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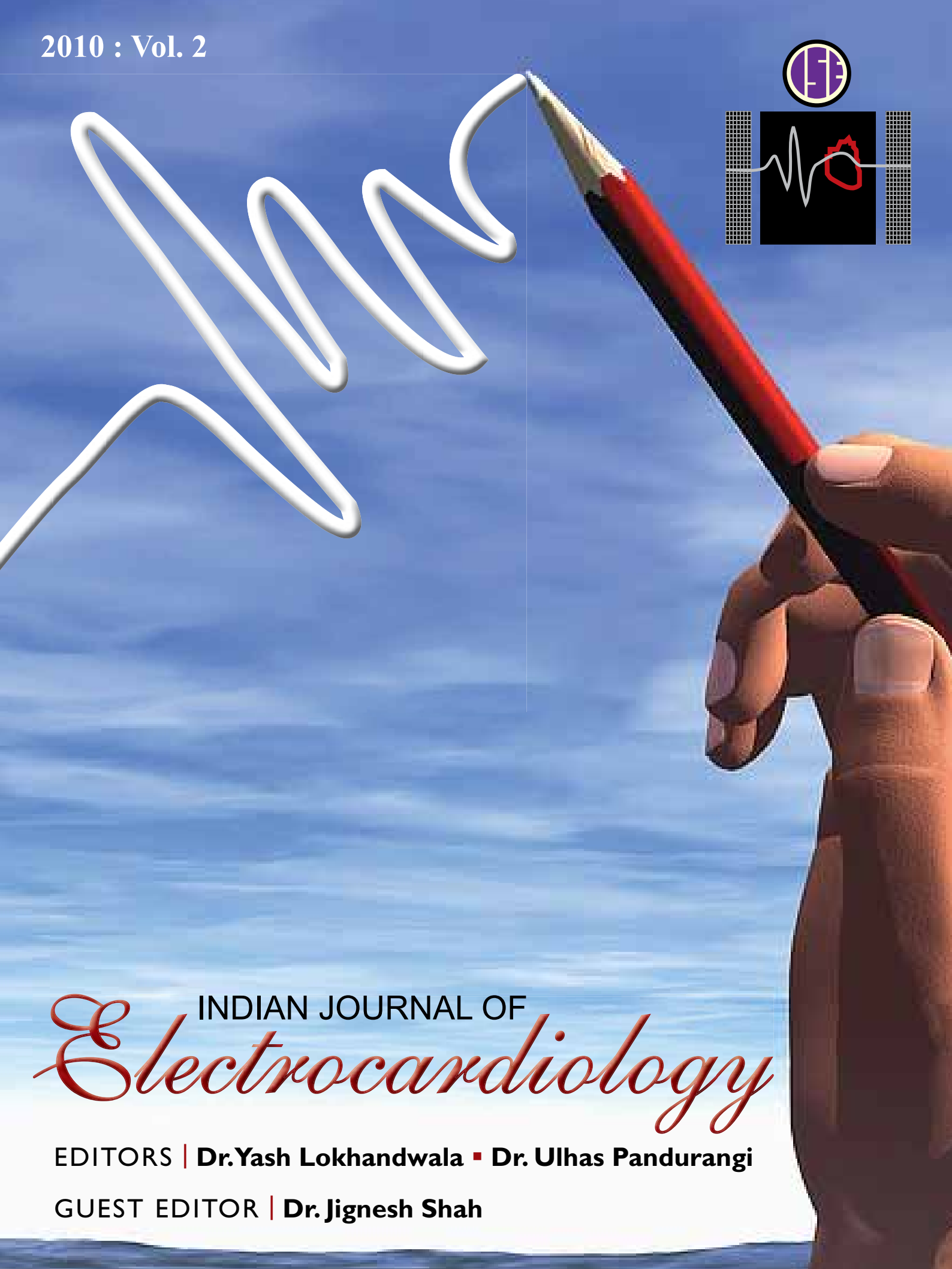

INDIAN JOURNAL OF Electrocardiology

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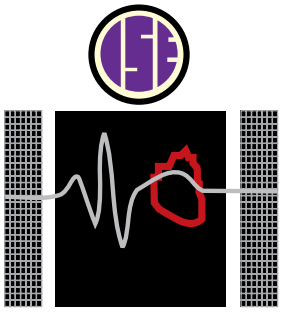

Welcomes

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 ISECON 2010, Mumbai.

The scientific committee has prepared an exciting academic program. I am sure their organization, faculty members' 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. Drs. Shomu Bohra and Chandrasekaran have written a comprehensive review on left bundle branch block. The anatomy, pathophysiology, diagnosis, prognosis and relevance in health and disease states have been comprehensively covered in this article. Dr. Lokhandwala and myself have reviewed the literature on CRT and have given an overview of the data for utility of CRT in certain subgroup of patients keeping the Indian scenario in mind.

We are fortunate to have two excellent case reports: Dr. Meena and colleagues present a case of aluminum phosphide poisoning with its profound cardiotoxicity. Dr. Monika Maheshwari and colleagues present the first case report of sinus node dysfunction in single ventricle with L transposition of great vessels.

Dr. Todd et al have presented an excellent review of various ECG findings in pulmonary embolism. Their comprehensive review of literature will assist all of us utilize ECG: the most commonly available test well to diagnosis this life threatening emergency. 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. These have been selected from those recently shown at the APICON in Jaipur.

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.

Yash Lokhandwala
Editor

Jignesh Shah
Guest 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 ISECON 2010 – Annual Conference of Indian Society of Electrocardiology.

Dr Ajay Naik and his team organized ISECON-2009 at Ahmedabad from 20th to 22nd February 2009. It was a great scientific bonanza.

Dr P Sahoo and his team members organized Bhuvaneshwar Arrhythmia Course – Mid Term Conference of Indian Society of Electrocardiology on 12th and 13th September 2009 – A real academic feast.

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

a. "ECG Learning Course" for postgraduate students at Mumbai on 3rd and 4th January 2009, at Ahmedabad on 11th January 2009, at Lucknow on 30th and 31st May 2009, at Guwahati on 20th and 21st June 2009, at Udaipur on 7th and 8th November 2009 and at Chandigarh on 23rd and 24th January 2010. About 70-80 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading

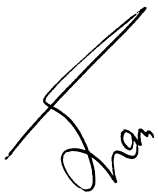
b. Satellite Symposia were organized at Mumbai on 7th June 2009, at Kolkata on 14th June 2009 under the leadership of Dr Rabin Chakraborty, at Ludhiana on 5th July 2009 under the leadership of Dr Vanita Arora and were appreciated by one and all.

c. A meeting of "Training the Trainers" was organized at Mumbai on 17th January 2010 to finalize the presentations of the "ECG Learning Course".

ISECON 2010 has arrived and we wish to re-write history again. Dr Yash Lokhandwala and Dr Amit Vora have laid down an academic feast for all of you. We wish ISECON 2010 to be a great memorable event.

My sincere thanks to Dr Yash Lokhandwala, Dr Ulhas Pandurangi, Dr Jignesh Shah (Guest Editor) and the Editorial Team for bringing out the ISE Journal – 2010, 1st Volume.

Long Live Indian Society of Electrocardiology



Dr SB Gupta
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Left Bundle Branch Block in Health and Disease

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Introduction

Left bundle branch block (LBBB) is an occasional finding seen on the electrocardiogram (ECG). It often occurs in patients with underlying heart disease and may also be associated with progressive conducting system disease. Presence of LBBB interferes in diagnosis of acute coronary syndrome on ECG and with interpretation of exercise testing. Presence of LBBB in heart failure is currently a hot topic, for it induces ventricular dyssynchrony, thereby worsening left ventricular function.. This article provides an overview of the anatomy, epidemiology, recognition on ECG and importance of LBBB in health and disease.

Anatomy and Physiology of Left Bundle Branch

The bundle of His divides at the junction of the fibrous and muscular boundaries of the interventricular septum into the right and left bundle branches. The main left bundle branch penetrates the membranous portion of the interventricular septum under the aortic ring and then divides into several fairly discrete branches. The components of the left bundle branch are[1-5] :

1. A pre-divisional segment.
2. An anterior fascicle that crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle.
3. A posterior fascicle that fans out extensively inferiorly and posteriorly.
4. In some hearts, a median fascicle to the interventricular septum.

The left bundle and its fascicles consist of Purkinje fibers which transmit impulses at a rate of 1 to 3 m/sec. This results in simultaneous activation of nearly all left ventricular myocardium in normal people. Pathological studies in LBBB have suggested that the block may be proximal (particularly in diffuse myocardial disease), distal or a combination of both [6].

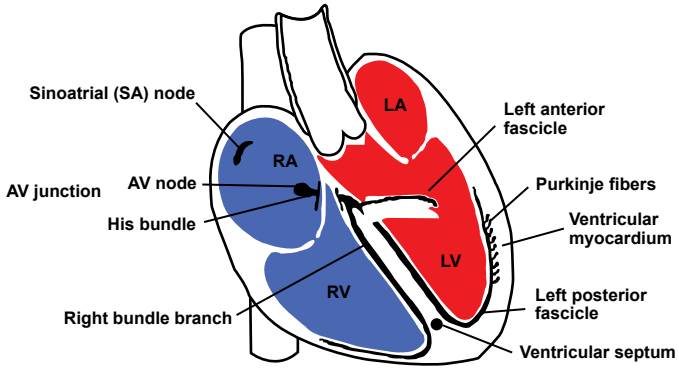

Figure 1 : Schematic diagram of conduction system of the heart

The left anterior descending artery through its septal branches, provides the primary blood supply for the left bundle branch, particularly for the initial portion. As is true for the right bundle branch, there may be some collateral flow from the right and circumflex coronary systems.

Causes of LBBB [6-8]

1. Degenerative
2. Hypertension
3. Coronary Artery Disease
4. Myocarditis
5. Cardiomyopathy
6. Valvular Heart Disease
7. Hyperkalemia
8. Drug intoxication
9. Bacterial endocarditis.

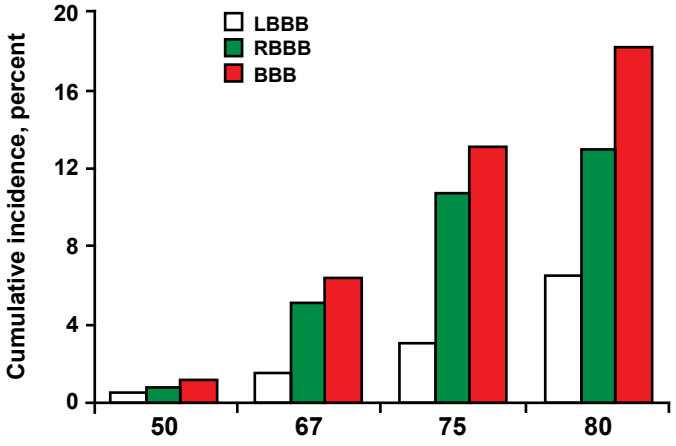

Figure 2 : In men, the cumulative incidence of right (RBBB), left (LBBB), or any bundle branch block (BBB) increases with age (upper panel). [9]

Incidence

The incidence of LBBB increases with age [9,10] as was shown in a 30 year prospective study of 855 Swedish men. The incidence of LBBB was 0.4 percent in at age 50, 2.3 percent by age 75, and 5.7 percent by age 80 [9] . In this otherwise healthy population, with no co-morbidities such an increased incidence suggests that LBBB is usually a marker of a slowly progressive degenerative disease of the conduction system (Figure 2).

However, there are conflicting data regarding association of LBBB with cardiovascular disease. During an 18 year follow-up from the prospective Framingham Heart Study [11], 55 subjects developed LBBB. These subjects had a mean age of 62 years, most had antecedent hypertension (62 percent) and/or coronary disease and almost one-half had cardiac enlargement on chest x-ray. Coincident with or subsequent to the onset of the LBBB, 48 percent developed clinically apparent coronary disease or heart failure. Over the period of follow-up, only 11 percent remained free of cardiovascular disease compared 50 percent in an age-matched control group without LBBB.

LBBB is infrequent in young healthy subjects with an incidence of 0.05% below the age of 30 [12,13]. The co-existence of underlying cardiovascular disease is much lower among these young patients with LBBB, nevertheless it should be ruled out [9,12].

Prognosis

The outcome is generally excellent in younger subjects with a LBBB [12,14], while LBBB in older individuals is associated with an increase in mortality. In the report from the Framingham study cited above, one-half of patients with new LBBB died of cardiovascular disease within 10 years, much higher than the 12 percent mortality rate in the control group [11]. The prognosis of LBBB is related largely to the underlying heart disease and the presence of other conduction disturbances.

A primary prevention study from Sweden followed 7392 middle-aged men with no cardiovascular disease for 28 years [15]. Compared to men without bundle branch block, the 46 patients with LBBB had significant increases in progression to high-degree AV block (adjusted hazard ratio 12.9) and all-cause mortality (adjusted hazard ratio 1.85) that was primarily due to out-of-hospital sudden death. Among the men with LBBB, one in six progressed to high-degree AV block or to requirement for a pacemaker. In contrast, right bundle branch block had no adverse effect on prognosis.

Among patients with coronary artery disease, LBBB is an independent predictor of all-cause mortality. This was illustrated in review of 7073 adults referred for nuclear exercise testing, 2 percent of whom had a complete LBBB [16]. After a mean follow-up of 6.7 years, those with LBBB had a greater mortality than those without (24 versus 11 percent). The presence of a LBBB in the setting of an acute myocardial infarction is associated with a significant increase in mortality, even when thrombolytic therapy has been administered.

Electrocardiogram in Common Left Bundle Branch Block

Normally, ventricular activation is initiated by ramifications of the left bundle in the left mid- and paraseptal regions. In LBBB, ventricular activation is initiated by rami of the right bundle branch that connect both to the right septum and right ventricle.

The electrocardiographic pattern in LBBB can be best understood in terms of three vectors:

- Early and usually leftward vector —The initial 10 msec (0.10 sec) vector in LBBB has two characteristics. First, the direction of activation is reversed, traveling from right to left. Second, activation also travels from apex to base and to the right ventricular apex and free ventricular wall. However, the septum is a larger structure than the right ventricular free wall; thus, septal activation predominates. The resultant vector is to the left and usually anterior, resulting in loss of the normal q wave and initiation of a wide, slurred R wave in I, aVL, and V6. In addition, an rS or QS pattern is seen in V1. “Pseudo-normalization” of septal depolarization in LBBB (ie, the reappearance of a q wave in I and V6) may reflect disease in or damage to the septum.
- Mid-temporal leftward and posterior vector — Depolarization continues in the myocardial cells of the septum from apex to base. From 20 msec through 60 msec, spatial vectors are oriented to the left and posteriorly since the left ventricle is a leftward and posterior structure. The spatial vector that appears at 80 msec represents the mass of left ventricular myocardial depolarization, resulting in a signal of large amplitude. The amplitude of this signal is further increased by two factors: the terminal vectors are not countered by right ventricular forces since the right ventricle has already been depolarized; and the thick posterobasal portion of the left ventricle is activated before the thinner anterolateral wall.
- Terminal leftward vector — The terminal 10 msec vector and beyond result from depolarization of the anterolateral wall of the left ventricle which, as mentioned, is thinner than the posterobasal region, producing a small vector that is also directed to the left and posteriorly. Damage to the anterolateral wall, as with a myocardial infarction, will decrease or actually reverse the direction of the terminal vector.

ECG pattern —Based on the abnormal initial vector, the relentless leftward progression of the initial, middle and terminal forces, the increase in amplitude due to the lack of counteracting right ventricular forces, and the conduction delay due to asynchronous activation two major changes occur in the ECG: loss of normal early septal forces; and the development of large and prolonged QRS complexes in the leftward leads (I,

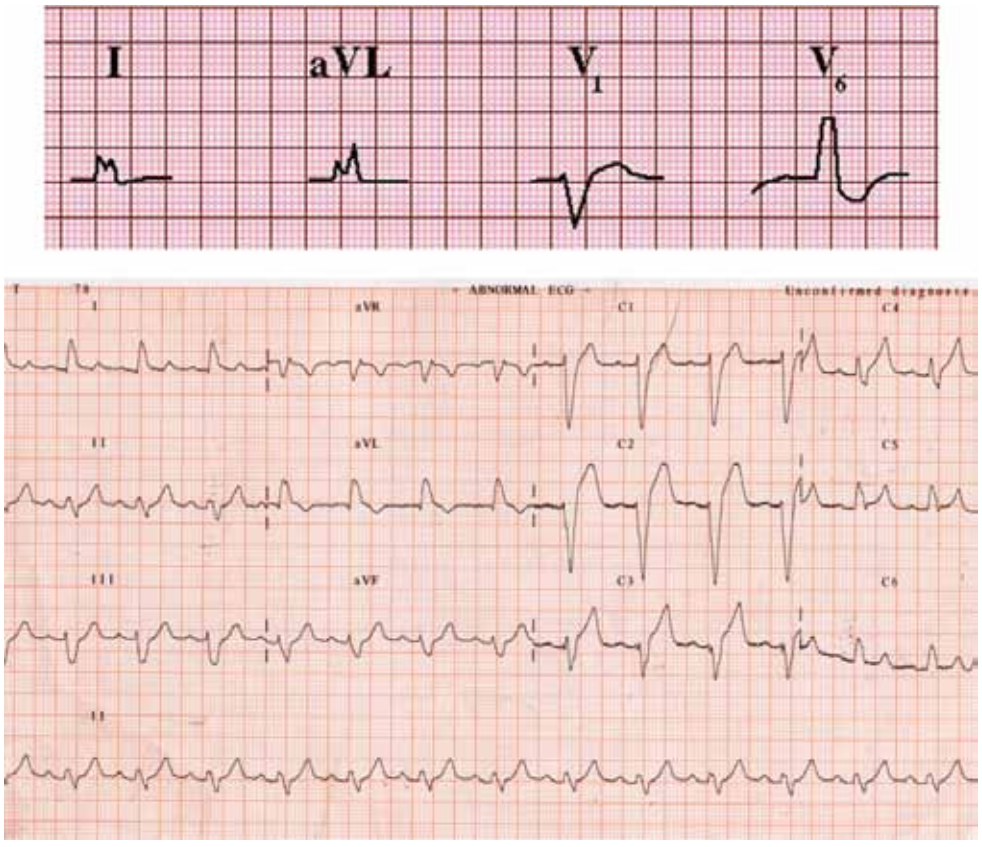

Figure 3 : Upper ECG summarizes the characteristics of LBBB in key leads as described in text. Lower ECG is a 12 lead ECG of LBBB in sinus rhythm.

