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## Editorial

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*Dear Colleagues,*

As we bring this issue of the IJE to you, we are at the threshold of the mid-term ISECON at Ranchi. Dr. Deepak Gupta, Dr. S. K. Bahl and their team have been sparing no efforts to ensure an informative and educative scientific programme. They have also tried to get a very good faculty and I am sure this programme will be a memorable one.

The last ISECON at Kolkata was a resounding success. Dr. Rabin Chakraborty, Dr. Aftab Khan and their team did a marvellous job in organizing this event. The scientific programme, the parallel EP session and especially the technician and nurses' training session all went off without a hitch. Importantly, the educational content and the presentation quality were of a very high order.

This issue of IJE has two articles taken from the web-based Indian Pacing and Electrophysiology Journal ([www.ipej.org](http://www.ipej.org)), with due permission. The first article on 'Commotio Cordis' and the second one on 'The History of Left Septal Fascicular Block' will both be of interest and utility. They are exhaustive reviews which could also be kept as a desk-reference for many of us. After that we have an ECG Quiz with ECGs selected from those presented at ISECON Kolkata.

Happy reading and we hope you send us more of your contributions. We also look forward to the next ISECON in 2009 at Ahmedabad. We thank Dr. Gopi Krishna Panicker from Quintiles ECG Services for his untiring effort in preparing this issue.

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

**Yash Lokhandwala**

*Editor*

## From Vice President's Desk

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*Dear Members,*

Indian Society of Electrocardiology has become a beaming society and it is proud to be associated and work under such a banner.

Dr Rabin Chakraborty and their team organized ISECON-2008 at Kolkata from 22<sup>nd</sup> to 24<sup>th</sup> February 2008. It was indeed a great scientific feast. Many Mid-term and ISECONs have been organized.

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

- a. Second seminar on “Global Approaches to Cardiac Safety” at Mumbai in collaboration with Quintiles on 8<sup>th</sup> and 9<sup>th</sup> March 2008
- b. “ECG Learning Course” for postgraduate students at Bangalore on 24<sup>th</sup> and 25<sup>th</sup> May 2008 and at Silvassa on 23<sup>rd</sup> and 24<sup>th</sup> August 2008. About 70-80 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading
- c. Satellite Symposia were organized at Mumbai on 18<sup>th</sup> May 2008, at Thane on 22<sup>nd</sup> June 2008 and on 6<sup>th</sup> July 2008 at Lucknow and were appreciated by one and all.
- d. A unique “Training the Trainers” was organized at Mumbai on 29<sup>th</sup> June 2008 to streamline the “ECG Learning Course. Most of the cardiologists in the field of Electrocardiophysiology attended the seminar.

Every one is eagerly waiting for Ranchi Arrhythmias Course organized by Dr Deepak Gupta and Dr Subir Pal and their team members on 27<sup>th</sup> and 28<sup>th</sup> September 2008 and ISECON 2009, to be held at Ahmedabad from 20<sup>th</sup> to 22<sup>nd</sup> February 2009 under the dynamic leadership of Dr Ajay Naik and the team.

I look forward to see you all at RAC-2008 and ISECON-2009, which will be a real treat.

My sincere thanks to Dr Yash Lokhandwala, Dr Ulhas Pandurangi and the Editorial Team for bringing out the ISE Journal – 2008, 2<sup>nd</sup> Volume.

Long Live Indian Society of Electrocardiology



**Dr. S.B. Gupta**

*Vice President, Indian Society of Electrocardiology*





## Review Article

### Commotio Cordis

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#### Abstract

Sudden arrhythmic death as a result of a blunt chest wall blow has been termed Commotio Cordis (CC). CC is being reported with increasing frequency with more than 180 cases now described in the United States Commotio Cordis Registry. The clinical spectrum is diverse; however young athletes tend to be most at risk, with victims commonly being struck by projectiles regarded as standard implements of the sport. Sudden death is instantaneous and victims are most often found in ventricular fibrillation (VF). Chest blows are not of sufficient magnitude to cause any significant damage to overlying thoracic structures and autopsy is notable for the absence of any structural cardiac injury. Development of an experimental model has allowed for substantial insights into the underlying mechanisms of sudden death. In anesthetized juvenile swine, induction of VF is instantaneous following chest impacts that occur during a vulnerable window before the T wave peak. Other critical variables, including the impact velocity and location, and the hardness of the impact object have also been identified. Rapid left ventricular pressure rise following chest impact likely results in activation of ion channels via mechano-electric coupling. The generation of inward current through mechano-sensitive ion channels results in augmentation of repolarization and non-uniform myocardial activation, and is the cause of premature ventricular depolarizations that are triggers of VF in CC. Currently available chest protectors commonly used in sport are not adequately designed to prevent CC. The development of more effective chest protectors and the widespread availability of automated external defibrillators at youth sporting events could improve the safety of young athletes.

**Key Words:** Commotio cordis, Ventricular Fibrillation, Athletes, Sudden Death, Mechano-electric coupling

#### Introduction

Sudden cardiac death as a result of a blunt and often innocent-appearing chest wall blow has been termed commotio cordis (CC). Derived from Latin and meaning *disturbance of the heart*, it is now apparent that CC is a primary electrical event, with the instantaneous induction of ventricular fibrillation (VF) resulting from non-penetrating chest wall impacts that are usually not of sufficient force to cause any significant structural injury to the heart<sup>1,2</sup>. This characteristic differentiates CC from *contusio cordis*, in which high impact blows result in direct myocardial tissue damage, often associated with injury to the overlying structures of the chest and thorax. Although the clinical context has proven diverse, many instances of CC occur as a result of chest impact with projectiles used in organized sports, such as baseball, hockey, and lacrosse<sup>3</sup>. In the United States, CC has now been recognized as a leading cause of sudden death in youth athletics and is being reported with increasing frequency. Over the last decade, CC has achieved broader visibility through a series of reports detailing its clinical spectrum<sup>3,4</sup>. At the same time, the development of a contemporary experimental model

has shed substantial light into the pathophysiology<sup>1</sup>. This article will review the clinical spectrum of CC, address recent insights into the underlying mechanisms, and discuss strategies to prevent this tragic event.

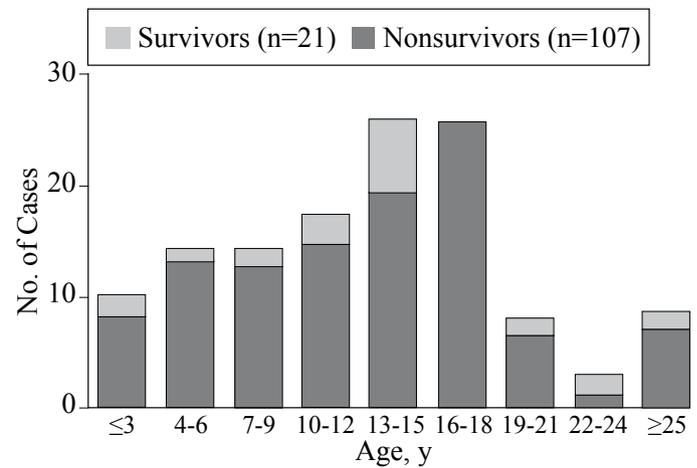
#### Clinical spectrum

Since being initiated in 1996, the United States Commotio Cordis Registry (USCCR -Minneapolis, Minnesota) has now accrued more than 180 cases<sup>3-5</sup>. As awareness of this phenomenon grows, CC is being reported with increasing frequency, with most cases in the registry (75%) clustered from the years 1988 to present<sup>6</sup>. However, the actual incidence remains unknown as many cases are still likely missed due to continued lack of recognition and underreporting. CC has most commonly been described in the setting of organized sport (**Table 1**), with most victims having been struck in the chest by standard projectiles used in the game<sup>3</sup>. Generally, projectiles that result in CC have a dense solid core, such as a baseball, hockey puck, or lacrosse ball. Only 2 cases in the USCCR have been attributed to impact with a cricket ball. However, this low incidence likely reflects the relative lack of popularity of cricket in the US and the fact

**Table 1** : Characteristics of Chest Blows in 128 Commotio Cordis Events.

Sports equipment	
Baseballs*	53
Softballs	14
Hockey pucks	10
Lacrosse balls	5
Cricket balls	2
Soccer ball	1
Hockey stick	1
Tennis ball (filled with coins)†	1
Bodily collisions in sports‡	
Knee or foot	5
Elbow or forearm	5
Shoulder	4
Fist§	2
Head (football helmet)	2
Goalpost	1
Daily and recreational activities	
Playful (“shadow”) boxing	6
Parent-to-child discipline	5
Gang rituals	3
Scuffle	1
Plastic sledding saucer (diameter, 91.44 cm [36 in])	1
Plastic (hollow) toy bat	1
Snowball	1
Playground swing carriage	1
Pet (collie) dog head	1
Hiccups remedy (by friend)	1
Fall on playground apparatus (“monkey bars”)	1

\*Includes 4 T-balls. †Training device for baseball pitchers. ‡Baseball, football, karate, soccer, basketball, and hockey. §Closed or open fist (including jab, push, slap). Reprinted with permission from: Maron BJ, Gohman TE, Kyle SB, Estes NA, 3rd, Link MS. Clinical profile and spectrum of commotio cordis. JAMA 2002;287(9):1142-6. Copyright © (2002), American Medical Association. All rights reserved.

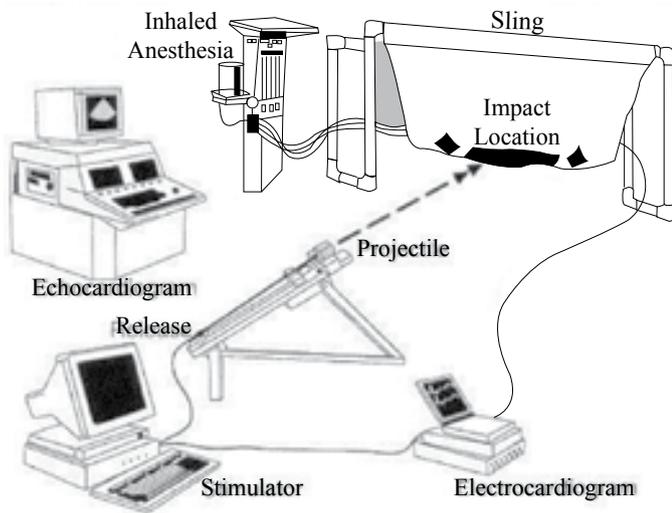


**Figure 1** : Age at the time of event in the United States Commotio Cordis Registry. Reprinted with permission from: Maron BJ, Gohman TE, Kyle SB, Estes NA, 3rd, Link MS. Clinical profile and spectrum of commotio cordis. JAMA 2002;287(9):1142-6. Copyright © (2002), American Medical Association. All rights reserved.

that chest impact in cricket is a rare event. Projectiles with a non-solid core tend to collapse on contact and absorb much of the impact energy. Only a single event has been attributed to chest impact with an air-filled soccer ball. In almost all cases, chest impacts that resulted in CC occurred to the left of the sternum, directly over the cardiac silhouette. Estimated velocities of pitched baseballs were 48 to 80 km/h (30-50 mph). Interestingly, 38% of the individuals competing in organized sports were wearing standard commercially available chest wall protection at the time of their event<sup>7</sup>. However, in 25 of these 32 cases, the chest wall protector did not adequately cover the left chest or precordium at the time of impact.

