ANNOUNCEMENT

Indian Society of Electrocardiology

ISECON 2005

Indian Society of Electrocardiology is organizing its
Annual Conference – **ISECON 2005**

on

2nd and 3rd April 2005

at

Bangalore

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C O N T E N T S

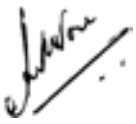
Editorial

Dear Readers,

Happy Diwali to all of you in advance. We are looking forward to NAC 2004 (Nagpur Arrhythmia Conference) which promises to be a sumptuous scientific feat. The organizing committee headed by Dr. Mahorkar has left no stone un-turned in trying to ensure a high quality for this meet. We are sure that the deliberations during the academic sessions will be of much use to one and all.

The last issue of the IJE was mainly dedicated to the ECGs of the interactive session conducted by Prof. HJJ Wellens. The feedback we received was very positive and we were told that this kind of presentation can be used for educational purpose. Keeping this in mind we are bringing forth this issue in a similar vein. The last ISECON in Delhi earlier this year had an ECG session prepared by Dr. Juneja R. & Dr. Pavri B. These ECGs are included in this issue with useful practical pointers discussed. We thank Dr. Swapna Athawale in helping us edit this issue.

Yash Lokhandwala
Editor



Amit Vora
Editor

From Hon. Secretary's Desk

Dear Members,

It is really heartening to see the progress of Indian Society of Electrocardiology. Membership is on rise so are the academic activities.

ISECON-2004 at New Delhi was a grand success under the leadership of Dr. K.K. Talwar and a very able Organizing Secretary Dr. Rajnish Juneja.

Seeing the interest of delegates and wish from the members of the society, it was planned that a mid-term CME will be organized in addition to our regular Annual Conference - ISECON. A result of this is Nagpur Arrhythmia Course (NAC), being organized in collaboration with Cardiological Society of India, Vidarbha Chapter. Dr. Uday Mahorkar, President ISE and Dr. Prashant Jagtap President Elect CSI Vidarbha Chapter have made extensive arrangements for a national academic feast.

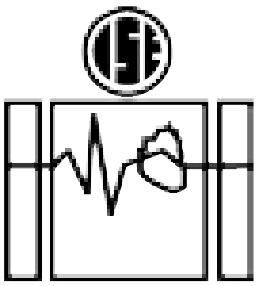
ISECON-2005 is planned at Bangalore on 2nd-3rd April, 2005. Dr. Ravi Kishore has taken the mantle to make it a roaring success.

An Advanced Cardiac Life Support Course was organized under the banner of ISE at Mumbai on 27th June 2004, which was a grand hit. We are getting enquiries for such many courses in future. A Pacemaker / Arrhythmias Update was organized at Kolkata on 19th September 2004 which was also well appreciated.

The popularity of ISE is on rise and it will be an endeavor that we will not look back.

With best wishes,

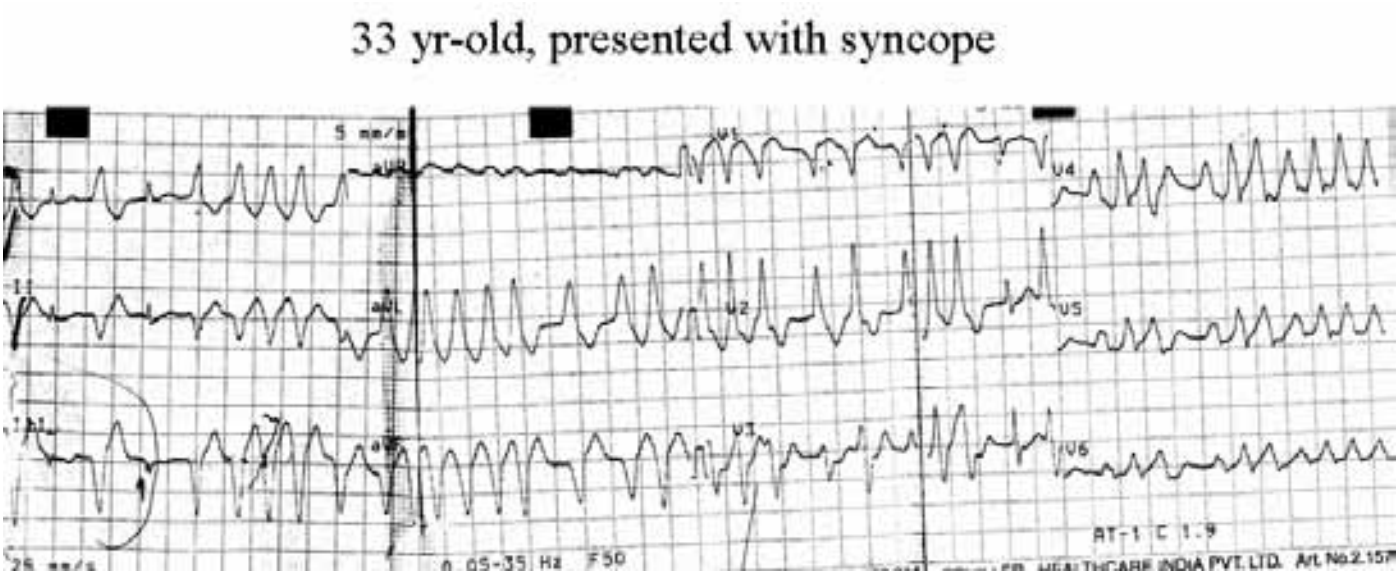
Dr S B Gupta
Hon. Secretary
Indian Society of Electrodardiology



Long live

Indian Society of Electrocardiology

ECG - 1



1. What is the diagnosis?

a. Old Inferior wall MI with VT/ VF
b. Polymorphic VT
c. WPW Syndrome with A Fib
d. A Fib with bundle branch block