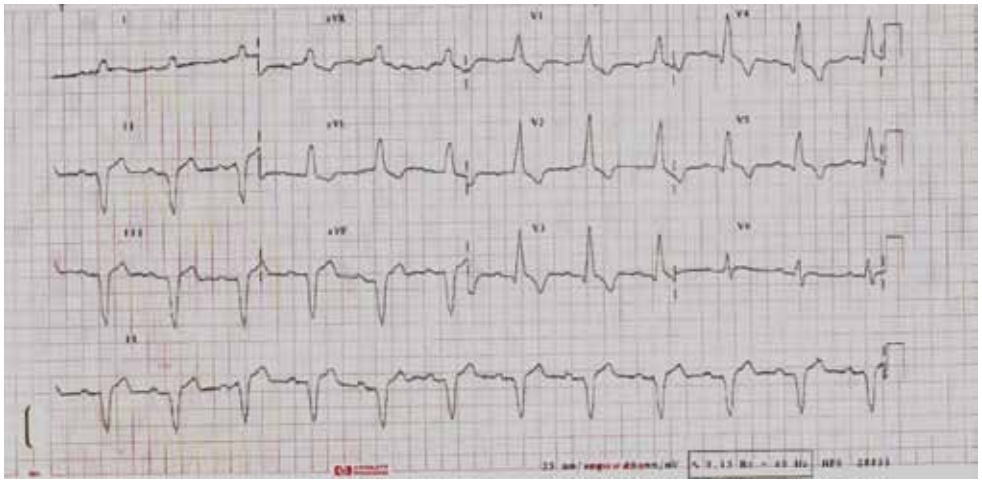

Figure 4 : ECG showing LBBB masquerading as RBBB in a patient with dilated cardiomyopathy.

avL, and V6). The changes can be summarized as follows.

1. Loss of the normal q waves in I, avL, and V6, and sometimes the appearance of a QS complex in V1 and V2.
2. A large, positive, and widened R wave without q or s waves in I, aVL, and V6.
3. Prolongation of the duration of the QRS complex to 0.12 sec or more. The duration is between 0.14 and 0.18 sec in most cases, but may be as long as 0.20 sec.
4. The mean electrical axis will be normal or somewhat to the left.
5. The altered activation sequence will also change the sequence of repolarization. Both the TQ-ST segment and T wave vectors are directed opposite to the QRS complex.

Incomplete left bundle branch block — Incomplete LBBB is characterized by a QRS duration of 0.10 to 0.12 sec; a diminutive or absent q in I and V6 that is frequently replaced by a slurred initial upstroke (pseudo-delta wave); a QRS morphology reminiscent of complete LBBB; a delayed intrinsic deflection (time from beginning of QRS to its maximal amplitude in V6); and usually increased voltage. Of interest, canine studies suggest that incomplete LBBB may be a form of left septal (median) fascicular block [17] .

Electrocardiogram in Variant Left Bundle Branch Block —

LBBB masquerading as right bundle branch block: - The major characteristic of variant LBBB is a superior and posterior displacement of the QRS loop with a terminal, often rightward, conduction delay similar to that in right bundle branch block. This results in marked left axis deviation and, due to the late rightward force, a terminal negativity (S wave) in V6.

Common LBBB is usually associated with a normal axis or only mild left axis deviation. Marked left axis deviation in LBBB suggests additional disease or concurrent left anterior fascicular block [18,19] .

A less frequent variant of LBBB is associated with right axis deviation, a combination that suggests dilated or congestive cardiomyopathy with biventricular enlargement [20,21] . Of interest, the right axis deviation is often episodic and on occasion is elicited by atrial premature beats as a rare form of

functional aberration [21].

LBBB in Disease

LBBB and Ventricular Hypertrophy

LBBB and LVH

- Echocardiography has shown that LVH is present in some patients with LBBB. However, the diagnosis of LVH can only be established by echocardiography in this setting; it cannot be made by ECG, since the two disorders produce similar changes.

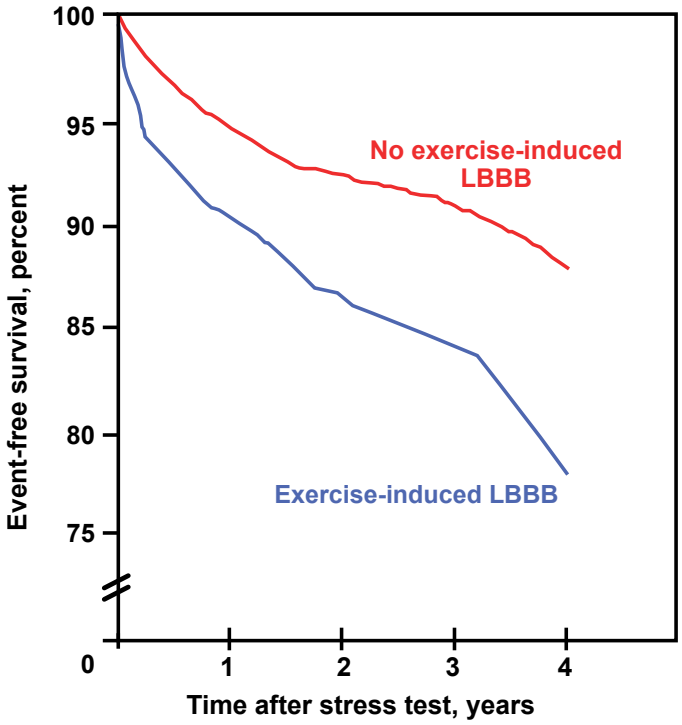

Figure 5: Among 17,277 patients who underwent exercise stress testing, the 40 patients who developed left bundle branch block (LBBB) during exercise testing had a significant reduction in cardiac event-free survival during follow-up. [22]

LBBB and RVH

Right ventricular hypertrophy may cause the leftward directed initial vector characteristic of LBBB to shift to the right, resulting in "pseudo-normalization" of the initial vector and the reappearance of q waves in I, aVL and V6. Echocardiography can be used to determine both ventricular thickness and function in this setting.

Coronary Artery Disease and Stress Testing in LBBB

Effect on stress testing — LBBB masks the ability to identify ischemia during exercise because of the associated ST and T wave abnormalities; as a result, exercise perfusion imaging or echocardiography is usually preferred when such patients are evaluated for ischemia.

Exercise-induced LBBB — Transient, exercise induced LBBB occurs in approximately 0.5 percent of exercise stress tests. One study analyzed the data of 17,277 patients undergoing exercise

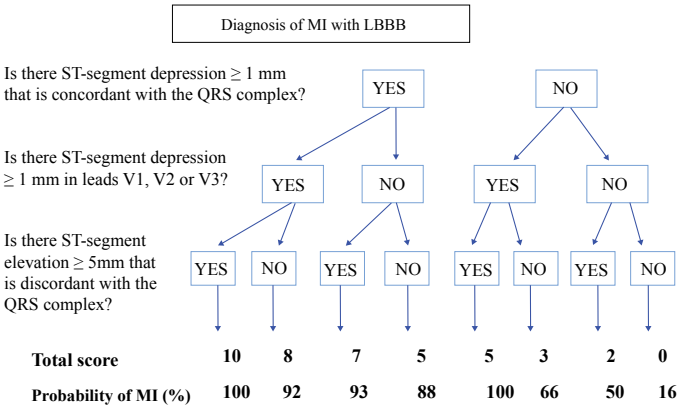

Figure 6 : Sgarbossa criteria for detecting acute myocardial infarction in presence of LBBB [24].

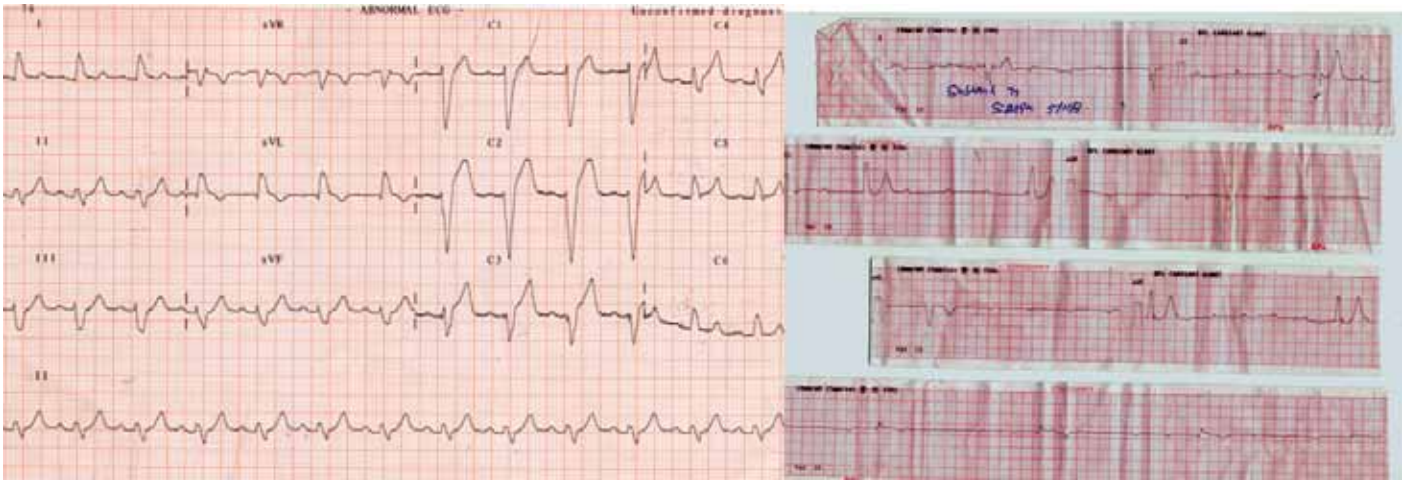

Figure 7 : A 64 year old gentleman who otherwise was thoroughly investigated for syncope had baseline LBBB. He presented with syncope and ECG during the episode was found to have complete heart block

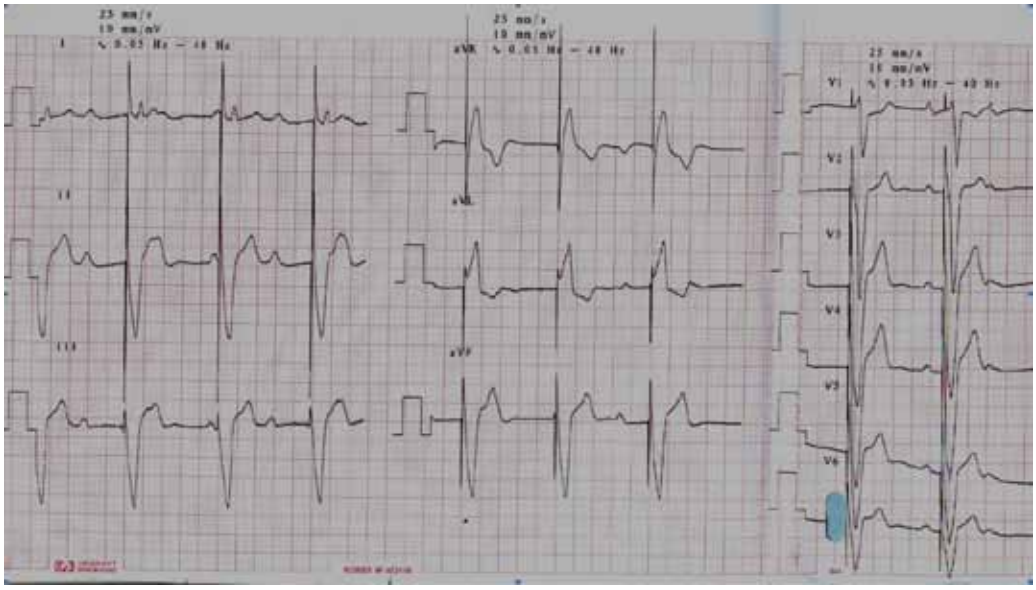

Figure 8 : Patient with single chamber pacemaker for complete heart block and pacing in unipolar mode, showing presence of LBBB morphology of QRS complex.

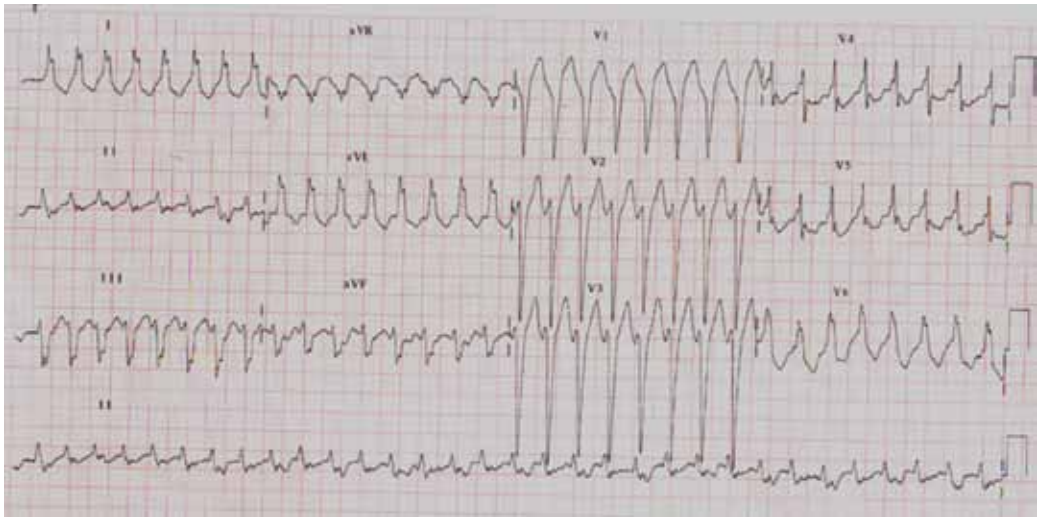

Figure 9 : 12 Lead ECG during tachycardia showing LBBB morphology.

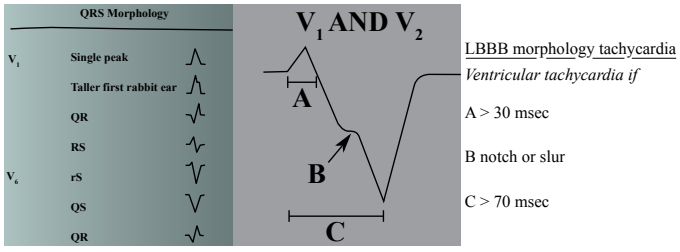

Figure 10 : QRS morphology suggesting Ventricular tachycardia.

test among whom 70 developed exercise-induced LBBB. They reported that exercise induced LBBB was an independent predictor of death and major cardiac events [22]. The four year cumulative event rate was 19 and 10 percent, respectively, for those with or without exercise-induced LBBB (relative risk 2.78) as shown in figure 5.

Diagnosis of acute myocardial infarction in common LBBB — LBBB also complicates and often prevents the electrocardiographic diagnosis of acute myocardial infarction. Figure 6 gives an algorithm for diagnosing acute myocardial infarction based on Sgarbossa criteria [24].

LBBB on ECG in Patients Having Syncope

Bradycardia: - In presence of syncope and with ECG presence of LBBB with normal or prolonged PR interval or in the presence of significant left or right axis deviation, patients need to be appropriately investigated for the presence of AV nodal or infra-nodal disease. Many of these patients may have intermittent complete heart block causing symptoms.

Tachycardia: - Patients who also complain of palpitations during the event may have a bundle branch reentrant tachycardia, especially when associated with cardiomyopathy.

24 hour Holter, Electrophysiology study or an event recorder may be needed for accurate diagnosis.

LBBB and Pacing

Right ventricular paced rhythm demonstrates LBBB morphology majority of the times (Figure 8). Pacing induced LBBB and wide QRS has been often considered as a source of concern for developing LV dysfunction in pacemaker dependant patients [25,26]. RV apical pacing typically shows QS or rS complexes in the precordial leads as shown in figure 8.

Patients with isolated LBBB are generally asymptomatic and do not require a pacemaker. However, this recommendation may change if syncope occurs, particularly if it reflects the concurrent presence of other conduction disturbances, such as third degree or type II second degree AV block or block in other fascicles.

A temporary pacemaker may be used for the patient undergoing right-sided cardiac catheterization because of the potential for transient complete heart block. Temporary pacing is also indicated for a LBBB, particularly when a first degree AV block is also present, in association with an acute myocardial infarction.

Biventricular pacing in patients with heart failure has been discussed under the next heading.

Impaired Left Ventricular Function and Heart Failure

LBBB is associated with asynchronous left ventricular activation, which reduces the efficiency of left ventricular contraction. Isolated LBBB is associated with dyssynchronous contraction and a significantly lower left ventricular ejection fraction than the controls (54 versus 62 percent). [27] The ventricular dyssynchrony associated with LBBB can modestly lower the left ventricular ejection fraction in patients with isolated conduction system disease [27]. The adverse effect of ventricular dyssynchrony is more important in the presence of heart failure.

LBBB is an independent risk factor for mortality in patients with heart failure, with increases in all-cause mortality and sudden death at one year [28]. The worse outcome is at least in part related to the association between LBBB and worsening hemodynamics due to asynchronous left ventricular activation [29,30]. This observation provides the rationale for the use of biventricular pacing in patients with heart failure who have an intraventricular conduction delay, primarily due to LBBB.

Biventricular pacing is recommended in patients having LBBB with QRS duration of $\geq$ 120 msec, left ventricular dysfunction with an ejection fraction of $\leq$ 35%, dilated left ventricular chambers who are NYHA functional class III or IV despite optimal medical therapy.[31] Biventricular pacing has been extensively evaluated in such patients and has proven to decrease both morbidity, including admissions for worsening heart failure and all cause mortality primarily resulting from reduction in heart failure related deaths [32]. Details regarding biventricular pacing are discussed elsewhere in this issue of the journal.

Patients Undergoing Noncardiac Surgery

In patients undergoing non-cardiac surgery, the presence of a LBBB without any structural heart disease is not associated with an increase in postoperative cardiac complications [33].

LBBB and Tachycardia

Many tachycardia's present with a LBBB morphology (Figure 9)

Differential diagnoses of LBBB tachycardia are as follows

1. SVT
    a. SVT with aberrancy (rate related or with baseline LBBB)
    b. Antidromic tachycardia through accessory pathway.
    c. Mahaim fiber related tachycardia.
2. VT
    a. Idiopathic Ventricular Tachycardia (RV outflow or inflow related tract Tachycardia)
    b. Arrhythmogenic Right Ventricular Dysplasia related tachycardia
    c. Bundle Branch Reentrant Tachycardia
    d. Scar Ventricular tachycardia (post ischemia or cardiomyopathy)

Presence of structural heart disease either in form of left or right ventricular dysfunction or aneurysm suggests likelihood of VT. Presence of signs of AV dissociation, absence of rS complex in any precordial lead, initial R in aVR, presence of typical QRS morphology in V1 and V6 as shown in figure 10 and initial slur in 1st 40 msec of the QRS when is slow as compared to the terminal slur in last 40 msec of the QRS complex, all point towards presence of ventricular tachycardia.

