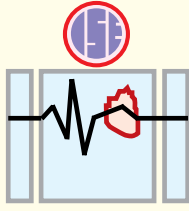


**Long live**

**Indian Society of Electrocardiology**

---



Executive Committee of  
INDIAN SOCIETY  
OF  
ELECTROCARDIOLOGY

**PRESIDENT**

Uday K Mahorkar

**HON. GENERAL-SECRETARY**

SB Gupta

**EXECUTIVE COMMITTEE**

**PAST PRESIDENT**

KK Talwar, *Chandigarh*

**PRESIDENT ELECT**

BV Agrawal, *Varanasi*

**VICE PRESIDENTS**

PTV Nair, *Mumbai*

SS Ramesh, *Bangalore*

HS Rissam, *New Delhi*

**TREASURER**

Amit Vora, *Mumbai*

**MEMBERS**

Vinod Vijan, *Nashik*

Uday Jadhav, *Navi Mumbai*

S Chandrasekharan, *Chennai*

Praveen Jain, *Jhansi*

Sunil Modi, *New Delhi*

Ramesh Dargad, *Mumbai*

NS Neiki, *Amritsar*

Sushum Sharma, *New Delhi*

**JOURNAL EDITORS**

Yash Lokhandwala, *Mumbai*

Amit Vora, *Mumbai*

## C O N T E N T S

---

<b>Editorial</b> .....	2
<b>Message from General Secretary</b> .....	3
<b>ECG Quiz</b> .....	5
<b>ISE Membership Form</b> .....	35

## Editorial

---



*Dear Colleagues,*

The Indian Society of Electrocardiology has shown a healthy growth over the last several years. The annual and mid-term ISECON meetings have become a regular affair. Mumbai, Delhi, Nagpur all saw well attended ISECON meetings with information & interaction sessions. The forthcoming ISECON in Bangalore promises to go a step higher in the academic pursuits of the ISE.

This issue of the IJE discusses the ECGs presented at Nagpur ISECON. We continue to receive feedback that such material helps reinforce ECG skills & serves as a useful teaching tool. Hence we hope to stimulate your diagnostic skills with this issue. We wish to thank Dr. Swapna Athawale for helping us to prepare this issue of IJE.

Please send in your articles & ECG vignettes for forthcoming issues of the IJE.

A handwritten signature in black ink, appearing to read 'Yash'.

**Yash Lokhandwala**  
*Editor*

A handwritten signature in black ink, appearing to read 'Amit Vora'.

**Amit Vora**  
*Editor*

## From Hon. Secretary's Desk

---



Dear Members,

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

Dr. K K Talwar, Dr. Rajnish Juneja and their team very well conducted ISECON-2004 from AIIMS New Delhi at Hotel Ashok New Delhi from 19<sup>th</sup> to 21<sup>st</sup> March 2004.

Then, Nagpur Arrhythmias Course (NAC 2004) was conducted on 9<sup>th</sup> and 10<sup>th</sup> October 2004 at Nagpur by Dr. Uday Mahorkar, Dr. Prashant Jagtap and their team. Those who attended the program, they can only tell the taste of the academic feast of NAC 2004.

Indian Society of Electrocardiology organized an ACLS Course at Mumbai on 27<sup>th</sup> June 2004. ISE also organized 2 satellite symposiums on arrhythmias - on 19<sup>th</sup> September 2004 at Kolkata and on 24<sup>th</sup> October 2004 at Chennai.

ISECON 2005 has arrived and Dr. A G Ravi Kishore and Dr. A S Chandrashekara Rao are leaving no stones unturned to make ISECON 2005 as memorable as the expectations of the ISE members.

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

My sincere thanks to Dr. Yash Lokhandwala, Dr. Amit Vora and the Editorial Team for bringing out the ISE Journal - 2005.

Long live Indian Society of Electrocardiology



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

## ECG - 1

14 yrs-old boy, h/s/o palpitations & CHF



1. **The correct diagnosis is**
- Atrial tachycardia
  - Sinus tachycardia
  - Atrial flutter
  - a + b

