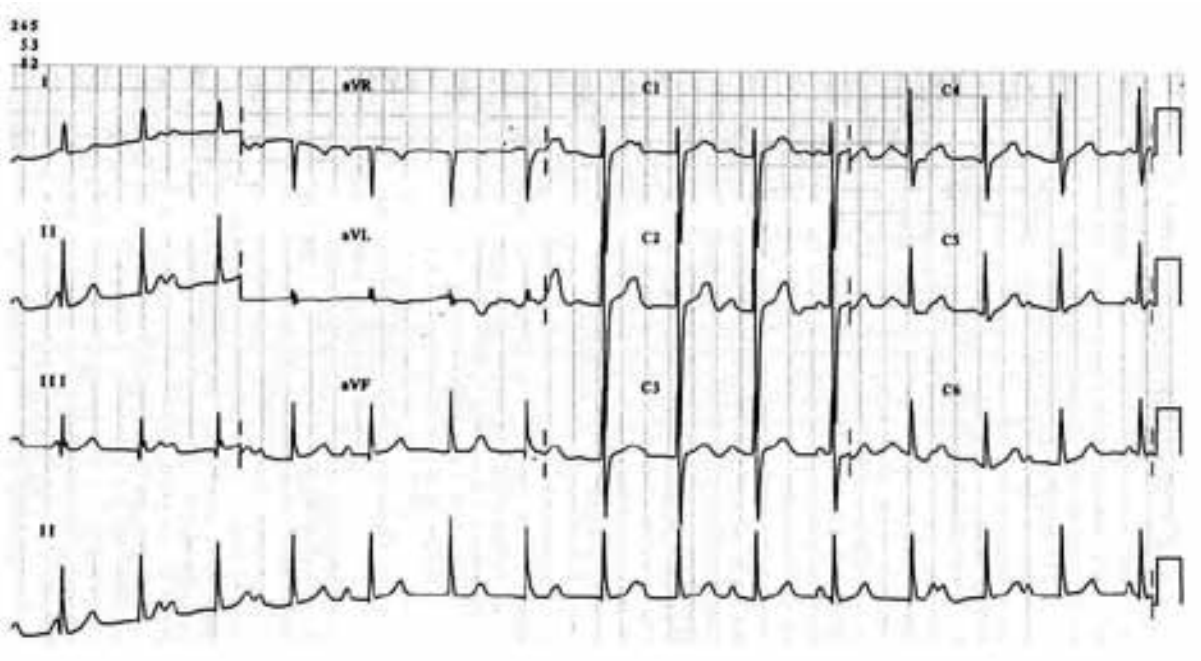
Fast-slow AVNRT also has an atrial exit near the coronary sinus ostium and would therefore have a similar appearance.

Slowly conducting accessory pathways (Coumel tachycardia, wrongly called PJRT) typically occur near the coronary sinus opening. Since they have a long conduction time the VA time is long, resulting in a long RP interval. The PR is normal because the AV conduction occurs through a normal conduction system.

Thus in these Coumel tachycardias, the surface ECG would be identical to that of low atrial tachycardia or fast-slow AVNRT. The fact that this is initiated by a PAC does not help to differentiate between any of these 3 differential diagnoses. The cardiac traces also show that the PAC after a basal train initiates this tachycardia. Further, complex EP manoeuvres and pharmacologic interventions are needed to differentiate between these 3 diagnoses.

ECG - 6

After recent VSD surgery, this ECG shows following abnormalities:



6. a. Sinus arrhythmia
b. Complete AV block
c. Accelerated junctional rhythm
d. All of the above