LBBB with inferior axis suggests idiopathic ventricular tachycardia. Initial sharp and relatively narrow r in lead V1 is more often associated with SVT with aberrancy or Mahaim fiber related tachycardia. Various algorithms including Brugada algorithm help to differentiate the SVT form VT. Baseline ECG in sinus rhythm if shows presence of LBBB, preexcitation, epsilon waves, PVC's of similar morphology as the tachycardia help to point towards the etiology. However sometimes electrophysiology study is warranted to diagnose the same.

Conclusion

LBBB is an infrequent finding in young healthy people, however, is not infrequently associated with structural heart disease. Presence of LBBB causes difficulty in assessing coronary artery disease and ventricular hypertrophy on ECG. In presence of structural heart disease, LBBB is associated with increased cardiac morbidity and mortality. LBBB in presence of heart failure and LV dysfunction increases the ventricular dyssynchrony and increases heart failure symptoms which can be corrected by biventricular pacing. Presence of LBBB in patients having syncope should possibly point towards presence of arrhythmia as a cause and should warrant further investigations.

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CRT: How useful is it in different subgroups?

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Congestive heart failure (CHF) is a major cause of mortality and mortality all over the world and India is no exception. In the United States, the prevalence of CHF is about 5 million and in the countries falling under the European Society of Cardiology, about 15 million patients suffer from this disease(1). It is estimated that a total of 1.2 million people in India suffer from CHF.

Significant strides have been made in understanding and treatment of coronary artery disease. Early revascularization has decreased the incidence of CHF in the Unites States as well as in Western Europe. However, the same is not true for the Indian subcontinent where various logistical issues have prevented widespread adoption of primary angioplasty for acute myocardial infarction. In addition, optimal treatment of CHF is marred by lack of resources and education among the primary care providers. Furthermore, heart transplant programs in India are in the infancy stage and hence few options are available to patients with end stage CHF.

Cardiac resynchronization therapy (CRT) is one of the therapies available in India for patients with end stage CHF. Based on multiple clinical trials over the past decade, the ACC/ AHA guidelines recommend this therapy for patients with EF ≤35%, NYHA Class III-IV, and QRS width greater than 120ms. These guidelines appear sweeping since they do not differentiate between LBBB and RBBB! Moreover, this therapy is available at a very high cost which is almost prohibitive in a large proportion of patients. The lack of wide spread adoption of third party payer system makes it difficult to offer this therapy to all patients who meet the clinical trials criteria for implantation of CRT. Hence, physicians are compelled to select patients with a very high likelihood of responding to this therapy and hence patient selection on the basis of available subgroup analysis becomes relevant. This review analyses the data from various randomized clinical trials as well as other studies to identify patients with high likelihood of responding to CRT. This will help recommend this expensive therapy to potential responders based on clinical evidence from various studies

Table 1 : Characteristics of Patients Included in Trials of CRT

	n	QRS, ms
MUSTIC	67	176 ±19
PATH-CHF	36	175 ±32
MIRACLE	4153	166 ±20
MIRACLE-ICD (severe)	369	164 ±22
CONTAK	490	158 ±26
COMPANION	1520	160
CARE-HF	813	160

QRS width

QRS width has been used as a marker of mechanical dysynchrony. Majority of the trials have used a QRS width ≥ 120 ms as inclusion criteria. We assess the data for benefit of CRT in patients with various QRS width.

The Cardiac Resynchronization Therapy in Patients with Heart Failure and Narrow QRS (RethinQ) trial was a double-blinded randomized study to assess the efficacy of CRT in patients with a standard indication for an implantable cardioverter–defibrillator with an ejection fraction of ≤ 35%, NHYA class III heart failure, a QRS interval <130 msec, and evidence of mechanical dyssynchrony (defined as an opposing- wall delay of ≥65 msec on tissue Doppler imaging or a mechanical dyssynchrony in the septal-to-posterior wall of ≥130 msec on M-mode). From among these 172 patients: 87 were randomly assigned to the CRT group and 85 to the control group. At 6 months, the CRT group and the control group did not differ significantly in change in peak oxygen consumption, mortality, quality-of-life scores, 6-minute walk test, or echocardiographic measures. At 6 months, cumulative survival was 94.2% (95% CI, 86.7 to 97.6) in the CRT group and 98.8% (95% CI, 91.9 to 99.8) in the control group (P = 0.11); cumulative freedom from death caused by worsening heart failure was 97.7% (95% CI, 91.1 to 99.4) in the CRT group and 98.8% (95% CI, 91.9 to 99.8) in the control group (P = 0.58). In a subgroup analysis, the peak oxygen consumption improved among patients with QRS >120ms but not among those with QRS <120ms (2).

Though most clinical trials showing benefit of CRT over medical management included patients with QRS width > 120msec, the median QRS duration in the COMPANION (Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure) trial was 160ms. The mean QRS duration ranged from 155 to 175 ms in the remaining trials (Table 1). This has prompted many to question the benefit of CRT in patients with an “intermediate” QRS duration between 120 and 150 ms. (3) In the CARE-HF and COMPANION trials, only those patients with QRS≥160 ms and ≥169 ms, respectively, experienced a significant risk reduction (4,5) Patients whose QRS was < 147 ms demonstrated essentially no benefit of treatment, whereas CRT had an intermediate effect in the patients whose QRS duration fell between these groups. Thus, it appears that the wider the QRS, more substantial the benefit from QRS.

Use of echocardiographic evidence of dyssynchrony in the presence of wide QRS:

Mechanical dyssynchrony may be assessed using conventional M-mode and Doppler echocardiography. Newer modalities include tissue Doppler imaging (TDI), tissue synchronization imaging (TSI), triplane TDI, real-time 3-dimensional echocardiography (RT3DE), strain rate imaging (SRI), and speckle tracking strain. Interventricular mechanical delay (IVMD) is the difference in left and right ventricular pre-ejection periods (LVPEP and RVPEP, respectively), measured from QRS onset to the beginning of aortic and pulmonary Doppler velocity curves, respectively. In the CARE HF trial, patients were considered candidates for CRT it they had a QRS width >150ms or QRS interval of 120 to 149 msec in addition to meeting two of three additional criteria for dyssynchrony: an aortic pre-ejection delay of more than 140 msec, an interventricular mechanical delay of more than 40 msec, or delayed activation of the posterolateral left ventricular wall. It was noted that there was only a minor difference among patients with QRS > 160ms (Hazard ratio 0.6) as compared to those with QRS < 160 (Hazard ratio 0.72) in terms of primary end point at 3 and 18 months (5). Hence, patients in the intermediate QRS width group appear to benefit as much as those with wider QRS if they have additional evidence of mechanical dysynchrony.

Thus, one can infer from these trials that patients with NYHA III-IV, EF <35% do not benefit from CRT if their QRS width is <120 msec, they appear to benefit significantly if QRS > 160msec. For those in the intermediate group, QRS width may not be enough and may need evidence of mechanical dysynchrony before CRT can be recommended.

Atrial fibrillation:

Lack of AV synchrony and an inability to consistently biventricularly pace secondary to irregular ventricular response challenge the utility of CRT in patients with AF. None of the major randomized clinical trials included patients with AF. However, we have some evidence from limited number of trials. Molhoek et al compared the effect of CRT in 30 patients with AF with that in 30 patients in SR. All sixty patients were in NYHA functional classes III to IV HF, LVEF ≤35%, QRS interval ≥120 ms, and a LBBB. After long-term follow-up of ≥2 years, NYHA functional class, Minnesota Quality of Life score, and 6-minute walking distance improved significantly in the 2 groups after 6 months of CRT. However, the number of non-responders was greater among patients who had AF. Despite a higher percentage of non-responders among patients who had AF, the long-term survival rate was comparable between both groups. (6)

In an observational study, Gasparini et al found that only 40% to 50% of AF patients achieve > 85% biventricular capture despite the usual pharmacologic and pacing programming efforts. Only patients in AF who underwent AV nodal ablation seem to benefit by increase in LVEF and decrease in LV end-systolic volume. The others had a very poor response (≤20%) to CRT (7) In a separate prospective study, Gasparini et al. assessed the efficacy of CRT on 162 patients with chronic AF. Of these, 48 patients were rate controlled using drugs and 114 underwent AVJ ablation to achieve rate control and resynchronization therapy delivery. The results among these chronic AF patients were compared with 511 SR patients treated with CRT. They observed that both SR and AF groups showed significant and sustained improvements of all assessed parameters. However, within the AF group, only those who underwent ablation showed a significant increase of ejection fraction ($p \leq 0.001$), reverse remodeling effect ($p \leq 0.001$), and improved exercise tolerance ($p \leq 0.001$); no improvements were observed in AF patients who did not undergo ablation. This difference was sustained over a four year follow up (8).

In another study, Delnoy et al. prospectively studied 263 CRT candidates (96 patients (37%) with chronic AF and 167 patients (63%) with sinus rhythm) meeting the standard criteria for CRT implant. New York Heart Association class, 6-minute walking distance, quality-of-life score, LV ejection fraction, and mitral regurgitation improved significantly to the same degree at 3 and 12 months in both groups. Reverse LV remodeling after 3 and 12 months was 74% and 82% (AF group) versus 77% and 83%, respectively (SR group, $p \leq 0.79$). In both groups, significant decreases in annual hospitalization rate for CHF in both groups (84% and 90%) were documented. Thus, the benefit of CRT in patients with chronic AF and heart failure is similar to that in patients with SR(9).

Overall, it appears from the current evidence that though patients with AF benefit from CRT, the benefit may be substantial only if after AVJ ablation is performed to ensure biventricular pacing.

Pulmonary hypertension

A significant proportion of patients with end stage CHF suffer from pulmonary hypertension. Whether patients afflicted with increased pulmonary pressure benefit from CRT considering that some of their dyspnea may be due to pulmonary hypertension rather than mechanical dysynchrony. In a study by Stern et al., 68 subjects with standard indications for CRT were retrospectively studied over a 12-month period. The patients were stratified into two groups: those with echocardiographic estimation of pulmonary artery pressure i.e., ePASP ≥ 50 mmHg (n = 27) and those with ePASP < 50 mmHg (n = 41). Even among this ideal patient population composed of 24 women and 44 men (age, mean ± SD; 70 ± 11 years), with a decreased LVEF ([25 ± 9]%) and a wide QRS (171 ± 54 ms), it was noted those with ePASP ≥ 50 mmHg had a significantly worse combined end point of hospitalization for heart failure and all cause mortality(P = 0.02). Patients with RV dysfunction had a trend toward an increased risk for an adverse outcome, regardless of ePASP (P = 0.06) (10)

Though further studies are awaited in this patient group, it appears that one needs to have a higher threshold for implanting CRT in patients with pulmonary hypertension.

RBBB:

Though majority of clinical trials related to CRT did not define any specific QRS morphology in the inclusion criteria, an overwhelming majority of the patients included in these trials were those with LBBB. A very minor proportion of patients enrolled in these trials had RBBB morphology.

In a canine model of tachycardia induced cardiomyopathy and RF ablation induced LBBB and RBBB, it was demonstrated that less mechanical dyssynchrony is induced by RBBB than LBBB. Furthermore, the corresponding impact of CRT in those with RBBB is reduced. Right ventricular-only pacing may be equally efficacious as BiV CRT in hearts with pure right bundle branch conduction delay (11).

To accurately characterize the activation sequence during RBBB and LBBB in human hearts, RV and LV activation sequences were studied in 100 consecutive HF patients using a 3D mapping system. The six patients with RBBB showed significantly longer time to RV breakthrough ($P < 0.001$), longer activation times of RV anterior and lateral regions ($P < 0.001$), and longer total RV endocardial activation time ($P < 0.02$) compared to ninety four patients with LBBB. Time to LV breakthrough was significantly shorter in patients with RBBB ($P<0.001$), while total and regional LV endocardial activation times were not significantly different between the two**.** However, detailed assessment of the ECG among patients with RBBB demonstrated that 4 out of these 6 patients had "RBBB masking the LBBB" pattern as evident by notched QRS in I, aVL and left axis deviation (LAD). The 2 patients who did not show this pattern had a distinctly shorter LV activation time(12). In an echocardiographic study among 200 consecutive patients with CHF and QRS > 120ms, the prevalence of an interventricular mechanical delay ≥40 ms was lower in patients with pure RBBB than that in those with RBBB plus LAHB and those with LBBB (33 vs. 50 vs. 54%, $P = 0.05$). A maximal difference in peak myocardial systolic velocity among all 12 segments (Ts)>100 ms was found in 63% of the patients with LBBB, whereas it was present in 31% of the patients with pure RBBB and in 42% of those with RBBB-LFH ($P < 0.001$). Thus, it appears that those with pure RBBB have a pathophysiologic basis for suboptimal response to CRT. (13)

Among the various CRT related clinical trials, COMPANION trial results sheds some light on the utility of CRT in patients with RBBB. Univariate analysis of COMPANION Trial indicated a hazard ratio close to 1 (no benefit) for patients with RBBB compared to that of 0.55 (significant benefit) for patients with LBBB (4) Likewise, in a MIRACLE trial substudy, 313 with LBBB, 43 with RBBB, and 35 with IVCD who received CRT, were assessed. It was demonstrated that significant improvement was achieved in functional class ($p = 0.001$) by patients with RBBB, and in quality of life ($p = 0.038$) by patients with IVCD. Patients in the RBBB and IVCD groups showed improvement in exercise time and peak oxygen consumption after CRT. Interestingly, 56% of patients with RBBB had an associated left anterior fascicular block (electrical axis 81.5 ± 33º) while 26% had left posterior fascicular block (electrical axis 125 ± 29.3º) (14) However, in a recent study by Adelstein et al., only 13.6% patients with RBBB showed any improvement in their heart failure symptoms. In this study, the QRS axis (indicative of additional fascicular block) showed no difference in rate of response. (15) However, meta-analysis of 2 clinical trials assessing the effects of CRT on RBBB and IVCD have demonstrated that CRT may indeed be detrimental to these patients.

Overall, it appears that patients with pure RBBB patients may not benefit from CRT. Patients with RBBB and additional fascicular block may have delay in LV activation and thereby improve with CRT.

Mitral regurgitation:

In clinical practice, significant proportion of patients with CHF have significant mitral regurgitation (MR). CRT reduces systolic MR by 30–40%, both at rest and during exercise, and abolishes diastolic MR. Its main mechanisms are apical and outward displacement of the papillary muscles, secondary to an enlarged and a more spherical left ventricle, causing increased subvalvar traction; mitral annular dilatation; and poor contraction of the left ventricle, with a slowed rate of rise of intraventricular pressure and slow closure of the leaflets. (16-18) In a study by Ypenburg et al. 68 patients consecutive (LV ejection fraction 23±8%) with at least moderate MR (≥grade 2) underwent CRT. The authors report that the majority of patients had improved MR after CRT, with 43% improving immediately after CRT, and 20% improving late (6 months) after CRT. The authors further note that the site of latest activation in early responders was mostly inferior or posterior (adjacent to the posterior papillary muscle), whereas the lateral wall was the latest activated segment in late responders. Furthermore, among responder group, if the CRT was discontinued there was an acute recurrence of MR (16, 17).

Based on multiple clinical trials, experts have concluded that patients more likely to have a significant reduction of MR by CRT if they have moderate-to-severe MR of nonischemic etiology and intra left- ventricular dyssynchrony, involving mainly papillary muscle apparatus (high interpapillary muscle dyssynchrony)(18).

Paced rhythm

In clinical practice wee often encounter patients with permanent pacemaker dependent with low EF and moderate to severe CHF. Several studies have demonstrated beneficial effects of the upgrade from RV apical pacing to CRT in this group of patients.

Reverse remodeling of the LV (defined as a reduction in LV end-diastolic or end-systolic volume) after upgrade from RV apical to CRT has been demonstrated in several studies (19-21)). In addition, the severity of mitral regurgitation may improve after an upgrade to CRT (22). Furthermore, LV hemodynamics and mechanical function may improve after an upgrade to CRT(22). Improvement in global LVEF has also been demonstrated in various studies, including 4 prospective studies with more than 110 patients with previous AV junction ablation and pacemaker implantation (23) Finally, it has been demonstrated that the upgrade from RV apical pacing to CRT may result in a significant improvement in exercise capacity and New York Heart Association functional class (24,25) Unfortunately, at present it remains uncertain if the upgrade to CRT in previously paced patients results in an improved survival.

It has been suggested in patients with low EF who need a pacemaker, it is better to implant a CRT device than allow the patient to pace via RV apical lead. Though some studies have demonstrated that LVEF is better over time in this group of patients with CRT than with RV pacing, other studies have failed to demonstrate such a benefit. Hopefully, larger studies in the future will clarify the role of CRT, if any, in this group of patients (23).

Conclusion:

Though clinical guidelines recommend implantation of CRT devices in patients with EF ≤35%, NYHA Class III- IV, QRS width of ≥120ms, certain subgroup of patients may not derive as much benefit as others. One needs to be cognizant of whether there is data supporting various subgroups of patients. Further studies will help us better identify responders from non-responders, thereby making us more confident in recommending it in the constraint-laden clinical environment of India.

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Case Report

Severe Myocardial Depression in a Patient with Aluminium Phosphide Poisoning : A Clinical and Electrocardiographical Correlation

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Introduction

Aluminium phosphide (ALP) poisoning (Celphos) has emerged as a common cause of accidental poisoning in children with a mortality ranging from 37-100%.[1] Since ALP is commonly used as a fungicide and rodenticide in India, many reports of accidental and intentional poisoning with severe consequences have been noted both in adults and children.However, only a few cases from outside India have been reported.

The spectrum of symptoms and signs and their severity depends upon the time lag between ALP ingestion and hospitalisation. The most common presentation is shock with cold and clammy skin, a weak thready pulse and severe hypotension often refractory to vasopressors. Cardiac, neurological, gastrointestinal and renal involvement is also common and documented in many case reports. We report a case of ALP poisoning with severe myocardial depression. Serial ECGs and all cardiac events are reported.