For correct answer see overleaf

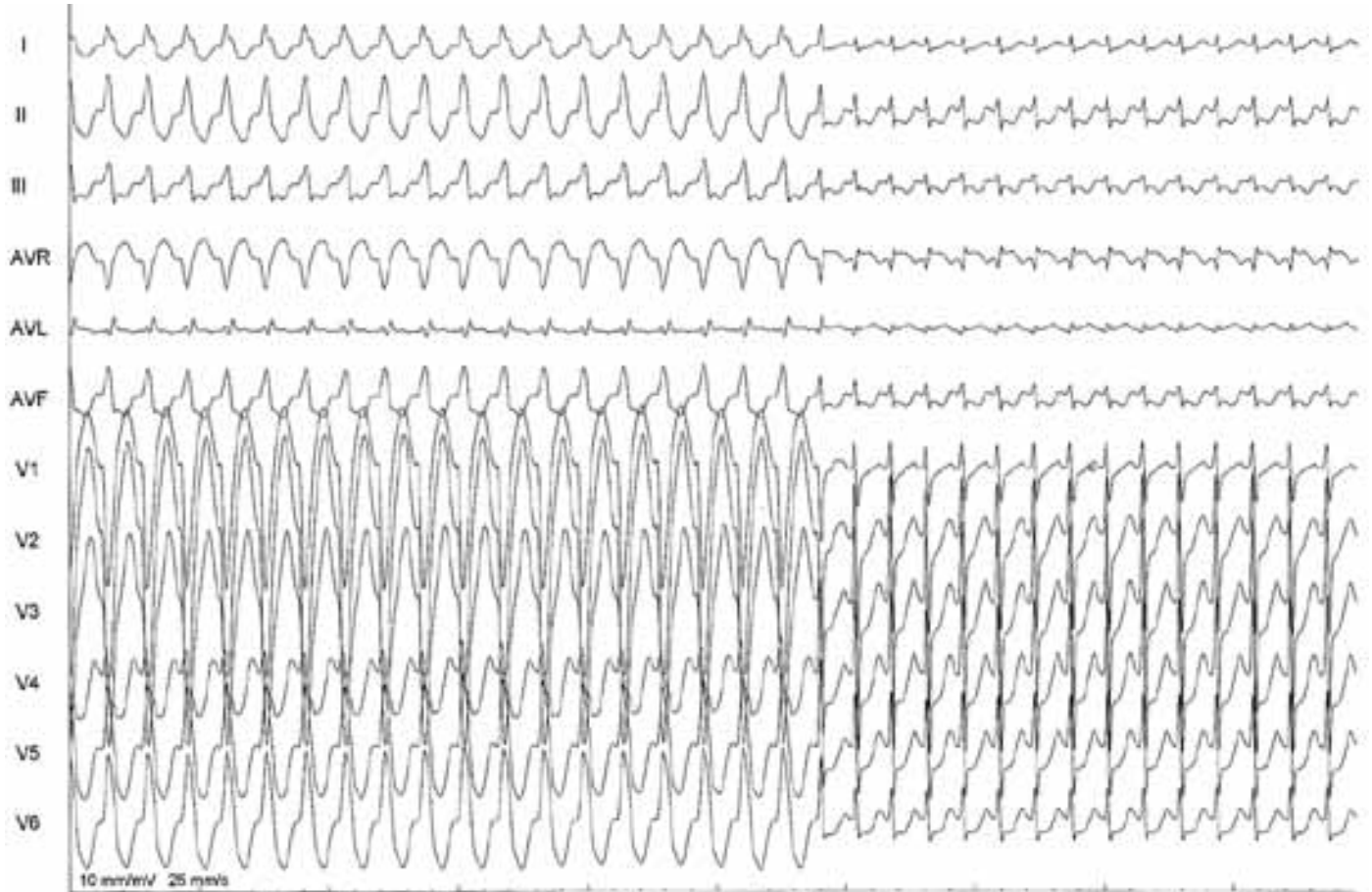
**ECG - 1**

**The correct answer is “d” - Atrial and sinus tachycardia.**

The 12 lead ECG shows two different tachycardias. In the beginning, the ventricular rate is 86 bpm and the underlying atrial rhythm shows two P' waves for every QRS complex, suggestive of atrial tachycardia. The atrial tachycardia rate is 172 bpm and there is 2:1 AV conduction. This is well seen in lead III. It is very likely to be atrial tachycardia especially if we observe the P wave morphology in leads avL and V<sub>1</sub>. One P' wave precedes the QRS complex with a prolonged PR interval and the next non-conducted P wave falls within the terminal part of the T wave. The 6th QRS complex occurs after a longer interval and is preceded by a distinctly different P' wave that is inverted in leads II, III and AVF, suggesting another low atrial ectopic focus. The rhythm subsequently shows 1:1 AV conduction with P wave morphology similar to sinus. Most likely the following rhythm is sinus tachycardia at a rate of 110 bpm. The unusual aspect in the latter half of the ECG is the P wave morphology in lead I, which appears inverted and hence could be another slow atrial tachycardia as well. This patient had clinically presented with heart failure and this was because of tachycardiomyopathy. Interestingly during the atrial tachycardia the ventricular rate is slower due to 2:1 AV conduction but with sinus restoration the rate is faster in view of heart failure.

## ECG - 2

29 yrs-old man with h/o paroxysmal palpitations



2. **The correct diagnosis is**
- AV nodal reentrant tachycardia (AVNRT)
  - VT changing into a SVT
  - Orthodromic tachycardia (AVRT)
  - None of the above

For correct answer see overleaf

**ECG - 2**

**The correct answer is “c” – Orthodromic tachycardia.**

This 12 lead ECG at the beginning shows a wide QRS tachycardia and later converts to a narrow QRS tachycardia. In presence of wide and narrow QRS tachycardia one should entertain the diagnosis of SVT with and without aberrancy. The wide QRS tachycardia in this ECG shows a rapid intrinsicoid deflection with a typical LBBB pattern favoring SVT with aberrancy & not ventricular tachycardia. Normally aberrancy during an SVT occurs with a faster rate – interestingly, the wide QRS tachycardia in this ECG shows a slower rate compared to the narrow QRS tachycardia. The narrow QRS tachycardia shows significant ST depression, suggesting that the P wave is after the QRS complex as in orthodromic AVRT. In an orthodromic tachycardia, the conduction is down the AVN via the normal conducting system and then up the accessory pathway. However, if there is BBB on the same side as the accessory pathway, the impulse going down the AVN gets blocked at the ipsilateral bundle and has to traverse via the opposite bundle branch and across the interventricular septum to reach the accessory pathway on the same side as the BBB. Thus, the length of the circuit of the orthodromic tachycardia during an ipsilateral BBB lengthens and the tachycardia rate is therefore lower. This ECG is thus, an orthodromic tachycardia involving a left sided accessory pathway with LBBB aberrancy in the initial strip. Slowing of a narrow QRS tachycardia on development of BBB aberrancy is indicative of an orthodromic tachycardia with the accessory pathway on the same side as the BBB.



## ECG - 3

In sinus rhythm....

During atrial pacing.....



3. The rhythm seen during atrial pacing in the right panel is suggestive of:
- Induction of polymorphic VT
  - Induction of two monomorphic VTs
  - SVT with aberrancy
  - None of the above

For correct answer see overleaf

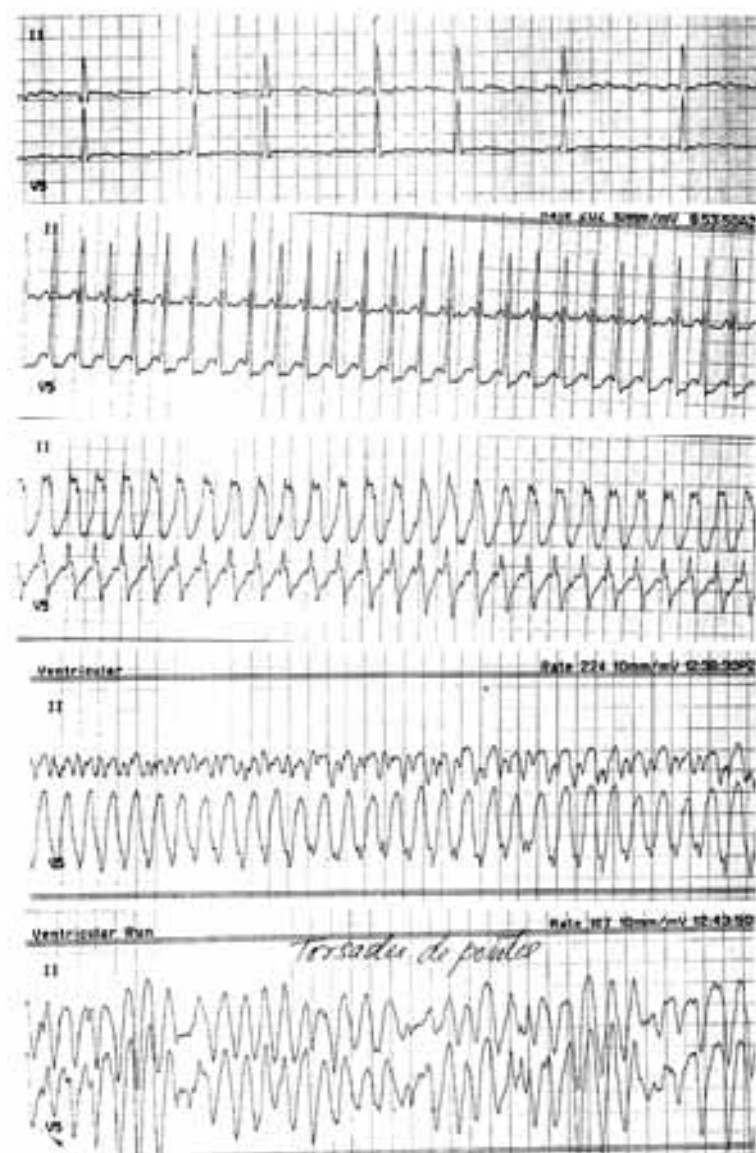
**ECG - 3**

**The correct answer is “d” - None of the above**

The left panel shows a normal sinus rhythm with pre-excitation as evident from short PR interval and presence of delta wave. The right panel shows wide QRS complexes with two different morphologies. Atrial pacing at fast rates would favor conduction down the accessory pathway as the refractory period of the accessory pathway is shorter than AVN. The right panel thus shows maximum preexcitation during rapid atrial pacing. However, there are two different QRS morphologies seen thereby indicating the presence of two independent accessory pathways. Since the QRS complexes are positive in leads  $V_1$  and  $V_2$  with RBBB like morphology, both the pathways are on the left side. The ECG during last 12 beats shows negative QRS complexes in leads I, avL and  $V_6$ , thus, indicating the presence of left lateral pathway. In the first half of the right panel, the QRS complexes are negative in leads III and avF and positive in leads I and AVL suggesting a left posterior pathway. However, the lead II has an equivocal QRS complex, thus indicating a fusion of two accessory pathways- left posterior and left lateral pathways. A polymorphic VT is ruled out, since the QRS morphology does not change from beat to beat. Also, there is no definite BBB morphology in the two tachycardias to suggest SVT with aberrancy.