Although commonly associated with sport, CC has now been reported in a diverse spectrum of non-sports related activities<sup>3</sup>. Many of these cases occurred in association with happenings of everyday life that resulted in often unintentional and innocuous-appearing chest blows. Some such examples include a 23 year old man fatally striking his friend in the chest as a mutually agreed upon remedy for hiccups. In two other cases, a 2-year old girl was incidentally struck in the chest by the head of her pet dog and a 5-year old boy died instantly after being struck in the chest by a circular plastic sledding saucer. Young males (median age of 14 years) appear to be most at risk from CC<sup>3</sup>. This susceptibility has been partially attributed to the compliant chest walls of children that allow for greater transmission of impact energy to the myocardium. Only 28% of the cases in the USCCR were aged over 18 years, with the oldest victim a 44 year-old woman (**Figure 1**).

The overall survival rate in known victims of CC is only 15%, with successful resuscitation often quite difficult<sup>3,8</sup>. Initial ECG data (recorded in the emergency room or by emergency medical

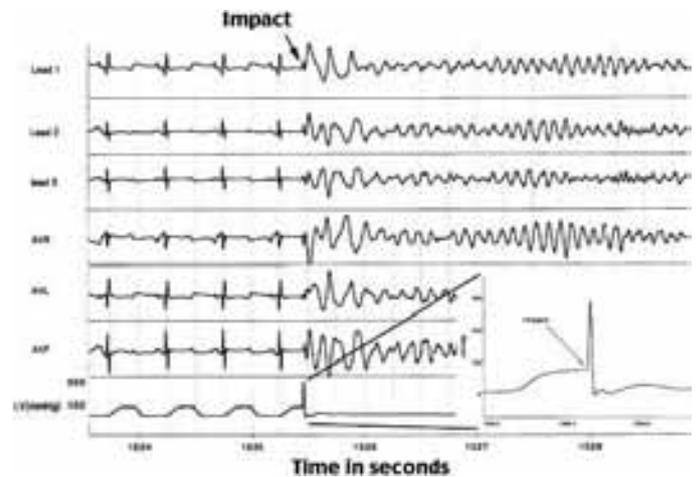


**Figure 2 :** Laboratory and study design for the commotio cordis model. An anesthetized and intubated animal is positioned prone in a sling. Under echocardiographic guidance, a ball affixed to an aluminum shaft is impacted on the chest directly over the base of the left ventricle. An electrophysiologic stimulator triggered from the surface electrocardiogram is used to gate impacts to the cardiac cycle. A chronograph measures the impact velocity. Reprinted with permission from: Link MS, Wang PJ, VanderBrink BA, et al. Selective activation of the K(+)(ATP) channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (Commotio cordis). *Circulation* 1999;100(4):413-8. Copyright © (1999), The American Heart Association.

technicians in the field) was available in 82 patients in the USCCR. Analysis revealed 33 cases of VF, 3 with ventricular tachycardia, 3 with bradyarrhythmias, 2 with idioventricular rhythm, and 1 with complete heart block. Forty of the cases documented asystole, which was unlikely to be the initial rhythm after impact, and is more likely a result of prolonged time from event to rhythm documentation. Application of early resuscitation and defibrillation appears to be the most important determinant of survival, as with other causes of VF. Cardiopulmonary resuscitation was known to have been performed in 106 of the individuals in the USCCR<sup>3</sup>. Of 68 cases in which early resuscitation was instituted (< 3 minutes), 17 survived (25%). In the cases where resuscitation was substantially delayed (> 3 minutes) only 1 out of 38 survived (3%).

### Pathophysiology

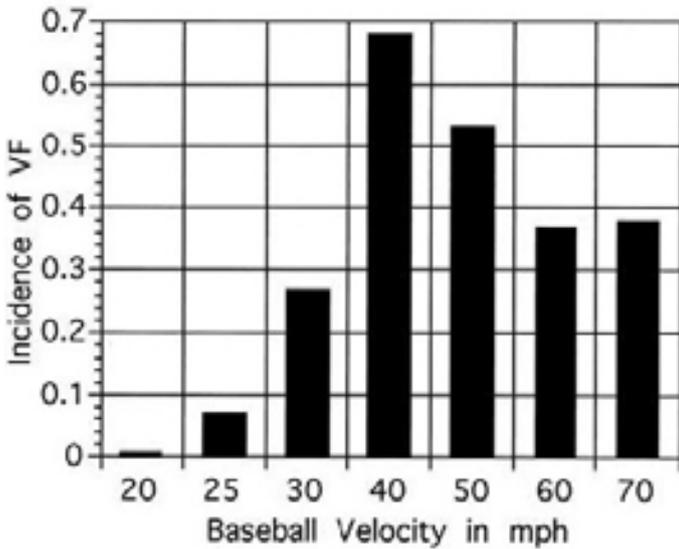
It is now apparent that CC is a primary electrical event, with sudden death resulting from the instantaneous induction of VF initiated by a chest wall blow. Autopsy data generally has not revealed any underlying congenital or acquired heart disease in victims. Available evidence suggests that the pathophysiology of impact-induced VF is multi-factorial and requires the precise confluence of several variables. Development of an experimental



**Figure 3 :** Six lead electrocardiogram and intraventricular pressure measurement from an 11 kg swine undergoing a 48 km/h (30 mph) chest wall impact with an object the shape and weight of a standard baseball. Ventricular fibrillation is produced immediately upon impact within the vulnerable zone of repolarization (10-30 ms prior to the peak of the T-wave). Reprinted from: Link MS, Maron BJ, VanderBrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J Am Coll Cardiol* 2001;37(2):649-54. Copyright 2001, with permission from Elsevier.

animal model has allowed for a deeper understanding of the underlying mechanisms. This model attempts to mimic the clinical profile of CC, and entails propelling projectiles commonly used in sport (baseballs and lacrosse balls) at the chest wall of anesthetized juvenile swine<sup>1</sup>. Release and subsequent impact of the balls are gated to the cardiac cycle by a cardiac stimulator with triggering from the surface electrocardiogram of the swine (**Figure 2**). Initial experiments involving this animal model defined a narrow window of vulnerability within the cardiac cycle that is critical for the development of CC. When impacts occurred precisely within 10 to 30 milliseconds before the peak of the T wave, VF was consistently produced (**Figure 3**). VF was instantaneous and was not preceded by premature ventricular contractions (PVC), ST-segment changes, or heart block. Chest impacts occurring in other portions of the cardiac cycle produced various other electrophysiologic effects - including ST-segment elevation, PVC, transient heart block, and left bundle branch block - but never resulted in VF.

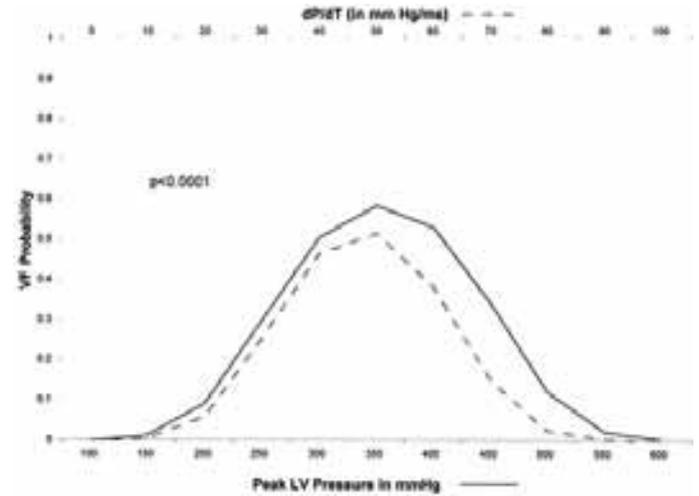
Several other factors have also been identified as crucial to the development of VF in this model of CC. Using echocardiographic guidance, the importance of impact location directly over the anatomic position of the heart was revealed<sup>9</sup>. VF occurred most commonly with blows directly over the center of the cardiac silhouette (30% of impacts) versus those over the left ventricular base (13% of impacts) or apex (4% of impacts). Thoracic impacts not overlaying the heart did not result in VF



**Figure 4 :** Incidence of ventricular fibrillation (VF) induced by chest wall impacts at the vulnerable period of repolarization (10-30 ms prior to the peak of the T-wave) with a regulation baseball propelled at velocities ranging from 32 to 113 km/h (20 to 70 mph) in the swine model of commotio cordis. The incidence of VF relative to the velocity of chest impact exhibited a Gaussian distribution with peak incidence at 64 km/h (40 mph) ( $p < 0.0001$  by logistic regression). Reprinted from: Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes NA, 3rd. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). *J Am Coll Cardiol* 2003;41(1):99-104. Copyright 2003, with permission from Elsevier.

or other electrophysiologic effects. In addition, a relationship between the hardness of the impact object and the likelihood of inducing VF was also identified. Impacts with softer safety baseballs were associated with a lower incidence of VF than impacts with harder standard balls<sup>10</sup>. Finally, the importance of the velocity of chest wall impacts was also systematically evaluated<sup>11</sup>. Baseballs were propelled with velocities ranging from 32 to 113 km/h (20 to 70 mph) and timed to impact on the vulnerable 20 ms window on the upstroke of the T wave. The plot of impact velocity relative to incidence of VF exhibited a Gaussian distribution. The threshold velocity to cause VF was 40 to 48 km/h (25 to 30 mph) and as impact velocity increased, the incidence of VF rose to a peak of nearly 70% of impacts at 64 km/h (40 mph). At velocities  $> 80$  km/h (50 mph), however, the likelihood of VF decreased (**Figure 4**). This observation is consistent with the observed clinical scenario of CC in youth baseball, where baseball velocities are estimated to range between 48 to 80 km/h (30 to 50 mph).

The importance of the location, hardness, and velocity of chest wall impacts in CC relate to the effects of these variables on induction of a critical left ventricular (LV) pressure that is necessary to produce VF. In experiments of impact velocity,



**Figure 5 :** The probability of ventricular fibrillation (VF) relative to the peak left ventricular (LV) pressure and LV pressure over time (dP/dt) in 8-12 kg swine undergoing 48 km/h (30 mph) chest wall impacts with a baseball. The data exhibit a Gaussian distribution ( $p < 0.0001$  by logistic regression). The highest incidence of VF was evident with peak LV pressures between 250 and 450 mmHg. Reprinted from: Link MS, Maron BJ, VanderBrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J Am Coll Cardiol* 2001;37(2):649-54. Copyright 2001, with permission from Elsevier.

higher velocities correlated with the generation of greater peak instantaneous LV pressures. As with impact velocities, the risk of VF correlated with the LV pressure rise created by the chest wall blow in a Gaussian distribution (**Figure 5**)<sup>8,11,12</sup>. The highest incidence was evident with peak LV pressures of 250 to 450 mmHg and decreased with pressures above and below this range. Thus, these data suggest that there is a lower and upper limit of vulnerability of LV pressures resulting in VF and that the instantaneous LV pressure rise produced by the chest blow mediates the electrophysiologic consequences of CC.

