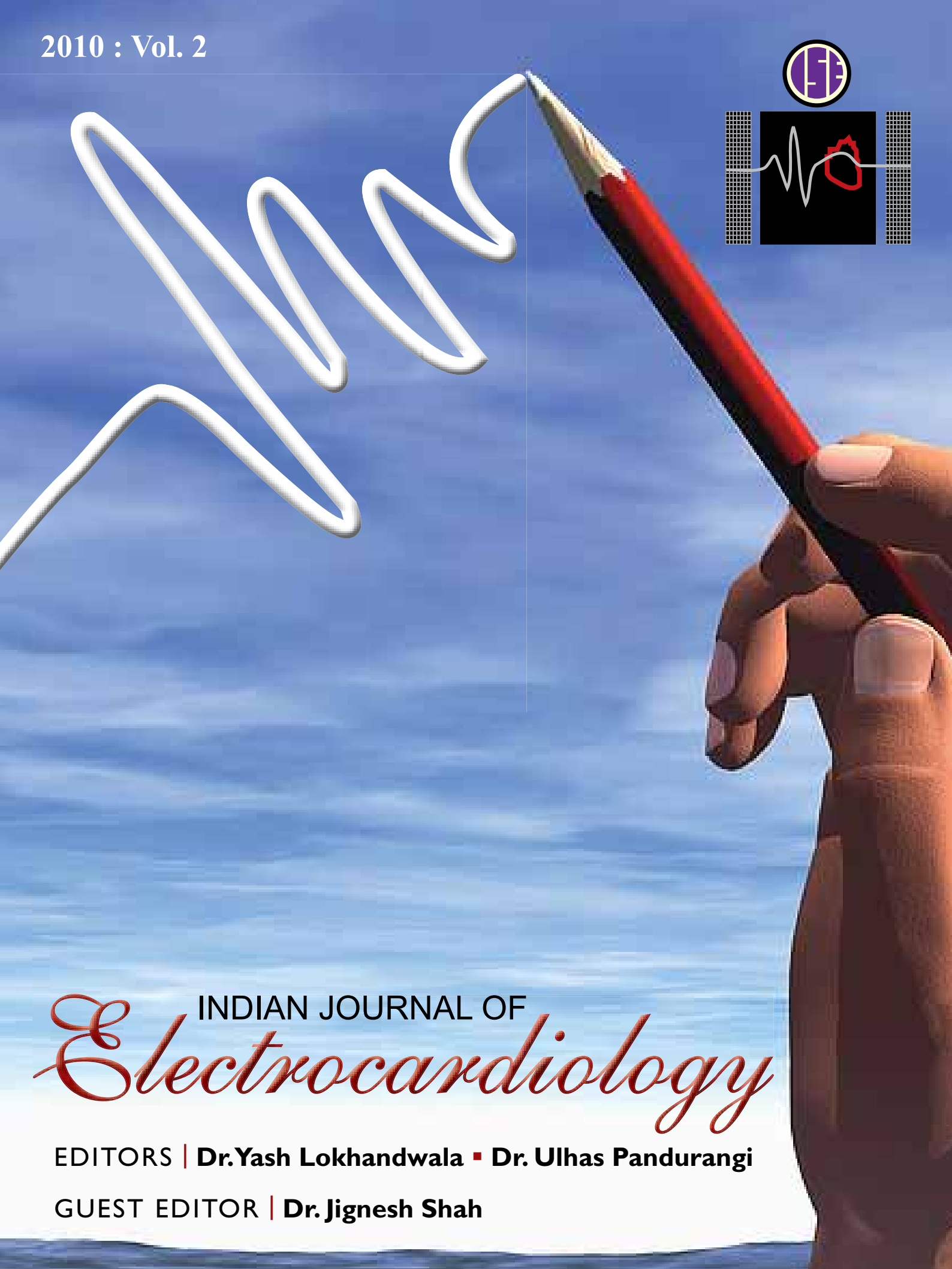
2010 : Vol. 2



INDIAN JOURNAL OF Electrocardiology

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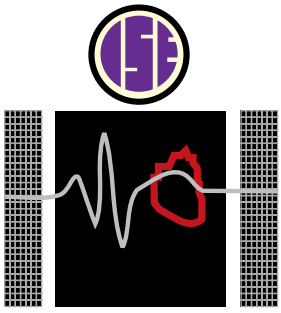

Welcomes

All Delegates to

PAC 2010

Patna

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 ISE Patna. The scientific committee has prepared an exciting academic program. I am sure their teachings and your interest will create the right mix for a very good learning experience.

The current issue of the IJE carries a wide range of interesting articles. Dr. Shomu Bohra and Dr. Sanjay Bindra have written a comprehensive review on Ivabradine, a drug that is being used with increasing frequency all over the country. A detailed review of the mechanism of action and its various uses will help in the management of patients ideally suited to use this drug. Dr. Aditya Kapoor has discussed various "ECGs in Critcal Care" settings. All of us face perplexing situations in critical care settings and a comprehensive review of ECGs in these settings help in optimal care of these patients. Dr. Pandurangi and colleagues have presented an excellent case of double tachycardia and have aptly utilized it to explain and understand mechanism of ST depression during regular narrow QRS tachycardias.

We are fortunate to have an excellent case report from Dr. Monika Maheshwari and colleagues. They present a case of Parkinson's tremors appearing as atrial fibrillation, atrial flutter as well as torsades in different leads.

Dr. Namboodri and Dr. Francis have presented an excellent review of ventricular arrhythmias in hypertrophic cardiomyopathy. Their thought provoking review will assist all of us in better risk assessment of patients with hypertrophic cardiomyopathy. 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.

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 assistance with this issue.

Jignesh Shah
Guest Editor

Yash Lokhandwala
Editor

Ulhas Pandurangi
Editor

From Vice President's Desk

Dear Members,

It is our great pleasure in bringing out the latest issue of Indian Journal of Electrocardiology on the eve of PAC 2010 – Mid Term Conference of Indian Society of Electrocardiology.

We organized ISECON-2010 at Mumbai from 19th to 21st February 2010. I am sure every one enjoyed the great scientific bonanza.

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

a. "ECG Learning Course" for postgraduate students at Mumbai on 1st and 2nd May 2010, at New Delhi on 22nd and 23rd May 2010 and on 10th July and 11th July 2010 at Chennai. About 80-100 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading

Patna Arrhythmia Course 2010 (PAC-2010) has been organized by Dr Ajay Sinha and his team and needs congratulations for organizing the event.

My sincere thanks to Dr. Yash Lokhandwala, Dr. Ulhas Pandurangi, Dr. Jignesh Shah, Dr. Gopi Panicker and the Editorial Team for bringing out the ISE Journal – 2010, 2nd Volume.

Long Live Indian Society of Electrocardiology

Dr. S.B. Gupta
Vice President
Indian Society of Electrocardiology

Ivabradine: Review of literature.

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Background

Epidemiologic and clinical data suggests that an elevated heart rate is a risk factor for ischemic cardiac events,[1] and heart rate reduction is associated with improved outcomes. Lowering of heart rate is thus one of the most important therapeutic approaches in cardiovascular disorders. Conventional pharmaco-therapeutic agents used to reduce heart rate are limited by their negative inotropic effects and various contraindications and intolerance. These agents have deleterious consequences on force of contraction, peripheral circulation, bronchial tone, bowel transit, glucose and triglyceride metabolism and neuropsychiatric symptoms including depression, insomnia and fatigue.

Searches to develop a pure heart rate lowering agent, led to the discovery of the I_f current, I_f channel and subsequently the selective and specific I_f channel blocker, ivabradine. Initially demonstrated as an effective antianginal and anti-ischemic agent for patients with angina,[2,3] ivabradine has found increasing application for management of patients with syndromes associated with increased sinus rates also. (Inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome)[4,5,6,7] This article provides short review of I_f channel, ivabradine and its current and future applications in cardiology practice.

Normal sinus rhythm and I_f channel:

In the normal, non-diseased state, heart rate is controlled by the sino-atrial node. Sino-atrial myocytes, the pacemaker cells in the heart, have the unique capacity to spontaneously generate slow diastolic depolarization. This drives the membrane voltage away from the hyperpolarized level reached at the completion of action potential and towards the threshold level for initiating a subsequent action potential. The rhythmic action potentials generated in this way propagate through the conducting systems of the heart and trigger myocardial contraction.

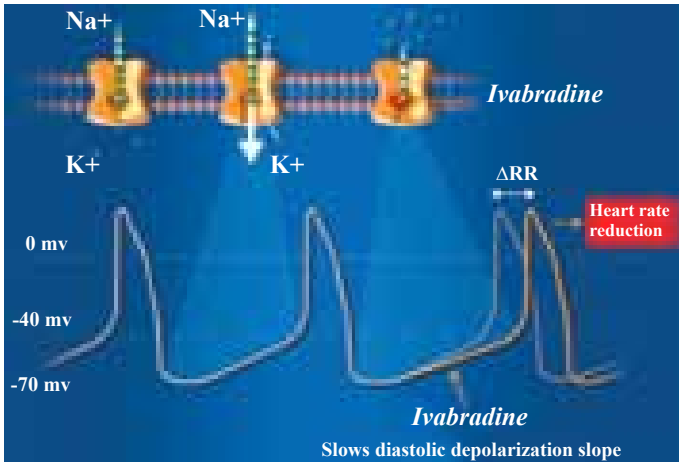

Figure 1: Schematic representation of mechanism of action of Ivabradine.

The 'f' denotes 'funny', so called because it has unusual properties. I_f current is an inward diastolic current activated on hyperpolarization, carried by both sodium and potassium ions across the sarcolemma.[8] It is characterized by unusually low single-channel conductance and slow activation kinetics. The I_f current is directly activated by intracellular cyclic adenosine monophosphate (cAMP), not linked to cAMP-dependent phosphorylation activity,[9] and is carried by the hyperpolarization-activated cyclic nucleotide-gated family of ion channels.[10]

I_f channels open and close in response to both ambient voltage and local intracellular cAMP concentrations. Adrenergic agonists (increased sympathetic states) activate adenylate cyclase, increasing local cAMP concentrations and thus increasing cAMP binding to the I_f channel.[11] Conversely, cholinergic transmitters (increased parasympathetic states) decrease local cAMP concentrations by inhibiting adenylate cyclase, thereby decreasing cAMP binding to the I_f channel. An I_f channel bound to cAMP is more likely to open, increasing the rate of slow diastolic depolarization and hence increasing the heart rate, whereas an unbound channel is more likely to remain closed, lowering the heart rate.[12]

Ivabradine

Ivabradine is a selective I_f channel blocker and was designated as **S-16257** during its development. It blocks the I_f channels by entering the channel pore from the intracellular side and reduces firing rate of pacemaker cells in the sinoatrial node without affecting the duration of the action potential (figure 1).[13] It acts on I_f channel at concentrations that have no effect on other ionic currents, making ivabradine a selective I_f channel inhibitor.[14,15]

This blockade is only possible when the I_f channel is open, and the magnitude of I_f inhibition is directly related to the frequency of channel opening. Thus, ivabradine action is expected to be most effective at higher heart rates, where its clinical usefulness would also be greatest. Ivabradine has >50% first pass metabolism with a CYP3A4 mediated metabolism. Half life is 2 hours. Excretion is renal as well as fecal. Recommended dosage has been 5 mg to 10 mg twice daily.

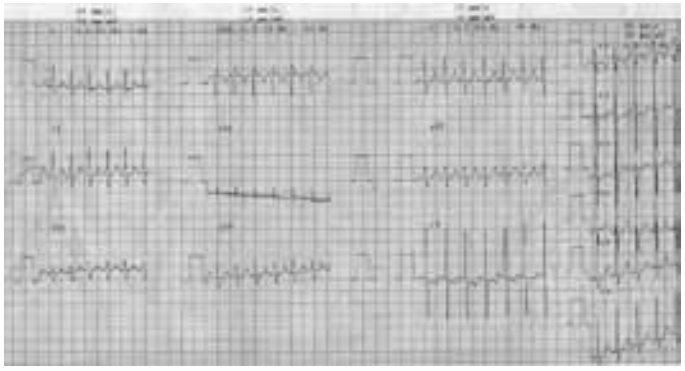

Figure 2 : Electrocardiogram of 11 year old boy having symptomatic tachycardia.