## ECG - 4

59 yrs-old,  
known IHD,  
c/o dyspnea,  
easy fatigue,  
palpitations &  
syncope



4. This Holter strip does 'not' show
- Atrial flutter
  - Monomorphic ventricular tachycardia
  - Sinus tachycardia
  - Polymorphic VT

For correct answer see overleaf

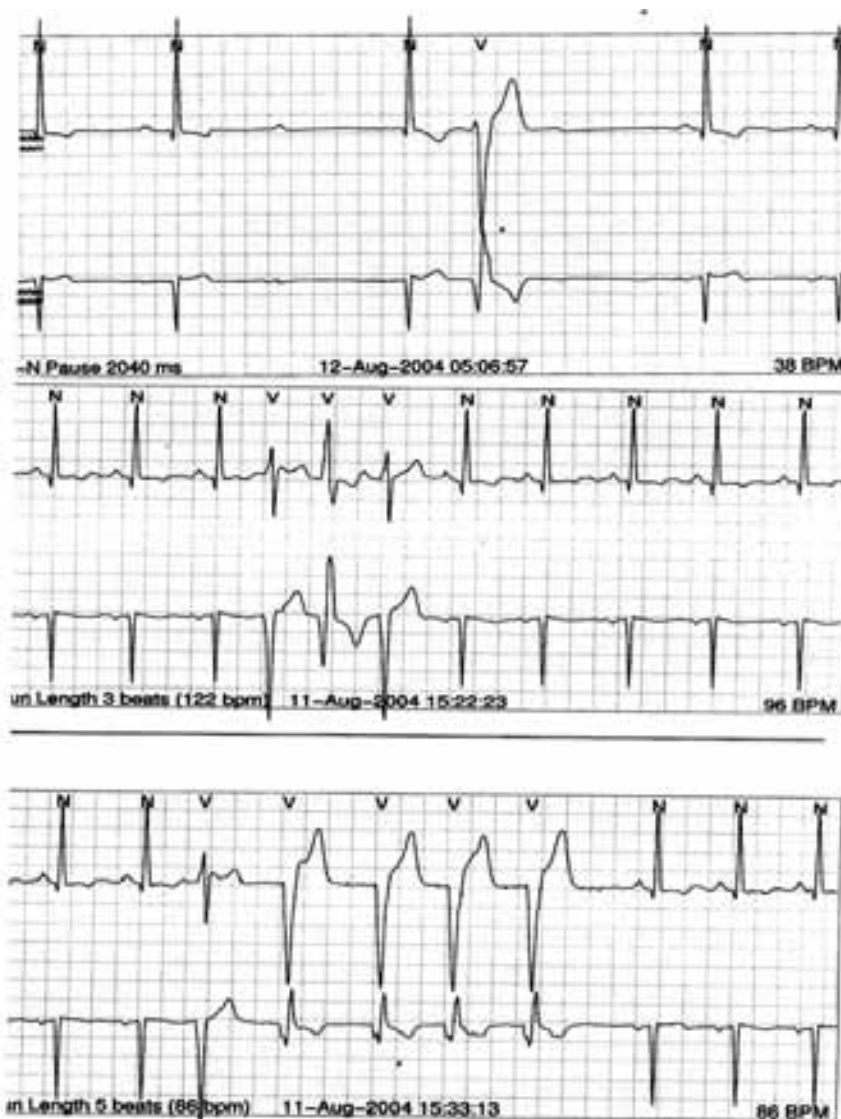
**ECG - 4**

**The correct answer is “c” - Sinus tachycardia**

There are five Holter strips each with a different rhythm in the same patient. The first strip shows atrial flutter with variable AV conduction as evident from the irregularly timed QRS complexes with distinct flutter waves intervening. The second strip shows a regular narrow QRS tachycardia at a rapid rate and appears to be atrial flutter with 1:1 AV conduction. This is suggested from the atrial flutter rate in first strip being exactly same as the tachycardia rate in the second strip. A sinus tachycardia at such rapid rates is very unlikely. The third and the fourth strips show a regular wide QRS tachycardia with bizarre QRS morphology, which are monomorphic VTs. The bottom strip shows an irregular wide QRS tachycardia with constantly changing QRS morphology suggesting a polymorphic VT.

## ECG - 5

52 yrs-old man  
past MI, now  
asymptomatic



5. The correct line of treatment in this patient with tachycardia-bradycardia is
- Pacemaker + Antiarrhythmic drugs
  - AICD
  - RF ablation
  - None of the above