For correct answer see overleaf

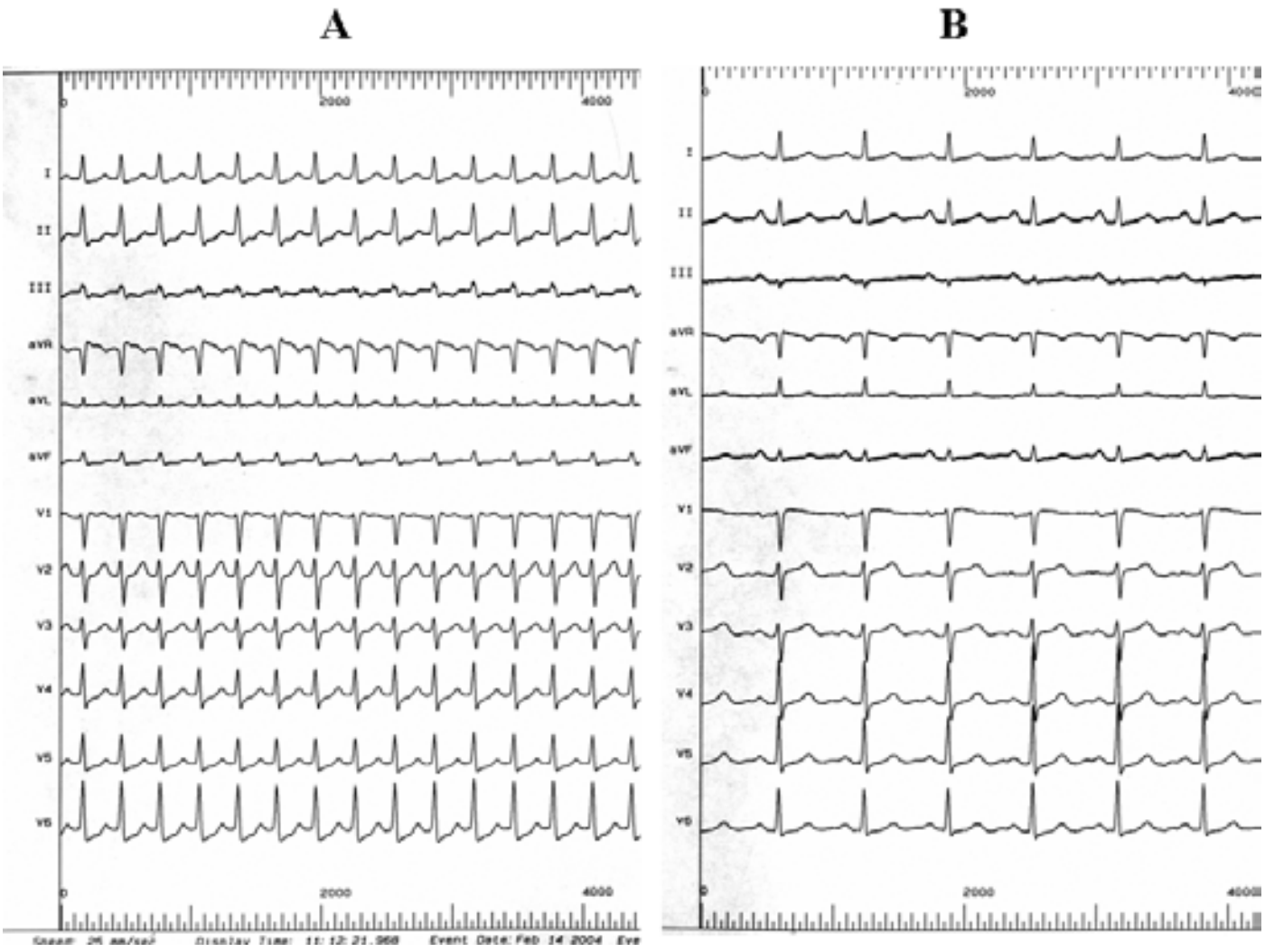
ECG - 1

The correct answer is "c" - WPW syndrome with AF

The 12 lead ECG show a fast, broad and irregularly-irregular *('FBI', as described by Dr. Wellens)* tachycardia with monomorphic QRS complexes. This is a classic ECG of AF with fast conduction over an accessory pathway. The inferior leads show a QS pattern but this is a pseudo-infarct pattern as recognized in the 2nd and 4th QRS complexes in lead II without any Q waves. These complexes show less or no preexcitation because of predominant conduction over the AV node. This is a monomorphic wide QRS tachycardia as evident in the individual leads. Polymorphic tachycardia would be defined when within the same lead there is differing QRS morphology. Also one cannot argue this to be atrial fibrillation with bundle branch block aberrancy because then the QRS complexes should show a typical bundle branch block pattern and also the intrinsicoid deflection should be sharp.

The location of the accessory pathway is right postero-septal. This is recognized by the of negative delta in inferior leads suggesting posterior pathway. The sudden transition of QRS complexes from V_1-V_2 support a septal location and likely right sided because of negative delta in lead V_1.

ECG - 2



2. The tachycardia in panel A is?

a. Typical AVNRT
b. Atypical AVNRT
c. Orthodromic AVRT
d. Ectopic atrial tachycardia

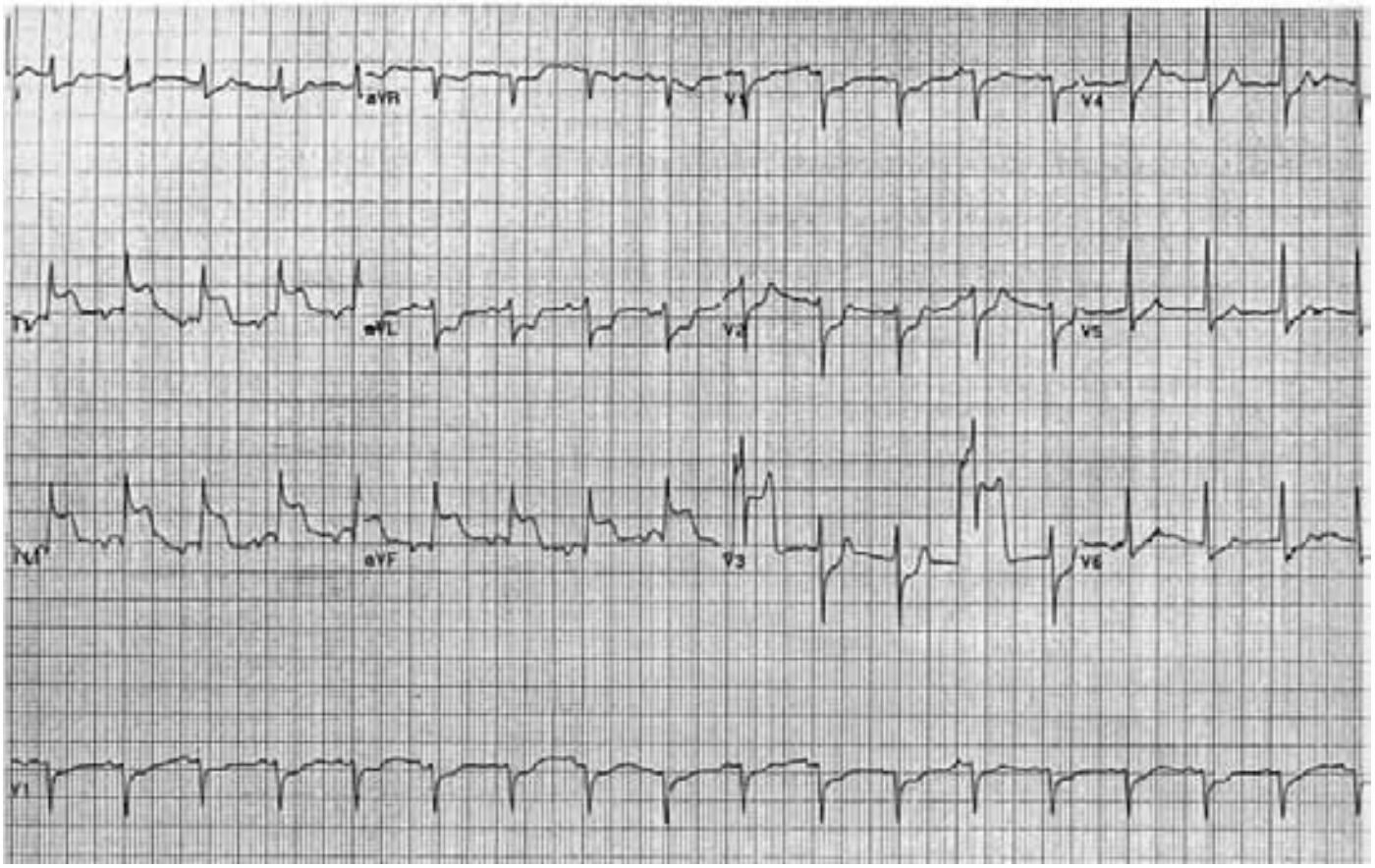
For correct answer see overleaf

ECG - 2

The correct answer is "a" - Typical AVNRT

Panel A shows a narrow QRS, regular tachycardia with no obvious identifiable P waves. On comparison with sinus rhythm ECG (panel B), there appears a pseudo S wave in inferior leads and pseudo R in lead V_1 during tachycardia. This suggests the P waves are buried within the QRS complex, only distorting the terminal portion of the QRS. P wave within the QRS means the atria and ventricles are activated simultaneously which classically happens with typical AVNRT. In atypical AVNRT, the retrograde limb within/around the AV node is the slow pathway, which results in a delayed activation of the atrium and has a longer RP interval. Orthodromic tachycardia should also show a distinct P waves outside the QRS complex although the RP interval is less than the PR interval. Ectopic atrial tachycardia would show presence of distinct ectopic P' waves with RP interval longer than the PR interval.

ECG - 3



3. Which vessel is occluded?

a. Proximal RCA
b. Mid RCA
c. Proximal Left Circumflex
d. Proximal RCA with LAD

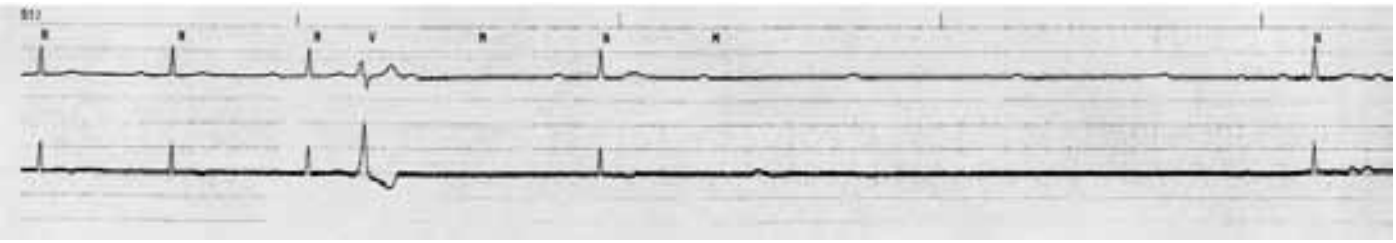
For correct answer see overleaf

ECG - 3

The correct answer is "a" - Proximal RCA

The 12 lead ECG shows hyperacute inferior wall MI, with ST elevations in leads II, III and aVF with ST segment depression in the anterior and lateral leads V_1-V_4, I, avL and V_5-V_6. The ST elevation in lead III is more than in lead II with ST depression in lead I; this is suggestive of a RCA occlusion. The supraventricular rhythm appears not sinus but low atrial, with inverted P waves in the inferior leads. Thus, the RCA occlusion is proximal before the origin of sinus nodal artery.