Unlike many rate-lowering agents, ivabradine reduces heart rate in a dose-dependent manner both at rest and during exercise without producing any negative inotropic or vasoconstrictor effect. Bradycardia due to ivabradine is proportional to the resting heart rate, such that the effect tends to plateau. Bradycardia occurred in 4.2% patients taking ivabradine, of which only 1.1% patients were symptomatic.[16] The QT interval after appropriate correction for heart rate showed no significant effect of ivabradine on ventricular repolarization duration. Other adverse drug reactions include gastrointestinal side effects and dizziness. 2.6-4.8% patients reported headaches. Because ivabradine also binds to hyperpolarization voltage-gated channels which carry the I(h) current in the eye, transient, dose-dependent changes of the electro-retinogram resulting in mild to moderate visual side effects like visual blurring and phenomes (which have been described as sensations of enhanced brightness in a fully maintained visual field) may occur in up to 15% of patients at higher doses.[3] These symptoms are mild, transient, fully reversible and non-severe.

Ivabradine is contraindicated in sick sinus syndrome, and should not be used concomitantly with inhibitors of CYP3A4 such as azole antifungals (such as ketoconazole), macrolide antibiotics, nefazodone and the anti-HIV drugs nelfinavir and ritonavir.

The advantage of ivabradine in compared to other heart rate lowering drugs has been its lack of cardiac side effects. Ivabradine does not significantly prolong QT interval and does not have negative inotropic or lusitropic effects. There is no effect on coronary and peripheral arteries.

Ivabradine in clinical practice

Ivabradine based on its effect on the I_f channels is an effective drug in decreasing heart rates. A decrease in heart rate can be beneficial in patients with angina or in certain subgroups of arrhythmia. The current indications and other possible uses are as discussed

- Ivabradine in chronic stable angina

  Angina results from an imbalance between myocardial perfusion and myocardial metabolic demands. Heart rate reduction can alter both elements of this imbalance beneficially. The resulting increase in diastolic filling time improves myocardial perfusion and the myocardial oxygen demand which varies directly with heart rate, decreases with the decrease in heart rate. Ivabradine being a selective I_f channel blocker effectively decreases heart rate and hence should reduce myocardial ischemia.

  Ivabradine has been evaluated in chronic stable angina in various clinical trials and has been shown effective when compared to placebo, β-blockers and calcium channel antagonists and also in combination with most conventional anti-anginal therapies for angina including nitrates, antiplatelet agents, dihydropyridine calcium antagonists and β-blockers. In short and the long terms studies ivabradine maintains efficacy over 12 months without any acquired tolerance.[2,3,16-20]

  One of the major clinical trials was the BEAUTIFUL study,[3] in which 10917 patients were evaluated. Patients given ivabradine showed a reduction of mean heart rate of 6 beats per minute (bpm) at 12 months in comparison to a placebo. Reduction in heart rate with ivabradine however did not improve cardiac outcomes in all patients with stable coronary artery disease and left-ventricular systolic dysfunction, but reduced the incidence of coronary artery disease outcomes in a subgroup of patients who have heart rates of 70 bpm or greater. The REDUCTION study, evaluating 4954 patients with chronic stable angina, patients taking ivabradine showed a significant reduction in heart rate, angina pectoris attacks and consumption of short-acting nitrates.[20] Future trials are being planned based on clinical data obtained from the available trials.[21]

  In conclusion, based on the current clinical data, use of ivabradine in chronic stable angina, should be considered in patients who have still not achieved adequate heart rate control of less than 70 bpm, on standard line of management. For patients who have contraindications to beta-blockers due to PR prolongation, bronchial asthma, peripheral vascular disease or those who have unacceptable side effects on beta-blockers, ivabradine may represent a suitable alternative for long term therapy of chronic stable angina.

- Ivabradine in acute coronary syndrome.

  A short term study comparing metoprolol and ivabradine in patients with acute myocardial infarction with left ventricular dysfunction, when given after 12 hours, showed that ivabradine group had a significant increase in EF, (P=.0001), with concomitant reduction in ESV and EDV (P=.0001, and .048, respectively) on follow up.[22] Further trials to evaluate the role of ivabradine in acute coronary syndromes are planned.[23]

- Ivabradine in inappropriate sinus tachycardia/sinus nodal

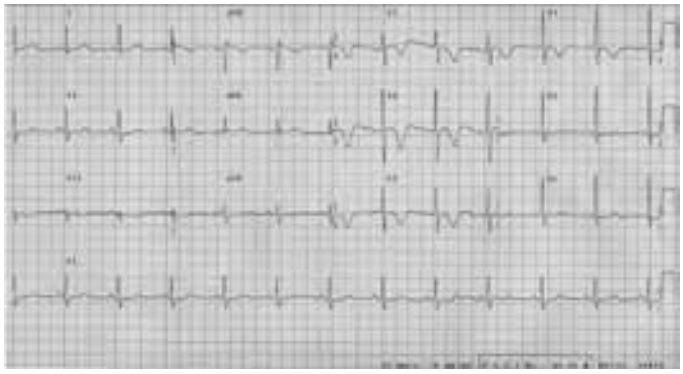

Figure 3: Electrocardiogram of the same patient after 2 days of starting ivabradine showing controlled rates.

related tachycardia.

Inappropriate sinus tachycardia and related presentations, including patients presenting with tachycardiomyopathy respond well to ivabradine.[4-6] Ivabradine treatment can be used in patients who do not agree or are not suitable candidates for sinus nodal modification by ablation techniques. Figure 2 represents electrocardiogram of an 11 year old boy with sinus nodal related tachycardia and tachycardiomyopathy, not responding to standard medications and who on being started with ivabradine 2.5 mg twice daily responded dramatically, with controlled heart rate (Figure 3) and normalization of heart function.[4]

- Ivabradine in Postural Orthostatic Tachycardia syndrome (POTS).

  Ivabradine is shown to be effective in patients having POTS as an alternative therapy. Effectiveness may be due to prevention of reflex sinus tachycardia. [7]

- Ivabradine in Chronic heart failure.

  Heart rate control in patients with chronic heart failure may be helpful to decrease morbidity and/or mortality. SHIFT study will evaluate the role of ivabradine in patients with chronic heart failure with heart rates of > or = 70/min. [24]

- Ivabradine in Atrial Tachycardia

  Mechanisms of atrial tachycardias like micro-reentry, automatic or triggered cannot be clearly differentiated on electrogram and is difficult to confirm the mechanism even on electrophysiology study. On a pilot basis, after appropriate consent, we have administered ivabradine in 11 patients having atrial tachycardia, who did not respond to or who had contraindications or did not tolerate standard drug therapy and who were unwilling or not suitable for ablation due to various causes (unpublished data). We found ivabradine to be effective in decreasing tachycardia rates in 3 patients all of whom were under 12 years of age. Ivabradine decreased the atrial rate in a patient with left atrial tachycardia thus relieving the patient's symptoms. [25] This raises the possibility of a role of sinus nodal like cells with I_f current as a molecular mechanism in the genesis of atrial arrhythmias or possibly of other mechanisms of ivabradine effectiveness. The effectiveness of ivabradine based on preliminary observation in pediatric age group suggests that in this patient population atrial tachycardias are more likely to be automatic as compared to adult arrhythmias and ivabradine may offer less toxic alternative in drug treatment of pediatric atrial tachycardia.

Conclusion

Ivabradine, a selective I_f channel blocker, effectively decreases heart rates in a dose dependant fashion and has no significant cardiac side effects. It has been proven to be effective in treatment of patients with chronic stable angina and inappropriate sinus tachycardia but possibly needs further evaluation in treatment of atrial tachycardia, chronic heart failure, acute coronary syndrome and POTS. Long term safety and possibly other antiarrhythmic properties needs to be further evaluated.

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ECGs in Critical Care

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Introduction

The most common diagnostic test done in emergency departments (EDs) and intensive care units is the electrocardiogram (ECG). Whether the ECG is a single-lead monitor, standard 12-lead, or sophisticated 80-lead body surface, the information can be critical in managing acutely ill patients. The ability to interpret an ECG is considered basic to all physicians, but particularly important for emergency physicians, cardiologists, and intensivists. The following review shall attempt to familiarize the physicians with ECG findings in common situations encountered in everyday critical care scenarios. Categories discussed include (a) Electrolyte abnormalities (b) Metabolic disorders: Hypothermia and related CNS conditions (c) Acute pulmonary embolism (d) Pericarditis (e) Artifacts

Table 1 : Electrocardiographic Findings Associated with Disturbances in Serum Electrolyte Levels

Electrolyte Abnormality	Electrocardiographic Findings
Hypokalemia	Decreased T wave amplitude T wave inversion ST segment U Wave Prolongation of QT(U) Interval Ventricular tachycardia Torsades de pointes
Hyperkalemia	
Mild	Large amplitude T waves "Peaked" or "tented" T waves
Moderate	PR interval prolongation Decreased P wave amplitude, disappearance QRS complex widening Conduction blocks with escape beats
Severe	Sine-wave pattern Venricular fibrillation Asystole
Hypocalcemia	Prolongation of QTc interval Ventricular dysrhythmias Torsades de pointes
Hypercalcemia	Shortening of QTc interval Bradydysrhythmias
Hypo / Hypermagnesemia	No unique electrocardiographic abnormalties, but often associated with calcium abnormalities

A. Electrolyte abnormalities

Changes in extracellular cations such as potassium, calcium, and magnesium levels can change myocardial membrane potential and hence alter the action potential. Because myocyte depolarization and repolarization depend on membrane potential gradients, abnormal serum electrolyte levels can have profound effects on myocardial conduction. These changes occasionally result in dramatic ECG findings, which if unrecognized, can eventually precipitate potentially life-threatening dysrhythmias. (Table 1) It is important to remember that often combined disorders occur and there is a dynamic interaction between these cations.

Hyperkalemia

Potassium is predominantly an intracellular cation that plays an important role in maintaining the electrical potential across the cellular membrane, as well as in depolarization and repolarization of the myocytes. Important causes of hyperkalemia include renal failure, mineralocorticoid deficiency or resistance, renal tubular acidosis, rhabdomyolysis, burns, hemolysis and medications such as non-steroidal anti-inflammatory agents, angiotensin-converting enzyme inhibitors, diuretics, and digitalis. Although in some patients EKG changes do not accompany serum potassium abnormalities, the ECG is a useful tool for assess the urgency of therapeutic intervention.

A. The earliest EKG correlate is ***T wave tenting*** ($K^+ > 5.5$ mEq/L), classically described as symmetrically narrow or peaked. Occasionally the inverted T waves associated with left ventricular hypertrophy can pseudonormalize (flip upright) with hyperkalaemia. These T wave changes occur as a result of the acceleration of the terminal phase of repolarization and are most prominently seen in the precordial leads. These tall T waves need to be differentiated from those seen in the hyepracute phase of acute MI wherein the T waves are often broad based, asymmetric and bulky, while those in hyperkalaemia are often narrow based and tented. However, these are soft criteria and as always clinical correlation is necessary.