For correct answer see overleaf

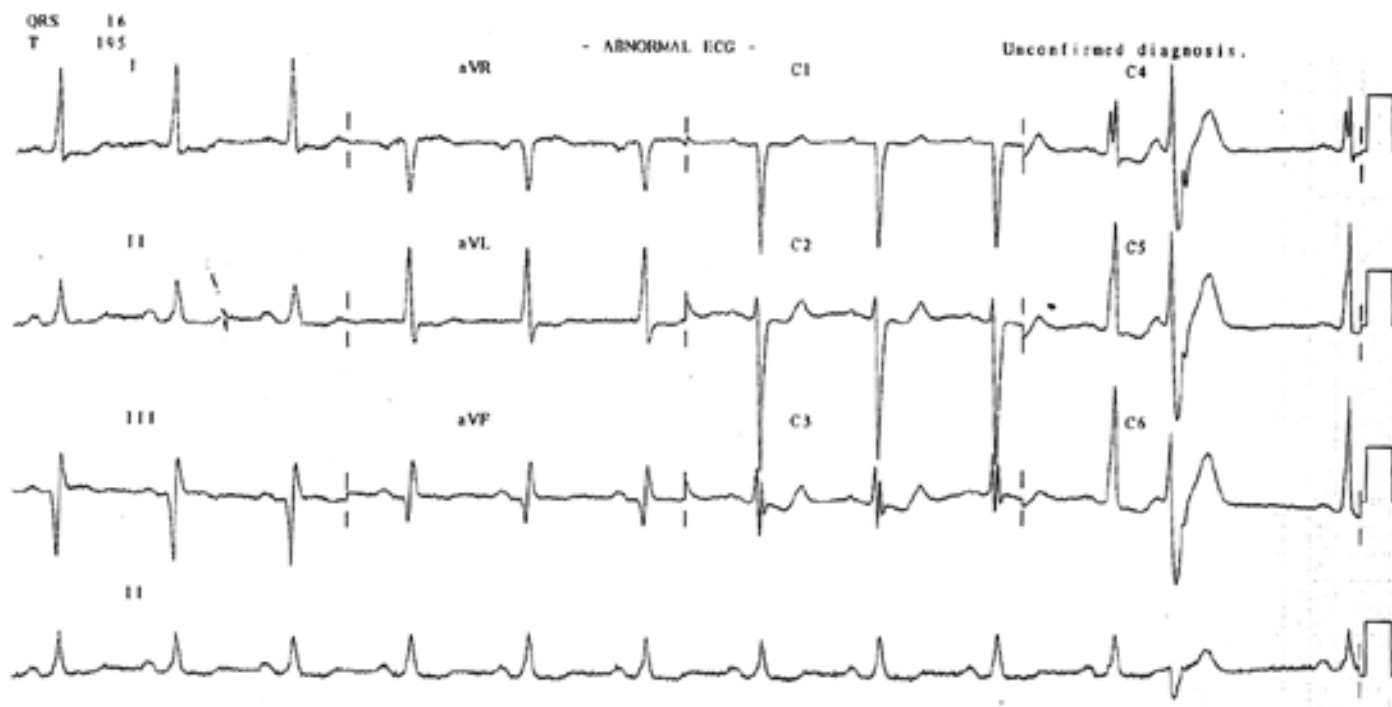
**ECG - 5**

**The correct answer is “d” - None of the above**

There are three Holter strips of this patient with old MI. The first strip shows apparent pauses after 2<sup>nd</sup> and 4<sup>th</sup> QRS complexes along with bradycardia. However, this ECG strip has been recorded during the early morning hours at 05.06.57 hours, at which time sinus bradycardia is physiological. There is a blocked P wave seen after the 2<sup>nd</sup> QRS complex followed by a junctional beat. Similarly, there is a ventricular ectopic beat just after the junctional beat followed by a compensatory pause. The AV block during sleep is likely physiological, related to vagal tone. Hence a pacemaker is not indicated. In the second strip, the 4<sup>th</sup> and the 6<sup>th</sup> complex are ventricular ectopics, while the 5<sup>th</sup> complex could be an aberrantly conducted supraventricular beat. The third strip shows an AIVR (accelerated idioventricular rhythm) @ 90/min. Thus, there are no malignant forms of ventricular ectopics or tachycardia to warrant use of AICD, RF ablation or antiarrhythmic drugs.

## ECG - 6

63 yrs-old, S/P CABG 1996, c/o syncope...



6. The likely cause of syncope in this gentleman is
- Vasovagal syncope
  - Intermittent AV block
  - Postural hypotension
  - VT

For correct answer see overleaf

**ECG - 6**

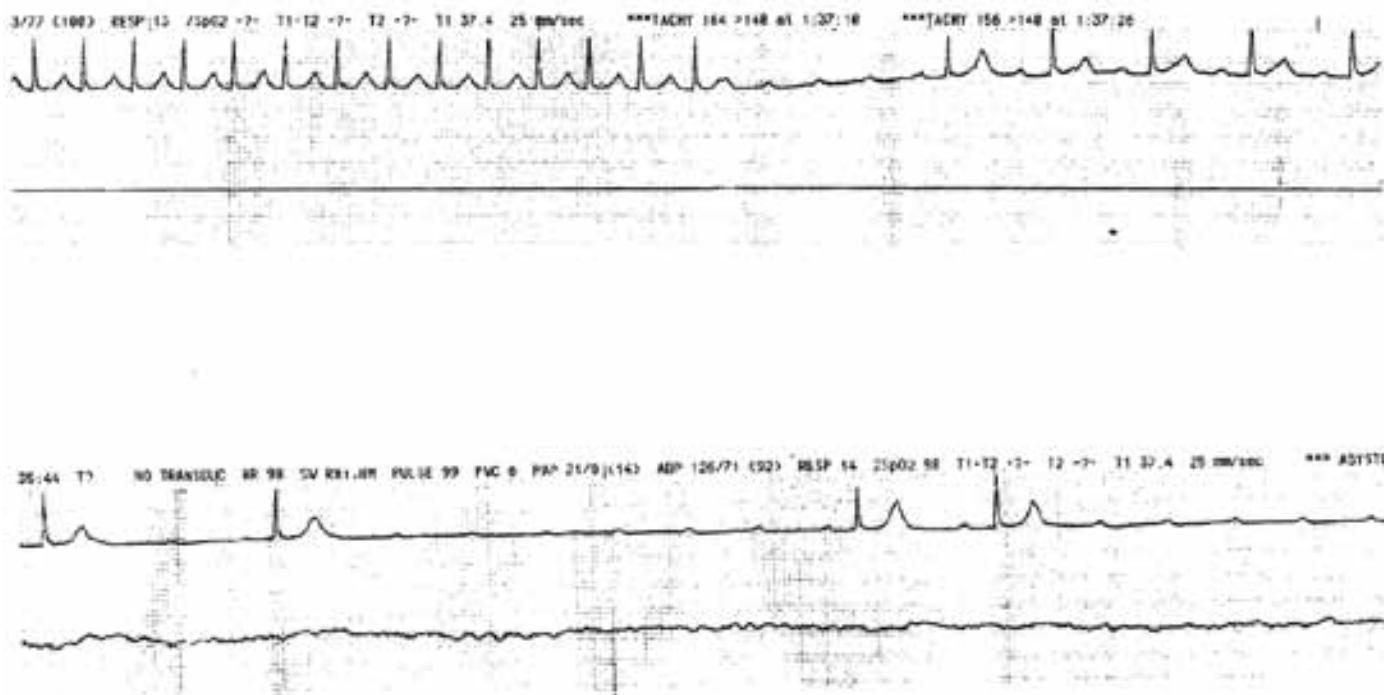
**The correct answer is “d” - VT**

The 12 lead ECG shows old inferior wall MI, normal sinus rhythm with a normal AV conduction. A single ventricular ectopic is seen with an R on T phenomenon. A diagnosis of vasovagal syncope should not be entertained in an elderly patient with structural heart disease (MI in this patient) unless all other malignant causes of syncope are ruled out. In view of the old inferior wall MI, the AV node conduction could suffer. However, this ECG shows no sign of abnormal AV conduction or prolonged PR to suggest intermittent AV block. Postural hypotension should be considered if the patient is on medications known to cause it and importantly the syncope should be postural and the hypotension clinically documented. Thus, VT is the most likely cause of syncope in the setting of old MI, CABG and VPBs with R on T phenomenon.



## ECG - 7

## Repeated episodes of syncope



7. The cause of bradycardia is
- Sinus node dysfunction
  - Paroxysmal AV block
  - a + b
  - Pseudo-bradycardia due to artifact