Mechanical stimulation of the myocardium resulting in electrical events is well-described, occurring in such circumstances as catheter induced ectopic beats and thumping of the chest wall during asystole to produce PVCs<sup>5,8</sup>. This phenomenon, termed mechano-electric coupling, has been attributed to the presence of mechano-sensitive ion channels that are activated by deformation of the myocardial cell membrane. In CC, rapid rise of ventricular pressure immediately following chest impact results in VF mediated through resultant myocardial stretch and the activation of ion channels. CC appears to share certain electrical similarities with myocardial ischemia, including ST-segment elevation and the phenomenon of R on T causing VF<sup>13,14</sup>. Activation of the  $K^+_{ATP}$  channel is primarily responsible for ST-segment elevation noted in myocardial infarction, and contributes to the increased risk of VF associated with ischemia.

In addition, mechano-sensitivity of the  $K_{ATP}^+$  channel has been previously demonstrated in a rat model<sup>15</sup>. In our model of CC, infusion of glibenclamide, a  $K_{ATP}^+$  channel inhibitor, reduced the magnitude of ST-elevation and the incidence of VF following chest blows<sup>13</sup>.

Our results suggest that the immediate activation of the mechano-sensitive  $K_{ATP}^+$  channel by chest wall impacts is in part responsible for the induction of VF in CC. Other stretch-sensitive ion channels are also likely to be involved. Interestingly, however, blockade of the non-selective cation stretch-activated channel (SAC) with streptomycin did not prevent induction of VF in our model<sup>12</sup>.

In CC, the inward current generated through the opening of mechano-sensitive ion channels results in ventricular depolarizations that in turn, trigger development of VF. However, ventricular depolarization alone is not sufficient to result in reentrant arrhythmia that underlies the mechanism of VF. Thus, initiation of VF in CC appears to require at least two features: (i) a *trigger* - premature ventricular depolarization - occurring in the setting of (ii) a *susceptible myocardial substrate*<sup>5</sup>. The necessity of both trigger and substrate is illustrated in experiments of impact velocity<sup>8</sup>. PVCs were observed in nearly 70% of impacts that did not result in VF. Thus, a trigger (ventricular depolarization) was produced, but did not result in VF, presumably due to the absence of appropriate substrate.

Interestingly, both trigger for CC and the susceptible myocardial substrate are in part created by a chest wall blow occurring in the vulnerable portion of the cardiac cycle. Susceptibility to development of CC relates to dispersion of repolarization that is present during the vulnerable period of the cardiac cycle when chest impact occurs. Recent data by Bode et al support this hypothesis<sup>16</sup>. Fluid-filled balloons were placed in the LV of Langendorff perfused rabbit hearts and increasing volume and pressure pulses were applied at different points of the cardiac cycle. VF was induced only when balloon inflation occurred within a vulnerable window of 35ms to 88ms after the initiation of an action potential. This vulnerable window corresponded to the time of spontaneous increase in repolarization dispersion. Even more interesting was the observation that as compared to baseline, pressure pulses that induced VF resulted in a *further increase* in repolarization dispersion. Thus, it appears that the upstroke of the T wave signifies a window of *potential* vulnerability for development of VF in CC, due to spontaneous increase in repolarization dispersion. The potential vulnerability for the induction of VF is *realized* when chest impact results in sudden elevation in LV pressure leading to further increase in repolarization dispersion. Analogous to this hypothesis is the R on T phenomenon. In non-ischemic myocardium, premature ventricular depolarization during the T wave does not normally induce VF. Thus, continuous ventricular pacing (VOO) is generally safe. However, with the increase in repolarization dispersion in the setting of ischemia, the potential for inducing

VF can be realized when a PVC falls on the vulnerable portion of the T wave<sup>17,18</sup>.

In addition, the experiments by Bode et al provide further insight into the electrical properties by which increase in repolarization dispersion might produce VF in CC<sup>16</sup>. In their model, it was observed that the LV myocardium was not excited simultaneously by a global pressure pulse. Instead it was noted that the earliest activation occurred at the LV site with the shortest repolarization time and occurred considerably later at sites with longer repolarization times. VF was induced when a sufficient electrical gradient was able to activate myocardium with early local recovery, but failed to activate myocardium that was refractory. Based on these findings, non-uniform excitation might thus form the basis for the initiation of reentry and the induction of VF in CC.

Although activation of the  $K_{ATP}^+$  channel is shared by both CC and ischemic myocardium, the mechanism of activation is quite different. In our model, angiography performed immediately after impact in animals that developed VF did not reveal any evidence of stenosis or spasm in epicardial coronary arteries<sup>1</sup>. Myocardial perfusion imaging with technetium 99m sestamibi performed after impact revealed only small mild apical defects in a minority (25%) of the animals tested. In addition, left ventriculograms and echocardiograms performed immediately after defibrillation revealed only mild apical or distal septal hypokinesis, regions distant from the area of precordial impact<sup>1</sup>. On pathologic examination, structural cardiac damage has not been observed with impact velocities of less than 80 km/h (50 mph)<sup>8</sup>.

### Prevention and Therapy

Deaths of young athletes from CC are tragic and often highly visible events. In the United States, several preventive strategies have been considered, including increasing the use of softer balls and chest wall protectors in organized youth sports. Safety baseballs with soft rubber cores have been designed to reduce the risk of head and other bodily injury from traumatic impacts. In our experimental model, safety baseballs significantly reduced, but did not eliminate, the risk of sudden cardiac death with chest impacts at 48 km/h and 64 km/h (30 and 40 mph)<sup>10</sup>. Despite these data, widespread use of safety baseballs in youth sports has yet to be adopted.

In the United States, chest protectors are marketed with claims of protecting athletes from chest wall trauma. However, in the USCCR, more than 38% of the fatal impacts occurring during organized sport involved individuals who were wearing a chest protector<sup>3,7</sup>. In several of these cases, migration of the protector exposed the chest to a direct impact. In other cases, however, projectiles were known to strike on the chest protector, which failed to prevent the fatal CC event. Recently, the effectiveness of commercially available lacrosse and baseball chest protectors was assessed for the prevention of sudden cardiac death<sup>19</sup>.

None of the currently available chest protectors was shown to significantly decrease the incidence of VF when compared to controls. In accord with our previous findings, peak LV pressure produced by chest wall impacts correlated linearly with the probability of VF in these experiments. Using a 3 rib biomechanical dummy model, Viano et al also demonstrated the ineffectiveness of several commercial chest protectors in reducing the potential risk of CC<sup>20</sup>. The majority of the chest protectors evaluated in these studies were composed of a compliant layer(s) of closed cell foam of varying thickness and density that is intended to dissipate the energy of an impact. The ineffectiveness of this design in reducing the incidence of VF is likely due to its inability to adequately reduce the peak LV pressure generated by chest wall impact. It is clear that further research on the development of an adequate chest protector for the prevention of CC is needed. Likely due to a lack of early recognition and the failure to initiate timely aggressive resuscitation and defibrillation, the survival rate in the USCCR is only about 15%<sup>3</sup>. Survival is most likely to occur with the institution of cardiopulmonary resuscitation and defibrillation within 3 minutes of the incident event. Similar outcomes were seen in our model of CC in which defibrillation with an automated external defibrillator (AED) within 1 or 2 minutes of VF resulted in successful resuscitation in 100% and 92% of animals, respectively<sup>21</sup>. Only 46% of shocks were successful after 4 minutes, and after 6 minutes survival decreased further to 25% ( $p < 0.0001$ ). The AED recognized VF with a 98% sensitivity and the specificity for non-shockable rhythms was 100%. Based on these data, more widespread access to AEDs at organized youth sporting events and the training of personnel in their early use would likely achieve substantial increase in the survival of CC victims. Indeed, successful resuscitation of victims of CC with AEDs has been observed. However, the importance of developing more effective primary prevention strategies and promoting their widespread use should also not be overlooked. This point is underscored by the recent tragic case reported in a 22-year-old student who suffered a CC event during an intercollegiate lacrosse game<sup>22</sup>. Despite early AED application and prompt defibrillation (within 2 minutes of his collapse), spontaneous circulation could not be restored. The student died an hour later in a neighboring emergency department.

### Conclusion

CC is being reported with increasing frequency and young athletes tend to be most at-risk. Sudden cardiac death results from the instantaneous induction of VF following a precordial chest wall blow. A narrow window of vulnerability corresponding to the time of intrinsic increase in repolarization dispersion, just before the peak of the T wave has been identified. In addition, several other variables, including the impact location and the hardness and velocity of the projectile relate to the production of a critical LV pressure that is necessary to induce VF. Rapid LV pressure rise following chest impact results in myocardial stretch

and activation of ion channels, including the  $K^+_{ATP}$  channel, via mechano-electric coupling. Inward current through

these mechano-sensitive ion channels results in further augmentation of repolarization and non-uniform myocardial activation, and is the cause of premature ventricular depolarizations that are the triggers of VF. Survival in the USCCR is quite poor and largely depends on institution of early defibrillation and resuscitation. Increasing the availability of AEDs at organized youth sporting events should enhance the safety of young athletes. In addition, the importance of developing more effective primary prevention strategies for CC - such as effective chest protectors and safety balls - and promoting their widespread use is needed.

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## Historical Review

# The History of Left Septal Fascicular Block : Chronological Considerations of a Reality Yet to be Universally Accepted

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### Abstract

There are several papers in literature that prove in a conclusive and incontestable way, that the left branch of the His bundle, in most instances (85% of the cases) splits into three fascicles of variable morphological pattern, and not into two: left anterior fascicle (LAF), left posterior fascicle (LPF), and left septal fascicle (LSF). The abovementioned papers have anatomical, histological, anatomic-pathological, electrocardiographic, and vectocardiographic, body surface potential mapping or ECG potential mapping and electrophysiological foundation.

Additionally, the mentioned papers have been performed both in animal models (dogs) and in the human heart.

Several clinical papers have shown that the left septal fascicular block (LSFB) may occur intermittently or transitorily as a consequence of a temporary dromotropic alteration, constituting an aberrant ventricular conduction, rate-dependent or by the application of atrial extra-stimuli, or naturally during the acute phase of infarction when this involves the anterior descending artery, before the septal perforating artery that supplies the central portion of the septum, where the mentioned LSF runs.

The ECG/VCG manifestation of LSFB consists in anterior shift of electromotive forces, known as Prominent Anterior Forces (PAF), which can hardly be diagnosed in the clinical absence of other causes capable of causing PAF, such as the normal variant by counterclockwise rotation of the heart on its longitudinal axis, in right ventricular enlargement, in the dorsal or lateral infarction of the new nomenclature, in type-A WPW, in CRBBB, and others. In this historical manuscript, we review in a sequential fashion, the main findings that confirmed the unequivocal existence of this unjustifiably "forgotten" dromotropic disorder.

In the developed countries, its most important cause is coronary insufficiency, particularly the proximal involvement of the left anterior descending coronary artery, and in Latin America, Chagas disease.