ECG - 4



4. Echocardiography in this patient shows evidence for a prior septal infarction. From the ECG, the only correct statement is:

a. The likely site of block is within the AV node.
b. The likely site of block is within the bundle of His.
c. An EP study is recommended before considering pacemaker implantation.
d. An EP study will likely show that this patient will not need a pacemaker.

For correct answer see overleaf

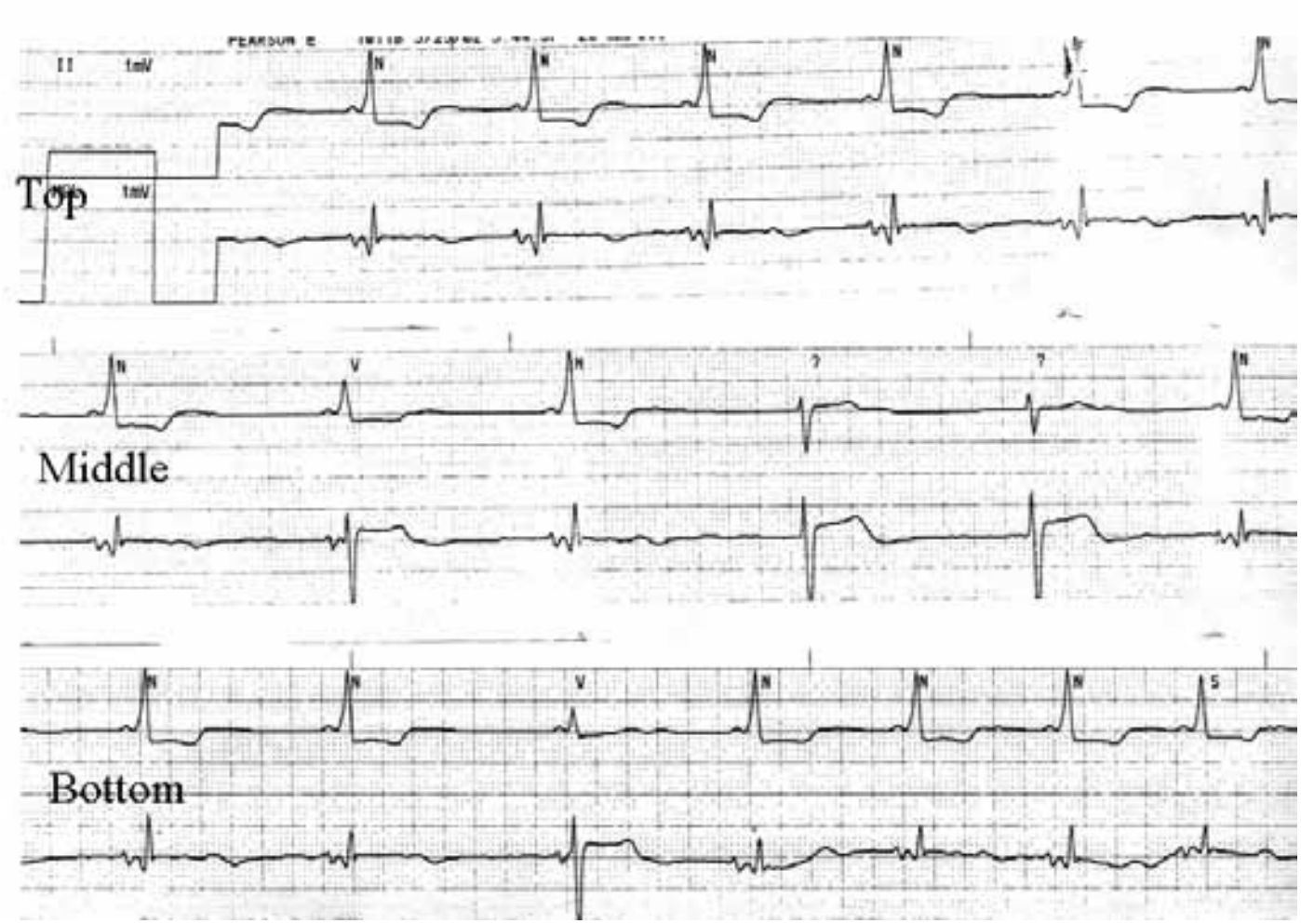
ECG - 4

The correct answer is "b" - The likely site of block is within the common bundle of His

The Holter ECG strips show first 3 sinus complexes with first degree AV block. The 4th complex is a ventricular premature complex, followed by complete AV block. This is classically described as a paroxysmal AV block. This is a result of phase 4 aberrancy in the His bundle fibers that occurs following lengthening of the cardiac cycle, most often following a ventricular ectopic. This phenomenon is seen in diseased hearts, in this case prior septal MI, suggesting ischemia of His bundle. The AV block though paroxysmal can be malignant and mandates permanent pacemaker implantation.

ECG - 5

Holter during sleep



5. This ECG suggests all of the following statements are correct, except:

a. Sinus bradycardia is physiologic during sleep
b. There is manifest preexcitation causing QRS fusion
c. There is likely a competing junction escape resulting in a non-preexcited (narrow) QRS complex after the longest RR intervals
d. An alternate explanation may be phase IV block in the accessory pathway (during long cycles).

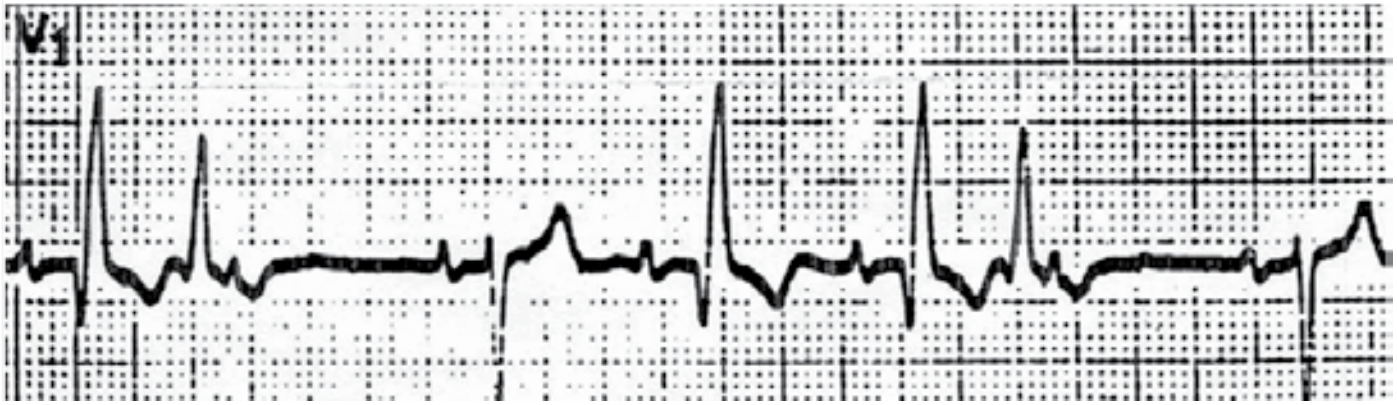
For correct answer see overleaf

ECG - 5

The correct answer is "d" - An alternate explanation may be phase IV block in the accessory pathway at long cycles

The ECG shows sinus bradycardia (at night time) with manifest but variable degree of preexcitation in the form of short PR interval and delta wave in most of the complexes (with varying degree of fusion between accessory pathway and AV nodal conduction). In termittently there is less preexcitation (2^{nd} complex in middle strip and 3^{rd} complex in bottom strip), especially following a longer RR interval and this is likely because of more AV nodal conduction and less of AP contribution to the QRS complex. The 4^{th} and 5^{th} complexes in the middle strip are escape junctional complexes as a result of significant sinus bradycardia. These junctional complexes are obviously not preexcited as there is no sinus activity and no AV conduction (over AP or AV node). Thus there is no evidence of any phase IV block in the AP.