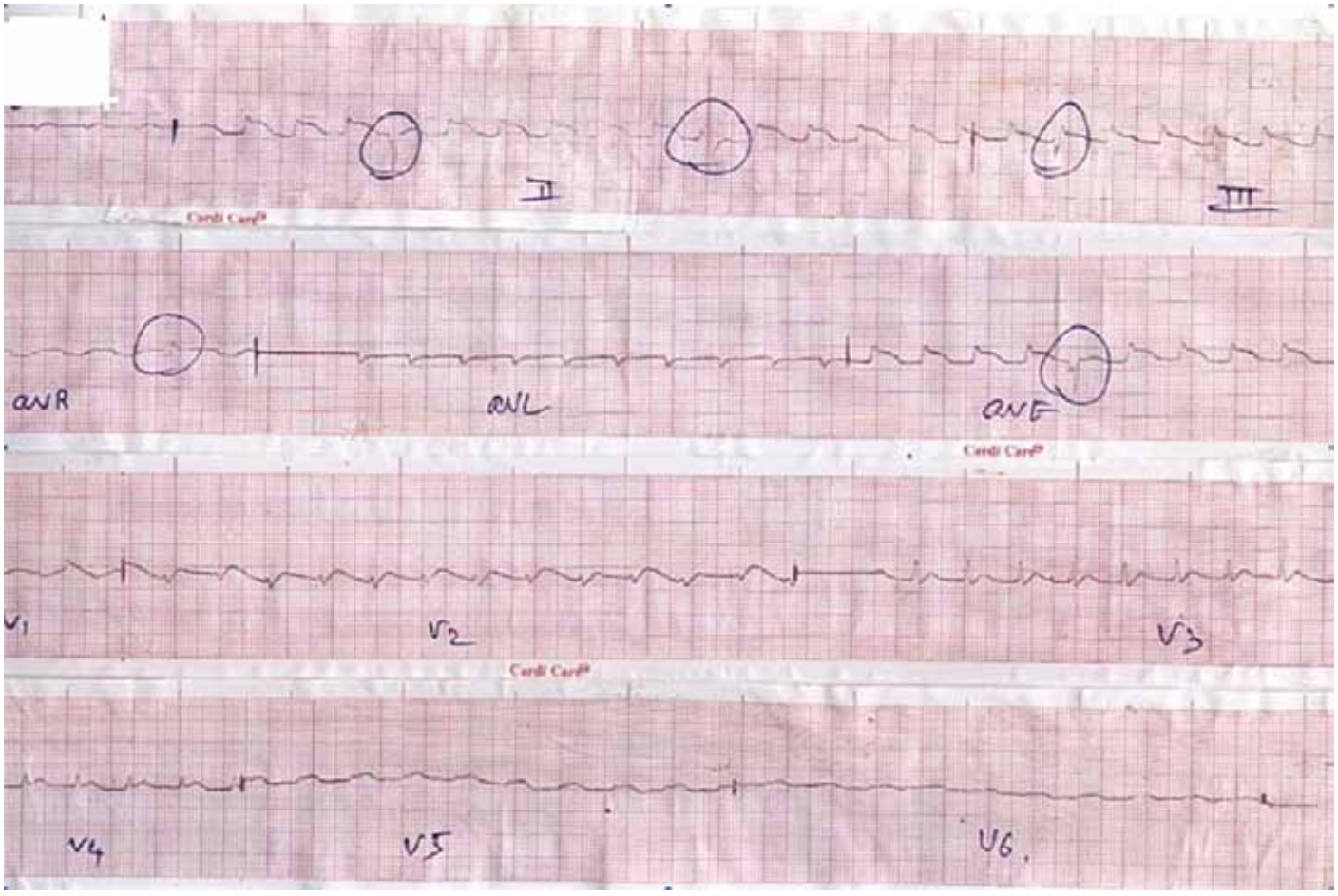

Figure 1

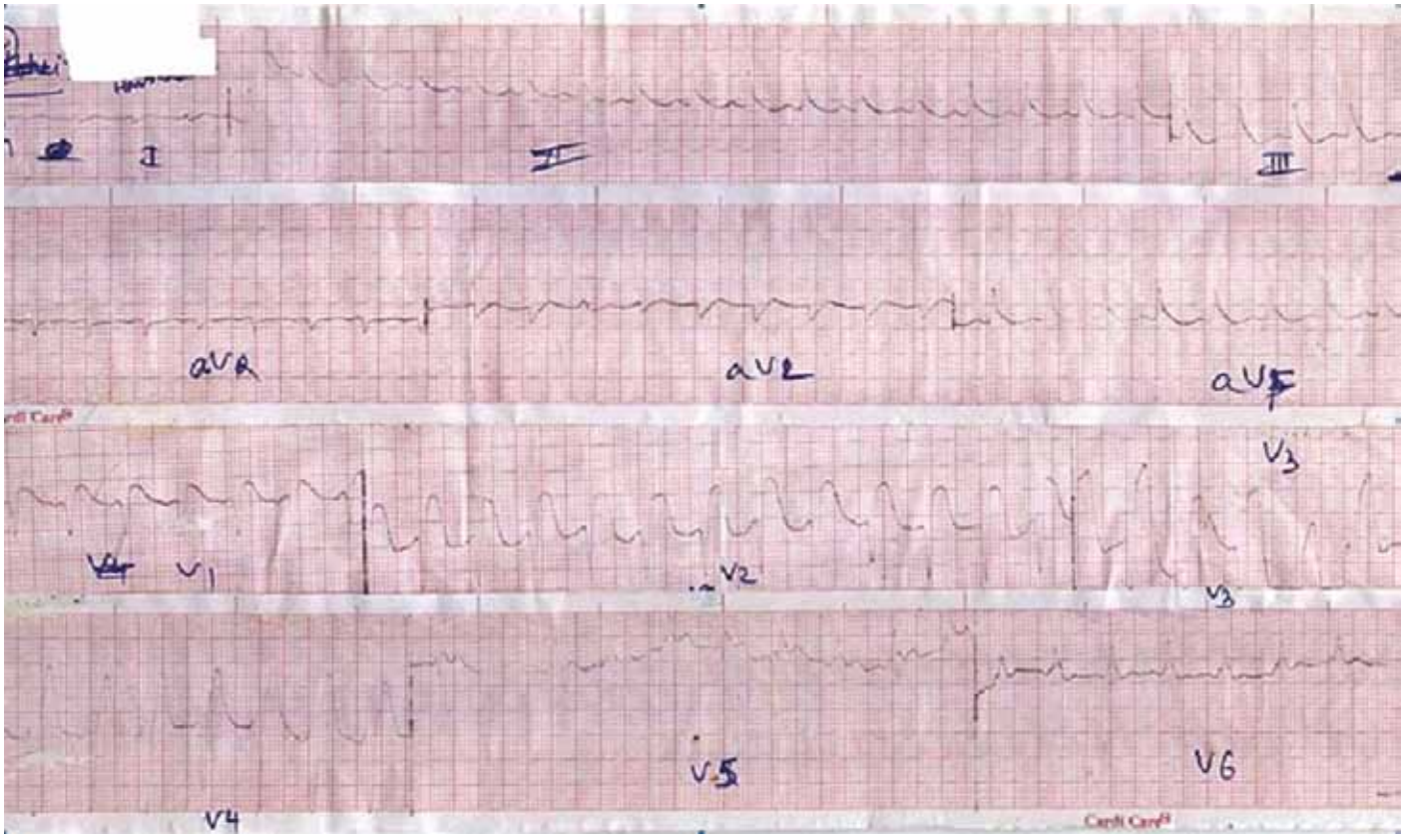

Figure 2

Case Report

A 40-year-old male patient came to the hospital 3 hours after ingestion of ALP with a suicidal intent (one tablet of Celphos - 3 gm). On arrival the patient complained of epigastric pain and tried to vomit three to four times in an attempt to remove the tablets. Though he was conscious and oriented, he appeared very unwell. His pulse rate was 110 bpm and his blood pressure was 70/30 mmHg. The oxygen saturation was 90% on room air. Examination of the respiratory system was unremarkable. . We began aggressively resuscitating the patient. Two peripheral lines for resuscitation were inserted and gastric lavage conducted with saline. Vasopressor norepinephrine was administered as per the standard dosage and one liter of normal saline was infused within one hour. On the first day, investigations showed a Hgb of 12 gm%, total white cell count of 7000/cumm. . Serum calcium was 9.2 mg/dl, serum magnesium was 2.33 mg/dl, serum sodium 143 meq/L, serum potassium 5.3 meq/L and serum bicarbonate was 20 mmol/L. Blood urea Nitrogen, serum creatinine and LFTs were normal. After two hours of gastric lavage we gave coconut oil through Ryle's tube. ECG recorded on arrival showed a broad QRS complex with ST elevation in mainly the inferior oriented leads mimicking inferior wall myocardial infarction [Figure 1]. Serum CPKMB was very high (290 U/L).

Over the next few hours the patient's blood pressure continued to fall despite being on maximum doses of norepinephrine infusion. We therefore started the patient on a dopamine infusion at 10 mcg/kg/min. Over the next six hours of aggressive supportive measures, the patient continued to deteriorate. An ECG six hours after admission showed ST elevation in the V1-V4 but some repolarization changes in II, III and aVF [Figure 2]. Electrolytes after six hours were normal except for a mild decrease in serum magnesium and hence, we started IV Magnesium therapy in the recommended dosage. On day 2, the patient was drowsy and could not maintain his saturation (80% saturation with oxygen) and therefore, the patient was intubated and ventilatory support was initiated. Blood pressure on the 2 nd day was 70/30 mm Hg and did not improve despite the maximum dose of dopamine and norepinephrine. ECG on the 2 nd day was suggestive of severe myocardial injury (ST elevation with broad QRS). Electrolytes were normal except for serum magnesium which was 3 mg/dl. Despite 48 hours of maximal resuscitative measures, the patient succumbed to myocardial depression.

Discussion

Aluminium phosphide (ALP) is used as a rodenticide and is a common agent used in suicide attempts in India. Most of the cases in India are reported from northern India. [2,3] Refractory myocardial depression from ALP toxicity is not uncommon and carries a mortality of up to 77% (37-100%).[1,4] Easy availability of the agent and lack of good antidote makes it an ideal suicidal

poison. Upon exposure to moisture, it liberates phosphine gas, which is absorbed rapidly by inhalation or through the cutaneous or enteral routes. Phosphine resembles cyanide in that it inhibits cytochrome oxidase and thereby hampers cellular oxygen utilization.[5]

The classical presentation of ALP is epigastric pain, nausea and cardiogenic shock reflected as severe refractory hypotension and is described in many case reports; [1,2,3,6,7] however, severe myocardial depression predominated in our case. There are a few case reports of survival in case of ALP poisoning when patients were treated with vegetable oils particularly with coconut oil and hence, we tried the same. [8,9]

Our patient had severe myocardial depression which could be correlated with serial ECG changes resembling myocardial infarction/ myocarditis or pericarditis. [Figure 1] in the initial hours resembles inferior wall myocardial infarction and later resembles extensive anterior wall myocardial infarction. We feel that the initially liberated phosphine may be absorbed through the stomach and diaphragm and affect the inferior wall first which rests on diaphragm and later the entire heart. ECG changes have been studied in detail in various studies [10,11,12,13] and include atrial fibrillation, supraventricular and ventricular tachycardia, ST-T changes, bundle branch blocks and AV conduction disturbances. We did not encounter any such ECG changes in our patient except ST-T changes and broad QRS complexes [Figure 1] and [Figure 2]. Broad QRS and ST-T changes along with raised cardiac marker CK-MB point to severe myocardial damage.

Controversies exist about the magnesium level and prognosis of poisoning. [14] There was no magnesium imbalance or any electrolyte disturbance seen in our patient though we administered magnesium to avoid hypomagnesemia induced arrhythmias and death.

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Case Report

Sinus Node Dysfunction in Univentricular Heart

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Introduction

Single ventricle, often referred as – double inlet or univentricular heart, is a rare cyanotic congenital heart disease, with an overall incidence of 1.1%[1]. It is almost always accompanied by abnormal great artery positions [2]. In approximately 10 to 25 percent of patients with L-Transposition of Great Vessels (L-TGA), electrocardiogram may demonstrate complete AV block.[3] This may occur spontaneously and unpredictably despite the absence of previous first or second degree AV block.[3]. However sinus node dysfunction has not been documented till date in these patients. By this case report we would like to draw attention of readers towards this rare entity seen recently in one of our patient.

Case Report

A 38 year old female presented in emergency department with complains of breathlessness on exertion & cyanosis. There was no childhood history of cyanotic spells.

On clinical examination, her pulse was 80bpm regular, blood pressure- 110/70 mmHg, respiratory rate – 20/minute, temperature 99.4 F. Jugular venous pulse showed prominent 'a' wave. There was central and peripheral cyanosis with bilateral, uniform clubbing of fingers & toes. A prominent second heart sound was palpated in pulmonary area. Apex was situated in left 5th intercostal space in mid-clavicular line. There was no parasternal lift or epigastric pulsations. On auscultation, S_1 was normal. S_2 was single & loud (A_2). A short, soft systolic murmur of grade 2/6 was present at left lower sternal edge. Chest X ray revealed reduced pulmonary vascularity, with an inverted outlet chamber forming bulge at the left upper cardiac border. Ascending aorta was lying convex and to the left. The right pulmonary hilus was prominent (waterfall appearance) (Figure-1) Electrocardiogram showed absence of regular P waves before each QRS complex and intermittent 'P'

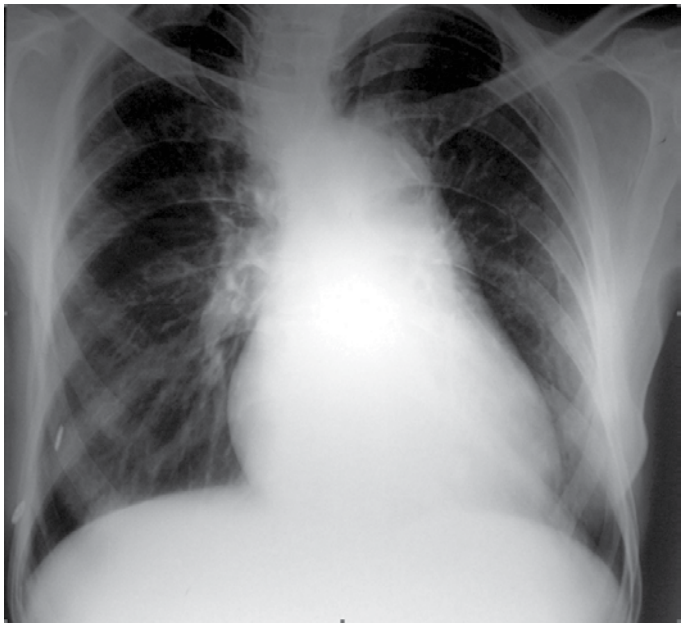

Figure 1 : Chest X-ray with an inverted outlet chamber forming bulge at the left upper cardiac border and ascending aorta lying convex and to the left.

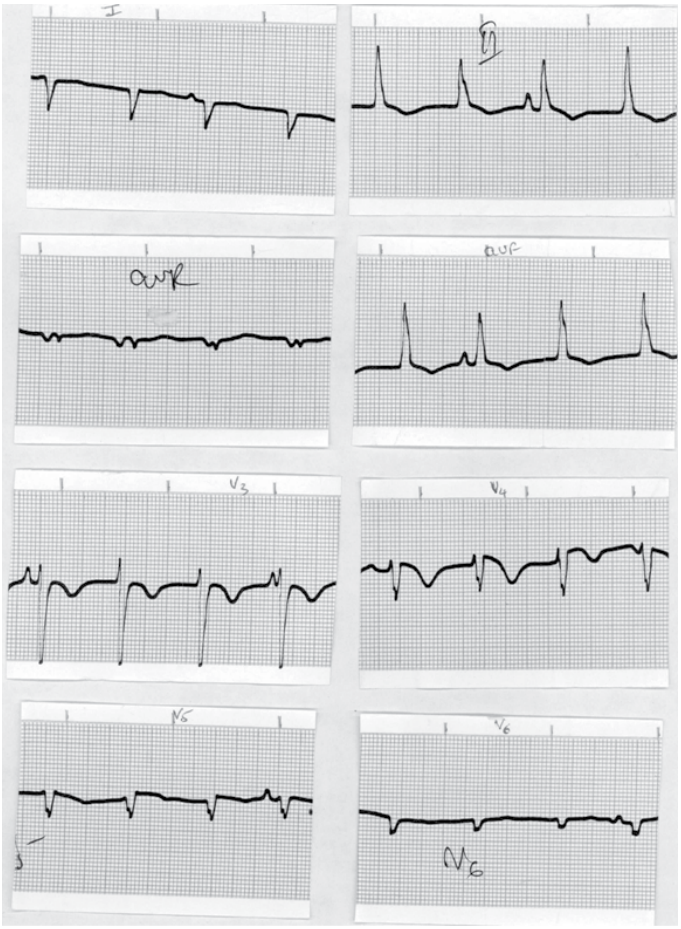

Figure 2 : ECG showing sinus node dysfunction.

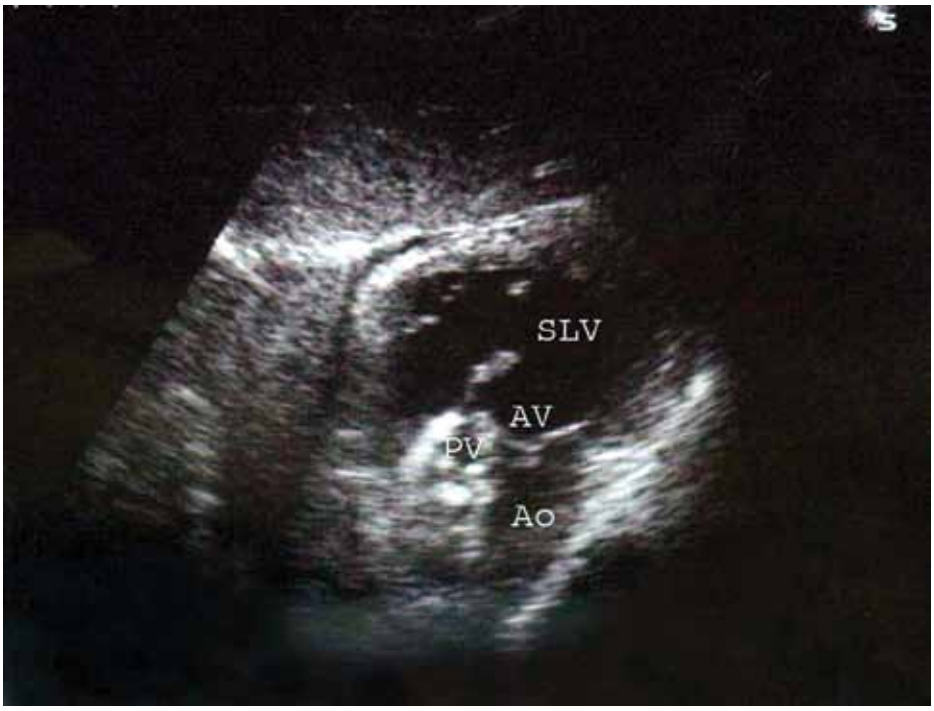

Figure 3 : Echocardiogram showing single left ventricle (SLV). Ascending Aorta (Ao) dilated and to left of pulmonary artery (PA). Pulmonary valves (PV) are thick & calcified.

waves suggestive of sinus node dysfunction (Figure-2). 2D–Echocardiography demonstrated a single left ventricle (finely trabeculated). Ascending aorta was dilated and situated to left of pulmonary artery. Pulmonary valves were thick & calcified with sub-pulmonic stenosis (gradient 70mmHg) (Figure-3). Patient was advised for corrective surgery. However she refused for it and so was discharged on medical therapy with diuretics & digoxin.