B. With higher levels of serum potassium (6-7 mEq/L), cardiac conduction between myocytes is depressed. Reduction in atrial and ventricular transmembrane potential causes slowing of the sodium channel, and since atrial tissue is

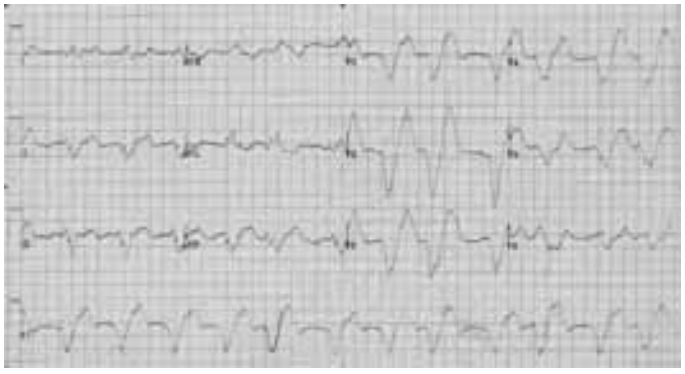

Figure 1: A patient of burns, with K of 7.5 meQ/L with signs of hyperkalemia :Tall T, absent P, broad QRS and Sine wave pattern

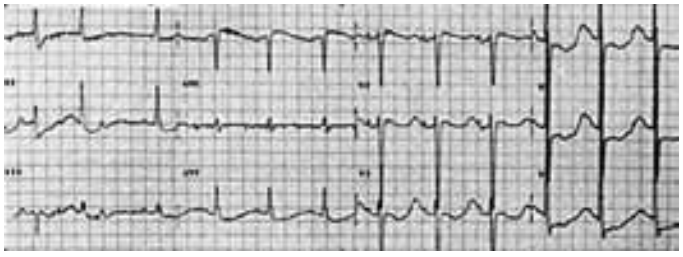

Figure 2: Patient of RHD on diuretics, K+ 2.9 mEq/L with ECG signs of hypokalemia: Sagging of ST segment, loss of T, prominent U and prolonged QT_U interval

more sensitive to these changes, ***P wave flattening and PR prolongation*** may be seen before QRS interval prolongation. As the serum potassium level increases further, there is eventual ***loss of the P wave***, and at even higher levels (> 2 times normal), there is suppression of sinoatrial (SA) and atrioventricular (AV) conduction, resulting in ***SA and AV blocks***. Interestingly, bypass tracts are more sensitive to delayed conduction from potassium elevation, which can result in the normalization of the EKG and loss of the delta wave in patients with Wolff-Parkinson-White syndrome

C. With levels of 7-9 mEq/L or higher, the markedly prolonged, wide QRS complex may fuse with the T wave, producing a slurred, "***sine-wave***" appearance on the EKG (Figure 1). This finding is a pre-terminal event culminating in ***VT/VF or asystole*** unless treatment is initiated immediately.

Although the above EKG progression is descriptive of the classic presentation of hyperkalemia, these changes do not always accompany corresponding serum potassium levels. Metabolic alterations such as alkalosis, hypernatremia, or hypercalcemia can antagonize the transmembrane effects of hyperkalemia and result in the blunting of the EKG changes associated with elevated potassium levels.

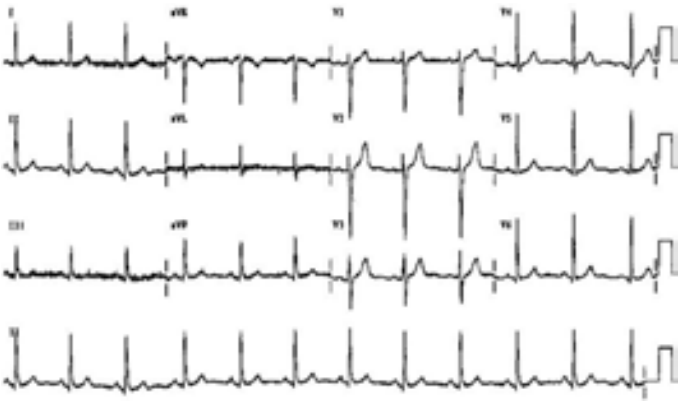

Figure 3: Hyper-parahytyrodism, Ca+ 12.1 ECG shows short QT with virtually absent ST

Hypokalemia (K+ < 3.5 mEq/L)

Hypokalemia may result from renal loss, gastrointestinal losses, extracellular to intracellular shift (as in diabetic ketoacidosis, metabolic acidosis, thyrotoxicosis), or inadequate potassium intake, and commonly due to drugs especially diuretics. As serum levels decline, the transmembrane potassium gradient is decreased, leading to prolongation of the action potential, particularly phase 3 repolarization and refractory periods. Roughly a decrease in serum K concentration of about 1 mEq/L indicates a total K deficit of about 200 to 400 mEq and patients with K < 3 mEq/L typically have a significant K deficit.

The earliest EKG change (K+ < 3 mEq/L) associated with hypokalemia is a ***decrease in the T wave amplitude***. Further decline in the potassium levels leads to ***ST segment sagging*** and ***prominence of U waves.*** (Figure 2) The ***PR interval can be prolonged*** and there may be an increase in the amplitude of the P wave. With even lower serum potassium levels, ***giant U waves*** may often mask the smaller preceding T waves, and bradycardia or high grade AV blocks can appear. As T waves become smaller and U waves become more prominent, there is prolongation of the QT, (or more correctly QT_U interval), which can precipitate ***torsades de pointes or ventricular tachycardia***.

Hypercalcemia (Figure 3)

The normal range of serum calcium levels is 8.7-10.4 mg/dL, with somewhat higher levels present in children. Hypercalcemia is associated with hyperparathyroidism, renal failure or in malignancy. Clinically significant rhythm disturbances associated with hypercalcemia are rare. Occasionally it produces abnormal ***shortening of the QTc interval*** on ECG, which is always at the ***expense of a shortened or even absent ST segment***. Bradydysrhythmias are also noted with hypercalcemia.

Hypocalcemia (Figure 4)

Hypocalcemia is classically seen with functional parathyroid

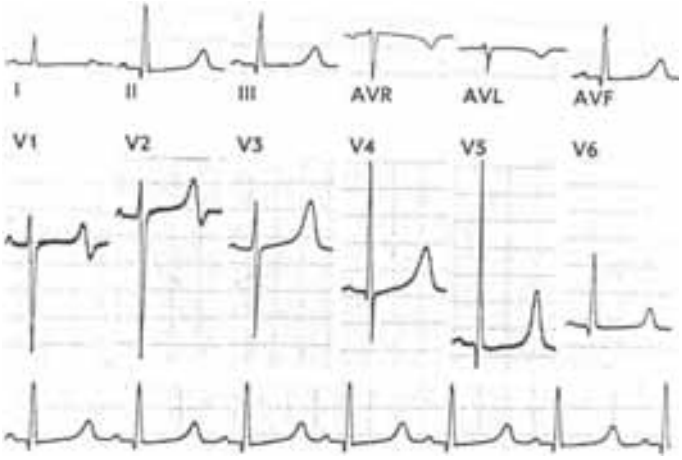

Figure 4: Hypo-parathyroisism and tetany, Ca+ 1.9 ECG shows prolonged QT and QTc

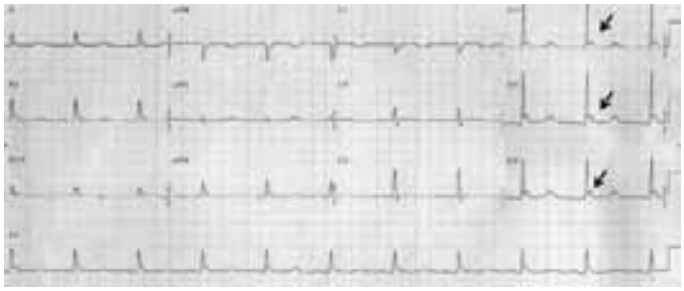

Figure 5: Hypothermia, body temperature 28.6 ECG showing Osborne waves (arrows),and bradycardia

hormone deficiency, either as absolute hormone deficiency (primary hypoparathyroidism), post-parathyroidectomy, or related to a pseudo-hypoparathyroid syndrome. Other causes of hypocalcemia include vitamin D deficiency, congenital disorders of calcium metabolism, chronic renal failure, acute pancreatitis, rhabdomyolysis, and sepsis. The primary ECG manifestation of hypocalcemia is ***lengthening of the QTc interval***. Prolongation of the QTc interval is associated with early after-repolarizations and triggered dysrhythmias. Torsades de pointes potentially can be triggered by hypocalcemia due to this lengthening of the QTc, however, it is much less common than with hypokalemia or hypomagnesemia.

B. Hypothermia

Hypothermia (body temperature <35°C) can be divided into three categories: mild (between 32 and 35°C), moderate (between 28 and 32°C), and severe (<28°C). The most serious complications occur with temperatures below 28°C and consist of hypotension, pulmonary oedema, areflexia, bradycardia, ventricular fibrillation and asystole. It produces typical changes on the ECG due to delayed conduction through the myocardium as well as due to artifacts which may be produced by intense shaking or shivering. However, the hallmark of hypothermia on ECG is the J point elevation leading to the classical ***Osborn waves***, best seen in precordial and inferior leads. (Fig 5) It results from the prominent transient outward potassium current resulting in a transmural voltage gradient in the epicardium, more pronounced under hypothermic conditions. These waves disappear after normalisation of the body temperature. Although the Osborn wave is associated with hypothermia, it is not pathognomonic. It is also seen in hypercalcemia, autonomic dysfunction, and cerebral injury.

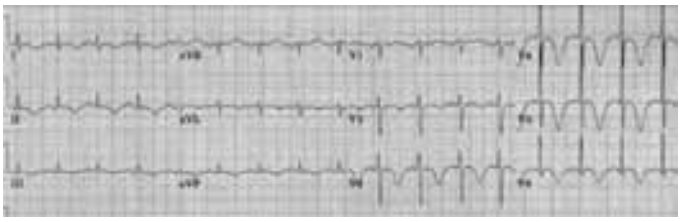

Figure 6: Widespread T wave inversion in a case of Subarachnoid hemorrhage

C. CNS diseases

Acute cerebrovascular accidents (C.V.A.) and subarachnoid hemorrahe (SAH) are often accompanied with characteristic ECG abnormalities which include prolongation of the Q-Tc interval, widespread ST-T inversions, (Fig 6) prominent U waves, sinus tachycardia, sinus bradycardia and rarely nodal rhythms, atrial fibrillation or malignant ventricular arrhythmias. Similar ECG changes have been described in meningitis and intracranial tumours. The mechanism by which these changes are produced is not clear though postulated mechanisms include direct myocardial damage by autonomic influences, and raised intracranial tension and vagotonicity.

D. Acute pulmonary embolism

The mechanism by which acute PE causes ECG changes remains unclear and have been attributed to ischemic, hemodynamic, anatomic, metabolic, and autonomic changes that affect the electrical pathways of cardiac tissue. These ECG changes are associated with increase in the PA pressures as well as RV size. Often ECG changes may persist well after PA pressures and ventricular size have returned to normal. The overall utility of the ECG is limited due to the variable presence, frequency, and poor sensitivity and specificity of the findings. .

Normal EKG: A completely normal EKG has been reported in anywhere from 9 to 26% of patients with acute PE.

Rhythm Disturbances. Sinus tachycardia is the commonest ECG finding while first degree AV block, premature atrial and ventricular beats, atrial fibrillation, and flutter have also been reported. **S1Q3T3**. The finding of a large S

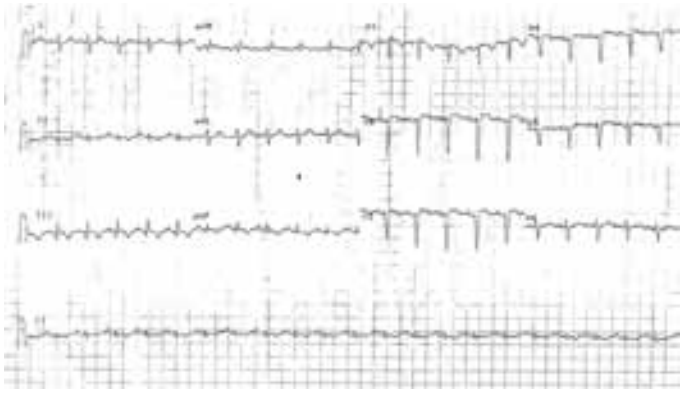

Figure 7: Massive PE presenting with giddiness, syncope. Note the S1Q3T3, sinus tachycardia, S in V6, and terminal positivity in avR

wave in lead I, Q wave and inverted T wave in lead III has often been mistaken as pathognomonic for acute PE. These findings are due to RV dilatation and RV strain. However the reported incidence of the S1Q3T3 combination varies from 10- 50% of acute PE patients accounting for poor sensitivity of this "pathognomic" sign.. (Fig 7)

Right Bundle Branch Block. Complete or incomplete RBBB is variably seen in 6-70% cases. In addition, RBBB can be associated with ST segment elevation and upright T waves in lead V1, potentially mimicking antero-septal or posterior infarct patterns. RBBB in the setting of acute PE is often transient and resolves in most cases. A QR pattern in V1may also be seen in those with massive PE.

Axis Changes. Right (most common), left, and indeterminate QRS axis shift have all been reported with variable frequency in acute PE patients.

Transition Zone Shift. Clockwise rotation and shift of the transition zone (the precordial lead site where R and S wave amplitudes are equivalent) to lead V5 has also been reported with variable frequency in PE patients.

Other changes like P pulmonale, low voltage QRS complexes, late R wave in avR, slurred S in V1/V2 and nonspecific ST depression or elevation or diffuse T wave inversions have also been reported.