For correct answer see overleaf

ECG - 6

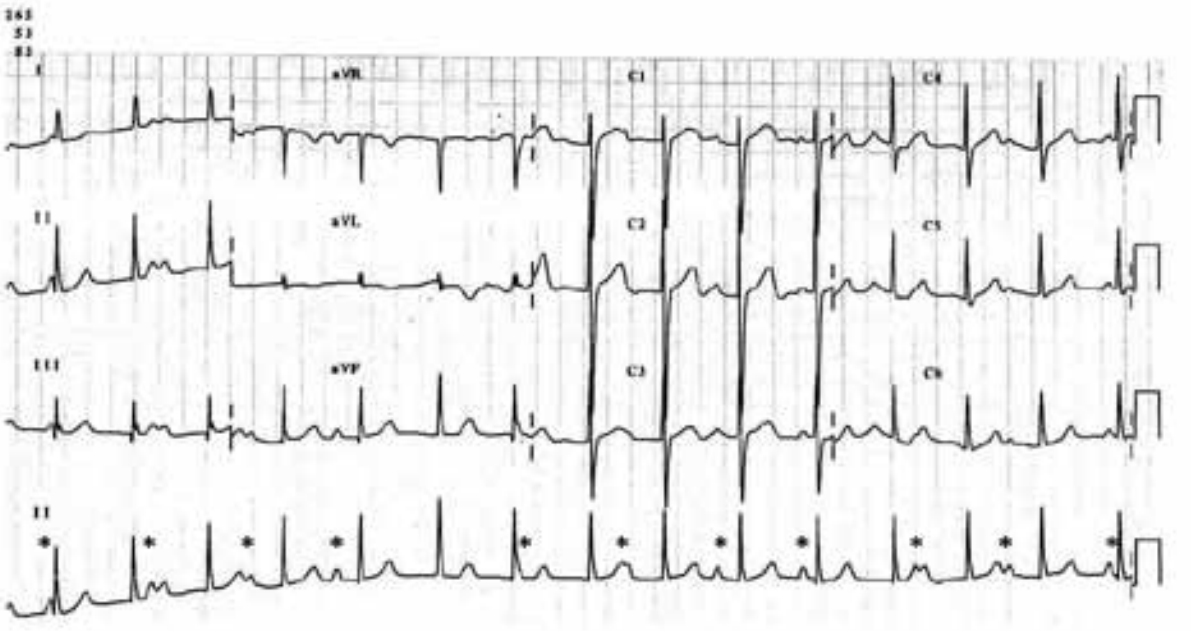
The correct answer is 'd' – All of the above

The ECG shows a regular ventricular rate of approximately 96 bpm.

The QRS complexes are narrow. The P waves are seen intermittently (*). At other times they are hidden within the QRS complexes are seen in long lead II. The PP interval is varying, suggestive of sinus arrhythmia. The atrial rate is approximately 80 bpm. The P waves have no relation to the QRS complexes. Even the P waves occurring after the T wave do not conduct to the ventricles. Thus, there is complete AV block.

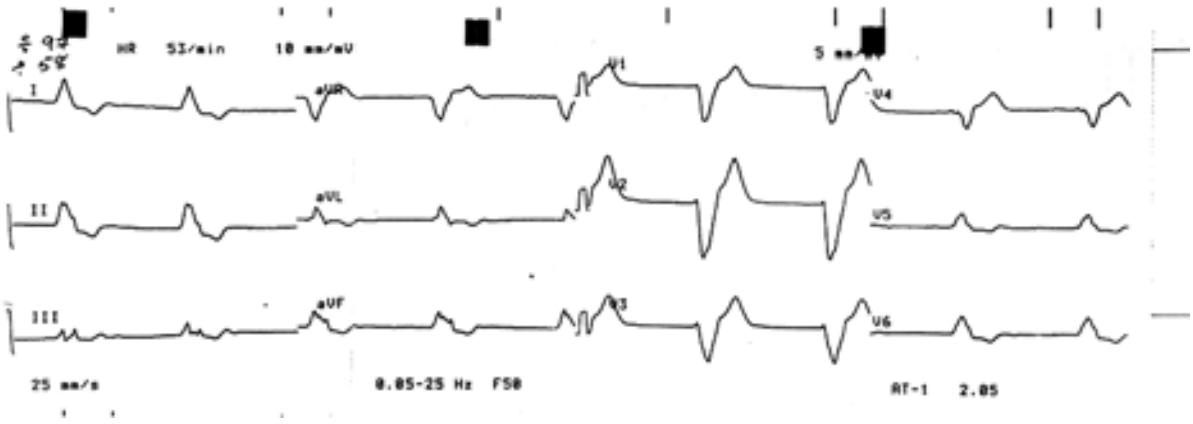
The ventricular rhythm then is generated by the AV junction, which is normally expected to have an intrinsic rate of 40 to 60 bpm. Since the ventricular rate is 96 bpm, this is an accelerated junctional rhythm. Hence the correct answer is **'d' – All of the above.**

Transient AV block associated with accelerated junctional rhythm is one of the known complications of VSD surgery.