Discussion

Atrio-ventricular (AV) conduction disturbances are commonly observed in patients of L-TGA. First degree AV block occurs in about 50% and complete heart block in 10-15% of patients .[2] Presence of dual AV nodes, one posterior and one anterior with an aberrantly located penetrating portion of the His Bundle is responsible for such AV conduction disturbance [4]. However sinus node dysfunction is not reported in patients of L-TGA. It is only described following atrial baffle corrective surgery (Mustard Operation), as one of the late complication of the surgery. The incidence varies from 50% at 5 years to 64% at 10 year.[5] Loss of sinus rhythm has been recognized as predictor of sudden cardiac death during follow up[6] and such patients may also require pacemaker / ICD.[7] The cause is probably damage to sinus node /interruption of sinus node blood flow during operative surgury.[4]

However in our patient presence of sinus node dysfunction without any past history of Mustard operation was unusual and draws attention to probable some additional conductive pathology affecting SA node in these patients besides the more commonly recognized involvement of AV node.

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ECG for the Diagnosis of Pulmonary Embolism when Conventional Imaging cannot be Utilized : A Case Report and Review of the Literature

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Abstract

The diagnosis of acute pulmonary embolism has always been challenging. However, it has recently been greatly assisted through advances in radiological imaging. While imaging techniques are widely available, they cannot always be utilized. We report a case of acute pulmonary embolism in a patient with several prior pulmonary resections that would likely result in a non-diagnostic V/Q scan and acute renal insufficiency that was a relative contraindication to CT pulmonary angiography. The patient's electrocardiogram displayed several features suggestive of acute pulmonary embolism, which in the absence of effective radiological imaging, were essential in her diagnosis and management.

Key words: Pulmonary Embolism; ECG

Introduction

Interest in diagnosis of acute pulmonary embolism (PE) utilizing the electrocardiogram (ECG) has decreased since the creation of imaging techniques such as V/Q scanning and CT pulmonary angiography. While these techniques provide superior sensitivity and specificity to diagnose PE, they cannot always be utilized. A small but significant number of patients have co-morbid conditions that make these imaging techniques either contraindicated or non-diagnostic. In these circumstances, the ECG in addition to clinical acumen can be essential in directing the physician towards the diagnosis. While no isolated ECG abnormality is definitively associated with PE, certain constellations of ECG abnormalities have been shown to be reasonably specific [1-3]. In this report, we describe a case where ECG abnormalities were essential in the initial diagnosis and management of acute PE, as imaging techniques were either contraindicated or likely to be non-diagnostic.

Case

A 64-year-old female with a remote history of right lower lobectomy for stage 1B non-small cell lung cancer presents with new onset confusion three weeks post excision of a left upper lobe lung nodule, subsequently determined to be metastases from her right lung. The confusion began one week after discharge and progressed over a two-week period prior to presentation, she had no prior history of cognitive dysfunction.

On initial assessment, the patient was agitated and confused and thus unable to provide a detailed history. Her current

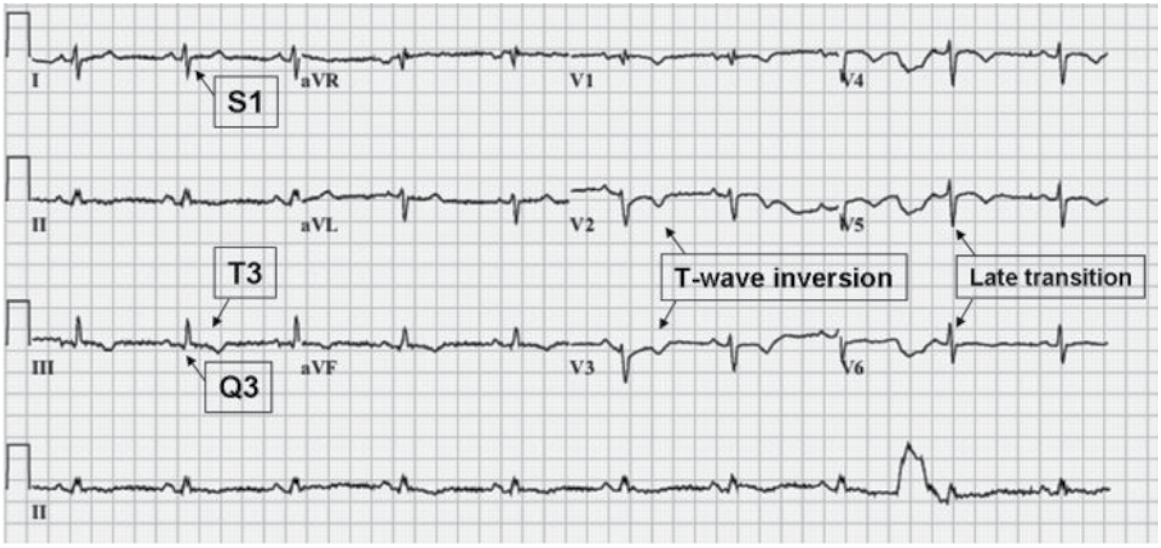

Figure 1

medications were; bisoprolol, methotrimeprazine, glatiramer acetate, trazodone, venlafaxine, controlled release morphine sulfate, lorazepam and dicolfenac/misoprostol. Physical examination showed vital signs as follows; heart rate 63 bpm, blood pressure 101/62mmHg, temperature 36ºC, respiratory rate 24 and SpO_2 94% on 5L 0_2. Cardiac exam revealed a normal S1, loud S2, no S3 or S4 and a I/IV holosystolic murmur that was more pronounced with inspiration. The meniscus of the JVP was not visible, however it was occludable, suggesting its level was above the angle of the jaw with the patient upright. No heaves, pedal edema, or pulsus paradoxus were observed. The lungs were clear with decreased air entry over the right lung base and left upper lobe consistent with prior lung resections. No focal neurological deficits were apparent and no other abnormalities were noted.

Laboratory values revealed acute renal insufficiency with serum creatinine of 149 μmol/L, up from baseline of 75μmol/L three weeks previously. Creatinine kinase and troponin I were within normal range, 84 and 0.055 respectively. The D-dimer was markedly elevated, >4.04ug FEU/ml. Blood glucose, white blood cell count and extended electrolytes were all within normal limits. Venous blood gas revealed pH 7.30, pO_2 29.3, pCO_2 52.7, HCO_3 25 on 5L 0_2. The respiratory acidosis and hypercarbia was attributed to her underlying COPD and reduced clearance of opiod metabolites, the slight HCO_3 elevation to volume contraction. The chest x-ray was not significantly changed from previous and a CT scan of the head did not reveal any acute intracranial abnormality.

Examination of the admission 12-lead ECG revealed: incomplete right bundle branch block (RBBB), right axis deviation (RAD), $S_1Q_3T_3$ pattern, T-wave inversion in leads V1-V6 and a late transition in the precordial leads (**Figure 1**).

The clinical presentation of hypoxia and relative hypotension on a background of malignancy, recent surgery and significant ECG changes were quite concerning for possible PE. The patient's confusion, while not typical of acute PE, has been recognized in the historical literature, with up to 5% displaying some form of neurological manifestation [4]. In this case the confusion was likely multifactorial being a product of hypoxia, hypercarbia and reduced renal clearance of her medications.

After initial stabilization, most treatment algorithms would advocate some form of imaging to confirm the diagnosis, usually a V/Q scan or CT pulmonary angiogram (CTPA). However, it was not possible to arrange a prompt V/Q scan, and given our patient's prior lung resections the result would likely be non-diagnostic. A CTPA would have been preferable but her acute renal insufficiency was a relative contraindication.

The use of lower limb Doppler ultrasound to help predict acute PE was considered, however the evidence to support its use is controversial, with some studies demonstrating as few as 29% of patients with confirmed PE having evidence of DVT [5]. A transesophageal echo would have been technically very difficult in this patient given her oxygen requirements and degree of agitation. A transthoracic echocardiogram (TTE) may have provided some indirect evidence of PE including signs of RV dysfunction and/or pulmonary hypertension, however, we already had strong ECG evidence for this and felt TTE would provide limited additional information.

Based on a high degree of clinical suspicion and significant ECG findings the patient was started on empiric anti-coagulation. She showed substantial improvement in her oxygen requirements and hemodynamic stability over the following 24 hour and the diagnosis of acute PE was confirmed by CTPA two days later after the resolution of her acute renal insufficiency.

Discussion

More than seven decades ago, McGinn and White described the first association between acute PE and specific ECG changes when they noted the familiar $S_1Q_3T_3$ pattern in 7 patients with acute cor pulmonale [6]. Numerous articles have been published since then describing the association between various ECG patterns and the diagnosis, severity and/or outcome of acute PE. Several of the more frequently described associations include: normal ECG, sinus tachycardia, complete and incomplete RBBB, axis changes, transition zone shift, low voltage, ST-segment and T-wave changes, $S_1Q_3T_3$ pattern, P-pulmonale and atrial arrhythmias [7,8].

The following is a brief review of the possible ECG changes associated with acute PE:

Normal ECG

A normal ECG has been reported in many studies of patients presenting with acute PE, with an incidence ranging from 9-30% [7-11]. Sreeram et al reported that the majority of patients presenting with a normal ECG maintained the rhythm throughout their hospitalization [2].

Sinus tachycardia

Sinus tachycardia is an abnormality frequently associated with acute PE, with a reported incidence of between 8%-69% [7].

Right Bundle Branch Block (RBBB)

Incomplete or complete RBBB has been associated with acute PE in a number of studies with variable incidence ranging from 6%-67% [7,8]. While this finding is felt to be fairly typical, a review by Ullman et al, estimated the overall incidence to be only 25% [8]. As well, the finding of PE associated RBBB is often transient in nature resolving within 3 months to 3 years [12]. Sorokina attributes the finding of RBBB to acute right ventricular overload and dilation, accompanied by subendocardial ischemia in the right bundle [13,14]. The appearance of a RBBB pattern has been noted to be more frequent in cases of massive trunk obstruction than peripheral

embolism [14]. This is likely a result of its greater potential to produce acute right ventricular overload.

Axis Changes

LAD, RAD and indeterminate QRS axis changes have all been associated with acute PE. While RAD has often been described as the 'classic'" axis deviation, LAD is observed more frequently [7,8]. However, this observation may be influenced by individuals with pre-existing cardiopulmonary disease. In the Urokinase-Pulmonary Embolism Trial (UPET) LAD was reported more frequently than RAD, however, when individuals with pre-existing cardiopulmonary disease were excluded, the incidence was equivalent [8,15].

Transition Zone Shift

The precordial lead in which the magnitude of R and S-waves become equivalent normally occurs at leads V3 or V4 and is often referred to as the 'transition zone'. A shift in the transition zone leftward so that the R and S-waves are equivalent at leads V5 or beyond has been reported by a number of studies [7,16] with an incidence of up to 51% reported in one study [2]. The cause of the leftward shift of the transition zone is not completely understood but has been postulated to be the result of biochemical abnormalities and hypoxia of the myocardium and His-Purkinje system slowing conduction in the RV, as well as through RV dilation itself [16]. RV dilation contributes to QRS axis shifts both anatomically and electrically, by physically altering the position of the heart in relation to the cardiac leads and by variably slowing conduction in the right bundle and RV such that the RV is responsible for greater proportion of the terminal depolarization vectors in the QRS complex. Thus, the QRS axis tends to shift rightward with a resultant left shift of the precordial transition zone.

Low Voltage

Several studies have noted low voltages, defined as greatest overall deflection of the QRS complex ≤ 5mm in all limb leads, to be associated with acute PE [1-3,9,15,17]. While the finding is frequently noted, it is quite variable with an incidence of 6-30% [3,10].

ST-segment and T-wave changes

ST-segment and T-wave changes are the most frequently noted abnormality associated with acute PE [2,7,15,18]. Non-specific ST-segment elevation and depression has been reported in up to 49% of all patients with acute PE [11]. In patients where the diagnosis of PE has been confirmed, some studies have used T-wave inversion as a diagnostic tool for predicting the severity of acute PE and likelihood of complicated course [9,19,20]. Kosuge et al showed that the rate of RV dysfunction correlated with the number of leads displaying T wave inversion [19]. In this study, all patients with T wave inversion in ≥ 7 leads had evidence of RV dysfunction on echocardiography. This was also found to be an independent predictor of in-hospital complicated events including: need for CPR, catecholamine or mechanical cardiovascular support due to hemodynamic instability.

$S_1Q_3T_3$ pattern

This 'classic' pattern is often considered the pathognomonic ECG abnormality associated with acute pulmonary embolism. However, its reported incidence in acute PE is quite variable from 10-50% and in some studies has been found to be equally likely in patients without PE [7,1].

P-pulmonale

P-pulmonale, defined as P-wave amplitude greater than 2.5 mV in lead II, has been associated with right atrial enlargement and/or hypertrophy secondary to acute PE [2,15]. It has been reported with variable frequency from 2 to 31% of patients with acute PE [7].

Atrial arrhythmias

A number of atrial arrhythmias can manifest from acute PE, these are thought to be related to increased atrial pressures and atrial enlargement [8]. Atrial fibrillation and flutter have been reported in up to 18% and 35% of acute PE patients, respectively [7]. However, other studies have reported the incidence to be as low as 0-5% [1,7,21].

Many of the ECG abnormalities associated with acute PE are thought to be the result of a right-sided heart strain pattern as well as possible myocardial ischemia at the cellular level [7]. Decreased perfusion within the lung secondary to the embolus is thought to cause a release of vasoconstrictive mediators such as serotonin and catecholamines [8,12,22]. Vasoconstriction along with mechanical obstruction from the thrombus, results in increased pulmonary artery pressures transmitted to the right heart. This increases right heart myocardial wall tension and can result in dilation of the right ventricle and right atrium, eventually resulting in contractile dysfunction and circulatory collapse.

As early as 1938, investigators attempted to explain the physiologic mechanism by which acute PE produces ECG abnormalities. Love et al. performed a series of experiments in which they mechanically obstructed the pulmonary artery (PA) producing a number of ECG changes similar to those found in clinical PE [21,23]. They noted that the ECG changes were ubiquitously preceded by RV dilation, leading them to conclude that RV dilation was responsible. While several other investigators [13,14,16] have drawn similar conclusions, it is unlikely to be the sole mechanism. Observations from other studies [2,9] have shown that RV dilation does not uniformly produce ECG abnormalities and, if they occur, there can be a significant lag time between initial RV dilation and onset of ECG abnormalities. As well, these abnormalities may persist long after the resolution of RV dilation and elevated pulmonary

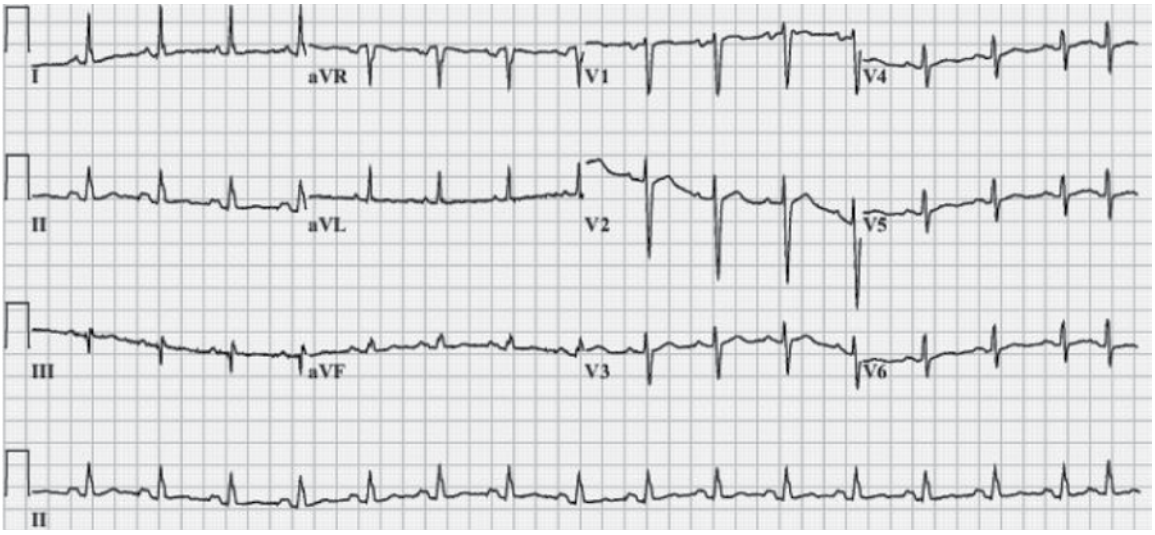

Figure 2

pressures making it difficult to suggest an isolated causal relationship [21]. As such, the physiologic explanation of the many ECG abnormalities associated with acute PE remains uncertain.

Much of the literature investigating the diagnosis of PE has focused on the limited sensitivity of the ECG. This has led many to suggest that it has little utility as a diagnostic screening tool for acute PE [7,8,10]. However, the usefulness of the ECG is not in its ability to identify all cases of PE, but in its ability to be reasonably specific given the appropriate ECG findings and clinical scenario.

While it has not been the focus of a clinical trial, it is reasonable to suggest that the specificity of the ECG is greatest when multiple abnormalities associated with PE are present concurrently and occur acutely. For instance, there are a number of disease processes that may produce a right-sided heart strain pattern over the course of months to years but there are few other than PE that are capable of doing so over the course of hours to weeks.