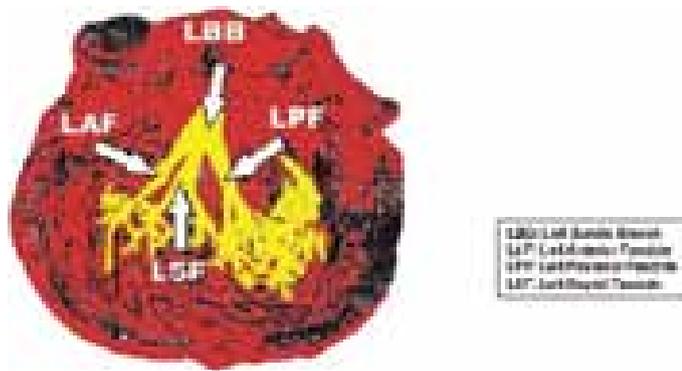
**Key Words:** Left Hissian intraventricular system; Fascicular blocks; Left Septal Fascicular Block; Hemiblocks.

### The trifascicular background of the left Hissian Intraventricular system

The story begins at the end of the 19<sup>th</sup> century with the initial description by Dr. Wilhelm His Jr. of the left Hissian

intraventricular system as being trifascicular<sup>1</sup>. He showed that a connective tissue sheet became a bundle connecting the upper and lower cardiac chambers, the bundle of His<sup>2</sup>.

At the beginning of the 20<sup>th</sup> century, Dr. Suano Tawara clearly



**Figure 1 :** The Trifascicular Nature of the Left His System. The trunk of the left bundle branch (LBB) of the His bundle split in three fascicles: Left anterior fascicle (LAF), Left septal Fascicle (LSF) and Left Posterior Fascicle (LPF). “The Conduction System of the Mammalian Heart” (1906)

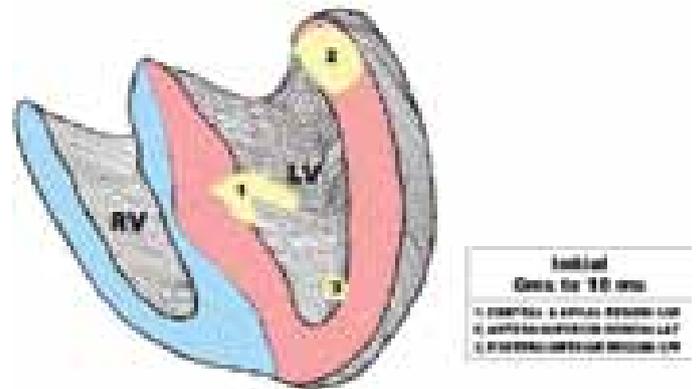
showed that the trunk of the left bundle branch (LBB) split in three fascicles and not in two<sup>3,4</sup> (**Figure 1**). Tawara’s pioneering work on the human conduction system still serves as an invaluable reference for basic and clinical research<sup>5</sup>.

The electrocardiographic study of divisional blocks of the left His system goes back to 1917 with the work by the German researchers Rothberger and Winterberg<sup>6</sup>. These authors observed that the injury in the divisions of the LBB produced significant deviations of SÂQRS in the frontal plane (FP), besides a discrete increase in QRS duration.

On the basis of experiments in dogs in 1956, Scher and Young<sup>7</sup> found that the initial activation of the septum occurred in three different areas.

In 1960, Uhley et al<sup>8,9</sup> described the trifascicular characteristic of the canine left His system and four years later, they showed the electrocardiographic manifestations (also in dogs) of the interruption of the trunk of the left branch and its three divisions. In the same year, Robb<sup>10</sup> reinforced the trifascicular characteristic of the left His system with his anatomical studies.

In 1967, Mauricio Rosenbaum<sup>11-15</sup> and his school made a great contribution to the study of this matter and had a great influence on the world of arrhythmology. He brilliantly described the electrocardiographic manifestations of blocks in the antero-superior (LAFB) and postero- inferior (LPFB) divisions of the LBB. This school considered that the human left His system was anatomically and functionally bifascicular, and for this reason the term hemiblocks was coined: left anterior hemiblock corresponding to the current LAFB and left posterior hemiblock corresponding to LPFB. These authors maintained the same nomenclature until to day. One year after the first Rosenbaum’s publication, and contrary to his school of thoughts, Barry and Pattern<sup>16</sup> showed the trifascicular structure of the left His system. In that same year, in an electrocardiographic and vectorcardiographic study Cohen et al<sup>17</sup> demonstrated



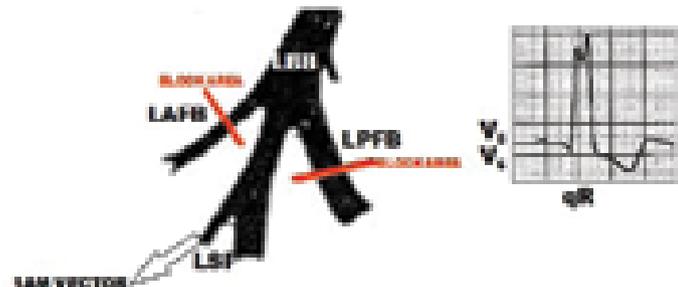
**Figure 2 :** Sequence of 10ms Initial Normal Ventricular Activation

the variations of aberrant ventricular conduction in man with evidence of isolated and combined block within the specialized conduction system.

In 1970, Dr. Dirk Durrer et al<sup>18</sup> from the Departments of Cardiology and Physiology, Amsterdam, established in a classical manuscript, using 870 intramural terminals in the isolated human heart that three endocardial areas were synchronously excited from 0 to 5 ms after the start of the left ventricle (LV) activity potential. To obtain information concerning the time course and instantaneous distribution of the normal excitatory process, the authors studied isolated hearts from seven individuals who died from cerebral conditions but had no history of cardiac disease (**Figure 2**).

The LV areas first excited were: 1) High on the anterior paraseptal wall just below the attachment of the anterolateral papillary muscle (ALPM) where end the LAF; 2) Central on the left surface of the intraventricular septum (IVS) where end the LSF. Septal activation started in the middle third of the left side of the IVS, somewhat anteriorly, and at the lower third at the junction of the IVS and posterior wall. The normally functioning LSF, the left middle septum surface and the inferior two-thirds of the septum originate the first vector, vector 1 or first anteromedial (IAM) vector and left inferior two-thirds of the IVS (second vector or vector the inferior two-thirds of IVS)<sup>19</sup>; 3) Posterior paraseptal, about one third of the distance from apex to base near the base of PMPM where end the LPF. The posterobasal area is the last part of the LV to be activated. Finally, endocardial activation of the right ventricle (RV) was found to start near the insertion of the anterior papillary muscle of the tricuspid valve 5 to 10ms after onset of the LV cavity potential. This initial activation of RV apex is coincident with the activation of inferior two-thirds of the left septum. Because the last one has opposite direction and has a bigger mass the RV apex activation is not manifested.

One year later in Italy (1971), Rossi<sup>20</sup> reinforces the trifascicular nature of the left intraventricular His system with a new anatomical and histopathological study. Demoulin and Kulbertus

**FASCICULAR LBBB: LAFB + LPFB**

**Figure 3 :** Atypical LBBB with initial q Waves in Left Precordial Leads.

et al<sup>21,22</sup> performed a histopathological viewpoint study of the LBB system in hearts from patients without conduction defects and found that the LBB would give off a third radiation or branch in 11 out of the 20 hearts. This structure traveled to the midseptal area and emerged either from the common left bundle (5 cases), from the anterior (3 cases) or posterior radiation (3 cases).

In 1972-73, Uhley<sup>23-25</sup> emphasized the same concepts in a classical editorial and a book chapter. In 1972, Myerburg et al<sup>26</sup>, working on isolated canine hearts, proved the trifascicular nature of the left His system by the mode of verified endocardial excitation. The same author would stress these ideas three years later<sup>27,28</sup>. The terminology of hemiblocks is criticized for the first time in 1973 by Hecht et al<sup>24</sup> coined the terms divisional/fascicular blocks as being more appropriate, since it was clear that the left branch split in three and not in two branches. In that same year, Gambeta<sup>29</sup> and Childers described the first electrocardiographic manifestations secondary to septal fascicular or septal focal block. These authors verified the intermittent, rate related appearance of Q waves in right precordial leads in the absence of antero-septal infarction. They also highlighted the frequent association with RBBB and/or LAFB.

Lazzara et al<sup>30</sup> in 1974 mapped canine left ventricular endocardial surface in vitro before and after lesions were placed in the proximal left bundle branch and proved the independent functional and anatomical behavior of the middle fibers of the LBB. In a subsequent publication, this author observed different types of refractoriness of the intraventricular system with three well-defined pathways. Additionally, the action potential and absolute, relative and functional refractory periods are significantly shorter in the LSF when compared with LAF and LPE. Phase 0 of the LSF is wider and consequently, conduction velocity is greater which justifies the centro-septal region activating 5 ms before the anterior and posterior ones. The three have an automatic phase 4, i.e. with discrete spontaneous elevation or diastolic depolarization<sup>31</sup>.

Kulbertus<sup>32,33</sup>, in successive publications from 1975 and 1976,

showed the distribution of the left branch and its three fascicles: anterior, posterior and septal. He studied 49 human heart specimens and found that only in 16 of them (15%) the third fascicle was missing, i.e. they were bifascicular. The remaining 85% was grouped in three types:

**Type I:** In 33 cases (74%), the septal division was easily identifiable, possible of a diameter greater than the other divisions, originating from the trunk of the left branch (18 cases), the antero-superior division (7 cases) or the postero-inferior division (9 cases).

1. Centro-septal area, in charge of a division of very variable and inconstant anatomy;
2. High anterior parasseptal area, just below the insertion of the septal fascicle of the mitral valve, which ends in the anterior papillary muscle.

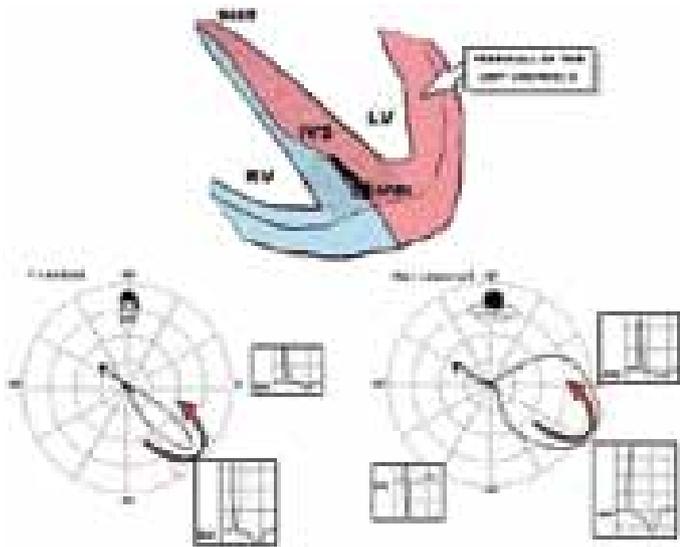
**Type II:** In 11 cases (23%), the author observed that there was a net made up by several fascicles coming from the antero-superior and postero-inferior divisions.

**Type III:** In 5 cases (2.4%), branch of the postero-inferior division with a prolongation of “false tendons” of the postero-inferior division.