For correct answer see overleaf

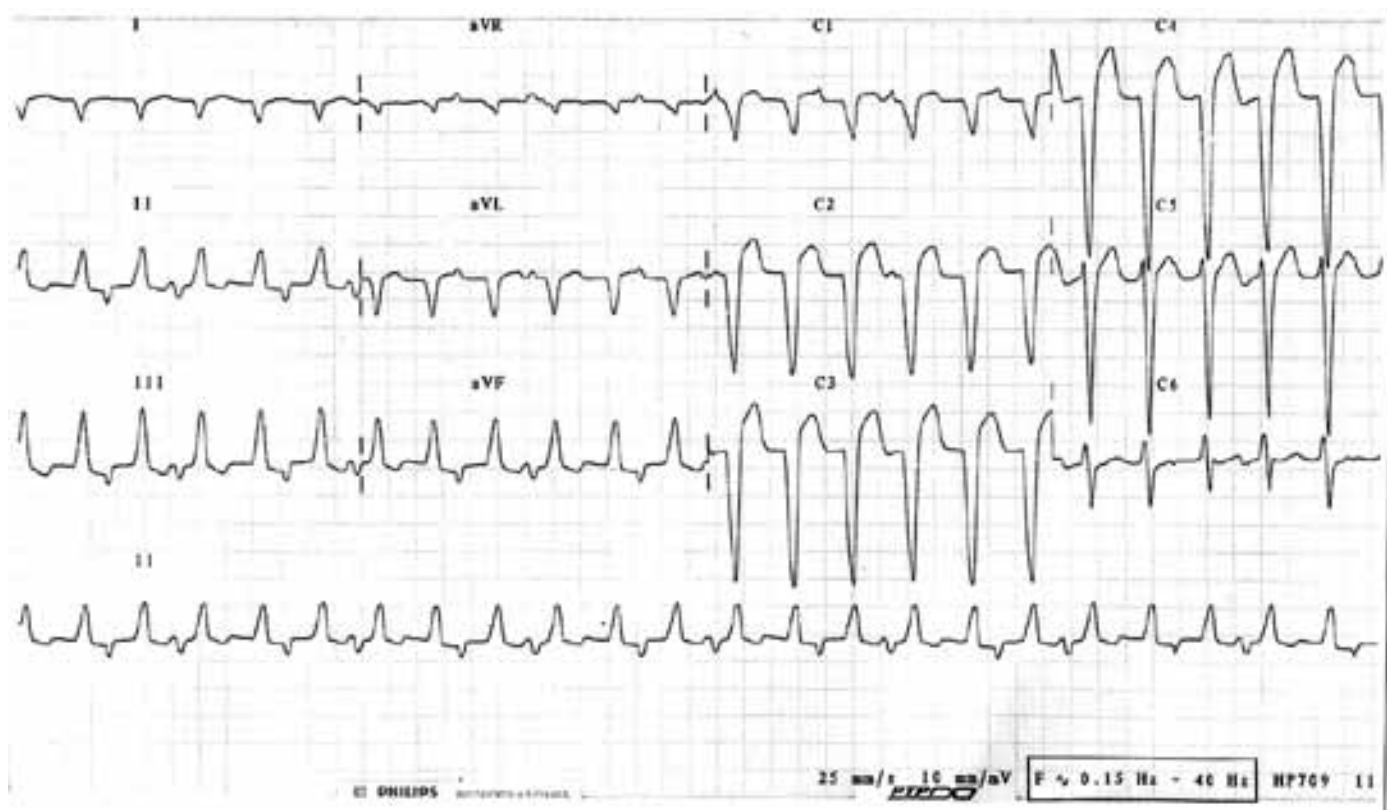
**ECG - 7**

**The correct answer is “b” - Paroxysmal AV block**

The ECG strip shows a regular atrial tachycardia with 1: 1 conduction, where P waves are embedded within the T waves. After the 14th QRS complex, there is a sudden apparent pause with no QRS complexes, but distinct non-conducted P waves are seen indicating a sudden AV block. A similar pause is seen in the 2nd strip with non-conducted P waves. This is called as paroxysmal AV block, occurring in the setting of otherwise normal AV conduction.

## ECG - 8

38 yrs-old, recurrent palpitations



8. This ECG shows
- SVT with aberrancy
  - VT with VA conduction
  - VT with VA dissociation
  - None of the above

For correct answer see overleaf

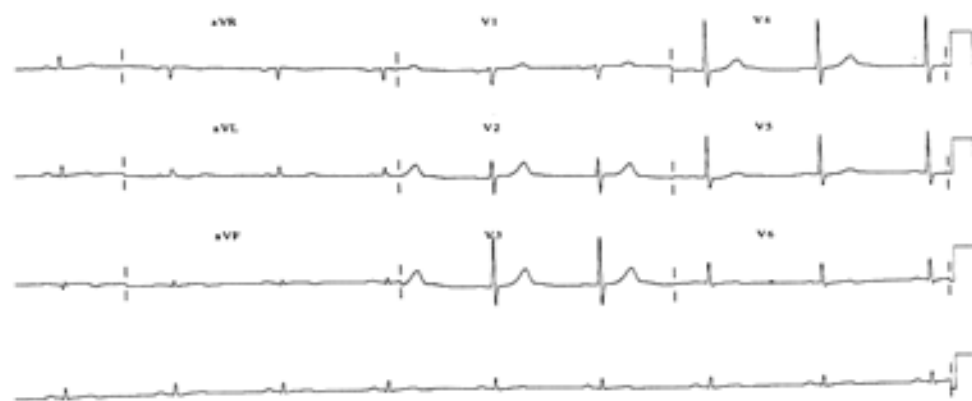
**ECG - 8**

**The correct answer is “b” - VT with VA conduction**

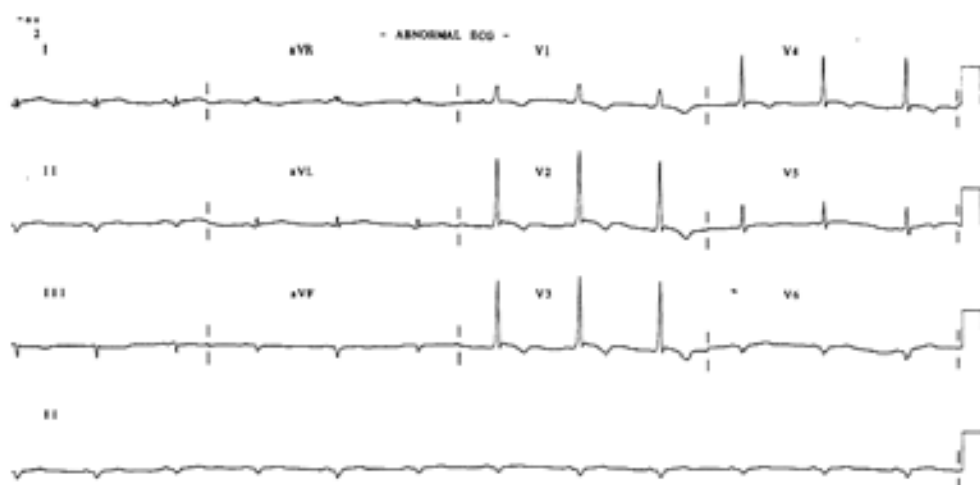
This 12 lead ECG shows a regular, wide QRS tachycardia. There is no definite BBB pattern and thus, an SVT with aberrancy is ruled out. Moreover, the LBBB-like QRS morphology (in lead V<sub>1</sub>) with right axis deviation as seen here, is a combination that is only seen in VT. Also, there are fewer P waves than QRS complexes. This is a VT with retrograde, inverted P waves interspersed between the QRS complexes. There is definite P-QRS relationship, wherein a P wave follows two consecutive QRS complexes with an increasing RP interval followed by the third QRS with no P wave conducted retrogradely. Thus, there is a VA conduction with VA Wenckebach phenomenon.