ECG - 6



6. Rhythm strip (lead V_1) from a 64 year-old man. Based on this strip, which of the following statements are true:

a. The patient has likely not had a prior septal MI.
b. The patient has frequent VPCs, the morphology of which suggest a prior septal MI.
c. The patient has frequent APCs, that conduct with aberrancy.
d. There is rate related RBBB due to "long-short" sequencing.
e. The occurrence of RBBB is associated with unmasking of a prior septal MI.

A. 1 is correct
B. 1 and 3 are correct
C. 2, 4 and 5 are correct
D. Only 3 is correct

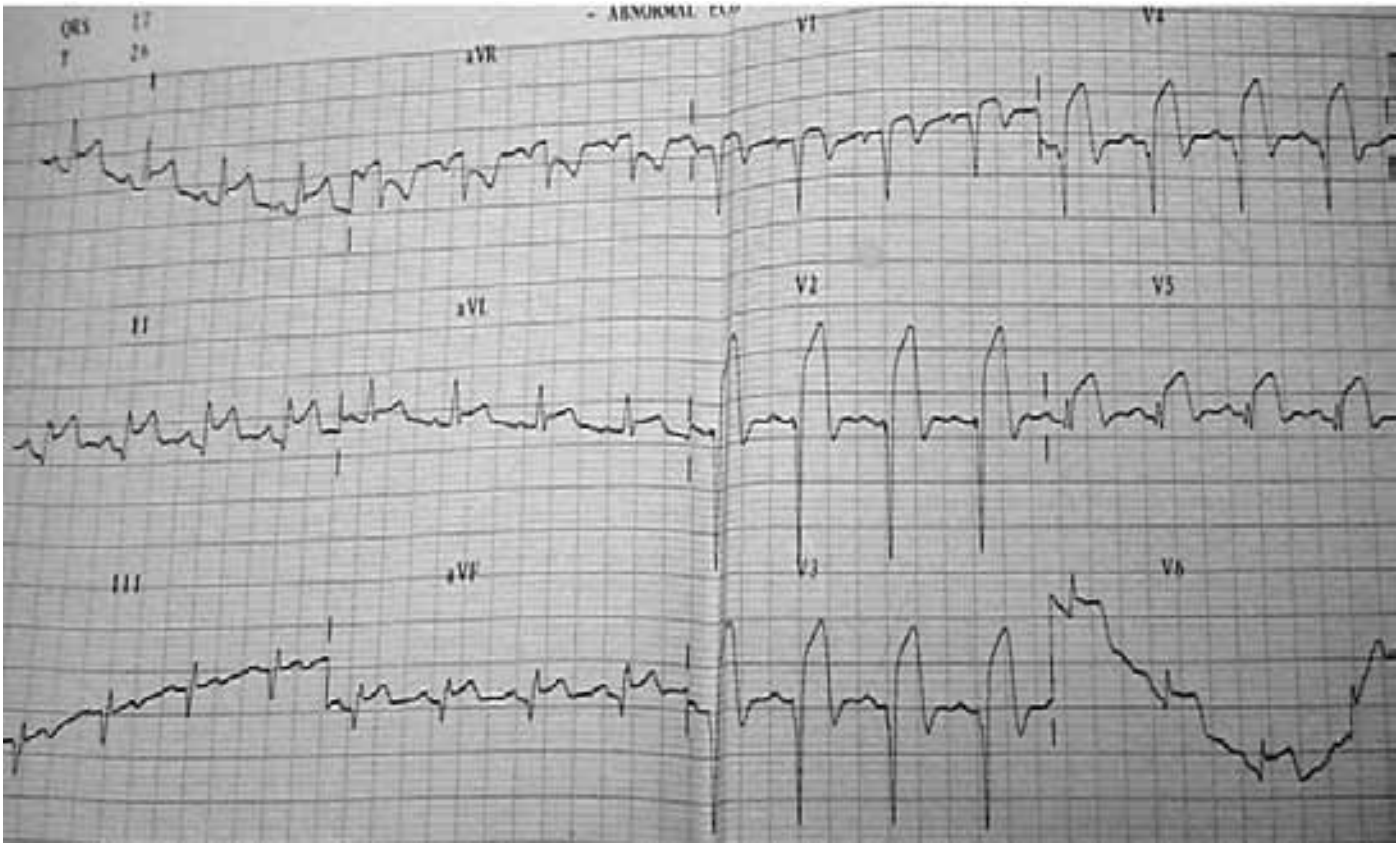
For correct answer see overleaf

ECG - 6

The correct answer is "c" - 2, 4 and 5 are correct.

This ECG strip shows 3 different QRS morphologies. The 3rd and the 7th complex are normally conducted whereas the 2nd and 6th complexes are VPCs with a qR morphology which is suggestive of underlying infarct. The compensatory pause following the VPCs results in a short-long-short sequence resulting in right bundle branch block aberrancy in the 4th & 5th complexes. The RBBB aberrancy also shows a qR pattern thus unmasking the underlying septal MI. There are no APCs.