E. Pericarditis

Acute pericarditis usually manifests as diffuse ST-segment elevation on the ECG, which can lead to diagnostic confusion with acute ST elevation MI, especially in a busy ICU setting. Hence physicians should be able to recognize the typical ECG manifestations of acute pericarditis. (Figure 8)

The classical four-stage evolution seen in acute myopericarditis was first described by Spodick and is outlined below:

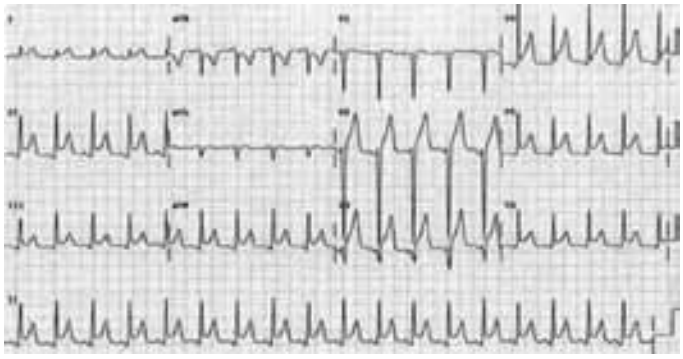

Figure 8: ECG of acute pericarditis showing diffuse concave upwards ST elevation, ST depression in aVr and PR segment depression (best seen in rhythm strip at the bottom)

Stage	Changes on ECG
Stage I	Diffuse concave-upward ST-segment elevation with concordance of T waves; ST-segment depression in aVR or V1; PR-segment depression; low voltage; absence of reciprocal ST-segment changes
Stage II	ST segments return to baseline; T-wave flattening
Stage III	T-wave inversion
Stage IV	Gradual resolution of T-wave inversion

The ST-segment elevation is diffuse and concave upwards, in contrast to acute MI which is region specific and convex upwards. The elevation reflects abnormal repolarization secondary to pericardial inflammation. There may also be ST-segment depression in leads aVR and V1. Usually there are no changes during depolarization, so the P wave and QRS complexes are normal. Depression of the PR segment is very specific of acute pericarditis and is attributed to subepicardial atrial injury and occurs in all leads except aVR and V1, which may show PR-segment elevation. The ST segments in acute pericarditis return to baseline in a few days and are then followed by diffuse T-wave inversion, while in acute MI the T wave inversion appears concomitant with the ST segment elevation. Loss of height of R wave and appearance of q waves are also not seen with pericarditis.

Differentiation from early repolarization variant may also be required in many cases. Early repolarization is characterized by ST-segment elevation limited to precordial leads, elevation of the ST segment in V1, an isoelectric ST segment in lead V6, notching of the terminal aspect of the QRS complex, and tall T waves. The ST segments may shift to baseline with exercise. The ST/T ratio in lead V6 may also be calculated by dividing ST-segment elevation in mm by height of T wave in mm. An ST/T ratio of greater than 0.25 in lead V6 suggests acute pericarditis, while lower values are seen in early repolarization variant.

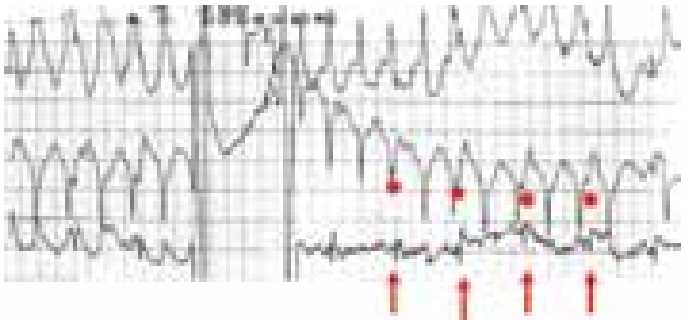

Figure 9: Artifacts appearing as VT, note the normal QRS (arrows) in the bottom strip continues to appear as a notch (dot) on the artifactual recording in the top tow leads

Electrocardiograpic artifacts

ECG artifacts are common and include tracings recorded from various sources. ECG artifacts have variable causes and include body motion of or near the skin electrodes, such as toothbrushing, scratching over an electrode, shivering; circuitry problems, such as inadequate electrode-recorder connections or frank disconnections; all these artifacts have been well documented to result in therapy, which is always inappropriate and on occasion frankly harmful to the patient. A clue to picking up artifacts is by noting that the normal QRS (though hidden) appears as a regularly occurring notch on the artifactual pattern (Figure 9) and as always keeping the clinical picture of the patient in mind.

Conclusion

Paying attention to simple ECG findings and always remembering to bear the clinical correlation in mind go a long way in interpreting ECG's correctly in critically ill patients in the ICU.

Case Report

ST Segment Depression During Regular Narrow QRS Tachycardia. What is the Mechanism?

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Madras Medical Mission, Institute of Cardiovascular Medicine, Chennai.

A 35 years old lady with a history of recurrent palpitations associated with pre-syncope and chest discomfort was found to have regular narrow QRS tachycardia during one of the episodes of palpitations (Figure 1). Significant ST segment depression (2 – 3 mm) appeared suddenly from the twelfth beat onwards which persisted till the tachycardia terminated spontaneously. The ECG obtained immediately after spontaneous termination of the tachycardia is shown (Figure 2).

What is the mechanism of ST segment depression?

It is to be noted that:

1. There is a sudden appearance of ST segment depression and the magnitude of this depression remains constant till the termination of tachycardia. ST segment depression due to cardiac ischemia is gradual in onset and the depression accentuates if the precipitating factor (the rapid heart rate as in this case) is not corrected.
2. The ST depression disappears immediately after the termination of the tachycardia. ST segment depression due to cardiac ischemia should have persisted atleast for a duration after the termination of the tachycardia.

   Based on these considerations, it is unlikely that the ST depression can be attributed to cardiac ischemia secondary to rapid heart rate and/or underlying coronary artery disease.

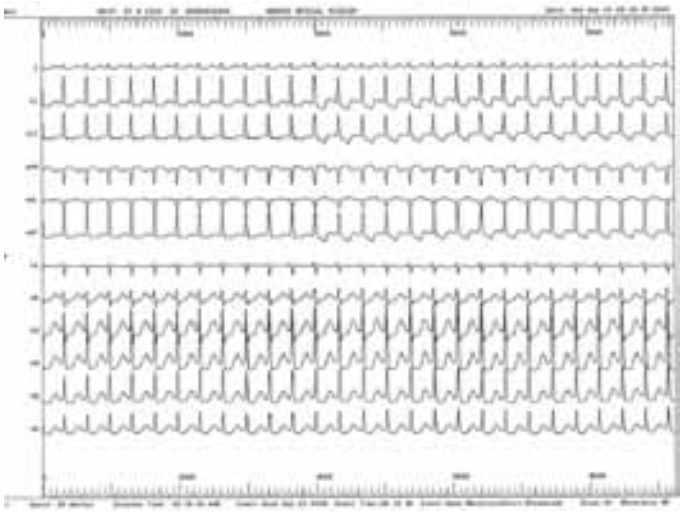

Figure 1 : A regular narrow QRS tachycardia, sudden appearance of ST segment depression from the twelfth beat onwards.

The commonest mechanism of ST segment depression seen during regular narrow QRS tachycardia is atrial activation (depolarization) during repolarization phase of ventricles (represented partly by ST segment) thereby leading to depression of ST segment, as seen classically in orthodromic tachycardia (AVRT – Atrio Ventricular Re-enterant Tachycardia). The antegrade limb of AVRT re-entry circuit is across the A-V node which activates the ventricles and the retrograde limb is across the accessory pathway, which activates the atria. The onset of activation of atria (depolarization) is usually 90 – 100 milliseconds after the inscription of QRS and hence falling on the ST segment (ventricular repolarization) and thereby deforming the ST segment, usually in the form of depression.[1]

In contrast, during AVNRT (Atrio-Ventricular Nodal Re-Enterant Tachycardia) the atria and the ventricles are activated almost simultaneously. Hence, the "P" waves formed by atrial

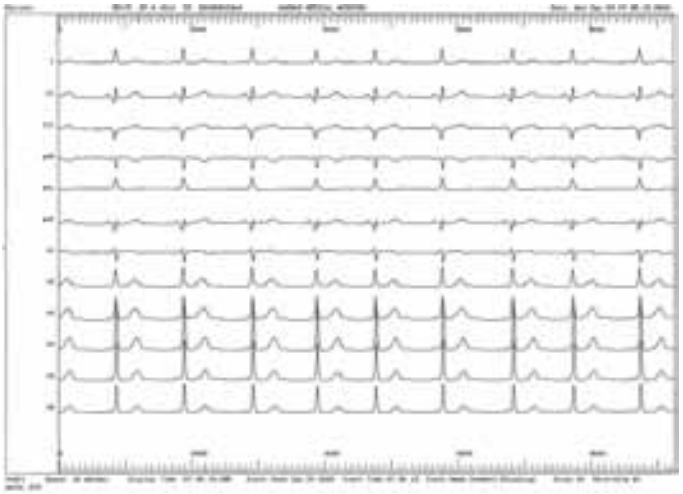

Figure 2 : WPW pattern – short PR interval and delta waves.

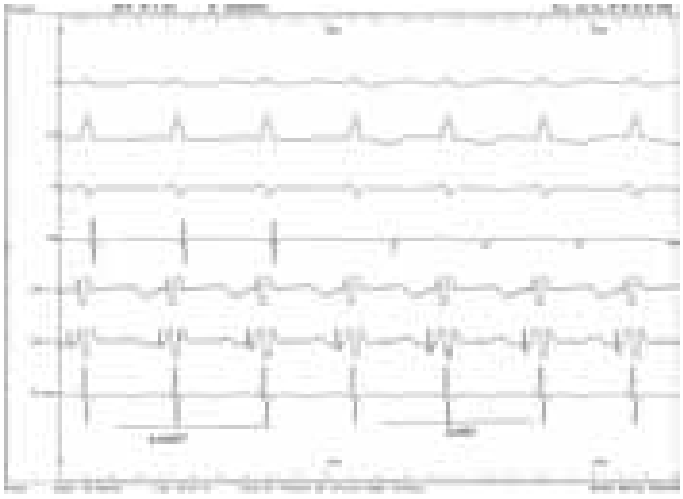

Figure 3 : Intra cardiac electrograms during transition between AVNRT and AVRT. ST segment depression is seen from the onset of AVRT.

activation are either buried inside the QRS complex and are not seen clearly or they may deform the terminal component of QRS complex producing pseudo r (r´) or pseudo s pattern.

The sinus rhythm ECG (Figure 2) shows Wolff-Parkinson-White syndrome pattern – short PR interval, negative delta waves in inferior leads, positive delta wave in V1 and transition in V2 suggestive of left posterior accessory pathway (which possibly participates in the AVRT associated with ST segment depression).

Pseudo r (r´) and pseudo s are seen clearly when the tachycardia was not associated with ST segment depression thereby suggesting the tachycardia to be typical AVNRT.

The intracardiac electrograms obtained during tachycardia when ST segment depression occurred suddenly is shown (Figure 3). During the first three beats (AVNRT), the atrial activation as seen in electrocardiograms recorded in HRA is simultaneous with ventricular activation as seen in RV apex recording. From the fourth beat onwards (AVRT), atrial activation has occurred 190 milliseconds after the ventricular activation thereby deforming ST segment in the form of depression.

This case illustration is also a demonstration of a rare phenomenon of transition of AVNRT to AVRT.[2]

Rule of Thumb

Regular narrow QRS tachycardia when "P" waves are not clearly seen is most likely AVNRT.

Regular narrow QRS tachycardia if associated with significant ST depression is most likely AVRT especially in the absence of clinical indicators of coronary artery disease.

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Case Report

Pseudo Atrial Flutter, Atrial Fibrillation and Torsades De Pointes : Parkinson's Tremors

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*Assistant Professor, Department of Medicine, **Ex. Senior Professor and Head, Department of Cardiology, J.L.N. Medical College, Ajmer

Abstract

We report a case of an elderly male with Parkinson's tremors whose electrocardiogram simulated atrial flutter / fibrillation and torsades-de-pointes. We also highlight diagnostic clues to correctly identify such pseudo-arrhythmias induced by tremors.