## ECG - 9

Pre-CABG



Post-CABG



9. After CABG, this patient had developed,

- Inferolateral MI
- RBBB
- Pulmonary thromboembolism
- Pre-excitation pattern

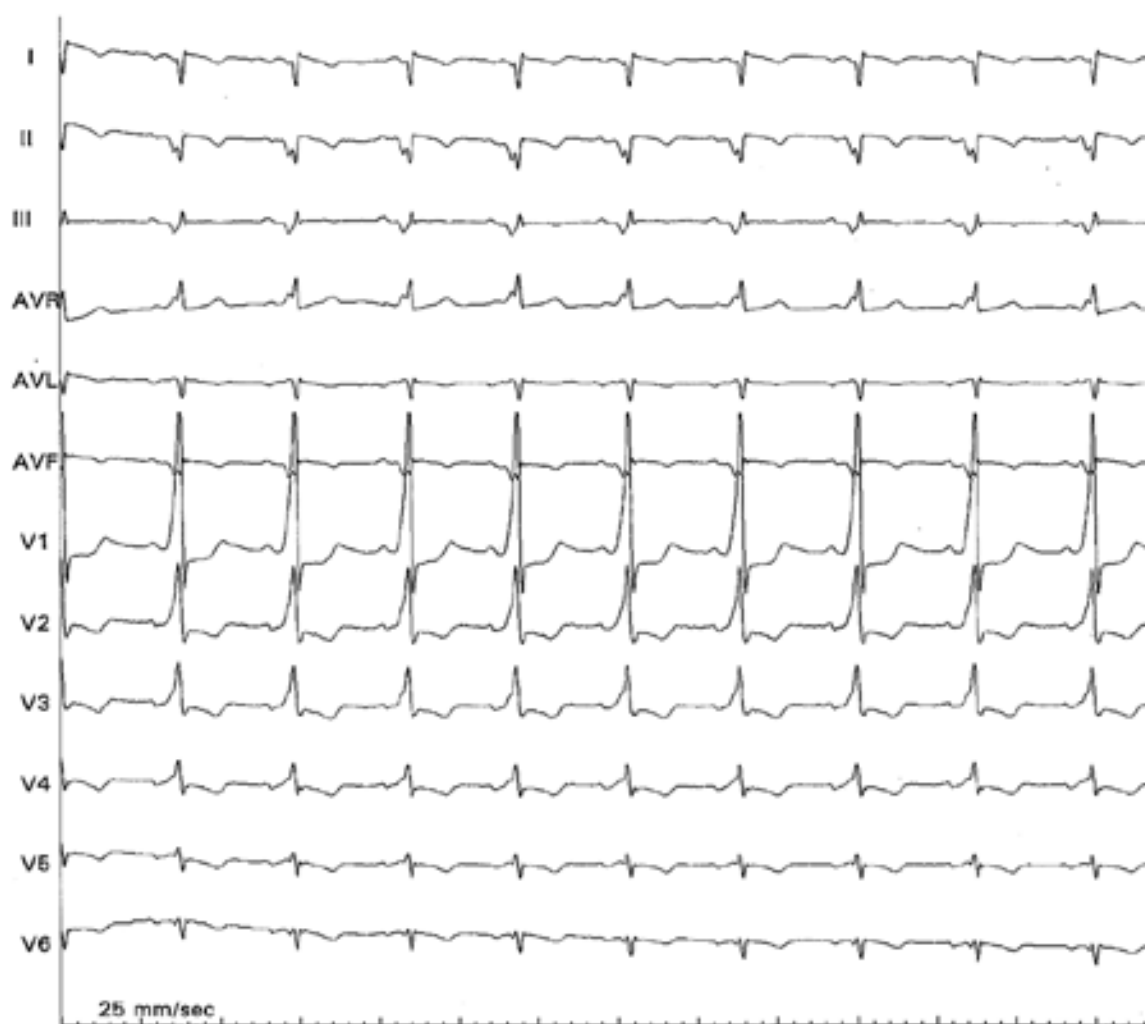
For correct answer see overleaf

**ECG - 9**

**The correct answer is “d” - Pre-excitation pattern**

As compared to the pre-CABG ECG, the post CABG 12 lead ECG shows a change in the QRS axis with prominent R waves in lead V<sub>1</sub>. The inferior leads show “Q” waves after CABG, the PR interval is short, the QRS complexes show a slurring in the initial upstroke followed by a normal conduction. This is a delta wave seen distinctly in precordial leads suggesting unmasking of pre-excitation post CABG.

## ECG - 10



10. The diagnosis is:

- a. RVH
- b. WPW pattern
- c. Dextrocardia
- d. b + c

For correct answer see overleaf

**ECG - 10**

**The correct answer is “d” - WPW pattern and dextrocardia**

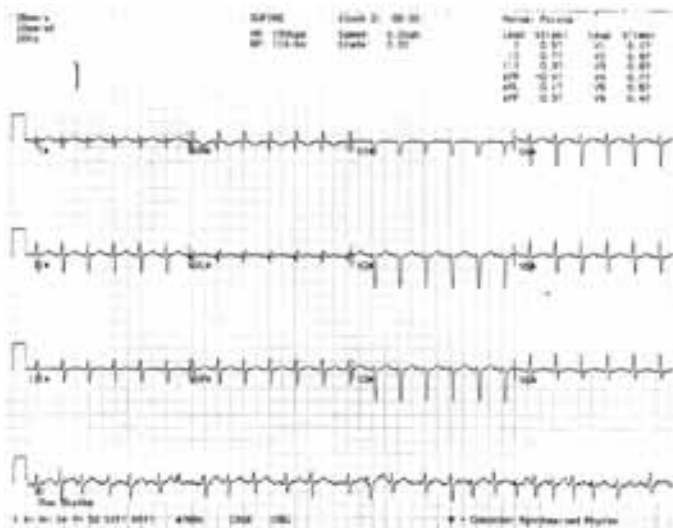
There is clearly preexcitation – the short PR, delta wave and wide QRS with secondary ST changes (best seen in leads  $V_2$ - $V_4$ ). It is important to look for the shortest PR; lead avL &  $V_5$ - $V_6$  would misleadingly show a normal PR interval due to an isoelectric delta wave. The P waves are inverted in leads I & avL suggesting dextrocardia with atrial situs inversus. The reverse R wave progression from  $V_6$  to  $V_1$  confirms dextrocardia.



## ECG - 11

36 yrs-old lady with 6 months h/o palpitations

Panel A



Panel B



11. The ECG in Panel B during stress test shows
- Atrial fibrillation
  - Atrial fibrillation with NSVT
  - Sinus tachycardia
  - AF with conduction down accessory pathway

For correct answer see overleaf

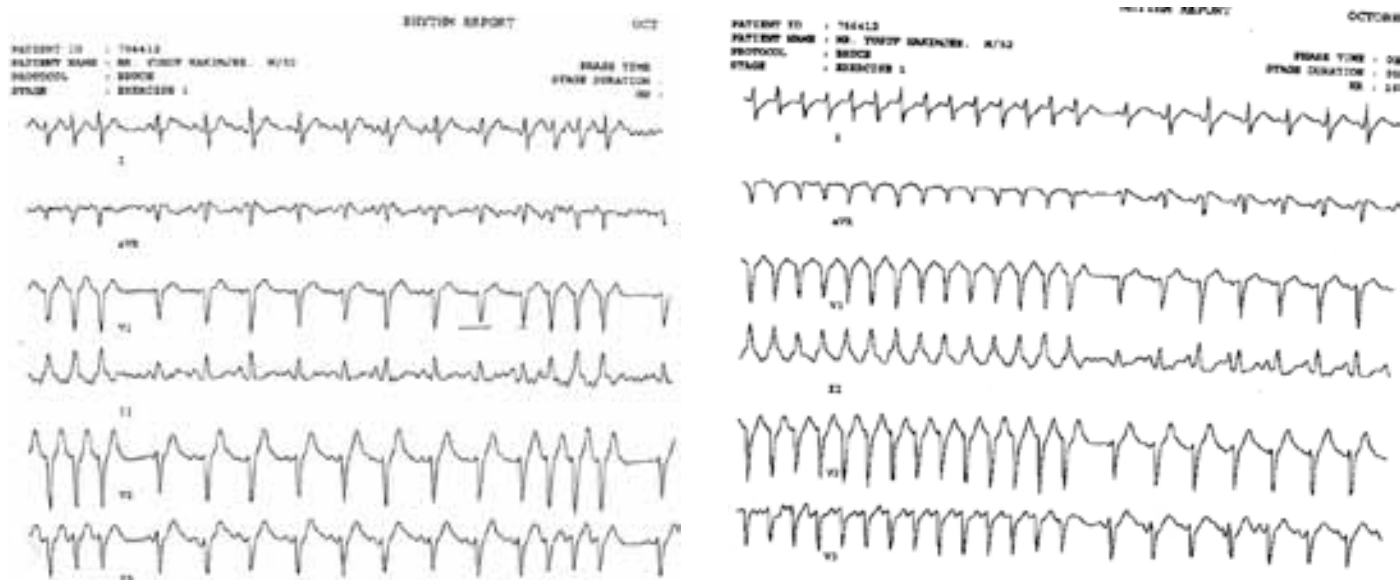
**ECG - 11**

**The correct answer is “a” - Atrial fibrillation**

Panel A ECG shows atrial fibrillation as the underlying rhythm with a fast ventricular rate. This is clear in the raw rhythm strip below. The “computer synthesized rhythm” produces an artefactual regular rhythm. Panel B ECG shows persistence of atrial fibrillation with a short ill-sustained run of wide QRS complexes. As seen in Panel B, after the 5<sup>th</sup> QRS complex there is a longer R-R interval and this is subsequently followed by a wide QRS complex but, now at a shorter interval. This short-long-short cycle sequencing in AF favors phase 3 aberrancy in the left bundle. Here, the run of wide QRS complexes is thus due to LBBB. This is also called as Ashman’s phenomenon.