ECG - 7



7. The culprit vessel/s causing the infarct is:

a. Proximal LAD
b. Proximal LCX
c. LAD + RCA
d. Mid LAD

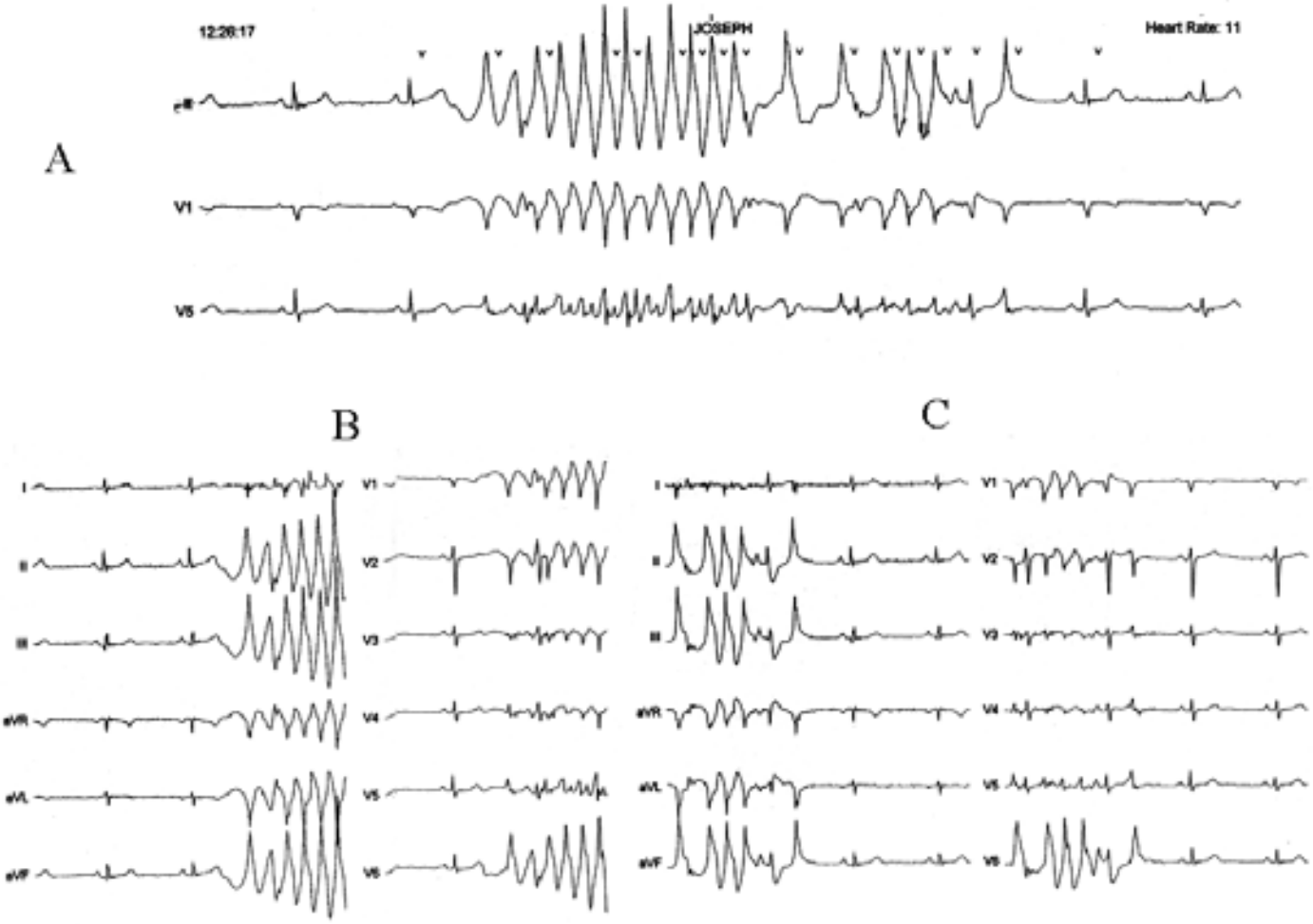
For correct answer see overleaf

ECG - 7

The correct answer is "d" - Mid LAD

The 12 lead ECG shows acute anterior and inferior wall MI with Q waves and ST elevations in leads V_1-V_6, I, aVL and II, III & aVF. The LAD is culprit for the anterior wall MI and the inferior wall MI is most often due to a RCA or LCx occlusion. It would be rare to have a total occlusion of two major vessels simultaneously. Lead aVR show ST depression and leads I & aVL ST elevation suggesting the LAD occlusion after the first major septal artery and before the diagonal (mid-LAD). It has been observed that in the setting of an acute anterior wall MI (& no known previous CAD), presence of Q waves in inferior leads does not indicate a large infarct. The lesion was in mid LAD, with a relatively preserved LV systolic function (> 40%). The LAD was wrapping around the ape (type III) and supplying the inferior wall as well explaining the ST elevation in those leads. In a proximal LAD occlusion and type III LAD, the Q waves in inferior leads are masked.

ECG - 8



8. The Holter shows:

a. Polymorphic VT/VF
b. Atrial fibrillation with rapid conduction over AP (WPW syndrome)
c. Torsade de Pointes
d. Holter Artifact

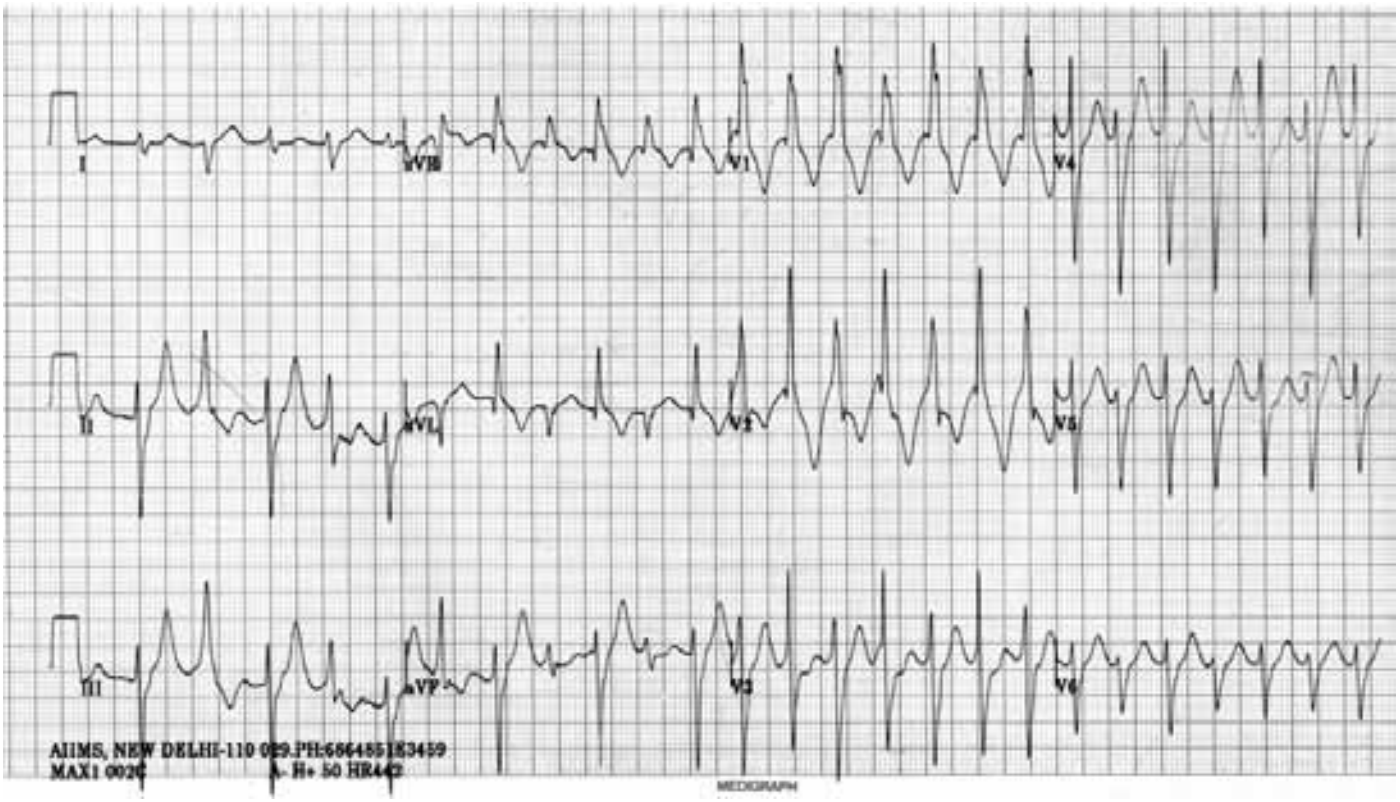
For correct answer see overleaf

ECG - 8

The correct answer is "d" - Holter Artifact

Panel A, B and C apparently show a run of wide and bizzare complexes, which appear polymorphic in nature, with an irregularly, irregular rhythm and an unsteady baseline. A close look at all the leads, especially lead V_5 in Panel A, leads V_2-V_5 in Panel B and leads V_1-V_5 in Panel C reveal a regularly timed QRS complexes at exactly the same rate as the preceding and following normal sinus rhythm. Also, the sinus rhythm do not show any evidence of abnormality with a normal QT interval as well. This is a Holter artifact.

ECG - 9



9. What is the likely diagnosis?

a. Catecholaminergic VT
b. Digitalis Toxicity
c. SVT with aberrancy
d. Ventricular bigeminy