Introduction

Tremor – induced artifact may mimic supraventicular arrhythmias (atrial flutter or atrial fibrillation)[1] or if the artifact has sufficient amplitude, ventricular tachycardia or ventricular fibrillation.[2] We present a rare and interesting single electrocardiogram (ECG) mimicking atrial flutter, atrial fibrillation and torsades-de-pointes due to Parkinson's tremors.

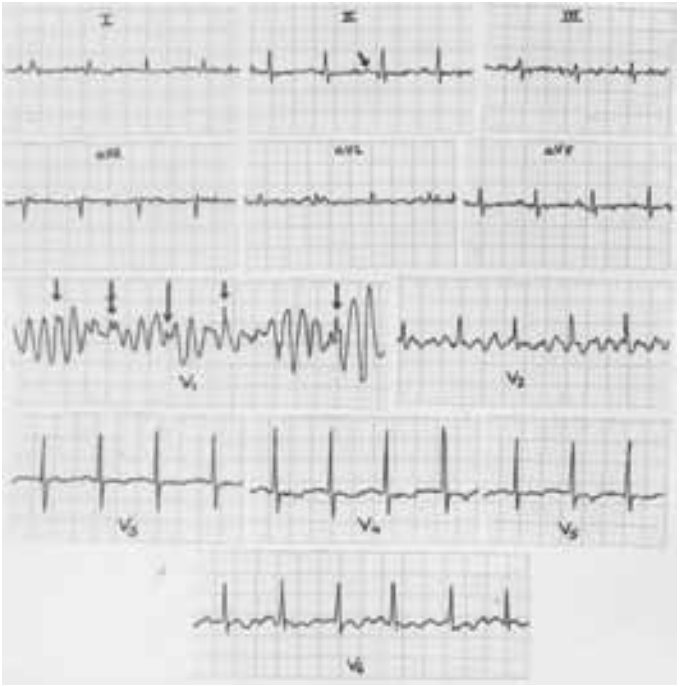

Figure 1 : 12 lead electrocardiogram of a patient with Parkinson's disease. Note pseudo atrial flutter waves in lead V_2 and V_6, pseudo torsades-de-pointes in lead V_1 and wavy baseline of pseudo atrial fibrillation in limb leads. Visible sinus P wave in lead III (arrow) with normal R waves spikes in lead V_1 (arrows) orient the diagnosis to tremor induced artifacts.

Case-Report

A 81 year old man with trochanteric fracture of left side femur was referred to outdoor for routine pre-operative cardiac evaluation. His pulse was 100 bpm, and blood pressure 120/76 mmHg. He was non-hypertensive, non-diabetic and was not taking any medications. Cardiovascular examination was unremarkable with normal heart sounds and no audible murmur. His central nervous system examination revealed resting tremors in fingers of both upper limbs with characteristic pill rolling movements. He also had mask like expressionless facies. On laboratory investigations serum electrolytes and thyroid profile was within normal limits. Chest Xray showed normal sized cardiac shadow. His 12 lead ECG revealed base line oscillations mimicking atrial flutter and fibrillation with well formed flutter waves visible in leads V_2 and V_6. A run of polymorphic ventricular tachycardia was also recorded in lead V_1 (Figure 1). However haemodynamic stability of the patient with regular pulse rate on clinical examination raised suspicion of pseudoarrhythmia, probably induced by Parkinson's tremors. Further diagnostic clues for these electrocardiographic artifacts were presence of (1) regular R-R intervals inspite of wavy baseline (2) intervening spikes of R waves of normal QRS complex (arrows) in lead V_1 (3) sinus morphology P waves visible in lead II (arrow) which confirmed Parkinson's tremor induced pseudo atrial flutter and fibrillation with torsades-de-pointes. He was treated with anti Parkinson's medication as per neurologist's advice.

Discussion

Body movements, poor skin-electrode contact, recorder malfunction, and electro- magnetic interference resulting in electrocardiographic artifacts mimicking atrial or ventricular tachycardia , are well described in literature.[3,4,5] However electrocardiographic changes of atrial flutter, and fibrillation with torsade-de-pointes simultaneously during single time recording of a 12 lead electrocardiogram has never been reported.

This case also highlights the importance of assessing a 12 lead ECG instead of depending on single channel telemonitor to make the diagnosis. If this patient was on a single channel telemonitor/ Holter monitor, mis-diagnosis of either Torsades-de-pointes or atrial flutter/ atrial fibrillation would have been made depending on the channel used. Such misinterpretation can further lead to unnecessary diagnostic and therapeutic interventions such as initiation of long term antiarrhythmic and anticoagulant drugs,[6] diagnostic cardiac catheterization and even implantation of permanent pacemakers or cardioverter defibrillators. Therefore ECG should always be interpreted in the context of the patient's condition and any other extraneous factors present at the time of recording. Physicians need to recognize and include artifact in the differential diagnosis so as to minimize unnecessary diagnostic and therapeutic procedures and provide optimal care to the patient.

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Ventricular Arrhythmias in Hypertrophic Cardiomyopathy- Can We Ever Predict Them?

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Key words: hypertrophic cardiomyopathy; ventricular arrhythmias

Hypertrophic cardiomyopathy (HCM) is characterized by gross cardiac and myocyte hypertrophy, myocyte disarray, and interstitial fibrosis. This condition is relatively common, with a prevalence of about 1:500 in the general population. Most patients with HCM are either asymptomatic or have only minimal symptoms. In general, HCM is a relatively benign disease with an annual mortality rate of slightly less than 1% in unselected HCM populations [1,2]. However, sudden cardiac death (SCD) may be the first manifestation of the disease. Approximately 60% to 70% of all patients with HCM die suddenly [3], and the fatal event is generally assumed, though not proven so far, due to ventricular arrhythmias.

The high arrhythmic propensity in HCM is contributed by a combination of many primary substrate abnormalities like hypertrophy, myocardial fibre disarray, interstitial fibrosis etc, and possible secondary triggers like ischemia, physical exercise, and excessive sympathetic stimulation. Marked heterogeneity in substrate and triggers would substantially reduce the predictive accuracy of any risk stratification model as known to occur in patients with many other cardiac arrhythmic conditions. To compound this, heterogeneity in the studied population, low event rate, effect of drug therapy etc would also limit the application of any predictive model.

However, a few revelations in the last decade have contributed to the better understanding of the fatal arrhythmia risk. Nonsustained ventricular tachycardia (NSVT) detected on Holter monitoring in younger (less than 30 years) patients have been identified to be associated with higher mortality rate [4]. Unlike their older counterparts where myocyte loss and fibrosis contribute incrementally to arrhythmogenicity, the higher mortality risk in younger patients probably reflects a more potent arrhythmogenic substrate caused by myocyte disarray, myocardial ischemia, and abnormal autonomic function. No doubt, the association between SCD and NSVT in young patients is striking; however, the majority of SCDs, even in young patients, occurred in patients without NSVT. This clearly shows that Holter monitoring identifies only a subset of subjects at higher risk. Clearly we need to rule out other contributing risk factors before reassuring an individual patient based on this non-invasive modality only.

Is there any role for 12-lead electrocardiogram to predict arrhythmia risk in them? In a cohort of patients with HCM selected because of their high risk for SCD, none of the studied electrocardiographic features (markedly increased voltages, QRS duration, left or rightward QRS axis, abnormal Q waves, and QTc or QT dispersion) did not predict subsequent appropriate implantable defibrillator intervention for ventricular tachyarrhythmias and was not useful in risk stratification for SCD [5]. Similarly, T wave alternans was also not useful to predict SCD in patients with HCM [6].

Another possible predictor could be exercise-induced arrhythmias. Though occurrence of ventricular arrhythmias during exercise is rare in HCM, its presence has long been identified to be associated with an increased SCD risk. In a recent study, the presence exercise-induced nonsustained VT or ventricular fibrillation was associated with a 3.73-fold increase in the risk of SCD or hemodynamically compromising sustained VT during follow-up [7].

Of late, a few studies with cardiac magnetic resonance (CMR) imaging in HCM also have shown some promising insights into risk stratification [8-12]. Delayed enhancement in CMR in HCM correlates with the histological finding of fibrosis and thus represents a likely substrate for ventricular tachyarrhythmias. In asymptomatic or mildly symptomatic HCM patients this finding in CMR had a significantly increased frequency of ventricular tachyarrhythmias on Holter monitoring compared with those without it. However, larger studies are required before establishing the role this non-invasive tool in the risk stratification in HCM.

Is there a role of genotype in deciding risk of SCD in these patients? Since the discovery of first causal gene and mutation for HCM in 1990, more than a dozen sarcomeric genes have been implicated in this disease [13]. Of these, certain mutations are considered high risk - for example, most mutations in TNNT2, R719Q and R403Q in MYH7, and double-causal mutations. However, a myriad of genetic (modifier genes, microRNAs, post-translational modifications of proteins, epigenetic factors etc) and nongenetic factors interplay to result in the complex phenotype in HCM. So a predictive model to assess the global risk of SCD in HCM based on genotype alone is still far from reality.

In conclusion, despite being half a century down the initial detailed clinical description of the entity, the predictors of fatal arrhythmias in this disease still remain largely elusive. However, in future, more advanced research into the causal genes and genotype-phenotype correlation, and larger natural history studies may likely enable us to predict the arrhythmia risk in HCM in a better way.

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ECG Quiz

Compiled by
Gopi Krishna Panicker[1], Yash Lokhandwala[2]

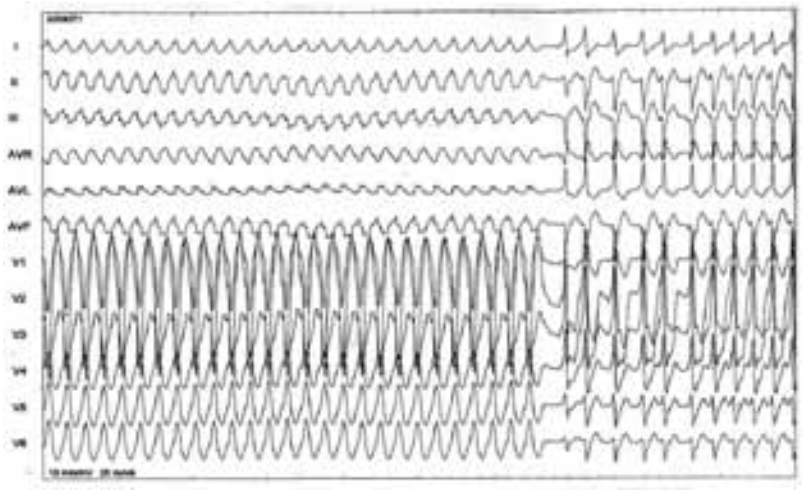
1: Quintiles Cardiac Safety Services, Mumbai
2: Arrhythmia Associates, Mumbai

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

ECG - 1

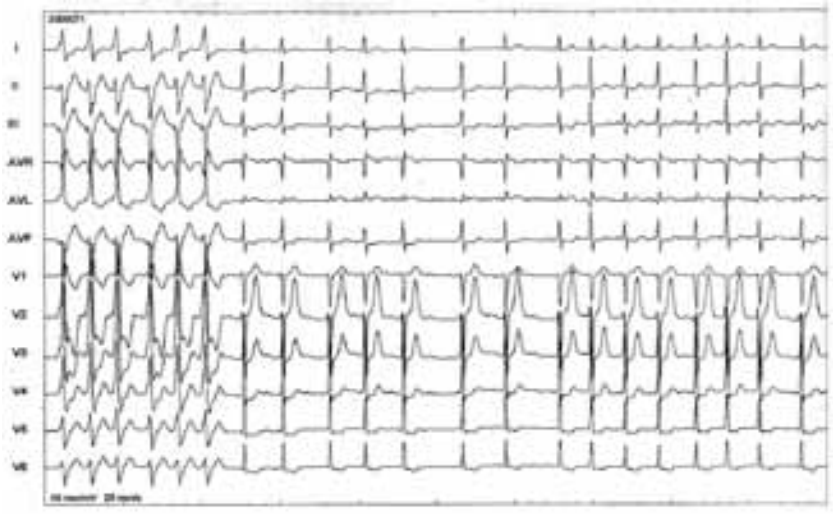
29 yr old man with recurrent rapid palpitations, sometimes with near-syncope

Figure 1



As the episodes continues…….