ECG - 7

A patient with heart failure who had received a biventricular pacemaker, presented with dyspnea and giddiness



7. The ECG shows :

a. Idioventricular rhythm
b. Ventricular paced rhythm
c. LBBB
d. None of the above

For correct answer see overleaf

ECG - 7

The correct answer is 'a' – Idioventricular rhythm

This ECG shows a regularly occurring wide QRS complex rhythm @ 53 bpm.

The width of the QRS complex is almost 200 ms as best seen in leads V3 and aVF. No P waves are seen.

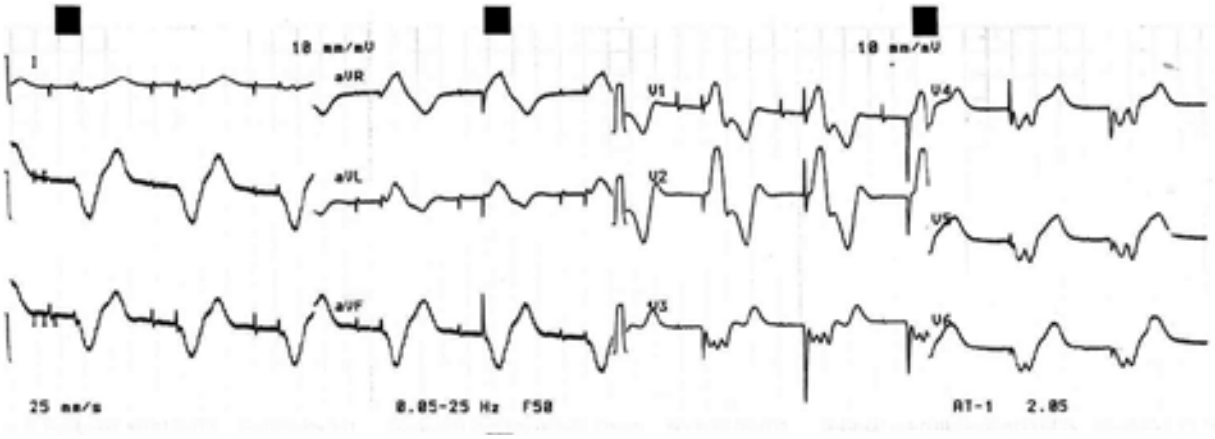
The morphology of the QRS and its width along with the absence of the P waves clearly indicates that this rhythm is generated from the ventricular myocardium. No pacing artifacts are seen. This could well be because the lower pacing rate is set at 50 bpm, whereas the ventricular rate is 53 bpm. Moreover, in the absence of P waves, the ventricles will not be able to track any atrial activity.

LBBB would be tenable only if there are P waves which are conducted with the typical LBBB pattern.

The story continues……

ECG - 8

The same patient with DDD pacing reprogrammed at a lower rate of 70 per minute



8. The investigation of choice will be :

a. Echocardiogram
b. X-ray
c. CAG
d. ABG, electrolytes

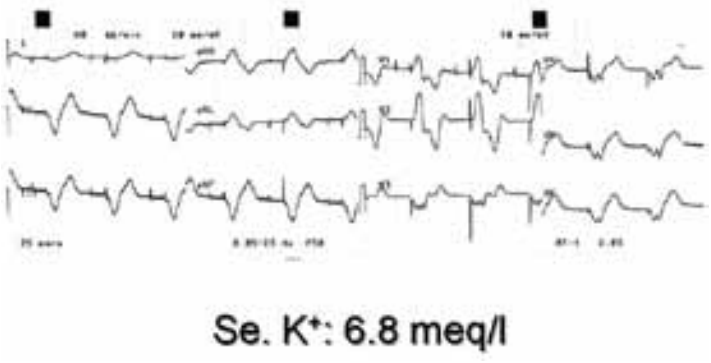
For correct answer see overleaf

ECG - 8

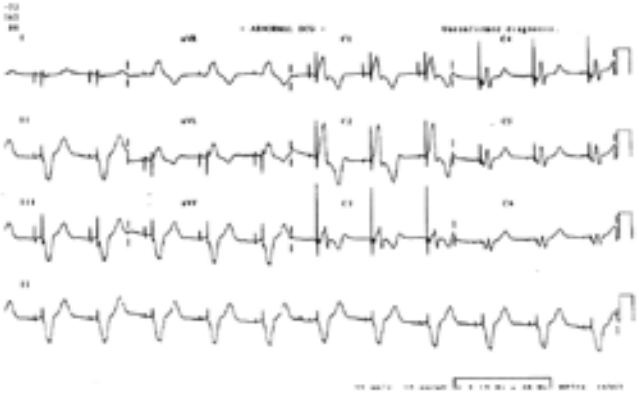
The correct answer is 'd' – ABG, electrolytes, especially the level of serum potassium.