There have been several studies that have shown the ECG to have a high specificity to predict pulmonary hypertension and/or RV strain secondary to PE. Sreeram et al. attempted to develop prediction rules in order to aid in the ECG diagnosis of PE by identifying several features suggestive of RV strain [2]. Forty-nine patients with proven PE and without previous lung disease were studied. Pulmonary embolism was considered probable (76%) if 3 or more of the following features were present:

1. Incomplete or complete RBBB, which was associated with ST-segment elevation and positive T-wave
2. S-wave in leads I and aVL >1.5mm.
3. A shift in the transition zone in the precordial leads to V5.
4. Q-wave in leads III and aVF, but not in lead II
5. Right-axis deviation, with a frontal QRS axis.
6. A low voltage QRS complex <5 mm in the limb leads
7. T-wave inversion in leads III and aVF or leads V1 to V4

The sensitivity of Sreeram's predictive rule to detect PE has not been validated by other studies, however they have shown it to be quite specific (94.2%) [1]. A study by Punukollu et al showed that T-wave inversion in leads V1 to V3 had a specificity of 88% and diagnostic accuracy of 81% for RV dysfunction in acute PE [24]. Daniel et al. developed an ECG scoring system based on typical features of acute PE such as presence of RBBB, T-wave inversions, and additional features of right heart strain that predicted severe pulmonary hypertension (sPAP > 50mm Hg) with a specificity of 97.7% if the patient's 'ECG score' ≥10 [20]. A study by Sukhija et al analyzed the prevalence of 18 ECG abnormalities in PE [25]. A logisitic regression analysis from the study showed that the presence of 2 of the following 5 ECG abnormalities (S_1, Q_3, $S_1Q_3T_3$ pattern, sinus tachycardia or supraventricular tachyarrhythmias) was associated with a specificity of 96% and a positive predictive value of 94%.

Comparison of our patient's admission ECG (**Figure 1**) with one obtained three weeks earlier, post surgical resection (**Figure 2**) shows many characteristic changes outlined by Sreeram [2]. These include: new incomplete right bundle branch block (RBBB), right axis deviation (RAD), $S_1Q_3T_3$, T wave inversion in leads V1-V6 and transition zone shift to V5. This constellation of ECG changes also produces an ECG score ≥ 10 as outlined by Daniel et al and meets the criteria outlined by Sukhija et al producing specificities of 97.7% and 96% respectively, and a PPV of 94% [20,25]. This number of ECG changes consistent with RV strain occurring over a 3-week period would be difficult to explain by any physiological mechanism other than acute PE.

Given the likelihood that a V/Q scan would be non-diagnostic and that we were initially unable to perform a CT pulmonary angiogram secondary to our patient's renal insufficiency, the application of Sreeram's predictive rule, Daniel's ECG score and Sukhija's regression analysis was important in raising our patient's pre-test probability of PE. On the basis of her clinical presentation and characteristic ECG changes we justified the use of anticoagulation. Several days later her renal insufficiency resolved and a CTPA confirmed the diagnosis of acute PE.

Conclusion

The diagnosis of acute pulmonary embolism has always been challenging and while imaging techniques have markedly improved our diagnostic accuracy, they cannot always be utilized. This case provides an example of how the ECG is still relevant, and in certain circumstances essential, to the diagnosis of acute pulmonary embolism.

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ECG Quiz

Compiled by
Gopi Krishna Panicker, C Narasimhan, S Jayprakash, Yash Lokhandwala

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

ECG - 1

Figure 1

From wide to narrow…

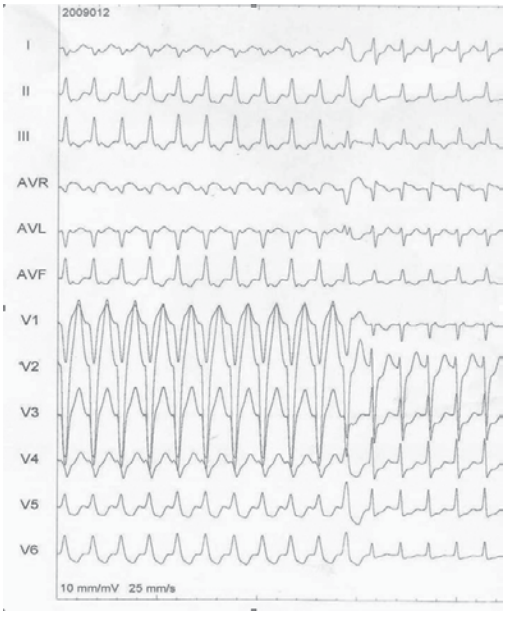
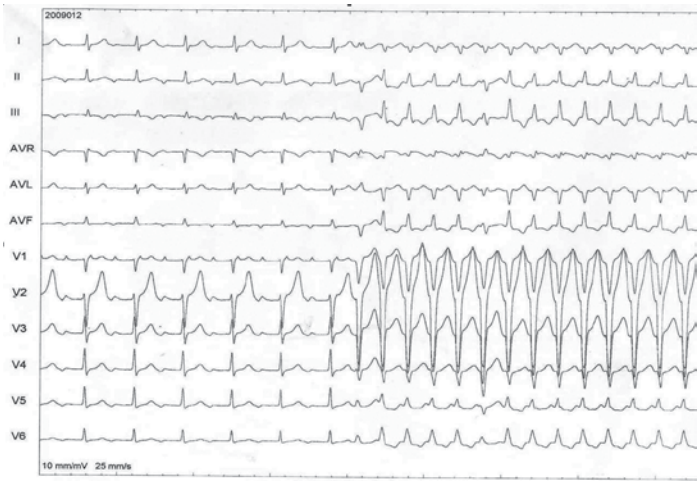


Figure 2

And back to wide…



What is the tachycardia mechanism?

a. Sinus tachycardia

b. Supraventricular tachycardia (SVT)

c. Supraventricular tachycardia and ventricular tachycardia (VT)

For correct answer see overleaf

ECG - 1

The correct answer is 'b' – Supraventricular tachycardia (SVT)

The first figure shows wide QRS tachycardia with LBBB pattern which changes to narrow QRS tachycardia. The heart rate remains exactly the same. Therefore, since the narrow QRS tachycardia is surely SVT, the wide QRS tachycardia must be SVT with LBBB. Also of note is that during the narrow QRS tachycardia, lead V1 shows a r'. The mechanism of SVT is therefore likely to be typical AVNRT. The 2nd figure shows that the initial half has a ventricular rate which is exactly half of the ventricular rate on the right side. The initial half also shows inverted P waves exactly between the two QRS complexes in the inferior leads. These P waves in lead V1 also time out perfectly with the r' in lead V1. Therefore, the mechanism here is the same SVT with 2:1 AV conduction. Later on a PVC (*) breaks the 2:1 conduction block and this is followed by 1:1 conduction during SVT. Because of a sudden doubling of HR, the left bundle is found to be refractory and therefore there is LBBB. Again, this type of pattern is classical in AVNRT with 2:1 AV conduction.

Figure 3

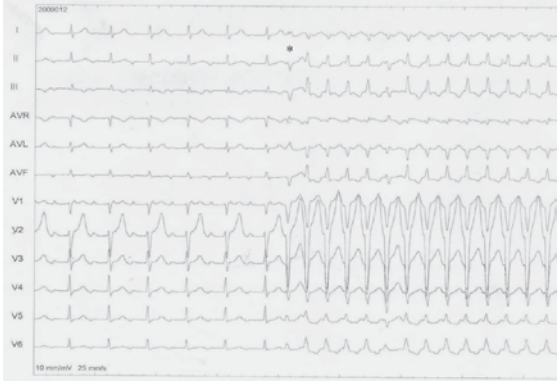
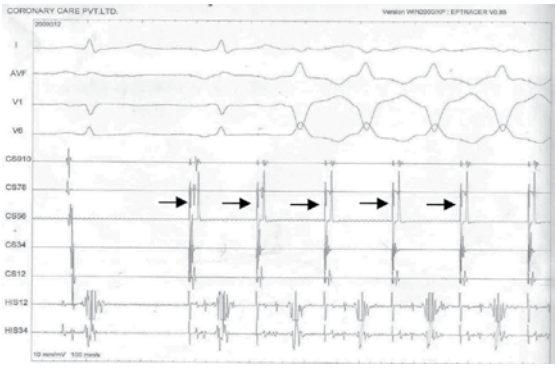


Figure 4



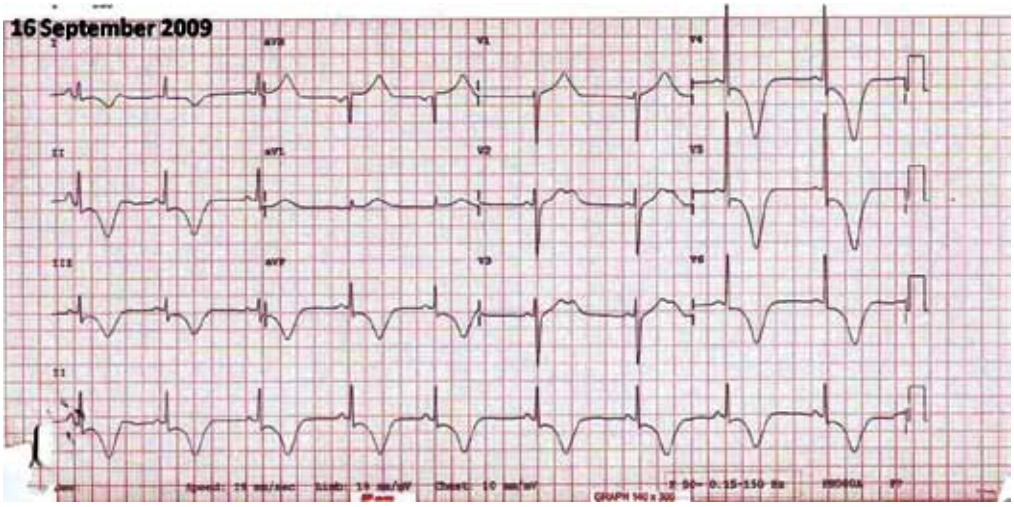
Typically, 2:1 AV conduction during AVNRT implies that the block is below the level of the AV node in the His bundle or below the His bundle. Since the 2:1 conduction spontaneously usually reverts within a few seconds to 1:1 conduction, this pattern is not seen commonly in clinical settings. This particular tracing was also recorded during an EP study.

During the EP study, rapid atrial pacing (Figure 4, arrows) also induces these LBBB. Therefore, clearly the LBBB in this case is rate-related.

ECG - 2

24 yr old man. Palpitations since several days. Recent admission, diagnosed to have SVT, terminated with diltiazem. Echo normal. What is the cause of the repolarisation abnormality?

Figure 1



What is the diagnosis?

a. Hypertrophic CMP

b. Ischemia

c. Memory sign

d. None of the above

For correct answer see overleaf

ECG - 2

The correct answer is 'c' – Memory sign

The ECG shows deep, symmetrical T wave inversion in the inferolateral leads. In fact, in leads V4 and V5, T inversion can be classified as giant (more than 10 mm). There is also mild sinus bradycardia. While such T waves can be seen in hypertrophic cardiomyopathy, they would be typically associated with voltage criteria of hypertrophy. In this ECG, neither the Estes nor the Cornell's criteria are met for hypertrophy by voltage. Also importantly the echocardiogram has been reported normal. (Though sometimes apical hypertrophy can be missed on echo).

There is nothing except the T waves to suggest ischemia. The young man did not have angina and came with paroxysmal tachycardia.

Infero-lateral T wave inversion following tachycardia has been well described many years ago as a memory sign following fascicular VT. A memory sign is seen in any condition where there is a prior wide QRS complex which later becomes narrow. Such changes can be seen with prior WPW pattern, LBBB or right ventricular pacing.

This patient subsequently underwent an EP study. During this, VT was easily induced (Figure 2). There was clearly VT as proved by the VA dissociation (Figure 3).

Fascicular VT is often misdiagnosed as SVT as in this case. The reasons for misdiagnosis are:

1. The QRS complexes are not very wide…sometimes even as less as 120 ms.

2. This tachycardia terminates with I.V. verapamil or diltiazem and

3. It occurs often in young people with normal hearts.

The memory sign lasts for a variable duration, ranging from minutes to days. In this patient, the T wave inversion disappeared after one month. Figure 3 shows the intracardiac recording during the wide QRS tachycardia.

Figure 2

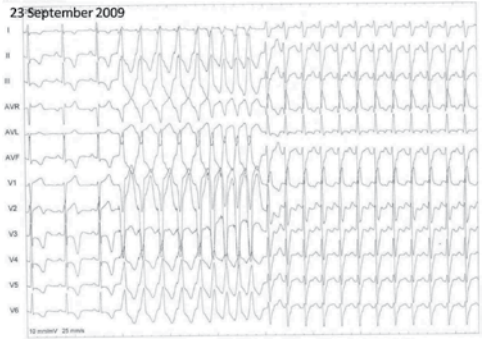
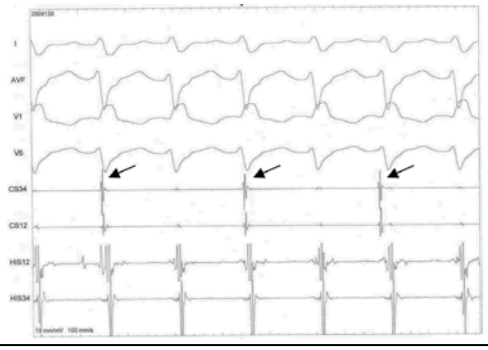


Figure 3

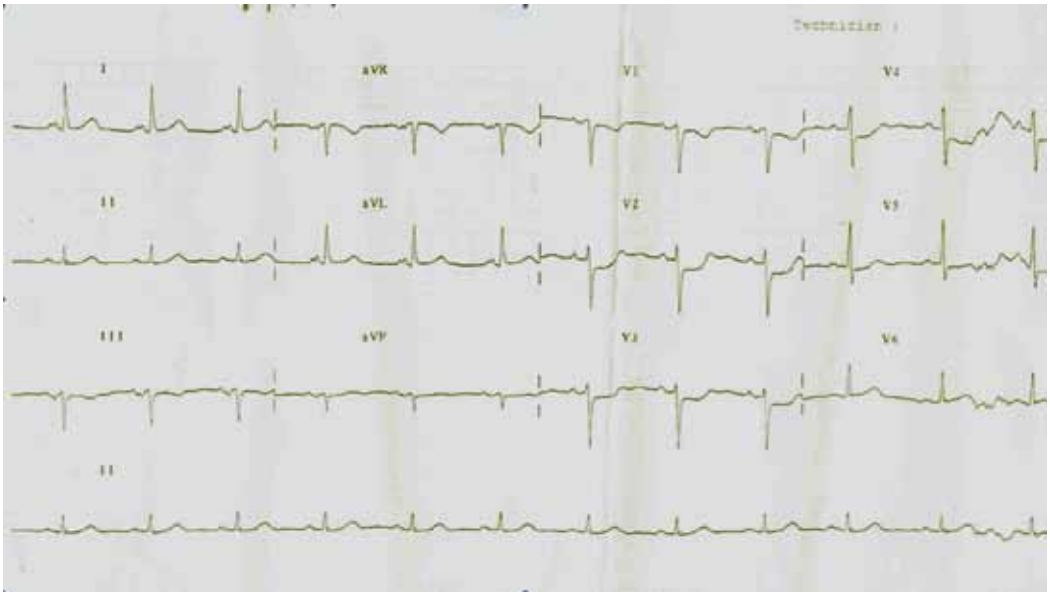


AV dissociation. The arrows point to the A waves

ECG - 3

72 yr old lady with chest discomfort for 2 hrs. What is your next step?…

Figure 1



Next Step

a. Lead V4R

b. Leads V7-V9

c. Echo

d. Troponin T

For correct answer see overleaf

ECG - 3

The correct answer is 'b' – leads V7-V9

The ECG in view of the clinical background is really suggestive of acute coronary syndrome. One sees around 2-4 mm of horizontal ST depression in leads V2-V4. The T wave is notched and the QRS axis is -20˚. The crucial decision to be made here is: are we dealing with unstable angina or acute MI'. This distinction is important because if there is STEMI, one would treat with thrombolytic therapy, where as if there is unstable angina one would treat with heparin.

Lead V4R in the absence of ST elevation in inferior leads is unlikely to be significant value in making this above distinction.

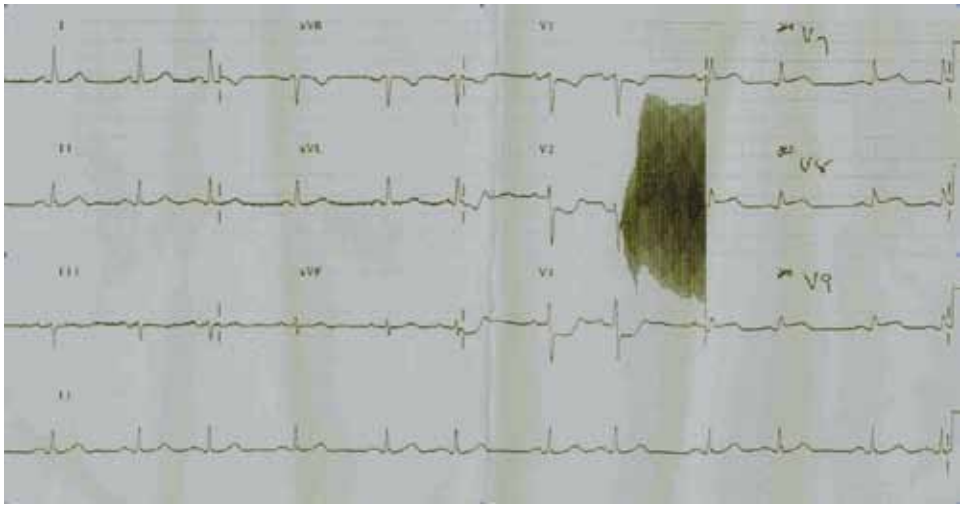
The echo could show a wall motion abnormality but again does not distinguish between unstable angina and acute MI.

The same holds true for Trop T, although it does help prognostically.

Occlusion of the posterior branches of the circumflex artery is known to not show any ST elevation in the conventional 12-lead ECG despite a large MI. In such cases, the MI process is purely on the posterior wall of the heart. This is best diagnosed in the acute setting by recording leads from the posterior wall, viz. V7-V9.

As seen in figure 2, these leads clearly show 2 mm of ST elevation… now clearly the diagnosis of STEMI and the patient received thrombolytic therapy with good results.