Then, we should ask why the initial ventricular activation (10 ms) occurs in three points of the left septal surface and not in two, as it should be expected if the left His system were functionally bifascicular? Moreover, how to explain the cases of left bundle branch block (LBBB), divisional blocks (LAFB + LPFB) that present q wave in left leads, outshining the typical electrocardiographic pattern of LBBB? Mauricio Rosenbaum called them “left intraventricular blocks without changes in the initial part of the QRS”. The great master, in his colossal work, states that these cases are “hard to explain”<sup>11</sup>. In 1970, Medrano et al<sup>34</sup> proposed that in these cases, the fibers of the septal division would originate before the location or area of block in the postero-inferior or antero-superior divisions. As a result, middle-septal activation is preserved (IAM vector) and is responsible for those q waves in left leads and concealing the LBBB pattern. In other words, by totally blocking the two antero-superior and postero-inferior divisions, LBBB did not occur, as it should be expected in a situation where there are only two branches (**Figure 3**).

In 1976, Massing and James<sup>35</sup> examine histologically the conduction system of 13 human hearts. They found that the LBB anatomy was extremely variable, demonstrating multiple fiber groups which fanned out over the entire left septal surface.

In that same year, Kulbertus<sup>36</sup> studied the phenomenon of aberrant ventricular conduction after the provocation of premature atrial extra-stimulation with transvenous catheter electrode. The most common types of aberration were as follows: isolated RBBB in 28 patients, RBBB with LAFB in 21, LAFB in 17, RBBB along



**Figure 4 :** VCG of a non-obstructive form of HCM. The septum (S) and left ventricular free wall (FW) are thicker in their apical portions (FW), i.e. there is absence of normal decrease in the thickness from the base to the apex. The author suggests that the anterior and left dislocation of the QRS loop in the horizontal plane, and inferior and to the left in the FP (translated by R waves of greater voltage in V4 and DII) are secondary to selective hypertrophy of the apical inferior third of the septum. The absence of q in left leads was explained by ILBBB or LSFB.

with LPFB in 10, LBBB in another 10 cases, and incomplete LBBB in 6 patients. In 5 patients, an important anterior displacement of vectocardiographic QRS loop was observed in the horizontal plane, maintaining counterclockwise rotation and with more than 75% of the area of the loop in front of the X line. Three of these five cases had normal previous VCG, one had a dorsal infarction and an anterior infarct was present in the last one.

Subsequently, Hoffman et al<sup>37</sup> showed in a series of clinical electro-angiographic cases, several examples of LSFB translated electro-vectocardiographically into significant displacement of the ventricular depolarization forces to the front in the horizontal plane. Interestingly, this author explained the mechanism as a conduction delay in an anterior division of the left bundle branch system.

The Portuguese school led by Fernando De Padua<sup>38,39</sup> mentioned that an anterior displacement of the QRS loop, independent of the axis deviation in the FP, can be observed and may be a result of the involvement of centroseptal fibers. The ECG pattern is supposed to show prominent R waves in V1-V2 similar to those found in dorsal infarction concomitant with no abnormal axis deviation in the FP<sup>40</sup>.

In 1977 a classical book of electro-vectocardiography gave a detailed explanation of the new concepts of septal activation

and conduction delays. The initial vector (first 0.01s) represents the activation of the middle third of the left septal surface, and the vector of the 0.02 s, the apical-anterior region, which is oriented to the front and the left, and the delay in the apical area could cause anterior displacement of the QRS loop<sup>41</sup>, meanwhile, during this same year, Alboni followed a case of aortic regurgitation that initially presented with an ECG pattern of left ventricular enlargement and repolarization abnormalities (“strain”)<sup>19</sup>. One year later, ECG and VCG revealed divisional LBBB (LAFB + LPFB); though, maintaining septal activation (Q waves in left leads). This phenomenon was attributed to preservation of conduction by the middle septal fascicle.

In the late 70s’ in Japan, Nakaya et al<sup>42,43</sup> demonstrated the presence of prominent anterior forces (interpreted as LSFB) in association with coronary artery disease. Later on, the same authors carried out experiments in dogs to prove this concept.

These authors have also proposed that these prominent anterior forces are frequently seen in hypertrophic cardiomyopathy (HCM), particularly in the *non-obstructive form*. These cases may also represent true LSFB. The broad R waves from V2 to V4 and QRS loop in the horizontal plane with more than 2/3 in front of the X line associated to absence of initial convexity (initial 20 ms) to the right is attributed to the involvement of the left branch divisions (LSF and LASF), caused by hypertrophy and fibrosis of the septum. Nakaya analyzed 1000 consecutive ECGs and VCGs and found three cases of HCM with prominent anterior forces, two being non-obstructive forms and one obstructive.

In 1978, Iwamura et al<sup>44</sup> performed experiments in isolated canine hearts and provided evidence for the different electrophysiological properties of the left septal Purkinje arborizations in the face of premature stimulation. During 1978-79, Dabrowska et al<sup>45,46</sup> attempted the standardization of diagnostic ECG criteria of LSFB in anesthetized and intubated dogs after incision of the interventricular septum.

In 1979, Cheng et al<sup>47</sup> observed that high QRS voltage in midprecordial leads was a frequent finding in non-obstructive forms of hypertrophic cardiomyopathy. The ECG/VCG patterns were very suggestive of Left Septal Fascicular Block with prominent anterior forces. The author attributed the absence of q waves in left leads (85% of the cases) to incomplete LBBB or LSFB (**Figure 4**).

Also in 1979, Athanassopoulos<sup>48</sup> presented a case of an acute myocardial infarction of the infero-anterior wall showing Q waves in the inferior leads and from V2 to V4, which disappeared twelve hours later. The myocardial infarction was confirmed by a typical enzymatic curve. The author attributed the ECG changes to transitory block of the septal region.

In the same year, the Brazilian school led by Prof. Joao Tranches<sup>49</sup> exemplified with great detail the electro-vectocardiographic aspects of isolated LSFB, and the more frequent cases seen in

association to other intraventricular blocks, both in coronary insufficiency and Chagas disease.

In 1980, followers of the Tranchesi School<sup>50</sup> published a clinical-electro-vecto-angiographic correlation of the dromotropic disorder of the septal division associated with critical injury of left anterior descending artery. The authors found prominent anterior forces in V1 and V2 (even R/S >1 in V1) in patients without posterior involvement and confirmed critical injury of the left anterior descending artery. In particular, if primary T wave abnormalities were present, ischemic LSFb should be suspected.

In 1981, Nakaya and Hiraga studied this phenomenon, by producing cuts in the Purkinje arborizations of the middle septal region of dogs<sup>43,51</sup>. They verified anterior and left dislocation of the QRS loop, along with activation delay in the apical region. Accordingly, the block in this area (11 dogs) resulted in a discrete delay in the apical area activation and in the Z lead (postero-anterior and corresponding to the V2 line), and an increase in voltage was observed from 1.4 to 2.6 mV by dislocation of the QRS loop to the front. Additionally, the block in the postero-inferior division (9 dogs) caused discrete delay in activation of the postero-basal region. On the other hand, the concomitant block in both areas (middle-septal and postero-inferior: 11 dogs) caused an important delay in the activation of an extensive area in the postero-basal and apical regions, significantly affecting the axis of X with the appearance of S and important deviation to the right of AQRS in the FP. The authors concluded that the Purkinje central septal block per se might cause significant electro-vectocardiographic changes by its capacity of interconnection. In all the cases that the left conduction system was anatomically analyzed, wide morphological variability was found. Interestingly enough, the LBB was never organized in two well-defined fascicles.

The main disciples of the Tranchesi School, Prof. Moffa et al<sup>52,53</sup> have showed in several publications the existence of LSFb, both in Chagas disease and in coronary artery disease. These authors have the merit of illustrating the intermittent character of the phenomenon.

In 1983, Inoue et al<sup>54</sup> in a vectocardiographic experimental work in dogs, verified that causing a block in the distribution area of the septal fascicle ("septal Purkinje network" called by the authors as SEP) a discrete delay is produced in epicardial activation of the septal area and in a 30% of instances an anterior dislocation of the QRS loop in the HP occurs. When the block was caused in the region of the antero-superior division, significant changes were not observed in VCG, with only superior and left deviation of the terminal vector of QRS being recorded. Finally, when the block was produced in both regions, the delay in activation reached an area much greater, from the anterior basal region up to the apical area and the direction of the maximal vector in the FP showed a significant superior and left dislocation. The authors concluded that the block in the SEP area per se, produces

electrical changes with sufficient traits to be characterized.

Sakaguchi et al<sup>55</sup> in 1988 performed an autopsy study of the left intraventricular system in 13 normal human hearts obtained from subjects aged 50 to 80 years. The samples were stained with hematoxylin-eosin or by the van Gieson method and examined by light microscopy. Reconstruction was performed using a two-dimensional system in order to histologically differentiate the bundle cells from Purkinje cells. The LBB bifurcated into the anterior and posterior radiations and the cells in the septal portion were almost all Purkinje cells except in two cases showing a septal branch between the two radiations. This difference in structure may explain why the centro-septal region is activated 5 ms before the others.

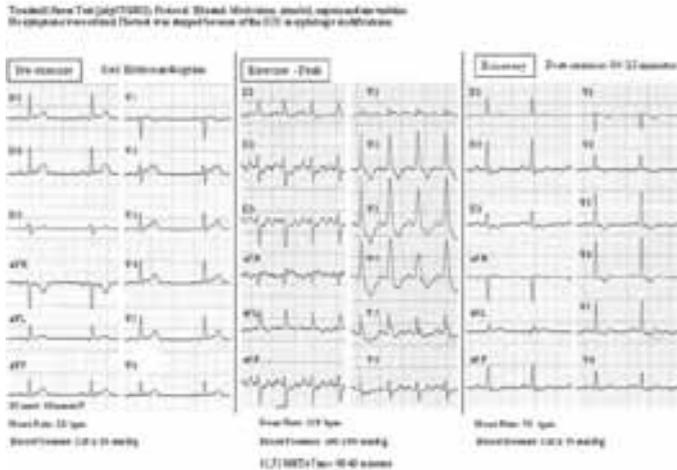
In 1992, Mori et al<sup>56</sup>, proposed criteria for the diagnosis of left septal fascicular block, based on the normal limits of the R and S waves and R/S of V1 and V2. The criteria can be summarize as follows:

1) Exclusion of other causes responsible of anterior QRS displacement, such as normal variant, chest anomalies (counterclockwise rotation of the heart in the longitudinal axis), type A RVH, RBBB and dorsal infarction.

2) One of the following two voltage criteria should also be satisfied. (i) R/S in V1 > 2, and R in V1 ≥ 5 mm, (ii) R/S in V2 > 2, and R in V2 ≥ 15 mm, or S in V2 < 5 mm. The frequency of left septal fascicular block diagnosed by these criteria was 3.5% among a large group of hospitalized patients. This frequency was less than that of the LAFB or RBBB, but it was higher than that of bilateral BBB.

In an elegant study published in 1996, Dhala et al<sup>57</sup>, demonstrated the trifascicular nature of the left His system during catheter ablation of the right branch. Twenty-five patients underwent catheter ablation of the RBB, either for bundle branch reentrant tachycardia, or inadvertent or deliberate right bundle ablation during atrioventricular junctional ablation for rate control. These authors divided all the patients (25) in two groups: Group I: the patients in this group (n=11) did not have previous signs of pre-ablation dromotropic disorder. After the ablation of the right branch only RBBB emerged. Group II: made up by those patients (n=14) with some right bundle branch conduction delay that was present before the ablation of the right branch. In 12 out of 14 a qR pattern in V1 was observed and interpreted as LSFb associated to AQRS deviation, either to the right (LPFB: 3 patients) or left (LSFB: 4 patients). The authors concluded that the trifascicular nature of left intraventricular conduction became apparent, or was unmasked, after involvement of the right branch.