Now what one sees in this strip is regularly occurring atrial and ventricular pacing artifacts. The QRS complex however is still very wide, measuring more than 240 ms in lead V5. The RS morphology in lead V1 suggests that both the left and the right ventricles are being paced, as expected. With biventricular pacing one actually hopes to narrow the QRS complex. Such a wide QRS complex indicates a severe conduction abnormality of the ventricles. This global conduction abnormality brings to mind hyperkalemia.

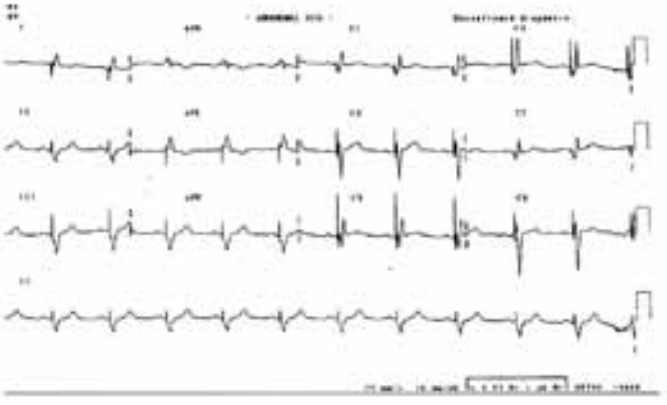
In hyperkalemia, the P waves also get blunted and lost. This explains why the atrial pacing artefacts are not followed by any visible P waves. The next three figures show successive normalization of the QRS complex and reappearance of P waves as the serum potassium under the effect of treatment gradually came back to normal.



After 10 cc Ca^{++} gluconate, 50 cc $NaHCO^3$ & GI drip...