Figure 2



What are these arrhythmias?

a. a. Monomorphic VT and A Fib

b. Polymorphic VT and A Fib

c. AVRT and A Fib

d. LQTS arrhythmias

For correct answer see overleaf

ECG - 1

The correct answer is 'c' – AVRT and A Fib

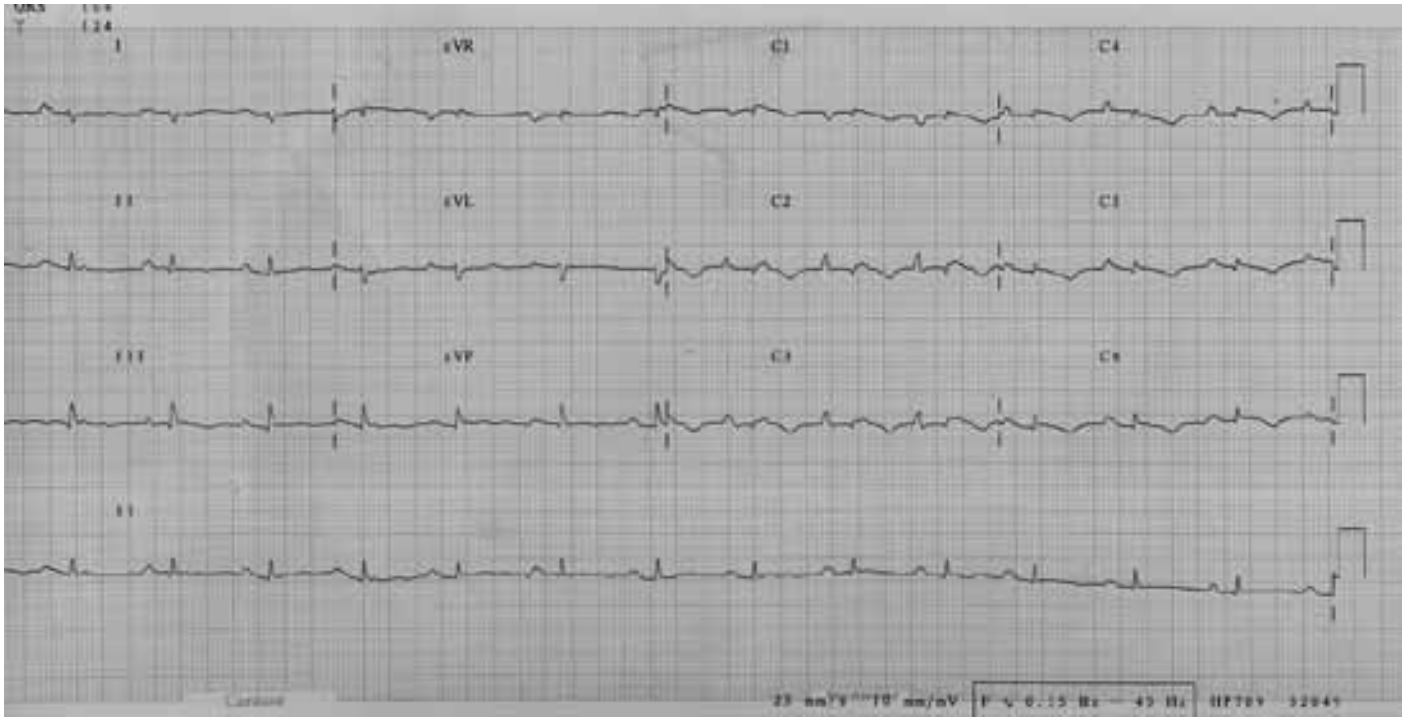
The first ECG shows initially a rapid regular wide QRS tachycardia @ 200 bpm. It shows a typical LBBB pattern. The latter portion of the trace shows an irregular wide QRS tachycardia with a different morphology. There is a delta wave which is seen in V2-V4. This is diagnostic of atrial fibrillation with pre-excitation (WPW). Slide 1A shows an abrupt cessation of pre-excitation with a sudden drop in ventricular rate. The A fib now continues with a normal QRS complex. Therefore, this is WPW syndrome with orthodromic AVRT which degenerates into A Fib. WPW syndrome patients have a high incidence of A Fib, the commonest mechanism being degeneration of AVRT as in this slide.

ECG - 2

24 yr old lady with a history of VT several years ago

What is the etiology?

Figure 1



a. ARVC

b. Ebstein's anomaly

c. PH

d. Addison's disease

For correct answer see overleaf

ECG - 2

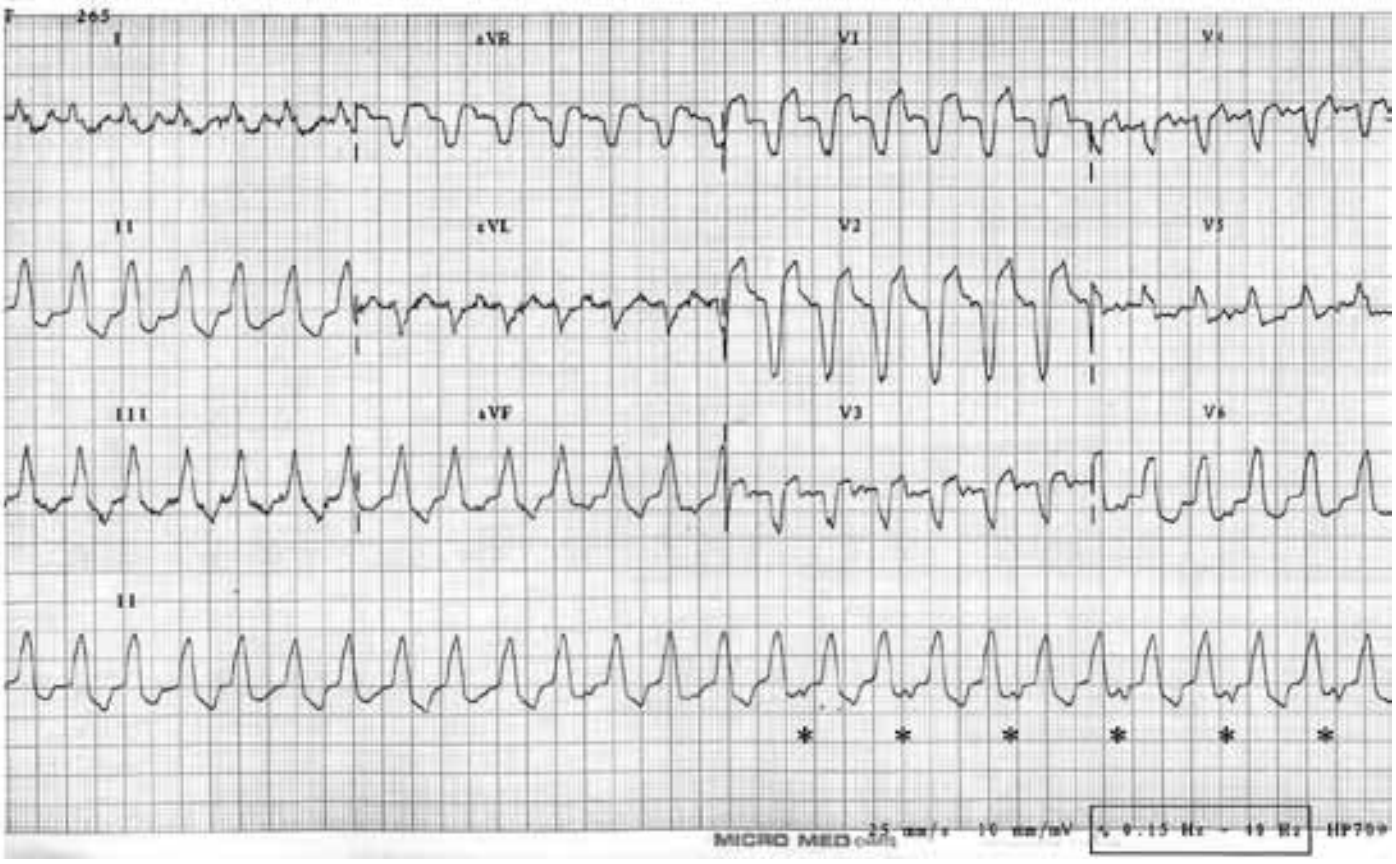
The correct answer is 'a' – ARVC

The ECG shows many abnormalities. The P waves are broad in lead lead I and sharp and prominent in lead V3-V4.

The QRS axis is rightward (120^{o}). There is low QRS voltage. The T waves are flat in inferior leads and gently inverted in the precordial leads. When taken in conjunction with a history of ventricular tachycardia, the diagnosis overwhelmingly points towards ARVC (Arrhythmogenic RV cardiomyopathy).

ARVC is often missed due to subtle abnormalities at echo. It is often familial and progressive. There are several ECG patterns in ARVC. Since VT in ARVC mainly arises from right ventricle, it shows LBBB type of morphology.

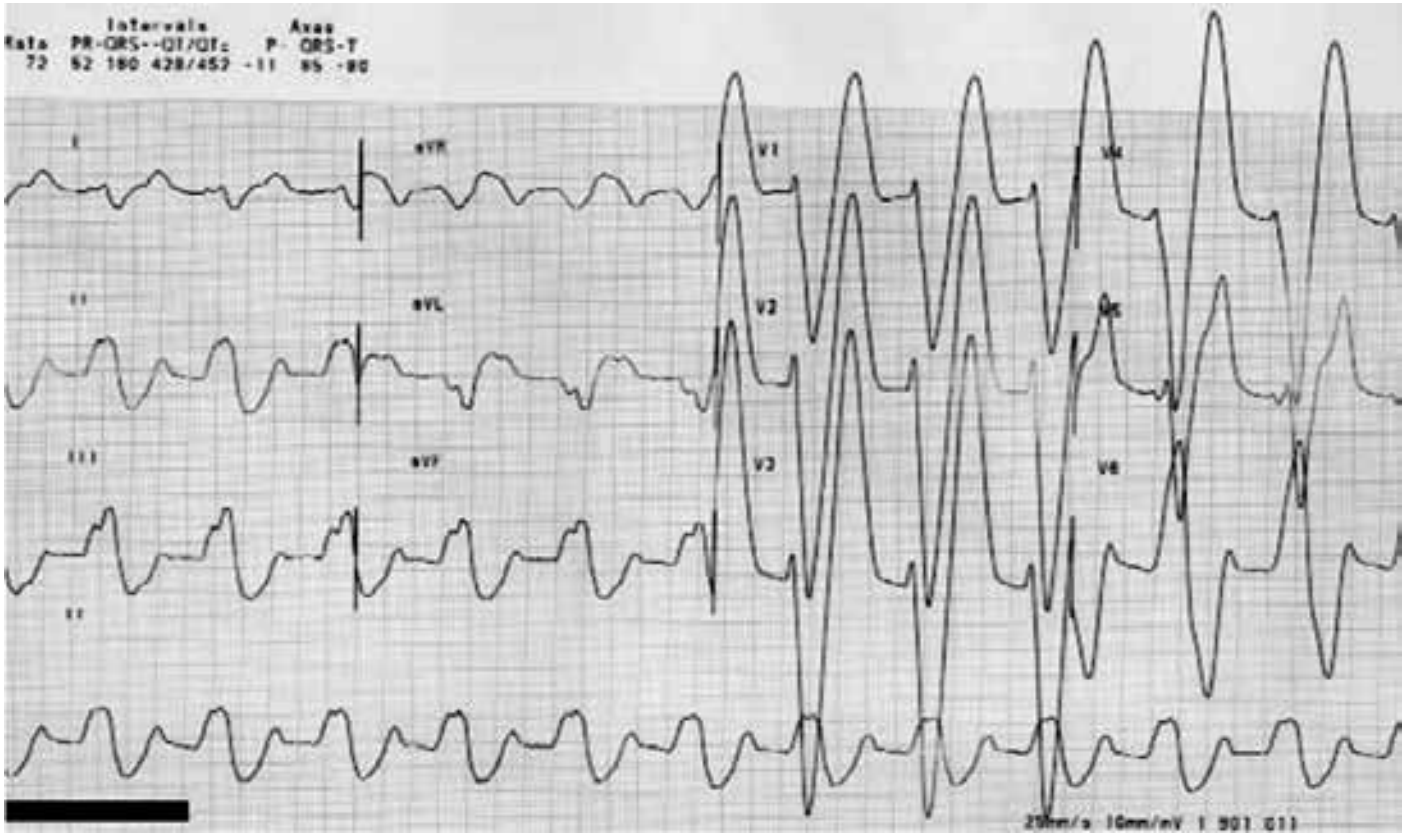
Figure 2. Note the 2:1 VA conduction in long lead II (* denote P waves)



ECG - 3

27 yr old lady, post burns, in ICU. What is the diagnosis?