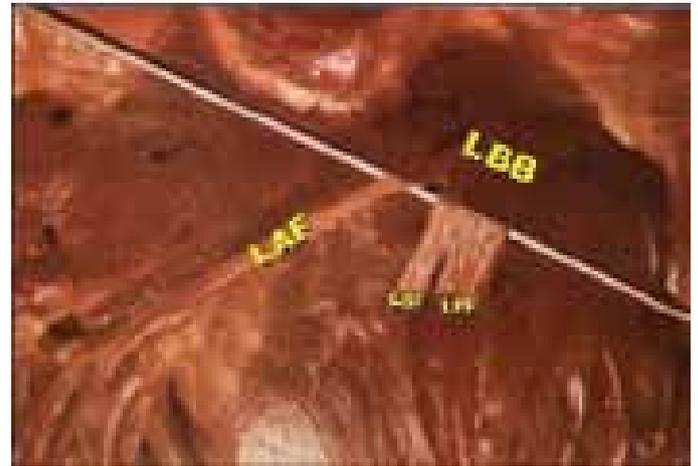
In 1997, Moffa<sup>58,59</sup> presented an undisputable case of a 69 year old patient with severe three-vessel disease who initially presented with the following ECG: sinus rhythm, first-degree AV block, QRS duration of 110 ms, QRS axis in 0°, Q waves from V1 to V6, followed by broad R waves from V1 to V4,



**Figure 5 :** Exercise-induced left septal fascicular block: an expression of critical obstruction of left anterior descending artery. Tall R waves in V1-V4 can be a normal variant in only 1% of patients and it is a hallmark ECG finding in left septal fascicular block. The proposed ECG criteria for LSF are: prominent R waves in V1-V3 (Prominent anterior forces or PAF), minimal QRS prolongation (QRS < 120 ms), T wave morphologic alteration (flat or inversion: very debatable and variable), frequent initial q wave in right and/or middle precordial leads and clinical absence of other causes of PAF<sup>64</sup>.

which decreased voltage in V5 and V6. The vectocardiogram presented prominent anterior forces of the QRS loop, with middle-area vector in the horizontal plane in +60°. This pattern was interpreted as LSF in absence of other causes that may determine prominent anterior forces. Two years after, the patient was readmitted with acute pulmonary edema and cardiogenic shock due to a myocardial infarction. The ECG had important changes after two days: QRS ( 160 ms), QRS vector of +100°, R waves in leads II, III and AVF that increase from II to III, QS in lead I, AVL, AVR and V1, rS in V2 and V3, R wave was notched in V5 and V6. This was a transient pattern that returned to the initial one in 24 hours.

In 2001, Sanches, Moffa and Sosa<sup>60</sup> from the InCor, Sao Paulo, Brazil studied five patients that met ECG/VCG criteria of LSF, who underwent biventricular endocardial catheter mapping. These ECG/VCG patterns were characterized by prominent anterior forces with increased ventricular activation time in V1 and V2, small initial q wave in V2-V3, R wave of V2 and V3 >15 mm, sharp-pointed R wave in V2-V3 leads with slow descendent ramp, absence of q wave in left precordial leads V5, V6 and I (by absence of the vector I AM), increasing voltage of R waves in V3-V4 and decreasing from V5 to V6. The QRS loop of VCG in the HP showed a marked anteriorization of the QRS loop, which is located predominantly on the left anterior quadrant and has a CW rotation. The authors observed conduction delay in the middle left septal surface and in the free wall of the LV - named anterior conduction delay - (sites 9, 10, 11 and 12 from the



**Figure 6 :** Demonstration of the type III LSF anatomic variation. Figure extracted from the original book by Rosenbaum MB, et al. (Modified from reference number 66 with permission). The LSF clearly originates from the LPF.

original description by Josephson et al<sup>61</sup>. These sites correspond to the area of distribution of the LSF.

Recently (2002-03), Prof. Rex MacAlpin<sup>62,63</sup> from Division of Cardiology at UCLA, presented possible models of LSF ventricular activations, proposed diagnosis criteria and showed illustrative cases of probable and possible LSF based on deductive reasoning. The author concludes that the LSF is a polymorphic conduction defect which may explain some previously inadequately understood electrocardiographic abnormalities.

Finally, we have recently presented a case report of intermittent exercise-induced left septal fascicular block as an expression of severe myocardial ischemia, with critical obstruction of left anterior descending artery<sup>64</sup> (**Figure 5**).

## Conclusions

The trifascicular nature of the human left His system has been a controversial matter. After conducting an extensive chronological review on this subject, we conclude that in most cases, the left His system is trifascicular. In fact, the LSF has distinct ECG and VCG patterns. It is characterized by prominent anterior forces on the horizontal plane, which can be distinguished from the normal variant with counterclockwise rotation of the heart around the longitudinal axis, types A or B patterns of RV enlargement, misplaced precordial leads, lateral myocardial infarction<sup>65</sup>, RBBB, type A WPW syndrome, HCM, Duchene's muscular dystrophy, and dextrocardia among other. Although the term "hemiblock" has been very appealing, it is also inappropriate. If this original bifascicular conception of the left His system were correct, as conceived by Rosenbaum and his school, how would it be possible to explain the following facts?

The activation of the middle third of the septum occurring 5 ms before the postero-inferior and antero-superior regions

Rosenbaum himself said that the middle-septal activation occurred in most cases from “false tendons” anterior to the postero-inferior division. For him, the distal postero-inferior division was a fan-like structure, and its anterior “pseudo-tendons” would be responsible for the activating the middle-septal region. Currently, it is known that one of the anatomical variations of the septal division is precisely the one that depends on the postero-inferior division (10% of the cases or type III). The intermittent anterior displacement of the QRS loop in the HP in certain cases of critical injury of the anterior descending artery in the absence of other factors that may cause the same VCG changes?

- Finally, in some photographs shown in Rosenbaum's masterpiece, the presence of a third septal branch is evident<sup>66</sup>. **(Figure 6)**

In a recent review article, Dr. Marcelo V. Elizari<sup>15</sup> used the old terminology (hemiblocks). The author wrote: ***“In fact the existence of middle septal fibers cannot be disregarded, and as such the functional and, probably clinical significance of the middle or septal fascicle cannot be totally ignored either”.***

In summary, we believe that the words of the Portuguese researcher Fernando de Padua are very eloquent: **“IF HEMIBLOCKS DO EXIST, THEY ARE ONLY TWO - IF A THIRD ONE IS POSTULATED, HEMIBLOCKS DO NOT EXIST!”**<sup>38,39</sup>

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# ECG Quiz

COMPILED BY

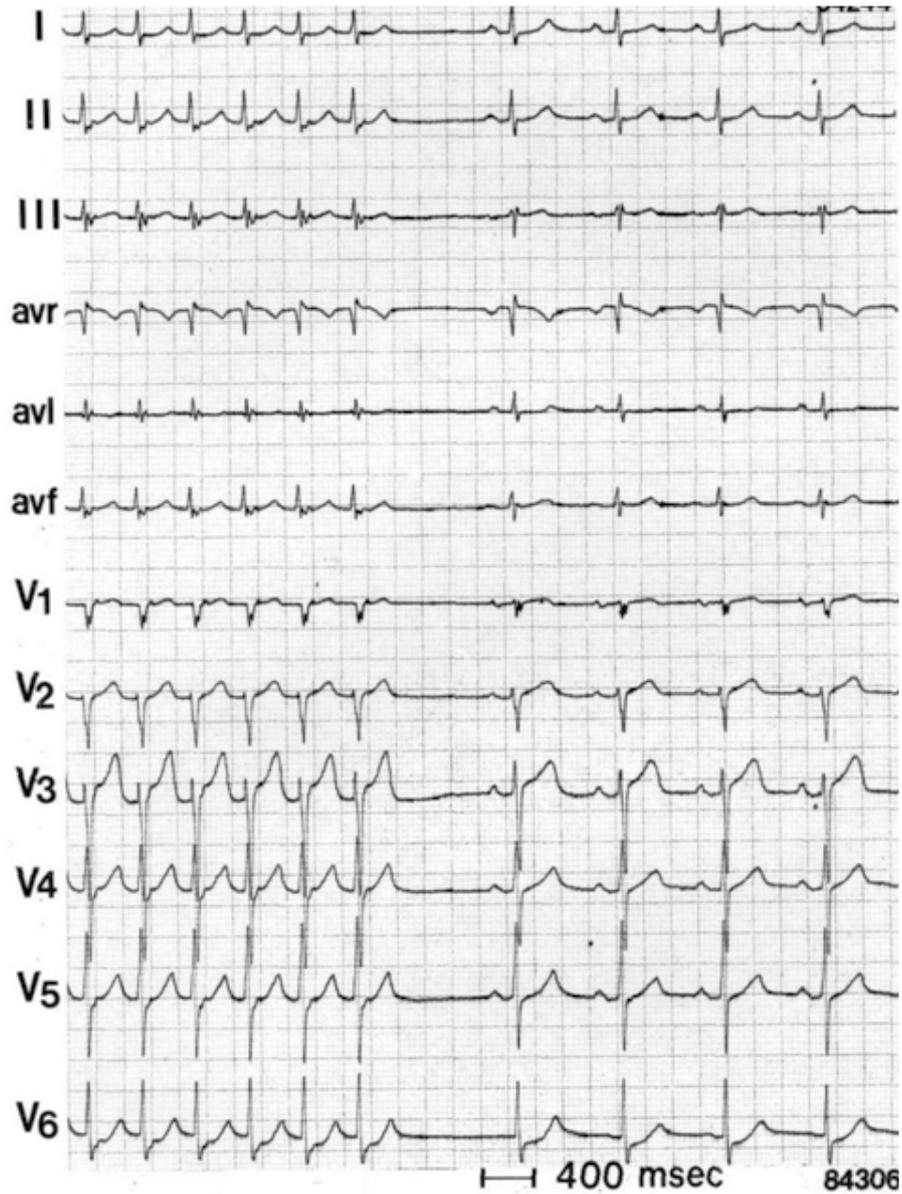
**Yash Lokhandwala\*, Gopi Krishna Panicker#**

\* Arrhythmia Associates

# Quintiles ECG Services

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

## ECG - 1



1. The ECG shows:
  - a. Atrial tachycardia
  - b. AVNRT
  - c. Atrial flutter
  - d. CMT using an Accessory Pathway for VA conduction