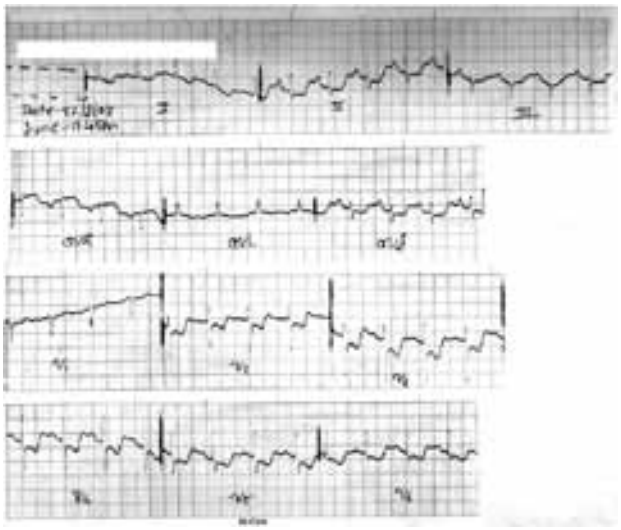


Se. K^+: 4.8 meq/l

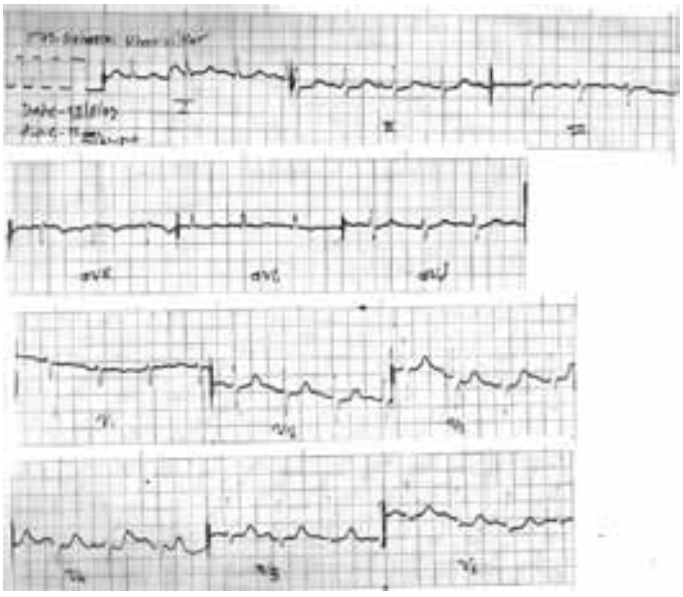


ECG - 9

A 70-year old woman, hypertensive, acutely breathless and collapses



After 2 hrs of supportive Rx with oxygen, IV fluids and dopamine



9. Is it acute coronary syndrome ?

a. Yes
b. No

For correct answer see overleaf

ECG - 9

The correct answer is 'b' – No

The ECG on presentation shows gross abnormalities. There is marked downsloping ST depression (upto 5 mm) seen best from lead V2 through to V6. ST depression is also seen in leads II and aVF and to some extent in lead I. Despite the changing baseline, there is also some ST elevation in lead aVR. This ECG pattern has been described typically in near-occlusion of the left main coronary artery (or a left main equivalent ACS). In elderly women, coronary ischemia may give the symptom of breathlessness and not classical angina. Therefore, the symptomatology along with this initial ECG certainly raises the first possibility of a large area of myocardial ischemia due to coronary artery disease.

However, the subsequent ECG taken without any specific anti-anginal, anti-thrombotic or thrombolytic therapy shows complete normalization of the ECG. This would be extremely unlikely in an ACS of this magnitude. This patient's Trop T was normal. A coronary angiogram performed on an urgent basis was normal. The echocardiogram done after this suggested mild dilatation of the right ventricle. Arterial blood gases had shown an oxygen saturation of 90% on high-flow oxygen. A subsequent CT pulmonary angiogram showed multiple sub-segmental pulmonary infarcts. So what was most likely is that the patient had initially a massive pulmonary embolism occluding the main pulmonary artery, which spontaneously fragmented along with the supportive treatment and went into the more distal pulmonary ciriculation.

Multiple ECG abnormalities have been described in acute pulmonary embolism. In fact the ECG could be even normal. But in major pulmonary embolism, RV strain patterns, pseudo-anteroseptal MI T wave patterns, bundle branch blocks and the classical S1Q3T3 pattern have been described.

Pulmonary thromboembolism is more often missed at the initial presentation. A high index of suspicion is required along with other relevant investigations.

Diagnosis of acute myocardial infarction in the presence of a right postero-septal accessory pathway

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Introduction

Diagnosis of myocardial infarction (MI) may be difficult or impossible in the presence of intra-ventricular conduction disturbances such as left bundle branch block (LBBB) or Wolff-Parkinson-White (WPW) syndrome[1]. Conversely these disorders may simulate infarction when the initial vectors of ventricular activation are oriented in such a way as to mimic a pathological Q wave or ST -T changes. The ECG findings of acute myocardial infarction in WPW syndrome, when the ventricular pre-excitation pattern is pronounced, are not well defined. Here we report two cases of WPW syndrome who presented to us with acute myocardial infarction. [1]

Case Reports

Case 1

A 52 years old male presented to our emergency department with angina for 8 hours. The ECG at admission showed sinus rhythm, short PR interval (90 ms), wide QRS (180 ms), superior QRS axis and QRS transition in V_2 with negative delta waves in V_1. The pattern was suggestive of ventricular pre-excitation by a right posteroseptal accessory pathway. There was significant ST elevation (3 – 5 mm) in the inferior leads (Fig. 1).

In view of the angina and elevated myocardial enzymes, fibrinolysis was instituted. The patient was symptom free after fibrinolysis. The ECG, post-fibrinolysis, showed resolution of ST segment elevation with persistent pre-excitation. However, the degree of preexcitation was lesser than before (Fig. 2). This could partly explain the changed QRS morphology (eg. QS to Qr in lead III) and the reduced QRS width (eg. 140 to 120 ms in aVF). The increased R wave amplitudes in the lateral leads could also be partly due to successful reperfusion. The patient underwent coronary angiography before discharge, which revealed significant thrombotic occlusion of the right coronary artery.