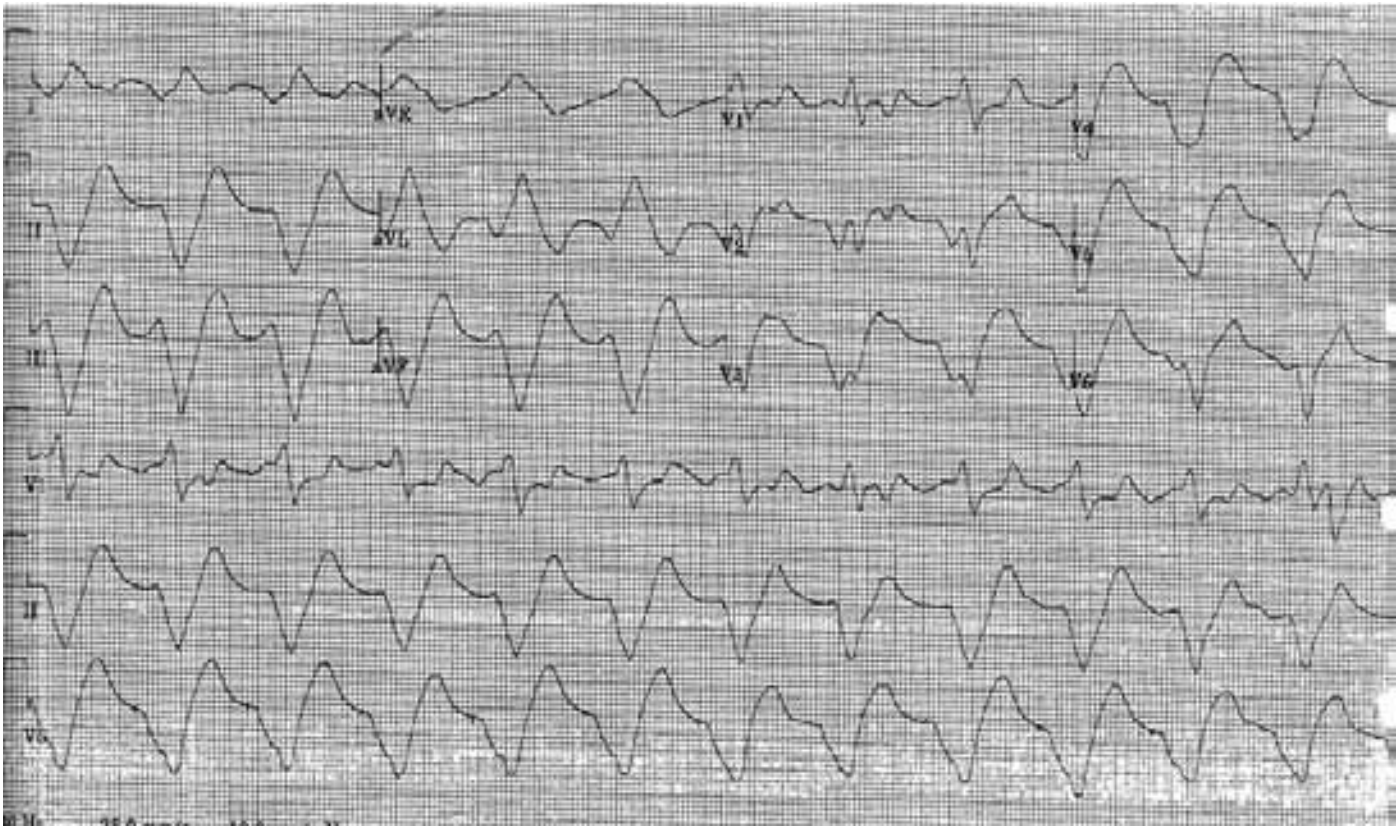
For correct answer see overleaf

ECG - 9

The correct answer is "b" - Digitalis toxicity.

The 12 lead ECG show a classic bi-directional tachycardia, best identified in the limb leads. It is a wide QRS tachycardia with 2 distinct morphologies alternating with each other. The origin of this VT is from the left posterior fascicle and is seen in digitalis toxicity. Catecholaminergic VT is exercise induced and most often polymorphic.

ECG - 10



10. What should you do?

a. Administer IV Xylocard
b. Emergent DC version
c. Administer IV Amiodarone
d. Administer IV Calcium

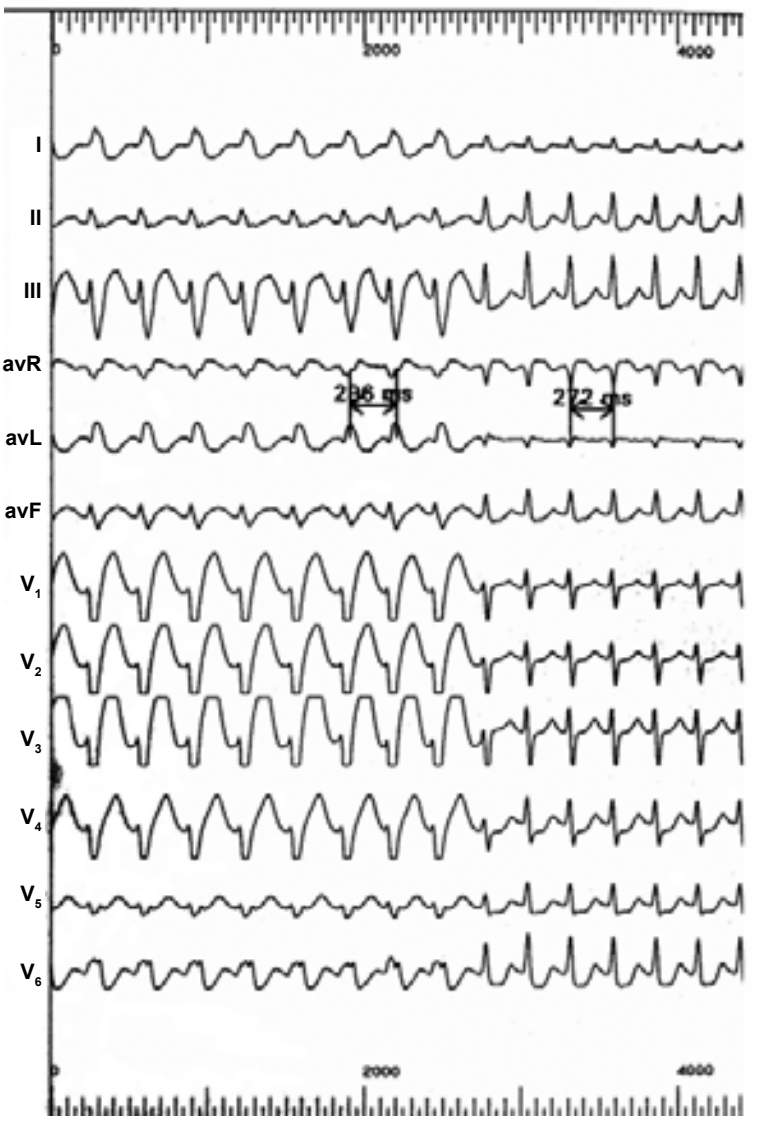
For correct answer see overleaf

ECG - 10

The correct answer is "d" - Administer IV calcium

The 12 lead ECG show a wide, bizarre QRS complexes. Two striking features of this ECG is the tall T waves in the infero-lateral leads with absent ST segment i.e. depolarization is delayed and meets repolarization. There are no P waves identifiable. All these changes are indicative of hyperkalemia. This is often mistaken as a slow VT, however the immediate life-saving treatment is to correct the hyperkalemia by administering IV calcium gluconate.

ECG - 11



11. What is your diagnosis?

a. AVNRT with aberrancy
b. VT changing into an AVNRT
c. Orthodromic AVRT
d. Mahaim bystander with AVNRT