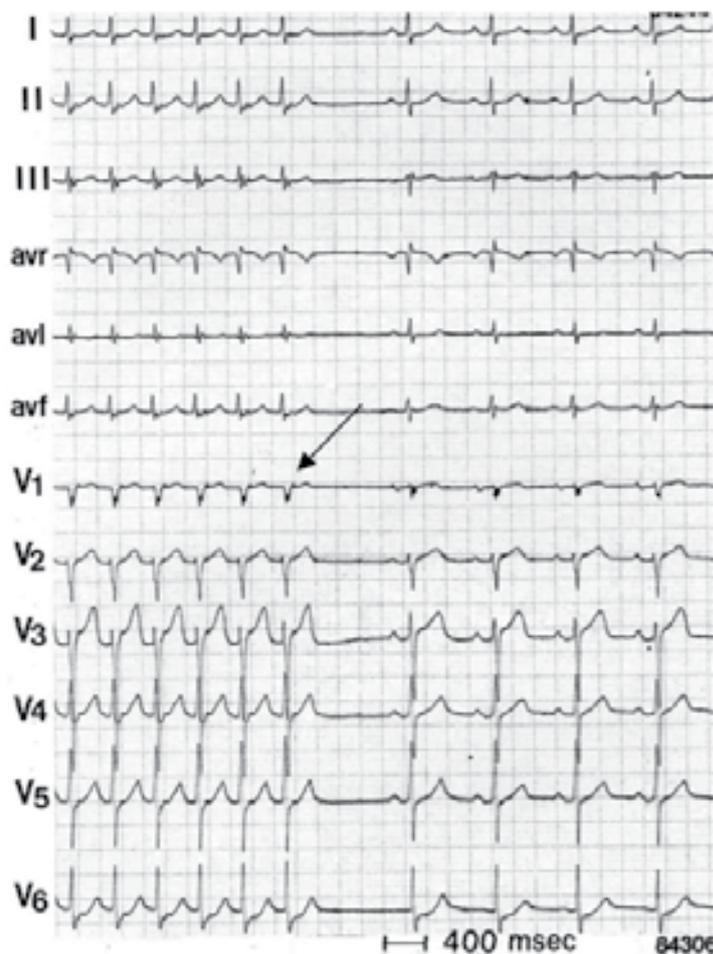
For correct answer see overleaf

**ECG - 1**

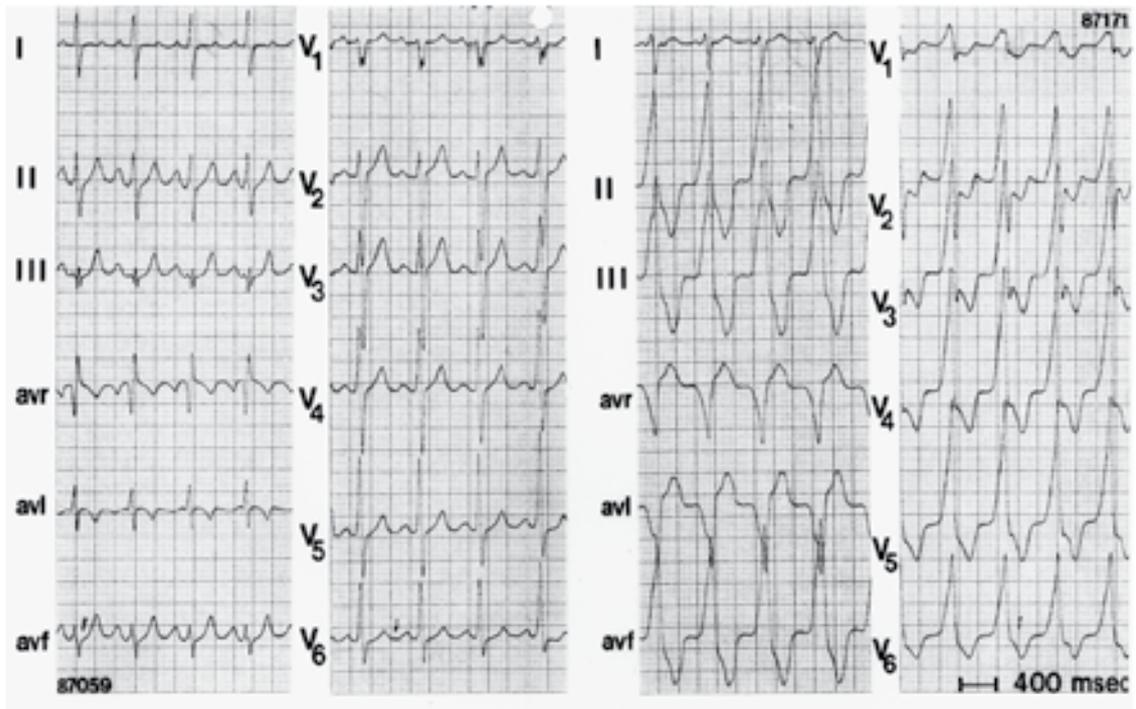
**The correct answer is 'b' – AVNRT**

The ECG shows a regular narrow QRS tachycardia on the left. There is spontaneous termination and the sinus rhythm on the right side appears unremarkable.

During the tachycardia, the QRS complex shows subtle differences from that in sinus rhythm. For instance, there is a clear-cut delayed r' seen in lead V1 (arrow). The end of QRS complex in the inferior leads also shows a wide, notched S wave which is absent in sinus rhythm. These findings suggest that the P wave is buried within the QRS complex. Atrial tachycardia will generally have a clear-cut P wave visible before the QRS complex. Atrial flutter would have an undulating baseline and an atrial rate to 200-300 bpm. Both these features are absent in this ECG. Orthodromic AVRT (choice d) would show P waves **AFTER** the QRS complexes. Then, there need not be pre-excitation during sinus rhythm through a concealed accessory pathway.



## ECG - 2



2. This ECG show:
- SVT with RBBB
  - VT
  - Ventricular pre-excitation
  - Don't know

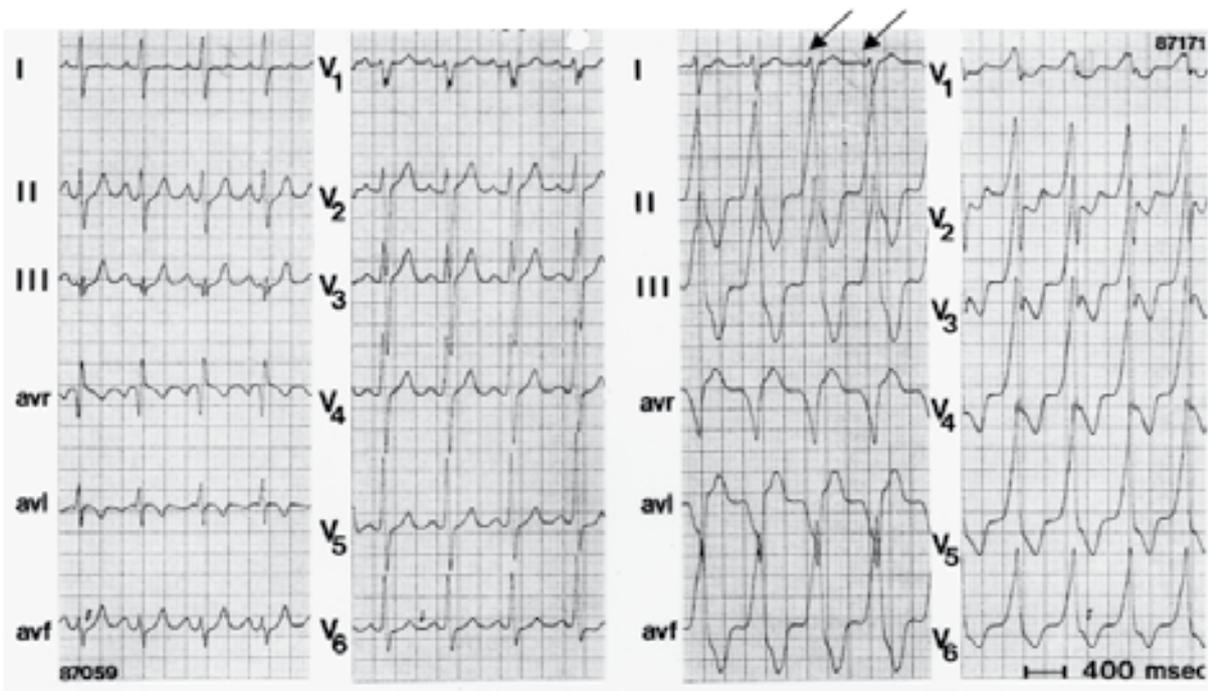
For correct answer see overleaf

## ECG - 2

The correct answer is 'b' – VT

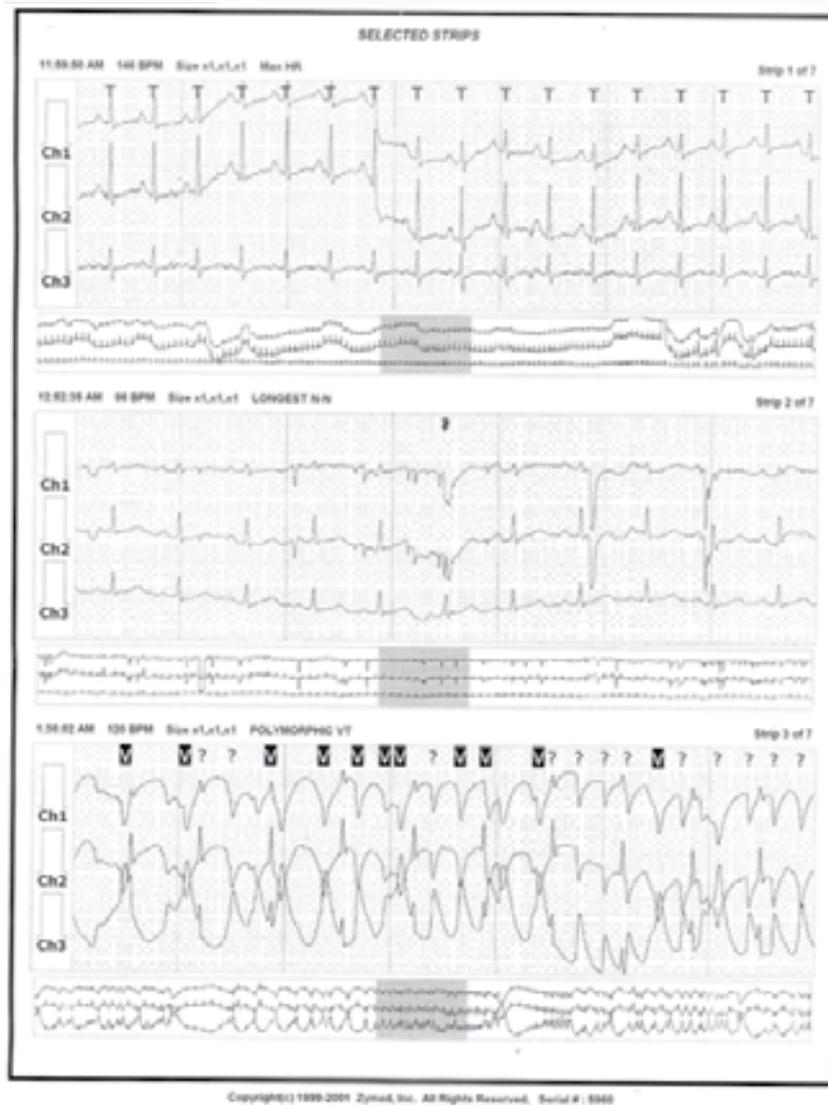
The panel on the left during sinus rhythm shows an indeterminate QRS axis and mild notching of the S wave in lead V1. Other than this, everything appears normal. The rhythm on the right shows wide QRS complexes at the rate of 120 per minute. P waves cannot be clearly seen. The complex which appears like a P wave in lead V1 (arrows) is actually a part of the QRS complex. This is confirmed by the onset of the QRS complex in the adjacent lead II. The QRS complex measures 200 ms and does not conform to any bundle branch block pattern. There is a monophasic R wave in lead V1 and a QRS axis of  $+120^\circ$ . The precordial leads nearly appear to have a positive stereotypy pattern. Since there is no typical bundle branch block pattern, SVT is excluded.