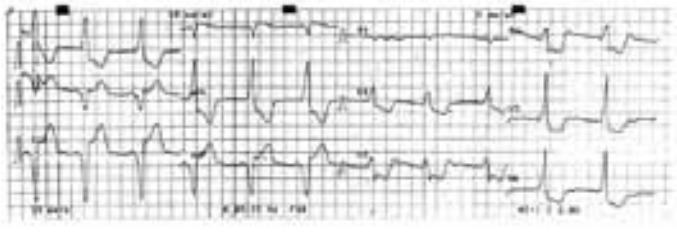

Figure 1: ECG of the case 1 on admission showing pre-excitation through right posteroseptal pathway and ST segment elevation in the inferior leads with ST segment depression in the anterolateral leads.

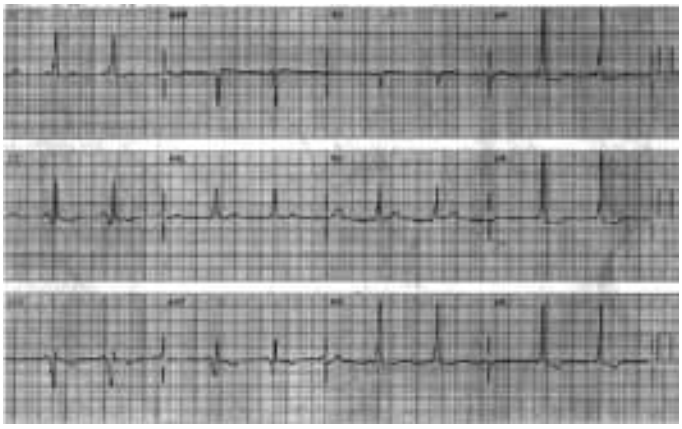

Figure 2: ECG of the same patient after fibrinolysis demonstrating pre-excitation and normalization of the ST segment

Case 2

A 63 year old man was admitted to the critical care unit with chest pain of 6 hours duration associated with profuse sweating. He had never experienced angina, palpitations or syncope earlier. The ECG at admission showed sinus rhythm with ventricular pre-excitation by a right posteroseptal accessory pathway. There was significant ST segment depression (8-12 mm) in the mid-precordial leads (V_2 - V_5) (Fig. 3). The R wave

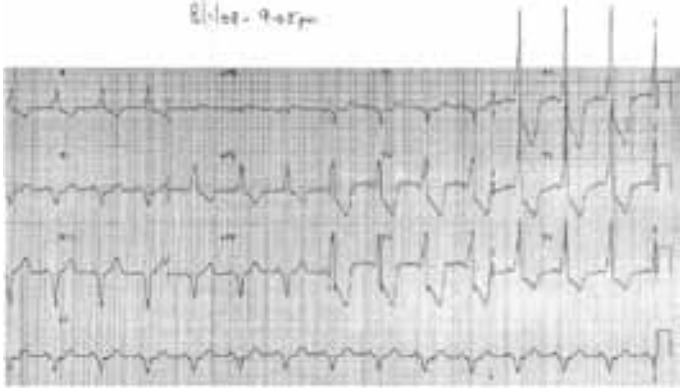

Figure 3: ECG of the Case 2 on admission showing pre-excitation through right posteroseptal pathway and significant ST segment depression in the anterolateral leads

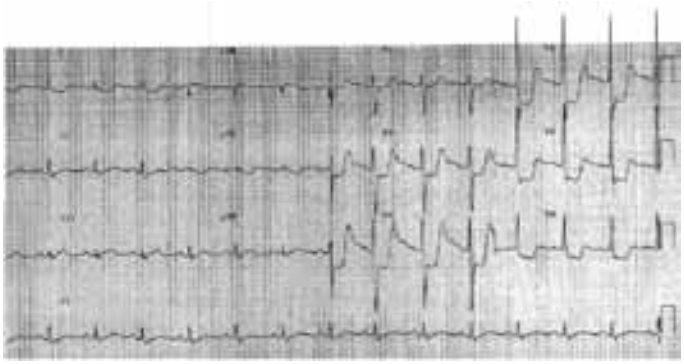

Figure 4: ECG of the same patient showing conduction through AV node after infusion of nitroglycerine but showing significant ST segment depression in the concerned leads