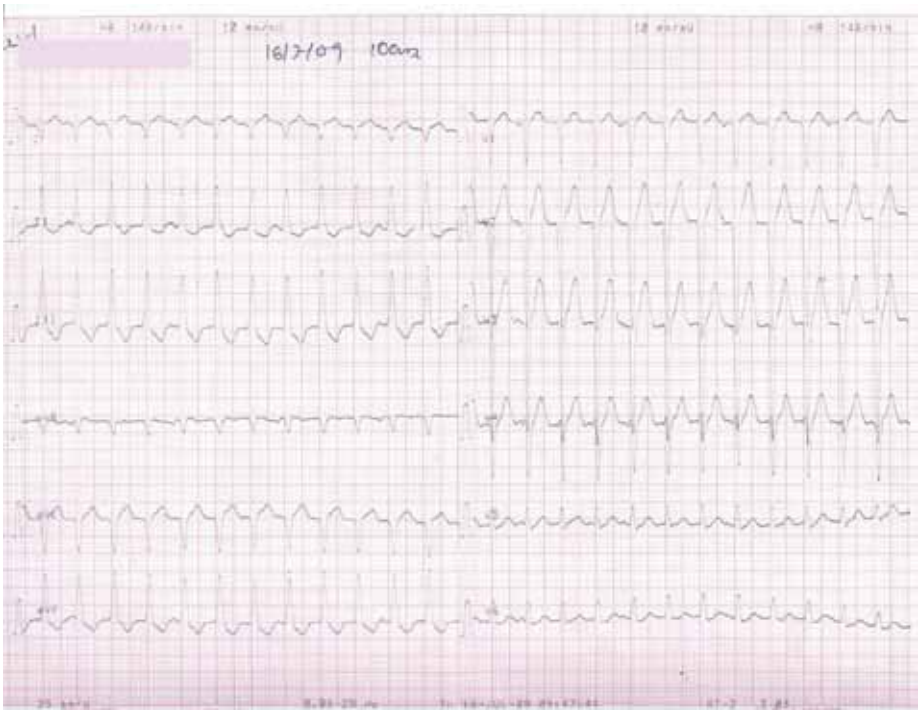
Figure 2 : Now qualifies for thrombolytic therapy!



ECG - 4

A 30 yr-old man…

Figure 1



The ECG shows:

a. PAT

b. Junctional tachycardia

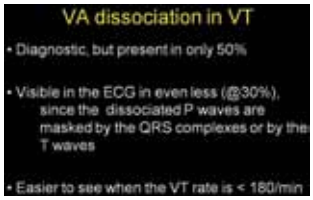
c. SVT

d. VT

For correct answer see overleaf

ECG - 4

The correct answer is 'd' – Ventricular tachycardia



The ECG shows a wide QRS tachycardia @ 148 bpm. While QRS width is not best appreciated in all the leads, if one looks carefully at lead V2 one sees that the QRS is 120 ms, which is the cutoff for a wide QRS tachycardia. The QRS pattern in precordial lead suggests an LBBB type of morphology in V1. However, in the limb leads there is RAD with QRS axis of 120˚. LBBB with RAD is almost invariably VT. The clincher in this case is the presence of AV dissociation, best seen in lead II (Figure 2). In sinus rhythm, the ECG showed similar morphology PVCs as the VT (Figure 3).

Figure 2

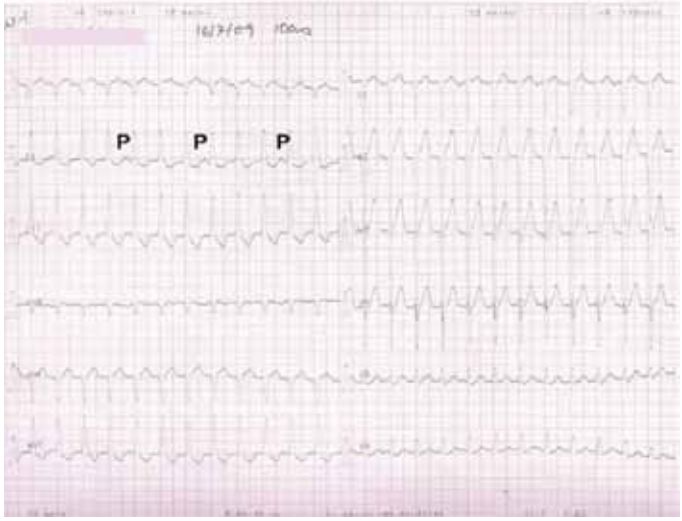
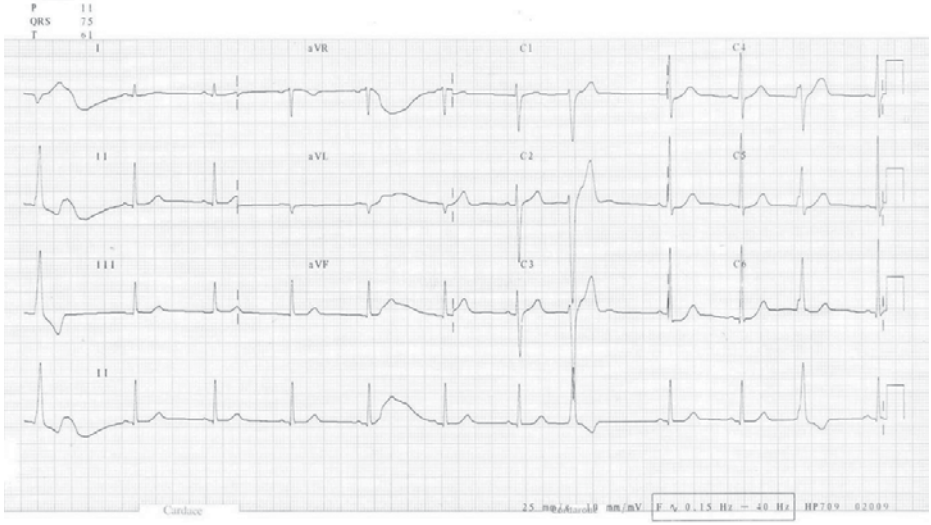


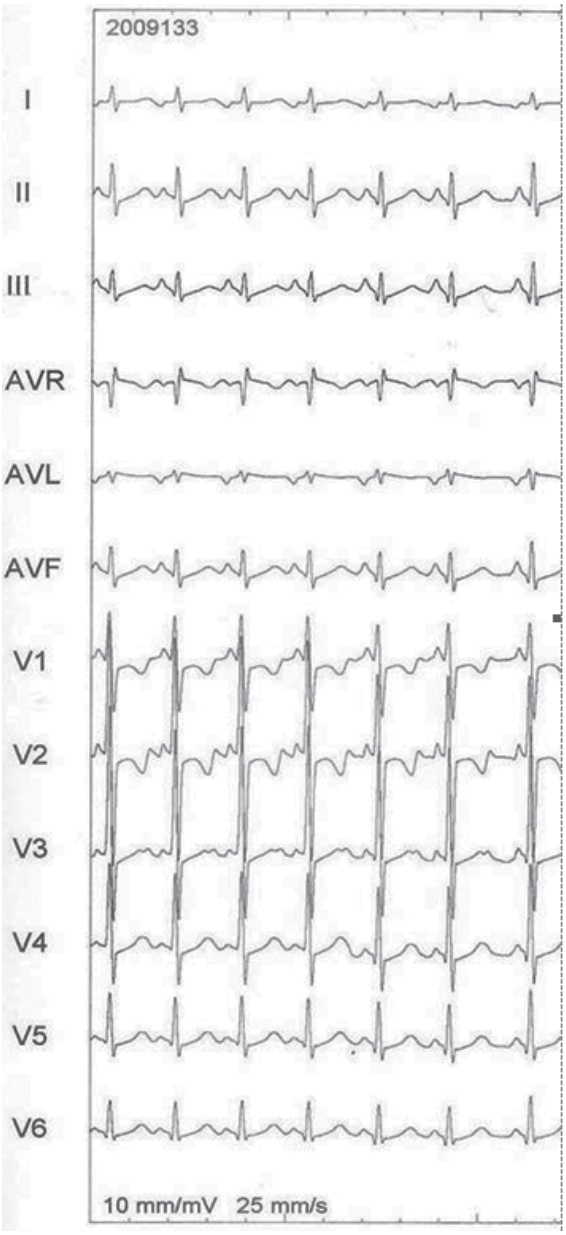
Figure 3 Similar PVCs in sinus rhythm



ECG - 5

12 yr. old boy. Palpitations noted several weeks. Mild LV dysfunction.

Figure 1



What is the diagnosis?

a. Inappropriate sinus tachycardia

b. Myocarditis

c. Atrial tachycardia

For correct answer see overleaf

ECG - 5

The correct answer is 'c' – Atrial tachycardia

The ECG shows a regular narrow QRS tachycardia with the HR being about 150 bpm. The clinical profile suggests that there has been incessant tachycardia. The LV dysfunction could have followed several weeks of tachycardia but of this we cannot be sure.

Myocarditis could have a similar clinical presentation but one would expect sinus tachycardia accompanying heart failure. This child had only mild LV dysfunction and crucially, P waves are negative in lead I. Sinus P waves will be, by rule, positive in lead I in addition to being positive in lead II and V4-V6. Therefore, this is clearly an incessant atrial tachycardia. The negative P wave in lead I and the positive P waves in inferior leads along with the positive P wave in lead V1 suggests that the site of origin of atrial tachycardia is high in the left atrium.

Figure 2 shows that this tachycardia is terminated during ablation. If one compares now the P waves in sinus rhythm from the 8th complex onwards, one sees that it has clearly become positive in lead I.

Figure 3 shows that this atrial tachycardia was arising near the origin of the left superior pulmonary vein. Over the period of the next 3 weeks, the LV dysfunction totally disappeared.

Figure 2 : During RF ablation…

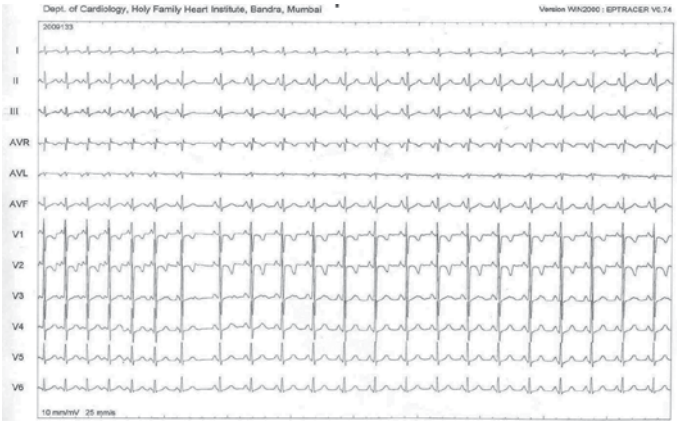
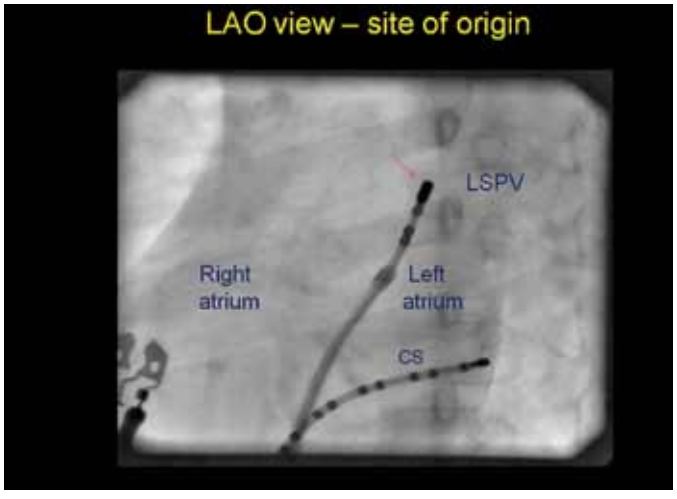


Figure 3



ECG - 6

56 yr old lady with easy fatiguability

Figure 1

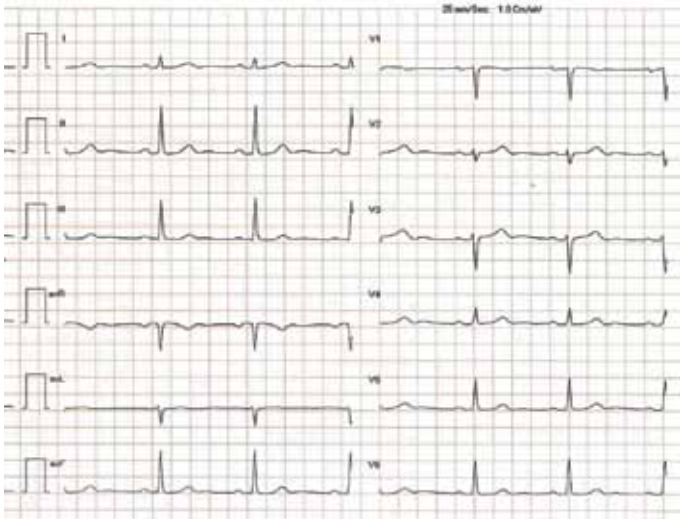
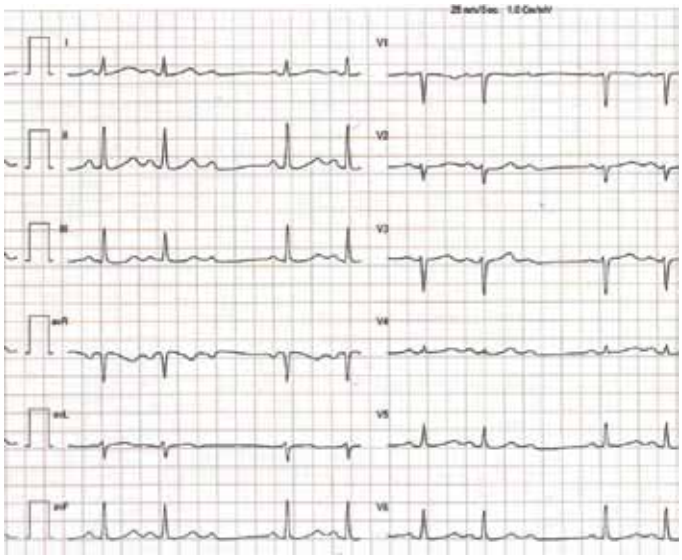


Figure 2 : During stress test…



What is the diagnosis?

a. Sinus pauses

b. AV nodal block

c. Intra-His block

d. Infra-His block

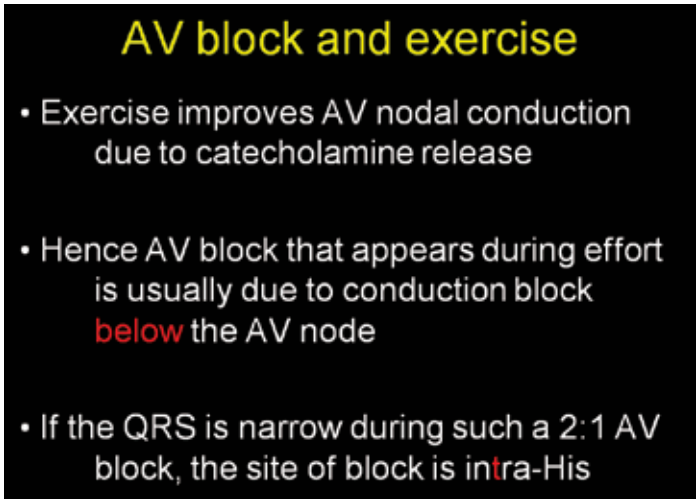
For correct answer see overleaf

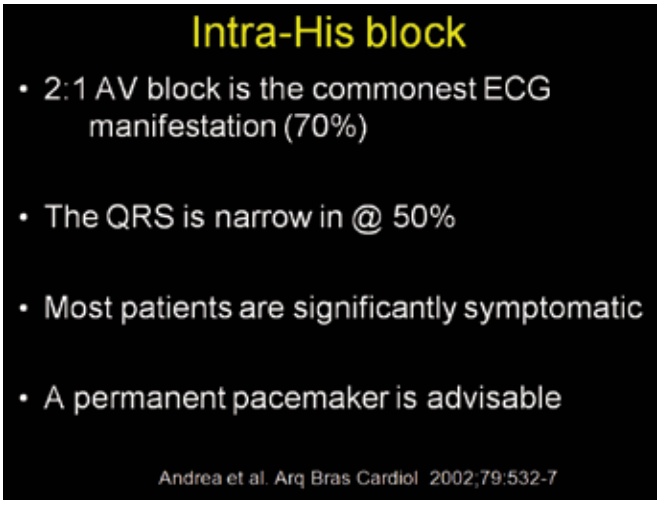
ECG - 6

The correct answer is 'c' – Intra-His block

The ECG in sinus rhythm before the stress test was normal. During the stress test as expected, there is some acceleration of sinus rate which has reached about 100 bpm. There are sudden pauses during which one can clearly see blocked sinus P waves. The QRS complexes throughout are narrow and this rules out infra-his block.

AV nodal block do not occur only during exercise. In fact, during exercise, the AV nodal conduction would improve because of the catecholamine release during physical effort. The His bundle region is largely unaffected by catecholamines and therefore with increased sinus rates, intra-his block could get unmasked as in this instance. Importantly, intra-His block should be tackled by permanent pacing.

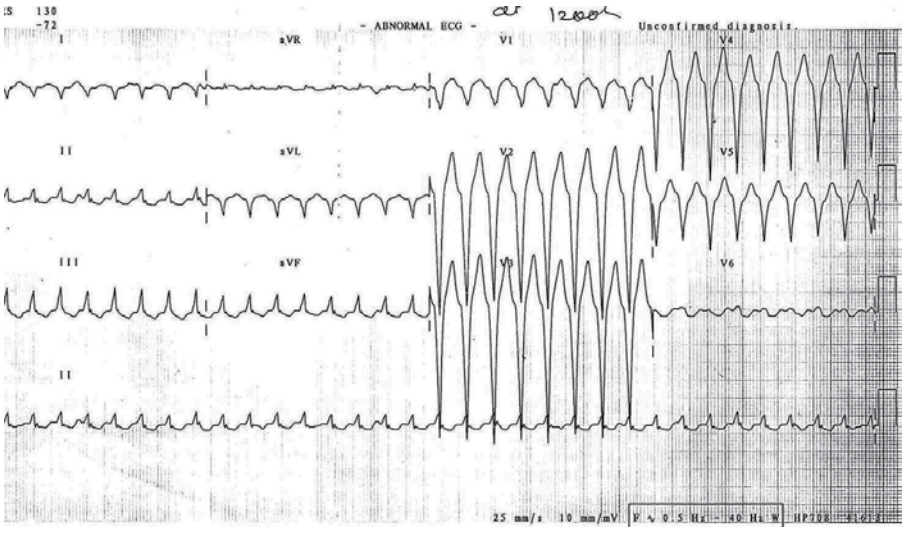




ECG - 7

65 yr old gentleman, admitted with palpitations…

Figure 1



Is this:

a. SVT

b. VT

For correct answer see overleaf

ECG - 7

The correct answer is 'b' – VT

There is a LBBB type of wide QRS tachycardia @ 200 bpm. There is a slow downstroke in lead V1 (from onset to nadir>80 ms) suggesting VT. To add to this there is RAD which supports this diagnosis. Clinching this diagnosis in the presence of dissociated P waves (*) as marked in lead I (Figure 2).