Figure 1



a. Hypocalcemia

b. Hyperkalemia

c. Hypothermia

d. Hypomagnesemia

For correct answer see overleaf

ECG - 3

The correct answer is 'b' – Hyperkalemia

The ECG shows wide and bizarre QRS complex. Even considering the wide QRS duration, it does not conform to and bundle branch block pattern.

As seen in lead V1-V4, the end of the QRS directly continues into the upstroke of the T waves. Such an abnormal conduction signifies severe diffuse His-Purkinje and myocardial conduction delays. The T waves are extraordinarily tall and peaked in V2-V4. No P waves are seen. The rate is 100 bpm. The patient was having sepsis and azotemia.

Cardiac action potential changes in hyperkalemia

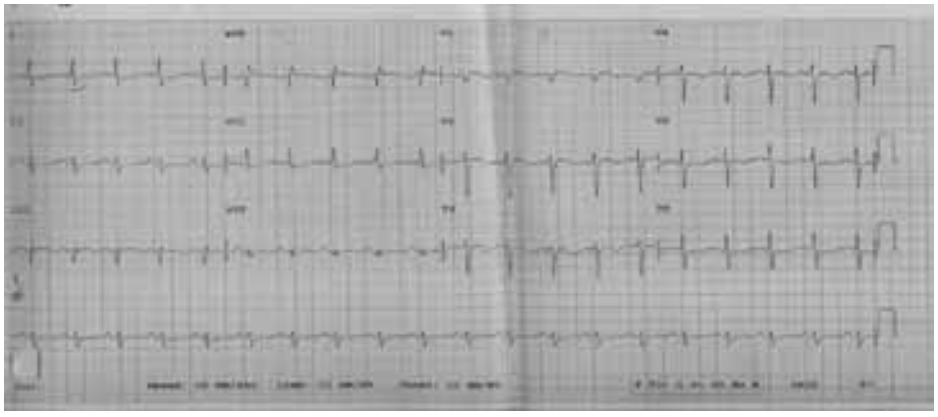
- The resting membrane potential is less negative
- The slope of depolarisation (phase 0) is decreased: results in conduction slowing and QRS/P widening
- Phase 3 velocity is increased: results in T wave changes
- Phase 4 slope is decreased: less automaticity
- The sinus node is less affected than the atrial myocardium: hence the P wave may not be visible

Dittrich et al. J Emerg Med 1986

ECG - 4

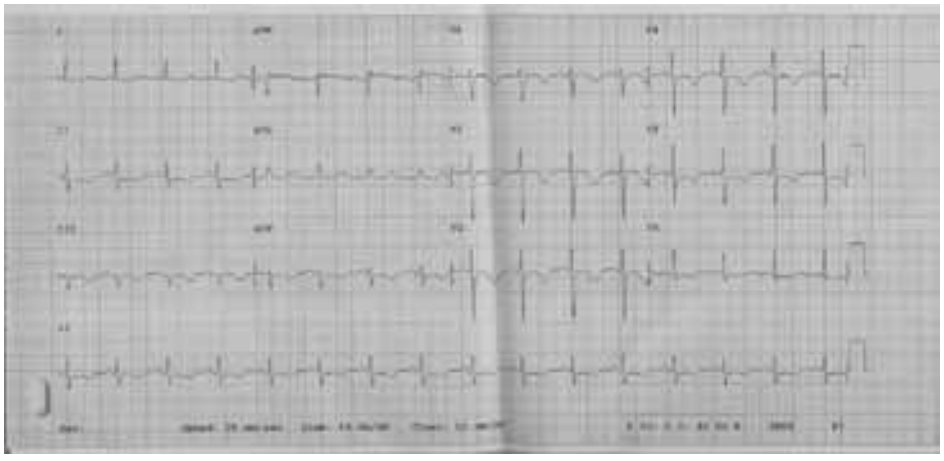
75 yr old lady, admitted with a history of syncope; bradycardia and hypotension when made to stand, oxygen saturation 88% on room air

Figure 1



2 days later…

Figure 2



The investigation of choice will be:

a. CT pulmonary angiogram

b. Coronary angiography

c. MR angio brain

d. Cardiac MRI

For correct answer see overleaf

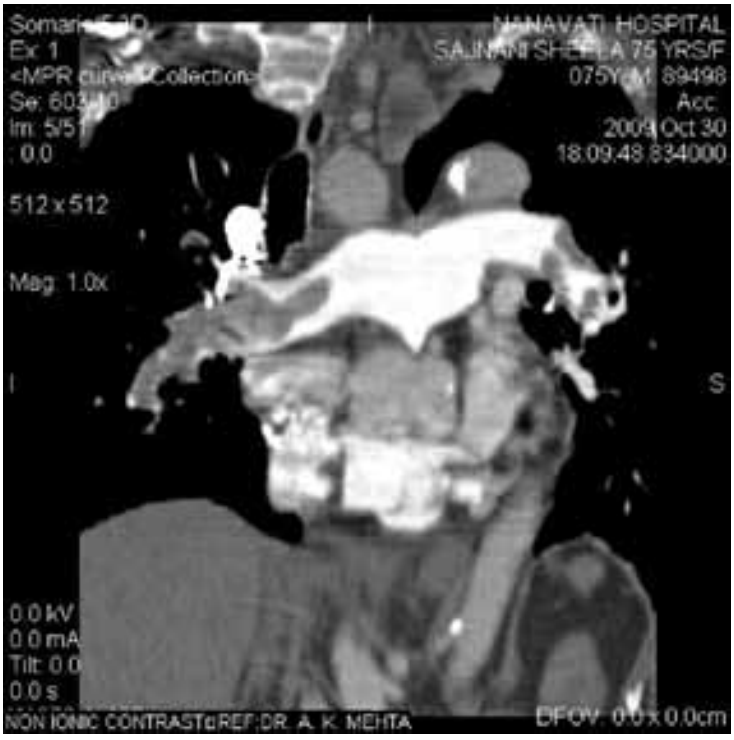
ECG - 4

The correct answer is 'a' – CT pulmonary angiogram

The ECG shows sinus tachycardia and gentle T inversion from V2 to V5. The QRS axis is horizontal. There are distinct S waves in lead I, prominent Q waves in lead III and gentle T wave inversion in lead III, constituting the S1Q3T3 pattern. The ECG 2 days later shows sinus tachycardia and accentuation of T inversion from lead V2-V5 as well as in inferior leads.

This constellation of ECG findings, arterial hypoxemia and the clinical presentation make it mandatory to rule out pulmonary thromboembolism.

A CT coronary angiogram showed large emboli in both the pulmonary arteries.



Syncope in Pulmonary Embolism

- More common in the elderly (19% vs 6%) -

  Punukollu et al. Int J Cardiol 2005

- Initial presentation in 10% -

  Koutkia et al. Heart Lung 1999

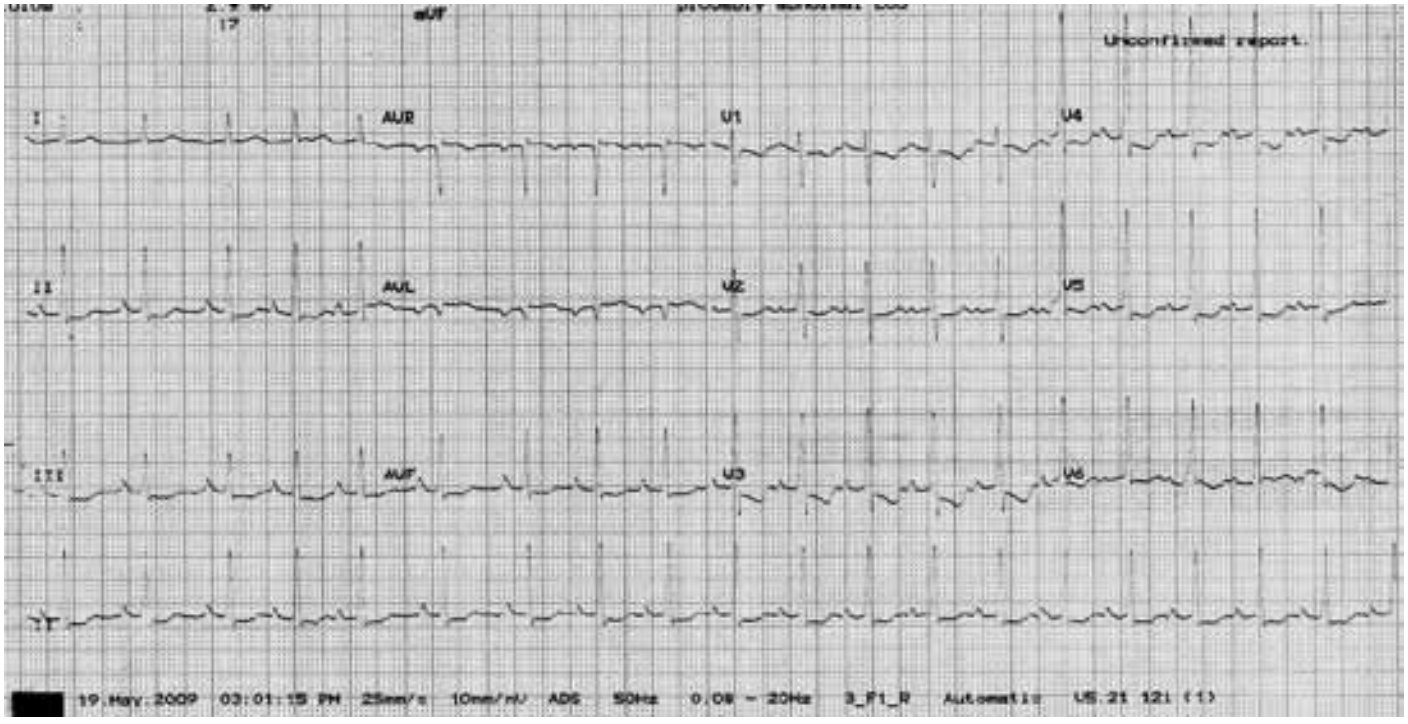
- Mechanisms
  - i. Central occlusion followed by fragmentation and distal migration;
  - ii. tachy/bradyarrhythmia,
  - iii. Vagal response

  Wolfe al. J Emerg Med 1998

ECG - 5

24 yr old nurse, heart rates usually 110-130 since 2 years. LVEF 45%. What is this incessant tachycardia?

Figure 1



a. Inappropriate sinus tachycardia

b. Atrial tachycardia

c. Fast-slow AVNRT

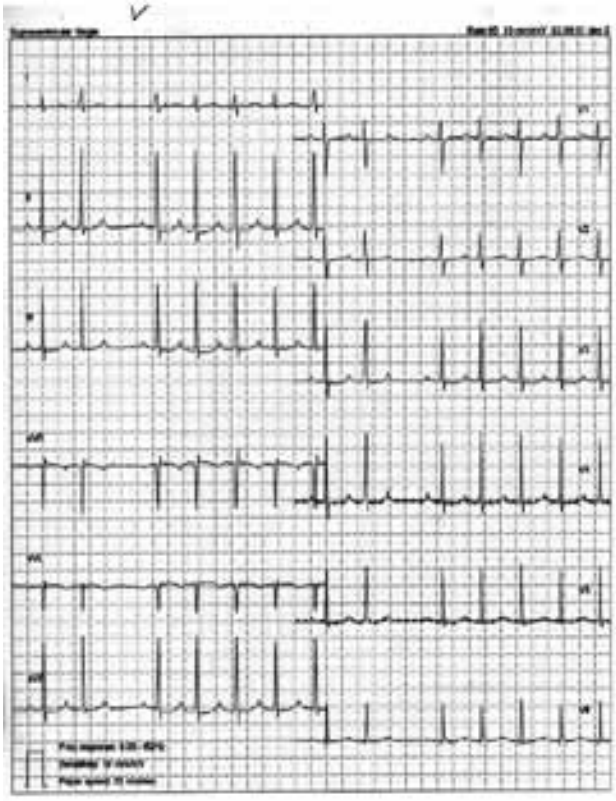
d. Coronary sinus rhythm

For correct answer see overleaf

ECG - 5

The correct answer is 'b' – Atrial tachycardia

The ECG shows tachycardia with a rate of 130 bpm. The P waves when viewed carefully are flat in lead I, inverted in lead aVL and positive in lead V1. The incessant tachycardia is therefore most likely an atrial tachycardia arising from high posterior left atrium. This would be consistent with a pulmonary vein focus. The LV dysfunction then is likely due to tachycardia-induced cardiomyopathy. A 12-lead Holter in this patient brought intermittent 2:1 AV conduction during tachycardia, again confirming the diagnosis of atrial tachycardia (sinus tachycardia will rarely ever show 2:1 AV conduction).