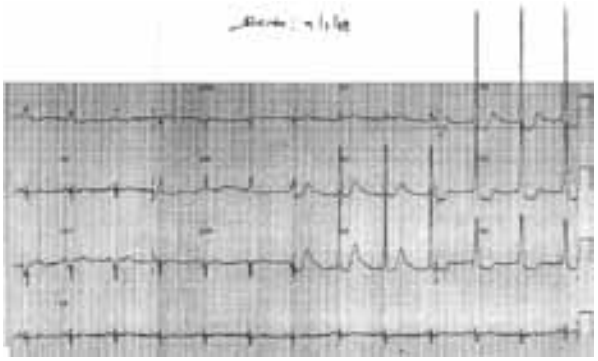

Figure 5: ECG of case 2 demonstrating normalization of ST segments in the presence of pre-excitation demonstrating that the changes in ST segment were secondary to ischaemia.

voltage in the lateral precordial leads (V_4 -V_6) was high (30 - 40 mm). The Troponin-I level was elevated. However fibrinolytic therapy was not considered because there was no ST segment elevation.

On infusion of intravenous nitroglycerine, pre-excitation disappeared (without an appreciable change in the heart rate), showing a normal of PR interval, narrow QRS and a normal QRS axis with persistence of significant ST segment depression in the precordial leads (Fig. 4). His coronary angiogram revealed a left dominant coronary system. The circumflex artery showed significant occlusion with TIMI II flow. Following revascularization the ECG showed ventricular preexcitation with the expected secondary ST-T changes in the lateral leads (Fig. 5).

Discussion

The WPW pattern is noted in 2-3 per 10,000 in the general population. Up to one third of these patients can present with palpitations (WPW syndrome). [2,4,5,6] The WPW pattern mimics MI. A WPW pattern resulting from right posteroseptal accessory pathway mimics inferior wall MI [1]. The waveform of WPW patterns resulting from left free wall accessory pathway or right free wall accessory pathways mimic posterior wall MI and anterior wall MI respectively.[1, 3] Concomitant secondary ST segment and T wave changes associated with WPW pattern may also mimic acute coronary syndromes.

Currently there are no specific guidelines in the literature for the diagnosis of acute MI in patients with a WPW pattern. There are no specific criteria defining the magnitude or the morphology of ST segment elevation or depression and also T wave changes to suggest acute MI in WPW pattern[1].

In our case report, one patient presented with ST segment elevation and the other patient presented with ST segment depression. In case 1, the combination of chest pain, ECG showing significant ST segment elevation (3-5 mm) and increased myocardial enzymes favoured the diagnosis of acute MI. The coronary angiogram confirmed the presence of thrombotic occlusion of right coronary artery. In contrast, in case 2, there was significant (8-12 mm) ST segment depression. These changes were attributed to acute coronary syndrome as confirmed by coronary angiography. With regard to the R waves, increase in R wave voltage after fibrinolysis in the first case and no change in the R wave voltage in the second case were observed.

Our case report suggests that the constellation of chest pain, significant ST segment changes (either elevation or depression) and positive cardiac enzymes in the presence of WPW pattern due to right postero-septal pathway should warrant appropriate treatment in the form of thrombolysis or primary percutaneous coronary interventions. The concomitant changes in the R wave amplitudes and T wave morphologies are nonspecific.

1. Thomas AB, James TD, Gerald WM, Serge SB, The diagnosis of myocardial infarction in the Wolff-Parkinson-White syndrome. Chest, 65: 5, May, 1974.

2. Ijaz AK, Izabela SS, Pseudo ventricular hypertrophy and pseudo myocardial infarction in Wolff-Parkinson-White syndrome. Am J Emerg Med 2000; 18: 807-809.

3. Loya YS, Pereira AC, Shah KD, WPW syndrome with myocardial infarction. JAPI 1988, Vol 36, No. 12, 724-725.

4. Rosner MH, Brady WJ Jr, Kefer MP, Martin ML. (1999). "Electrocardiography in the patient with the Wolff-Parkinson-White syndrome: diagnostic and initial therapeutic issues". *American Journal of Emergency Medicine* **17** (7): 705–14.

5. Sorbo MD, Buja GF, Miorelli M, Nistri S, Perrone C, Manca S, Grasso F, Giordano GM, Nava A. (1995). "The prevalence of the Wolff-Parkinson-White syndrome in a population of 116,542 young males" (in Italian). *Giornale Italiano di Cardiologia* **25** (6): 681–7.

6. Munger TM, Packer DL, Hammill SC, Feldman BJ, Bailey KR, Ballard DJ, Holmes DR Jr, Gersh BJ. (1993). "A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989". *Circulation.* **87** (3): 866–73.