Sinus rhythm was restored after amiodarone. The sinus complexes apart from flat T waves in lead II and aVF are unremarkable. There are unifocal PVCs with different morphology from that in VT (Figure 3). Here the QRS of the PVCs is positive with a LAD.

Coronary artery disease will produce monomorphic VT in the presence of an old infarct scar. There is no suggestion of an old MI; hence angiography is unlikely to throw light on the aetiology of VT.

An EP study would be superfluous because we already have the diagnosis of VT.

Since the VT and the PVCs suggests multiple foci, with no obvious etiology, a myocardial pathology needs to be looked into. A cardiac MRI is the best modality to look at the myocardium.

Figure 2

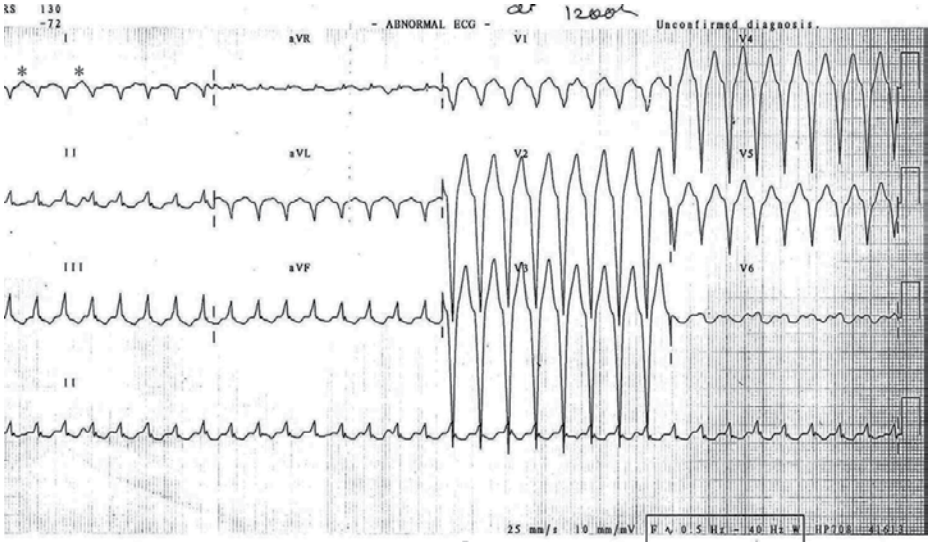
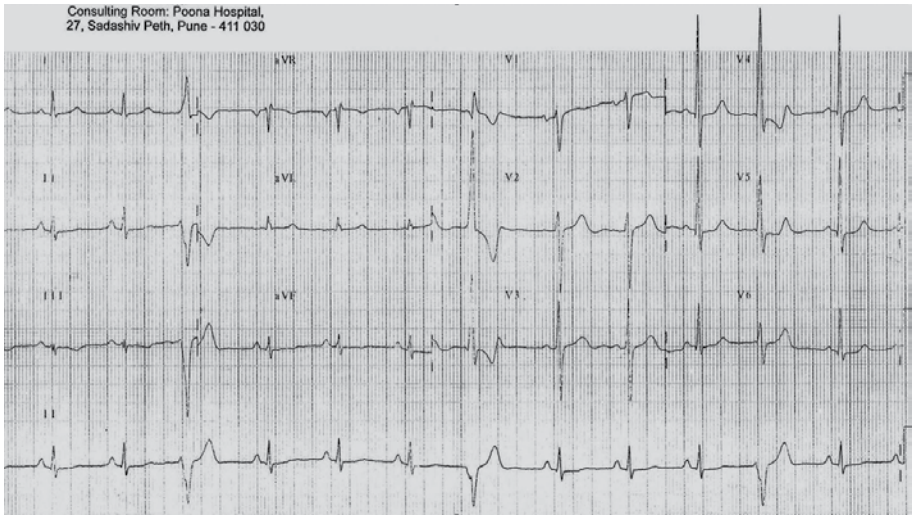


Figure 3 : After amiodarone…



The MRI showed several areas of enhancement suggesting infiltration in different areas of myocardium. Also there were grossly enlarged hilar, subcarinal and paratracheal lymph nodes (Figure 4). CT-guided biopsy of the lymph nodes (Figure 5) showed granulomas without caeseation and Castleman's bodies. The diagnosis of cardiac sarcoiodosis was made and the patient was started on aggressive immunosuppressant treatment.

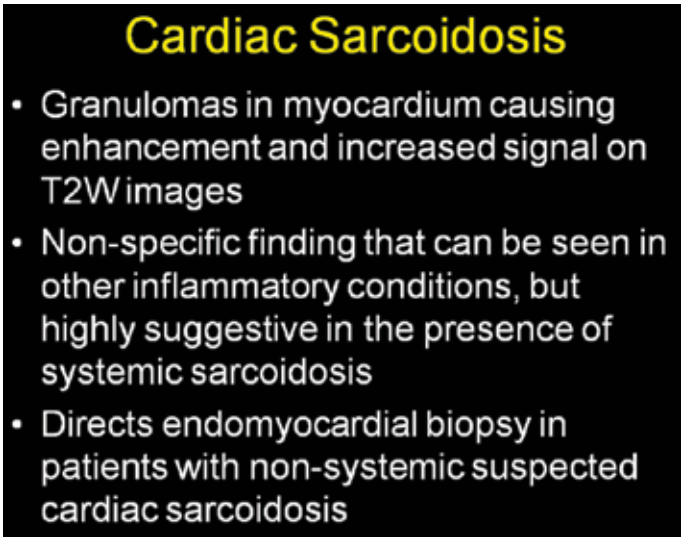


Figure 4 : Arrows point to enlarged lymph nodes

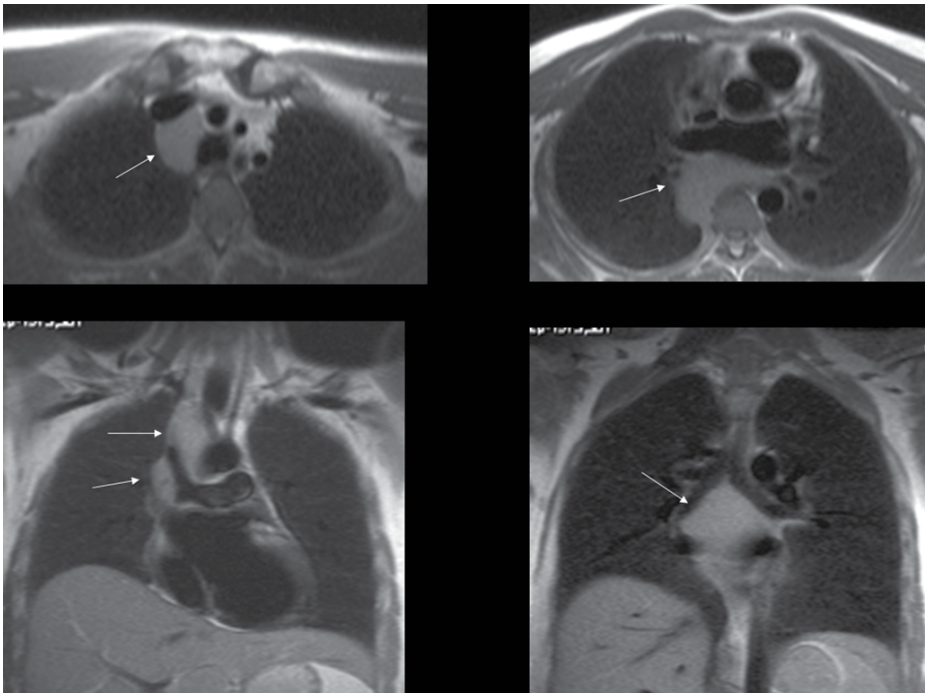
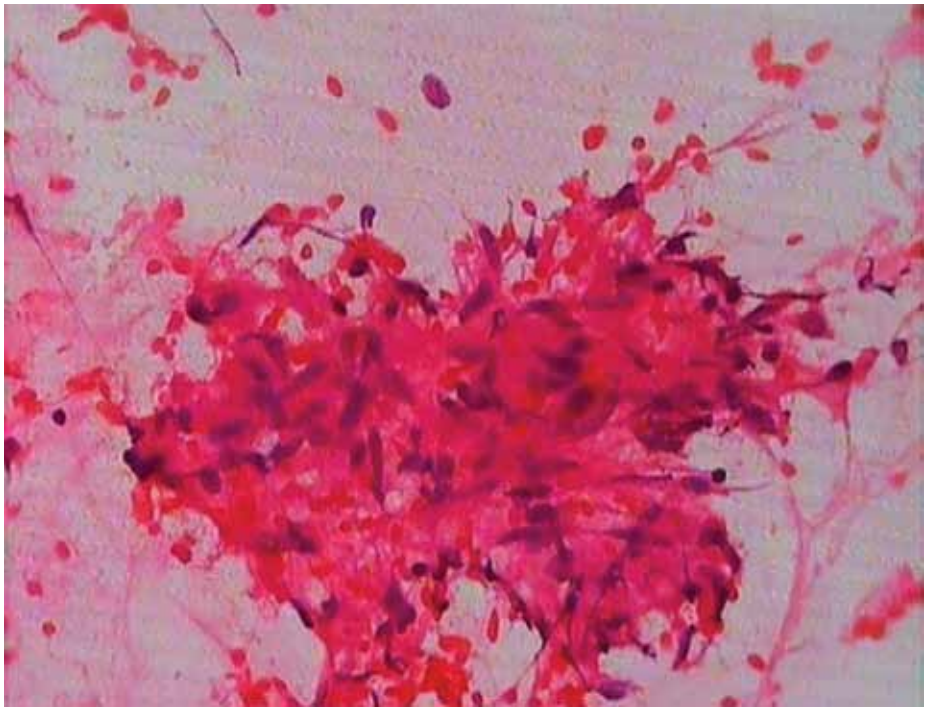


Figure 5 : CT-guided lymph node biopsy



Letter to the Editor

Dear Sir,

Primary angioplasty (PAMI) is now established to be superior to thrombolysis, in early stages of acute myocardial infarction (AMI)[1]. However, a vast majority of patients in India, do not have early access to PAMI due to geographical, technical and financial reasons. In many cases, a lot of precious time is wasted trying to arrange for a PAMI, leading to an increase in the myocardial damage due to irreversible necrosis. A prompt thrombolysis would definitely save more myocardium than a delayed PAMI, as illustrated by this case.

A 60 year old gentleman, presented to our casualty with a history of classical precordial chest pain, with dyspnoea and diaphoresis, since 2 hours. The ECG (Fig. 1) revealed a hyperacute extensive anterior wall MI with gross ST elevation (grade 3 ischemia) in leads I, AVL and V1 to V6. He was promptly administered Streptokinase (1.5 million units IV infusion over 30 minutes), along with other supportive treatment. The ST elevation resolved and he became pain free within 30 minutes. After 4 hours, Troponin I and CKMB levels were elevated and an echocardiogram showed hypokinesia of the anterior wall and the interventricular septum with an LVEF of 0.40. Coronary angiogram done after 2 weeks showed only a mild plaque in the proximal LAD which was well re-canalised with a TIMI 3

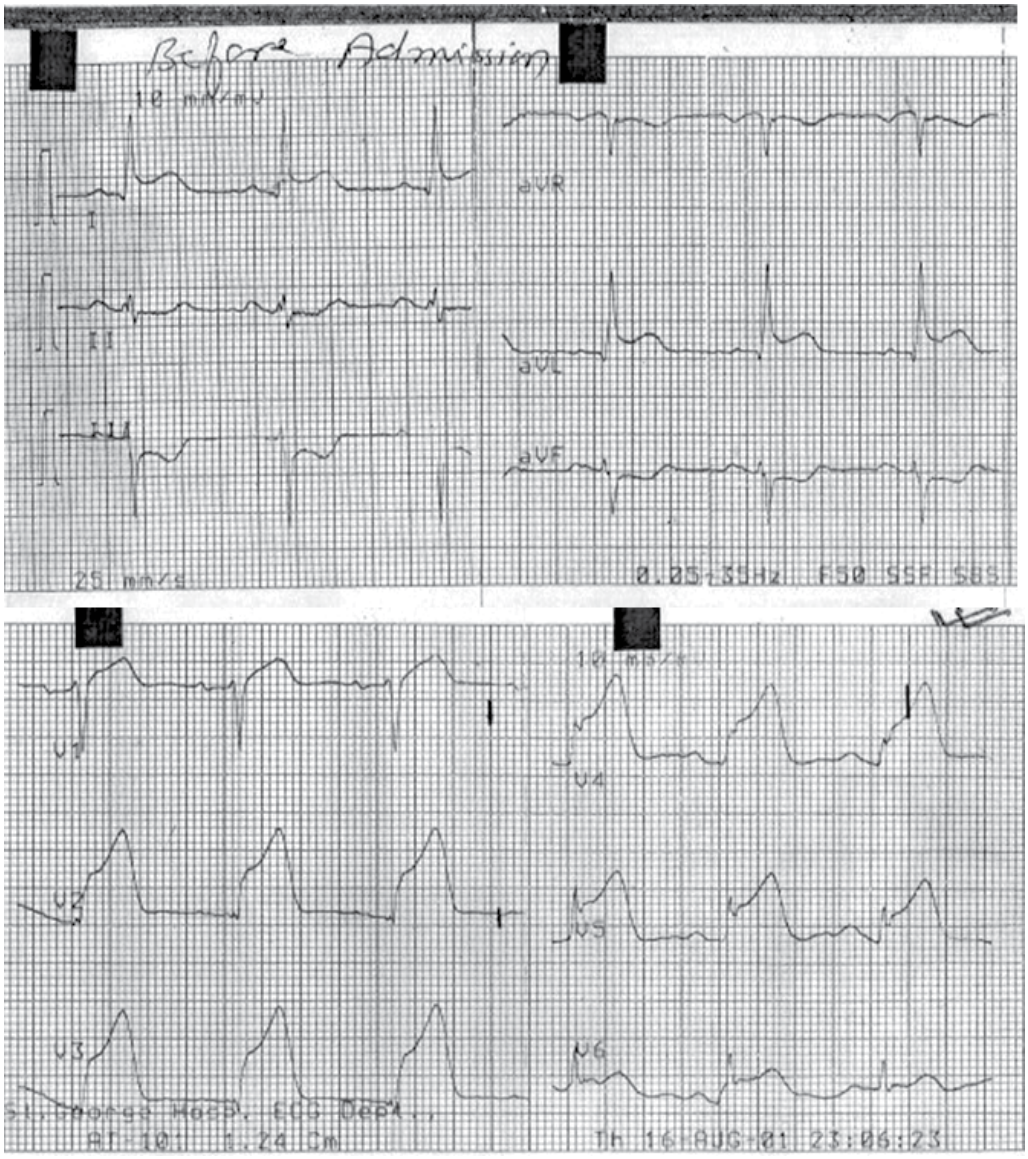


Figure 1 : ECG at presentation shows a hyperacute extensive anterior wall myocardial infarction with ST elevations in leads I, AVL and V1 to V6.

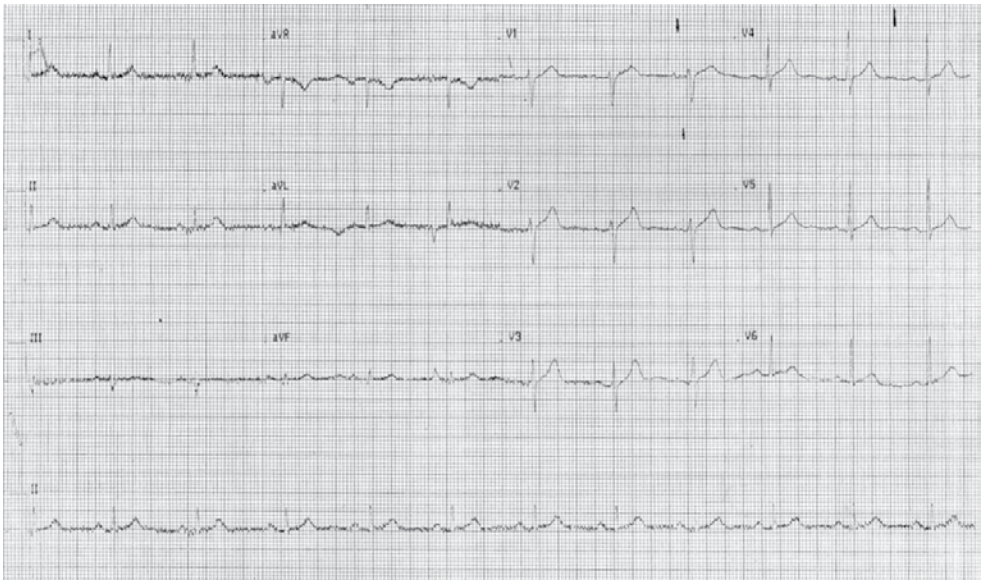


Figure 2 : ECG at 6 month follow-up reveals complete normalization with no residual evidence of myocardial damage.

flow. He had an asymptomatic course and at 6 months follow-up his ECG (Fig. 2) had completely normalized with no residual evidence of the extensive AMI. The echocardiogram showed that there was no regional wall motion abnormality and the LVEF was about 0.60.

This case highlights the fact that, if the patient presents early (within 2 hours of onset of chest pain), thrombolysis is as effective as PAMI[2]. In such cases, prompt thrombolysis should be initiated rather than wasting precious time trying to arrange for PAMI.

Dr. K. Bharti
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Dr. Bhavesh Vajifdar
Consultant Cardiologist, Glenmark Cardiac Center, Mumbai.

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2. ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction: July 2004.

INDIAN SOCIETY OF ELECTROCARDIOLOGY

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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.

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Qualifications _____

University (Post-Graduation obtained) _____

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Mailing Address _____

_____

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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

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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.

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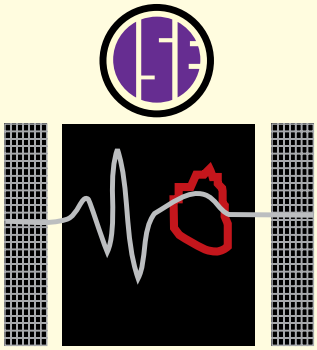
1. Person should be a Member of the Society.
2. He/She should be of atleast 7 years of standing after Post-Graduation.
3. He/She should have minimum 3 publications In Cardiology In Indexed Journals (Not Abstracts)
4. List of Publications to be submitted for the Fellowship.
5. Fellowship Fees: Rs.2,000/- (+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.

Friends of the Society



SECRETARIAT
S. B. GUPTA
VICE PRESIDENT

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