## ECG - 12

38 yrs-old with exertional palpitations



12. These ECGs show exercise induced

- Atrial tachycardia
- Ventricular tachycardia
- Atrial fibrillation
- AVNRT

For correct answer see overleaf

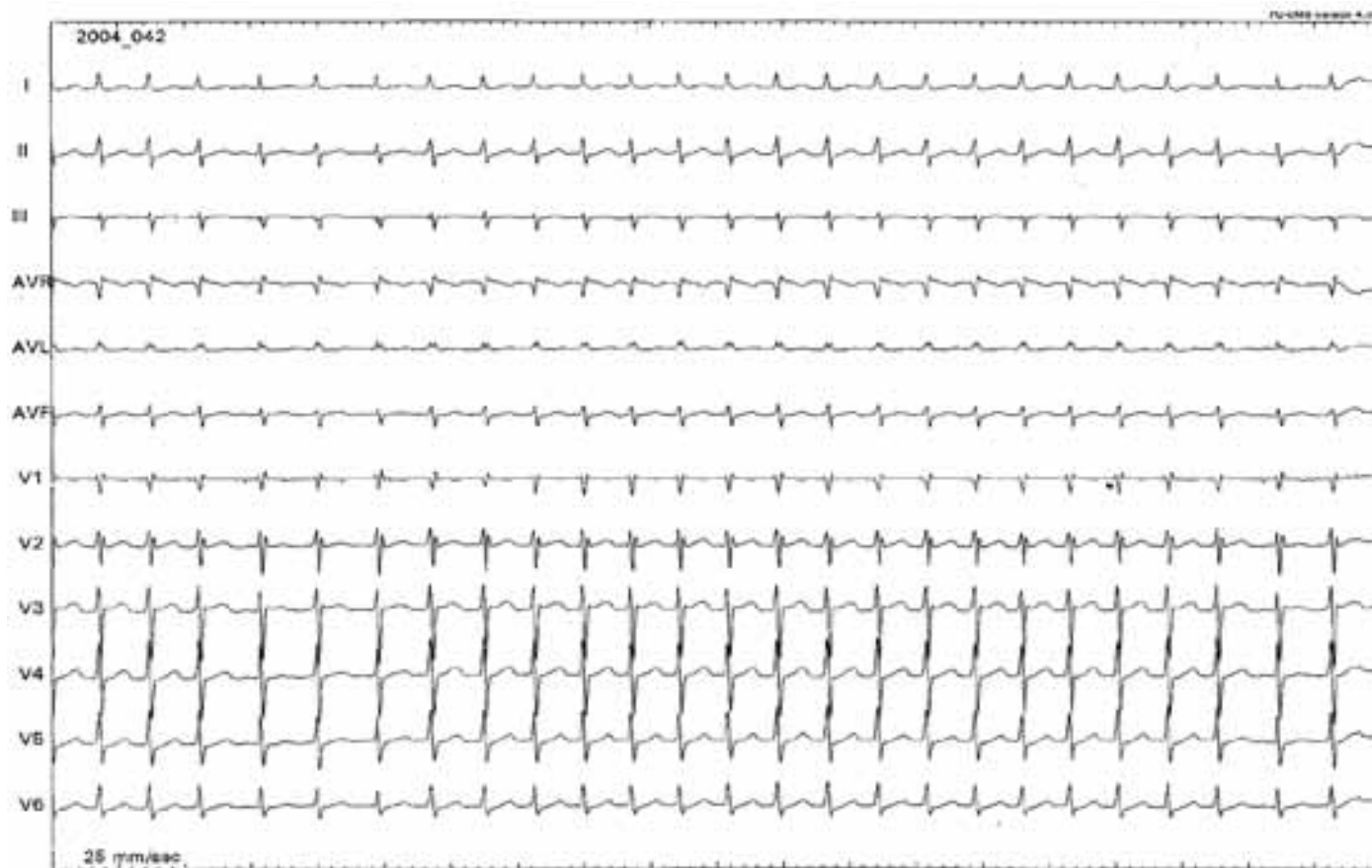
**ECG - 12**

**The correct answer is “a” - Atrial tachycardia**

The left panel shows a run of regularly spaced three and four rapid beats at the beginning and towards the end of the ECG respectively with a normal sinus rhythm intervening. The right panel also shows an ill-sustained regular tachycardia followed by a normal sinus rhythm. The QRS morphology during the tachycardia is similar to the one in normal sinus rhythm. Lead V<sub>1</sub> shows peaking of the T waves during tachycardia due to superimposed P waves. Thus, this regular tachycardia has a supraventricular origin and is exercise induced atrial tachycardia.