Hypokalemic Periodic Paresis

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A 44 year old gentleman presented with a history of low grade fever with chills since 3 days, weakness of limbs since 1 day and inability to walk for a few hours. He had no other symptom and had no past history of any significant medical illness or medication. Cardivascular, respiratory and abdominal examinations were unremarkable. His muscle power was reduced to grade 3/5 in all four limbs, but the reflexes were normal. There was generalized hypotonia. There was no other CNS abnormality. A blood sample was drawn and sent for analysis, and an ECG was taken which revealed (fig.1) sinus bradycardia at a rate of 57 bpm, normal PR interval (160 ms), a slightly broad QRS (114 ms) with a normal QRS axis and prominent U waves leading to a prolonged QT-Uc (680 ms).

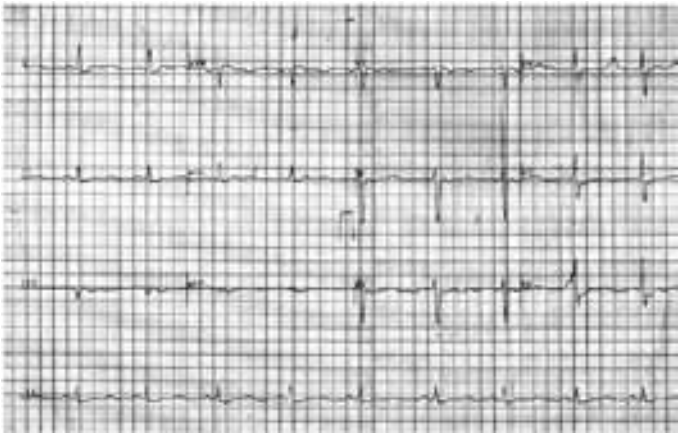

Fig. 1: ECG in a case of hypokalemic periodic paresis showing sinus bradycardia (57 bpm), normal PR interval (160 ms), a slightly broad QRS (114 ms) with a normal axis, normal QTc (304 ms) and presence of U waves. The serum potassium was 1.8 mEq. / Lit.

Based on these findings, a presumptive diagnosis of hypokalemic periodic paresis was considered and this was confirmed when the serum potassium level was found to be 1.8 mEq./L. Apart from the hypokalemia, there was no abnormality on the blood reports. An intravenous KCl infusion was started, and the patient's muscle power gradually revcovered and normalized to grade 5/5 on the next day. The ECG also improved with disappearance of the U waves and normalization of the QTc to 320 ms.

This ECG highlights a classic fact that in hypokalemia there is QT prolongation, usually with appearance of U waves; and that these ECG changes revert with correction of hypokalemia.

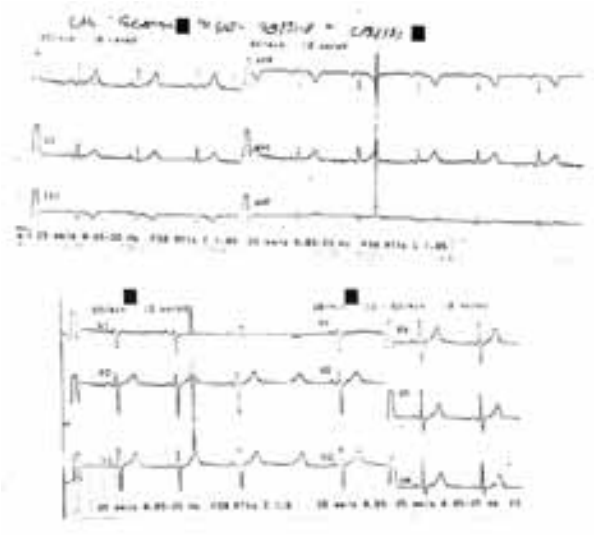

Fig. 2: ECG after correction of serum potassium level. Note that the ECG has normalized; showing sinus rhythm (68 bpm), normal PR interval (140 ms), a normal QRS duration (90 ms) with a normal axis, normal QTc (304 ms) and disappearance of the U waves.

INDIAN SOCIETY OF ELECTROCARDIOLOGY

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Thanking you,

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

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Signature _____

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Accepted / Not Accepted

Life Membership No.

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Hon. Secretary, ISE

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