For correct answer see overleaf

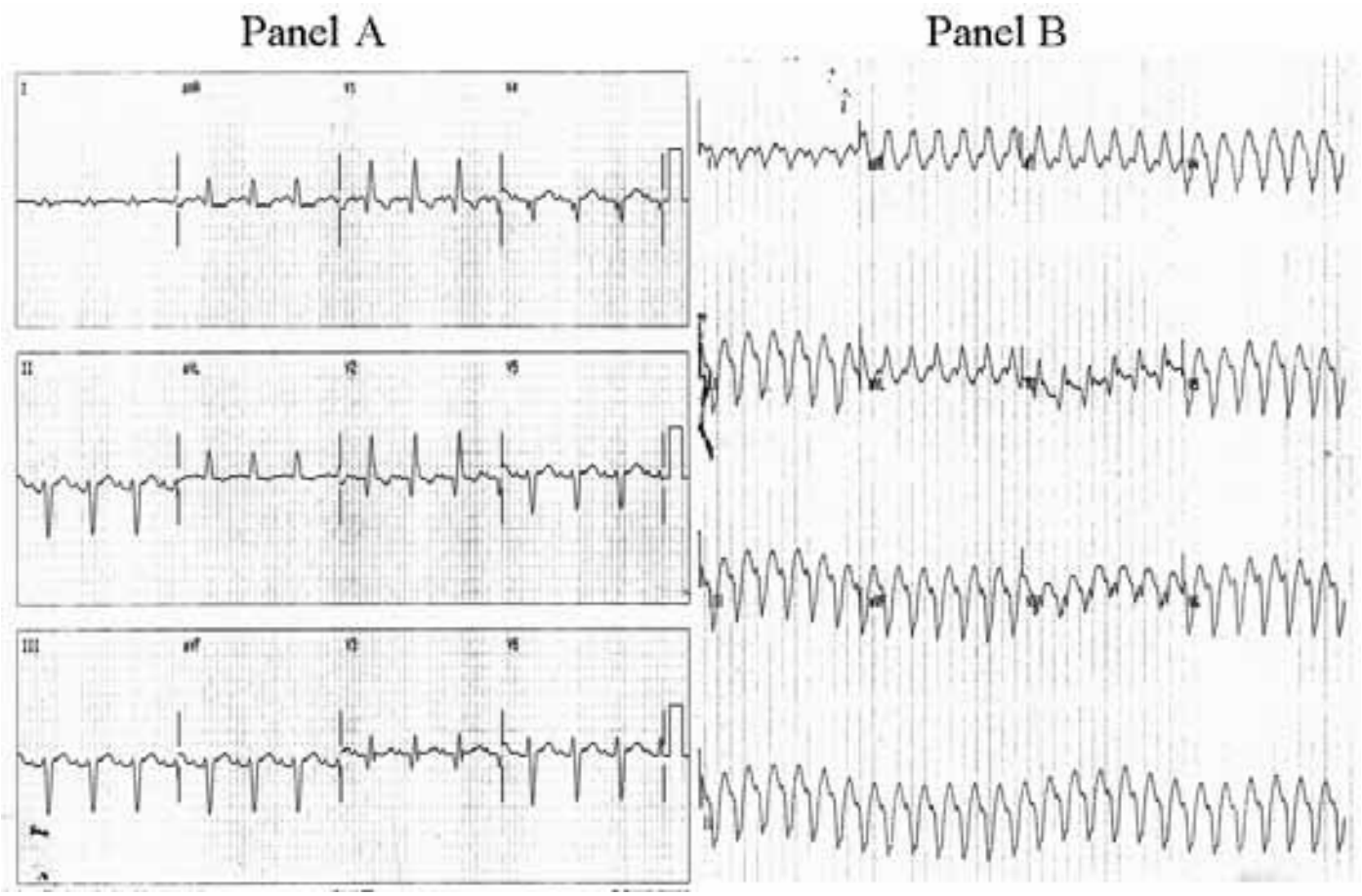
ECG - 11

The correct answer is "c" - Orthodromic AVRT

The 12 lead ECG shows a wide QRS tachycardia (first 8 complexes with a cycle length of 286 ms) changing to a faster, narrow QRS tachycardia (last 7 complexes with cycle length of 272 ms). In presence of a narrow QRS tachycardia, the wide QRS is most likely to be SVT with bundle branch block aberrancy. In this ECG it is LBBB aberrancy. Usually aberrant conduction occurs at a faster rate; instead in this ECG the tachycardia is *slower* during LBBB aberrancy. This phenomenon can occur with an orthodromic AVRT with retrograde conduction over an accessory pathway ipsilateral to the BBB. During narrow QRS tachycardia there is some ST depression in the lateral leads because of the P wave dragging the ST segment. There is some ST elevation (because of retrograde P) in lead aVR but not so in lead aVL, suggestive of a left to right atrial activation i.e. a left lateral accessory pathway conducting retrogradely during the orthodromic AVRT. The electrical impulse travels through the AV node into the left ventricle via the left bundle and ascends up the left lateral accessory pathway. During a left bundle branch block the impulse will have to activate the right bundle first and traverse across the inter-ventricular septum on to the left side and engage the left lateral accessory pathway. This is a longer circuit and hence the tachycardia cycle length is slower.

The fact that during LBBB aberrancy the tachycardia rate is slower than during narrow QRS tachycardia rules out AVNRT with aberrancy. The bundle branch block pattern is very typical with a rapid intrinsicoid deflection and thus VT is unlikely. A Mahaim or atrio-fascicular tachycardia has a left bundle branch block morphology with left axis deviation. A bystander Mahaim AP with AVNRT should not have a change in tachycardia cycle length.

ECG - 12



12. The ECG in panel B is:

a. SVT with aberrancy
b. VT
c. Atrial Flutter with aberrancy
d. WPW syndrome with antidromic AVRT

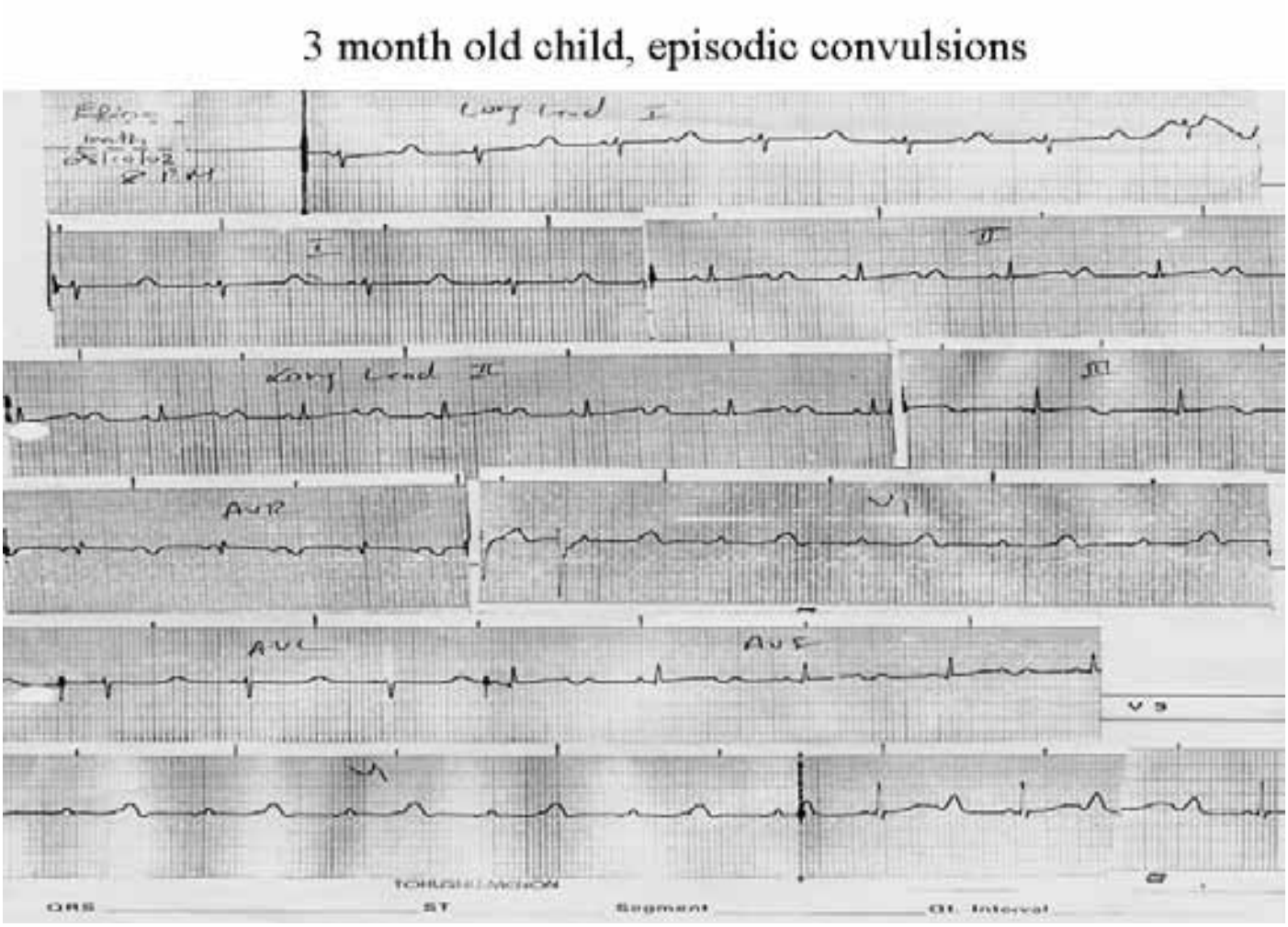
For correct answer see overleaf

ECG - 12

The correct answer is "b" - VT

Panel A, in sinus rhythm reveals anterior wall MI with RBBB. *A wide QRS tachycardia in this setting is assumed to be VT, until proved otherwise.* This is confirmed by the QR complexes in panel B, which is pathognomonic of a VT especially so in the setting of an old MI. SVT (or atrial flutter) with aberrancy is unlikely with a shift in QRS axis to north-west quadrant as opposed to the left axis deviation during sinus rhythm. At baseline in panel A there is no preexcitation whatsoever and this would rule out antidromic tachycardia.