## ECG - 13

65 yrs-old, repeated paroxysmal palpitations



13. This ECG shows
- Atrial tachycardia
  - AVNRT
  - Atrial fibrillation
  - AVRT

For correct answer see overleaf

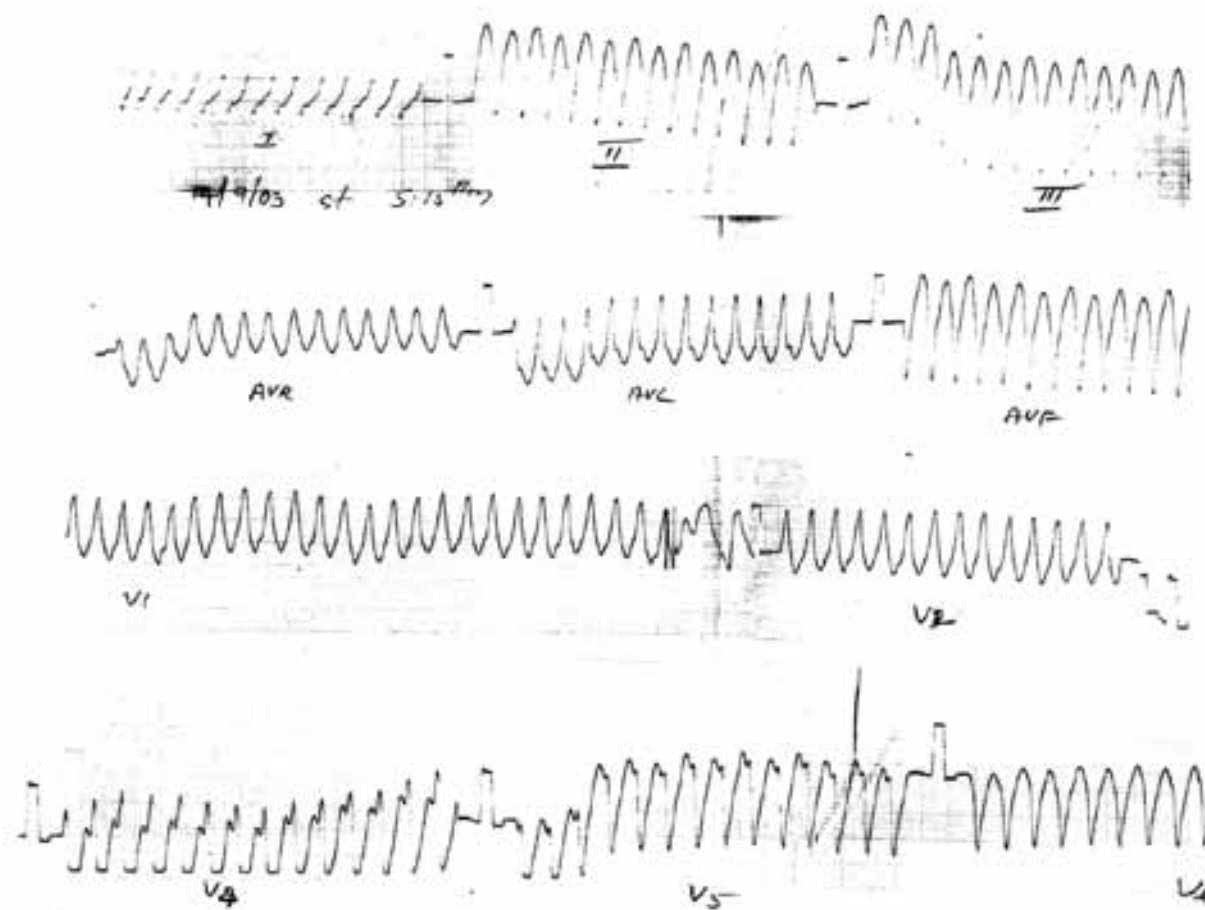
**ECG - 13**

**The correct answer is “a” - Atrial tachycardia**

This 12 lead ECG shows an irregular narrow QRS tachycardia. The rhythm being irregular rules out AVNRT and AVRT. The P waves are often within the QRST complexes and at some places seen distinctly separate from the QRS complexes as evident in lead V<sub>1</sub>. It is clear that there are more P waves than QRS complexes. Thus, this is atrial tachycardia with discernible P waves and variable AV conduction. In AF, P waves are not seen distinctly.

## ECG - 14

28 yr-old, first episode of palpitations, syncope



14. This ECG shows VT arising from

- RV apex
- LV outflow
- LV infero-apical
- RV outflow

For correct answer see overleaf

**ECG - 14**

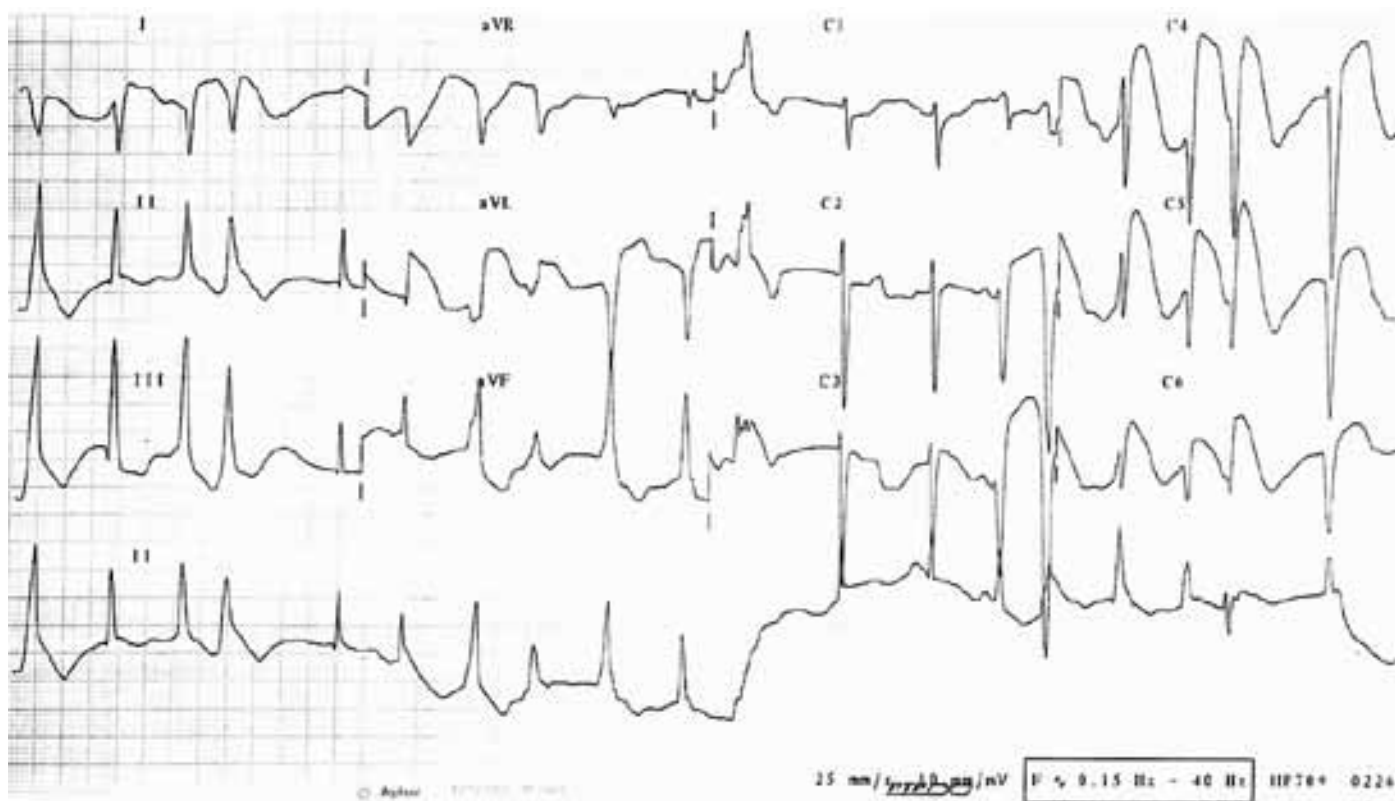
**The correct answer is “c” - LV infero-apical**

The 12 lead ECG shows a regular wide QRS tachycardia @ 250/min, which is a VT. The positive QRS complexes in leads V<sub>1</sub> and V<sub>2</sub> suggesting RBBB like morphology. Thus, the focus of VT is in the LV. The QRS in leads II, III and AVF are negative and those in leads I, avL and avR are positive. This indicates the focus in the inferior and apical portion of the LV. LVOT VT would have positive QRS complexes in the inferior leads.



## ECG - 15

32 yr-old lady, day 1 post-op (thyroid tumor), seizures



15. The cause of seizures is likely to be

- VT
- Coronary ischemia
- Sinus node dysfunction
- Non-cardiac

For correct answer see overleaf

**ECG - 15**

**The correct answer is “a” - VT.**

The 12 lead ECG shows significant QT prolongation with multiple PVCs. The QT prolongation is well appreciated following the narrow QRS complexes in lead  $V_1$ , where it is seen that the QT is much more than half the RR interval. The PVCs show an ‘injury’ pattern ST elevation in leads  $V_5$ - $V_6$ , but this cannot be used to diagnose coronary ischemia. In the setting of QT prolongation with multiple PVCs, torsade de pointes is a distinct possibility. Thus, the risk of VT is very high in this patient and the likely cause of seizure.