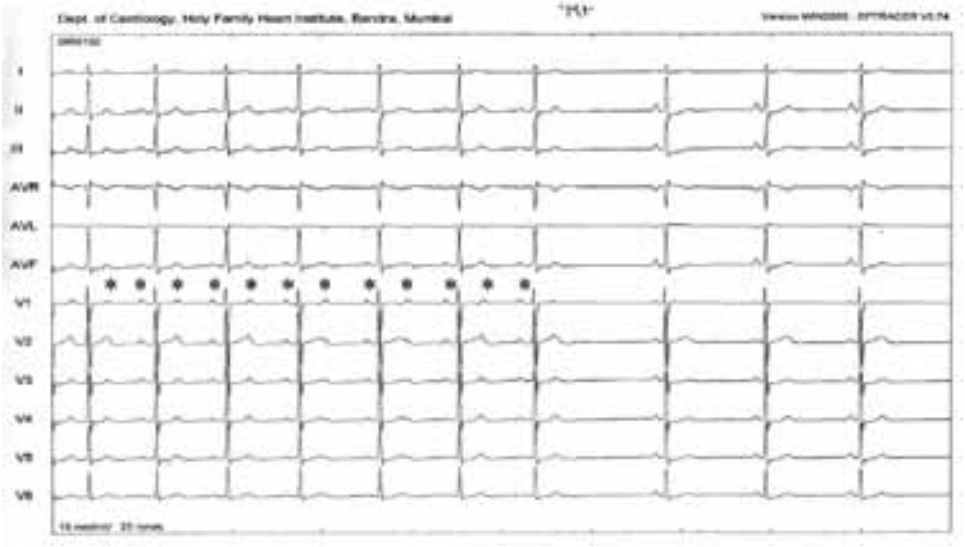


Mechanism of tachycardiomyopathy:

Incessant tachycardia over weeks, leading to:

1. Short diastole & this impairs reuptake of Ca++ by sarcoplasmic reticulum.
2. Depletion of mitochondria due to exhaustion of energy-generating capacity.
3. Remodeling of myocardium in response to long-lasting tachycardia. Altered expression of cytoskeletal protein, CK & ANP.

This nurse underwent successful cure by RF ablation. The ECG below shows atrial tachycardia with 2:1 AV conduction (* shows P waves), terminated by RF energy with restoration of normal sinus rhythm in last 3 complexes. The echocardiogram was normal after 1 month.



ECG - 6

68 yr old man, smoker, comes with a history of severe chest pain

Figure 1

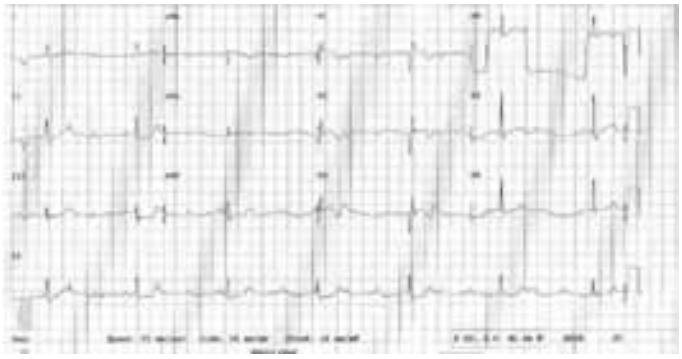


Figure 2

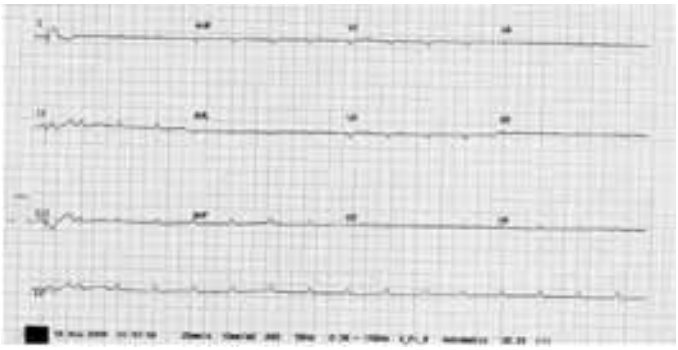
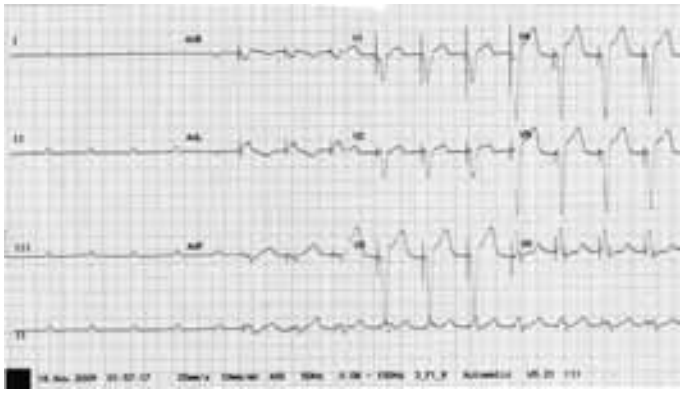
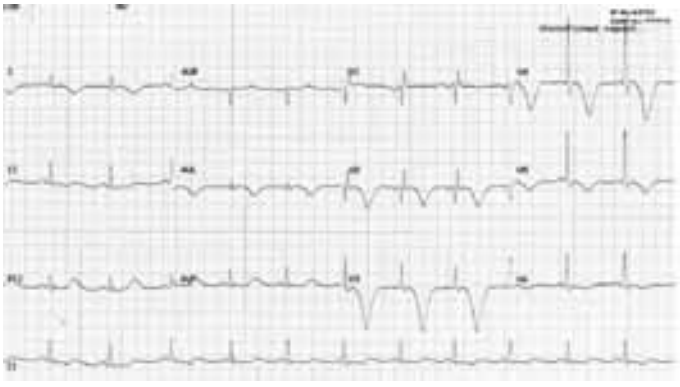


Figure 3

Soon after…



3 days later…..
What was the mechanism of AV bock?



a. Anterior MI with AV nodal block

b. Anterior MI with intra-His block

c. Anterior MI with infra-His block

d. Lev's disease

For correct answer see overleaf

ECG - 6

The correct answer is 'b' – Anterior MI with intra-His block

The first tracing shows sinus tachycardia with complete AV block and ***narrow QRS complexes***. There are QR complexes in leads V1/V2 with coving ST segments and terminal mild T inversion.

2nd ECG shows only ***'P waves' !!!***

The 3rd ECG shows ventricular pacing after the initial asystole. The ECG of 3 days later shows 1:1 AV conduction with deep symmetrical T wave inversion with leads V2-V4.

The cardiac enzymes were elevated, there was septal hypokinesia on the echocardiogram and the coronary angiogram showed a likely recanalised left anterior descending artery with ectasia.

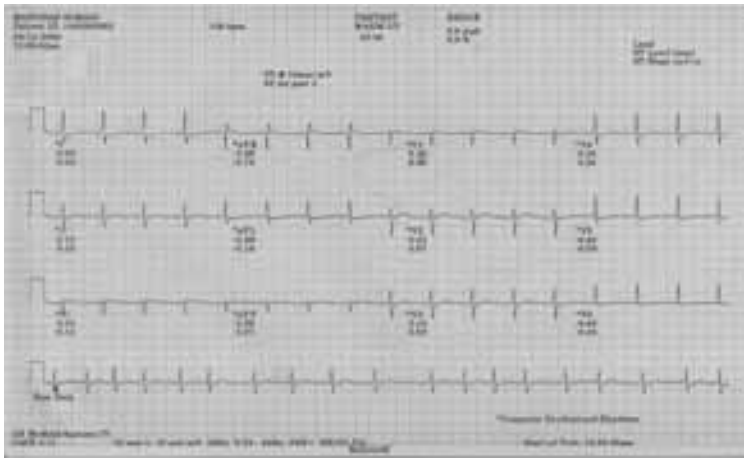
Complete AV block in acute MI

- Majority have inferior wall MI due to RCA occlusion
- In anterior MI, patients with CHB are generally unstable, with wide QRS escape rhythms
- Rarely in anterior MI CHB with a narrow QRS escape is seen; this occurs because of proximal LAD occlusion causing ischemia of the His bundle, which may receive supply from the 1st septal artery

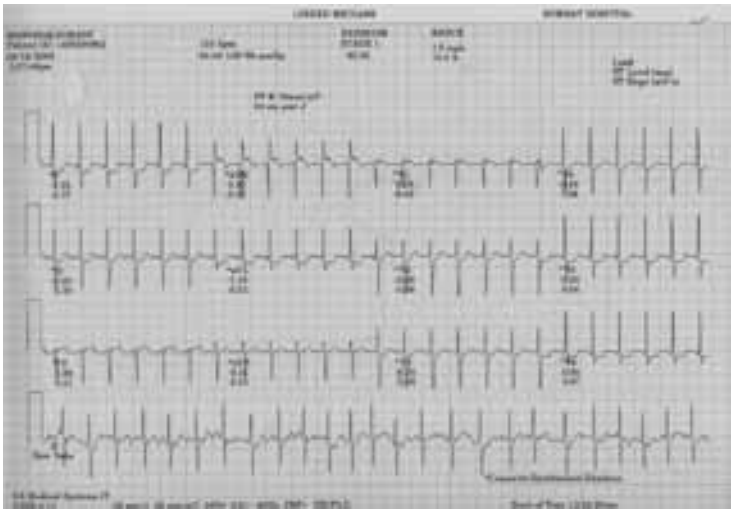
ECG - 7

58 yr old man undergoing a stress test ….

Figure 1



How does one interpret the ST changes?



a. Brugada syndrome

b. Ischemia

c. Digoxin effect

d. Normal

For correct answer see overleaf

ECG - 7

The correct answer is 'd' – Normal

The 12-lead presentation shows 'sinus tachycardia' along with horizontal ST depression of up to 2 mm in lead I, aVL and V5,V6. It also shows ST elevation in lead aVR. However, this is a Linked Median trace as denoted at the top. This type of trace is a computer synthesized ECG (as marked at the bottom right). Many stress test machines use various techniques to eliminate the baseline disturbance produced as the patient is moving through the stress test.

While this technique produces an apparently nice looking ECG, one must remember this is ***not the faithful representation.*** If one sees the raw, unadulterated trace as indicated below long lead II one sees that the patient is clearly having atrial fibrillation. Even in figure 1, while the long lead II shows atrial fibrillation, the linked median trace shows a regular rhythm. The apparent ST depression is similarly an artifactual phenomenon in this patient. The patient was not taking digitalis and had a normal coronary angiogram.

INDIAN SOCIETY OF ELECTROCARDIOLOGY

APPLICATION FORM FOR LIFE MEMBERSHIP/FELLOWSHIP

SECRETARIAT
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Dear Sir,

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

My particulars are as follows :

Name in full (Surname first) _____

Qualifications _____

University (Post-Graduation obtained) _____

Year of obtaining first Post-Graduation _____

Mailing Address _____

_____

Tel. No. Hospital _____ Clinic _____ Residence _____

Fax _____ E-Mail _____

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

No. _____ Dated _____ of _____

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

Thanking you,

Yours sincerely,

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

Name _____

Address _____

Signature _____

FOR OFFICE USE ONLY

Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No.

_____

Hon. Secretary, ISE

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

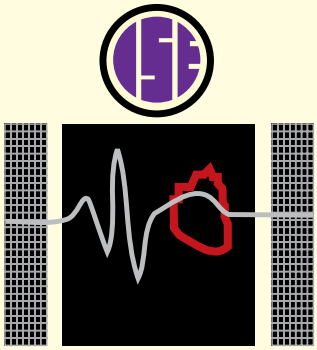
***Life Members :**

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

****Fellowship:**

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

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



SECRETARIAT
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