ECG - 13



13. What is your diagnosis?

a. Ventricular Bigeminy
b. Sinus bradycardia
c. Intermittent preexcitation
d. None of the above

For correct answer see overleaf

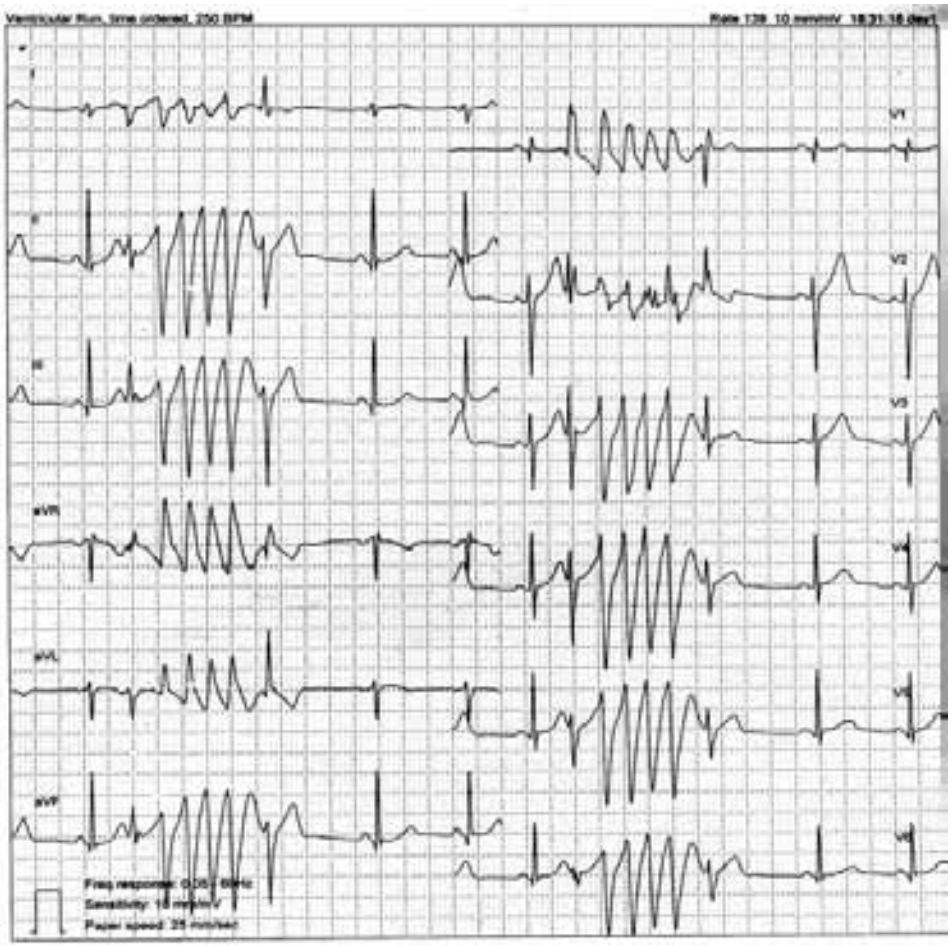
ECG - 13

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

The 12 lead ECG show regular, narrow but small QRS complexes. The wide complexes in between the RR raise the suspicion of ventricular ectopics; however, there is no ST-T wave following and therefore these are not ventricular premature complexes, but are late T waves. Thus the QT interval is markedly prolonged. Also, for a 3 month-old child the bradycardia is pronounced. However just before the T wave begins there is a distinct P wave of similar morphology as the sinus P, the timing is also regular but with a 2:1 AV block. The AV block occurs since the blocked P wave falls within the QT interval when the ventricles are still refractory. This ECG reveals a long QT interval with bizarre T wave and a 2:1 AV block.

ECG - 14

18 yr-old boy
presented with palpitations
& syncope



14. What would you advise the patient?

a. Reassure him and order a repeat Holter after 3 months
b. Put him on β blockers ± amiodarone
c. Advise an ICD
d. Advise coronary angiography and if negative give monitored IV Ajmaline

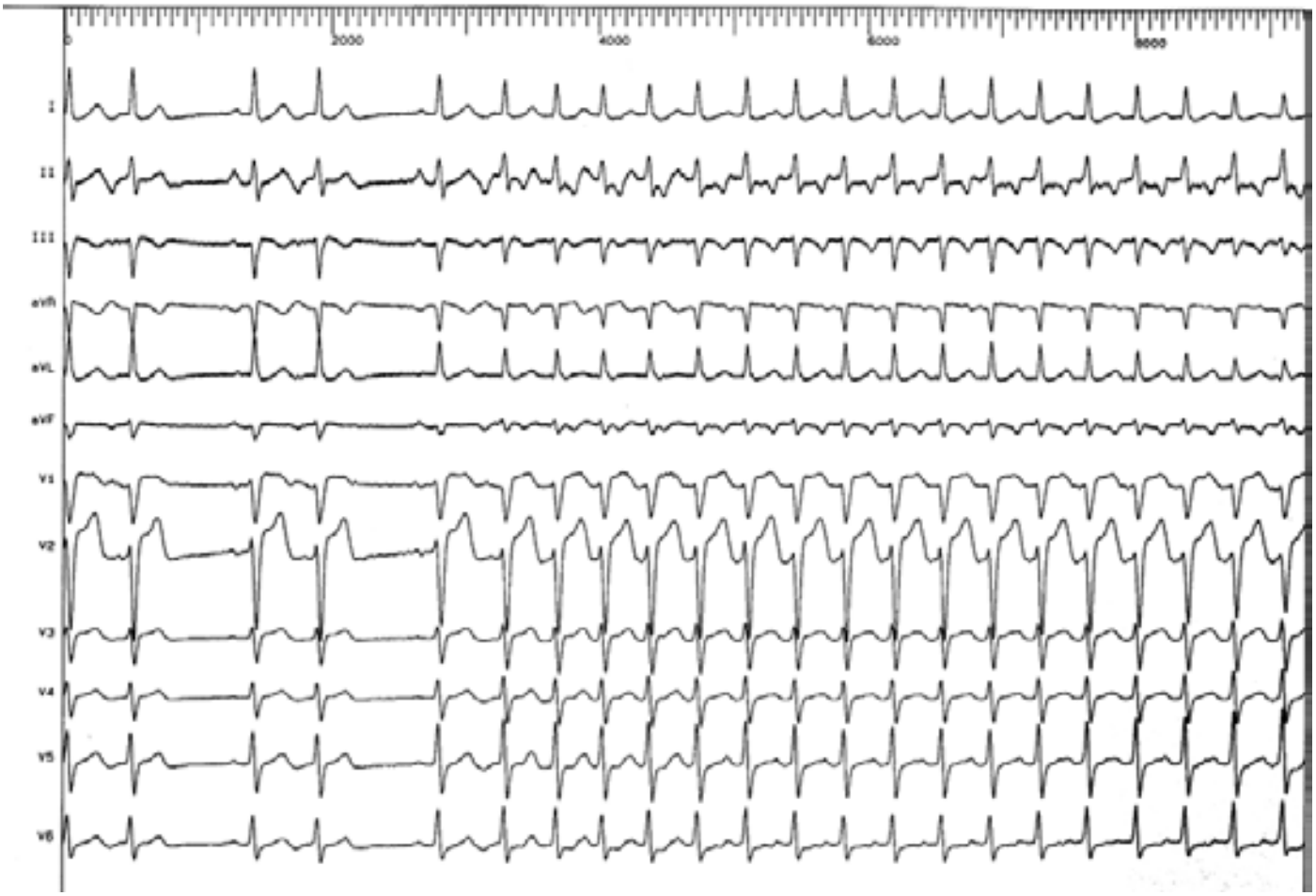
For correct answer see overleaf

ECG - 14

The correct answer is "c" - Advise an ICD.

The 12 lead ECG shows repetative episodes of non-sustained, very rapid, polymorphic ventricular tachycardia (VT). This VT is initiated by a ventricular ectopic beat, the QRS morphology of the ectopic beat suggests originating from the left anterior fascicle. This is a type of idiopathic VT/VF which can result in sudden cardiac death and hence ICD is the best choice. There are recent reports of ablating this arrhythmia, wherein the target is the Purkinje or fascicular ectopic beat which initiates the VT.

ECG - 15



15. What is your diagnosis?

a. AVNRT
b. Atypical AVNRT
c. Orthodromic tachycardia
d. Ectopic atrial tachycardia

For correct answer see overleaf

ECG - 15

The correct answer is "d" - Ectopic atrial tachycardia

The first 4 complexes in this 12 lead ECG suggest atrial bigeminy. The fifth complex is sinus and is followed by an atrial ectopic that initiates the narrow QRS tachycardia. The tachycardia shows distinct P' waves similar to the P' waves of the atrial ectopic beats. The RP interval during tachycardia is longer than the PR interval and the differential diagnoses of atypical AVNRT, PJRT or ectopic atrial tachycardia should be entertained. The P' wave morphology suggests origin in the left and inferior location (negative P' in inferior leads and flat in lead I & aVL). This rules out atypical AVNRT & PJRT as both would have an exit in to the atrium from the inferior and septal location. This ECG therefore with its early bigeminy pattern is suggestive of ectopic atrial tachycardia.

SECRETARIAT

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