As for pre-excitation, the only possibility will be a pre-excited anti-dromic tachycardia. However, this does not conform to any pre-excitation pattern. Most importantly, patients with pre-excited tachycardia also invariably show pre-excitation in sinus rhythm.



## ECG - 3

Middle aged lady with palpitations. Referred for further evaluation after this Holter....



3. Long-term Rx plan:
- Amiodarone
  - EPS ± RFA
  - ICD
  - CAG ± revascularization
  - None of the above

For correct answer see overleaf

## ECG - 3

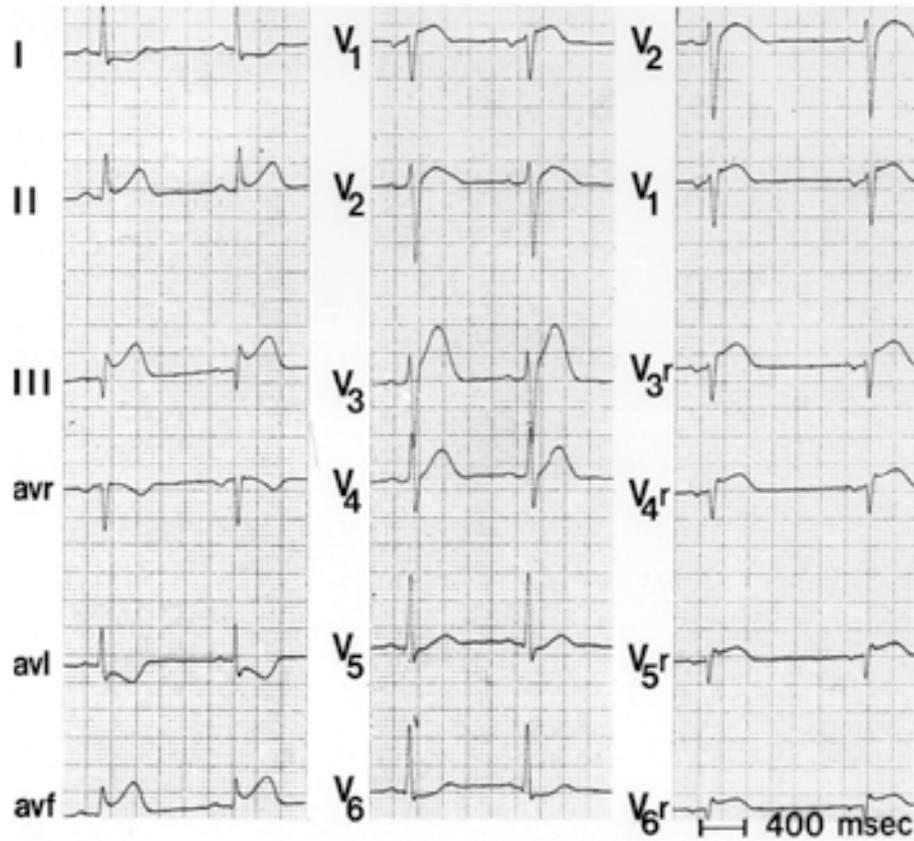
The correct answer is 'e' – None of the above

The top panel shows sinus tachycardia. The middle panel shows a normal sinus rate with several artefacts as pointed out by the asterisks. The bottom panel in channel 2 shows regularly occurring QRS complexes as marked by the arrows. The rest of the bizarre deflections are artefacts. During a Holter study, when the patient is moving, the electrodes are loose, if the patient is sweating or if there has been poor skin preparation, then one sees a lot of artefacts.

The computer software often does not recognise these, as seen in this example, where it has labelled these artefacts as polymorphic VT. In fact computers are largely useless for the interpretation of arrhythmias even in today's software technology. It is important to manually over-read rhythm diagnoses; otherwise one will be misled and wrongly treat the patient. All this patient required was reassurance.



## ECG - 4



4. **Where is the occlusion?**
- Proximal RCA
  - Cx
  - Proximal RCA with RV MI
  - Proximal LAD

For correct answer see overleaf

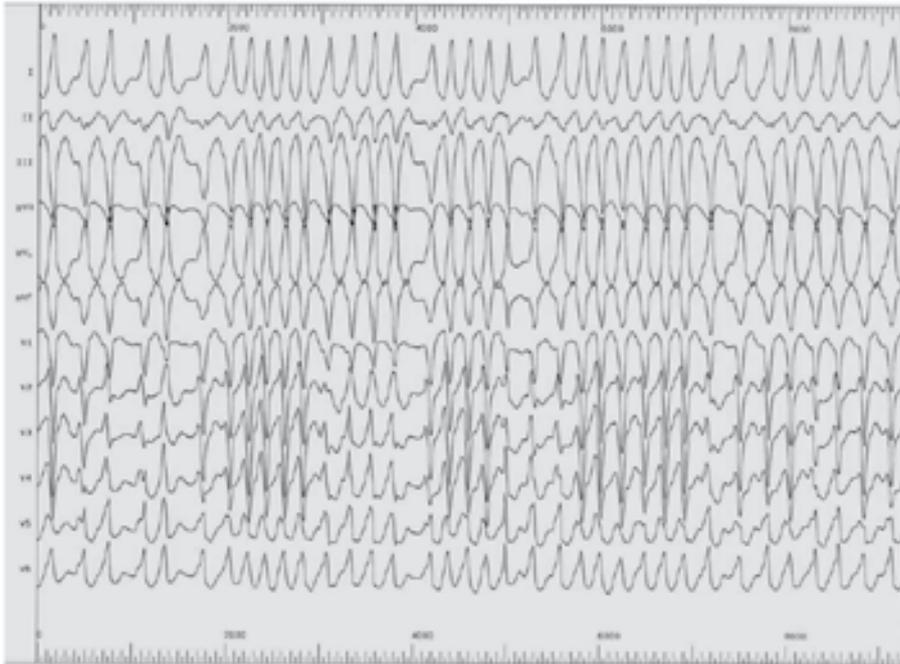
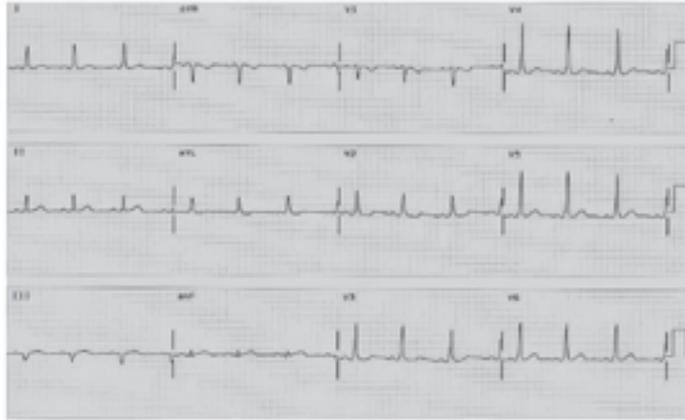
**ECG - 4**

**The correct answer is ‘c’ – Proximal RCA with RV MI**

There is gross ST elevation in the inferior leads maximally seen in lead III. There is also mild ST depression in lead I. This points to a right coronary artery occlusion. The precordial leads however show ST elevation from leads V1 to V3. One would instinctively think of an anteroseptal MI due to LAD occlusion. However, the right sided leads, specifically leads V3R to V5R show marked ST elevation alongwith a positive T wave. So clearly one is dealing with right ventricular infarction. Looking at it in entirety, the inferior and right ventricular wall would be infarcted only if the RCA is occluded proximally. Since the right ventricle lies anteriorly, the anterior precordial leads V1 to V3 could show ST elevation in this setting. Typically, in RV infarction, the ST elevation is most marked in lead V1. It is important to be aware of this pattern of RV infarction especially when one comes up with multiple vessel disease in unstable angina or acute MI setting, where urgent intervention is contemplated for the culprit vessel. Also this has bearing on the medical management as regards fluids, nitroglycerine and inotropic agents.

## ECG - 5

19-year old with history of palpitations and a syncopal episode.



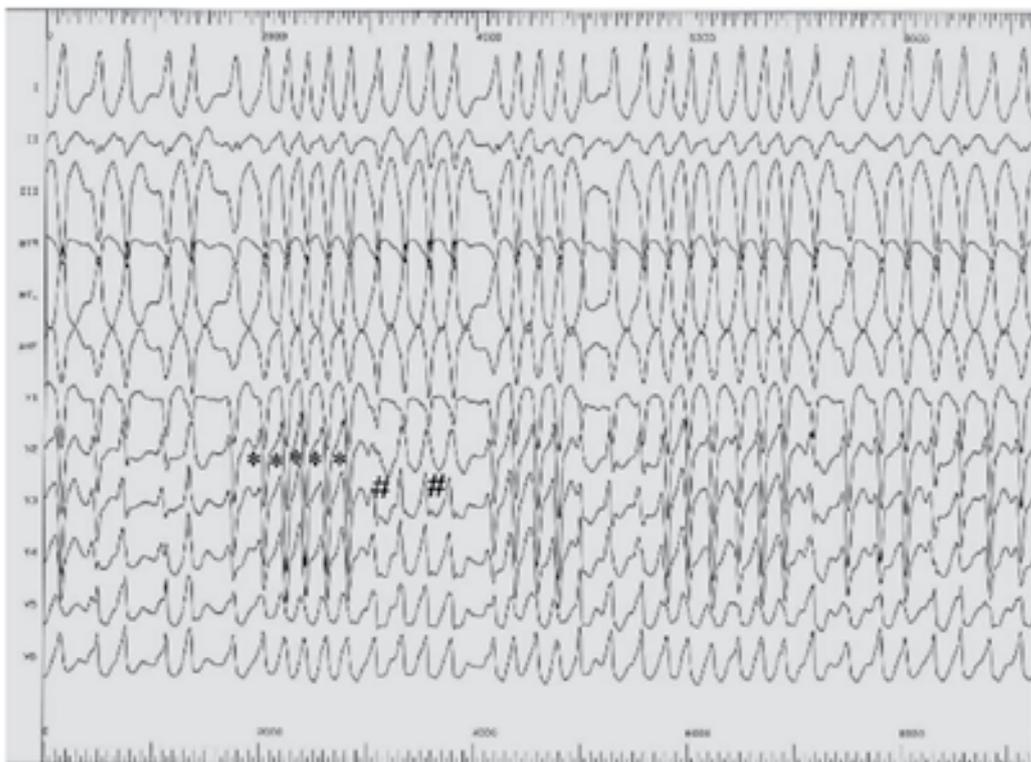
5. **This ECG shows:**
- Polymorphic VT
  - Atrial fibrillation
  - Monomorphic VT

For correct answer see overleaf

**ECG - 5**

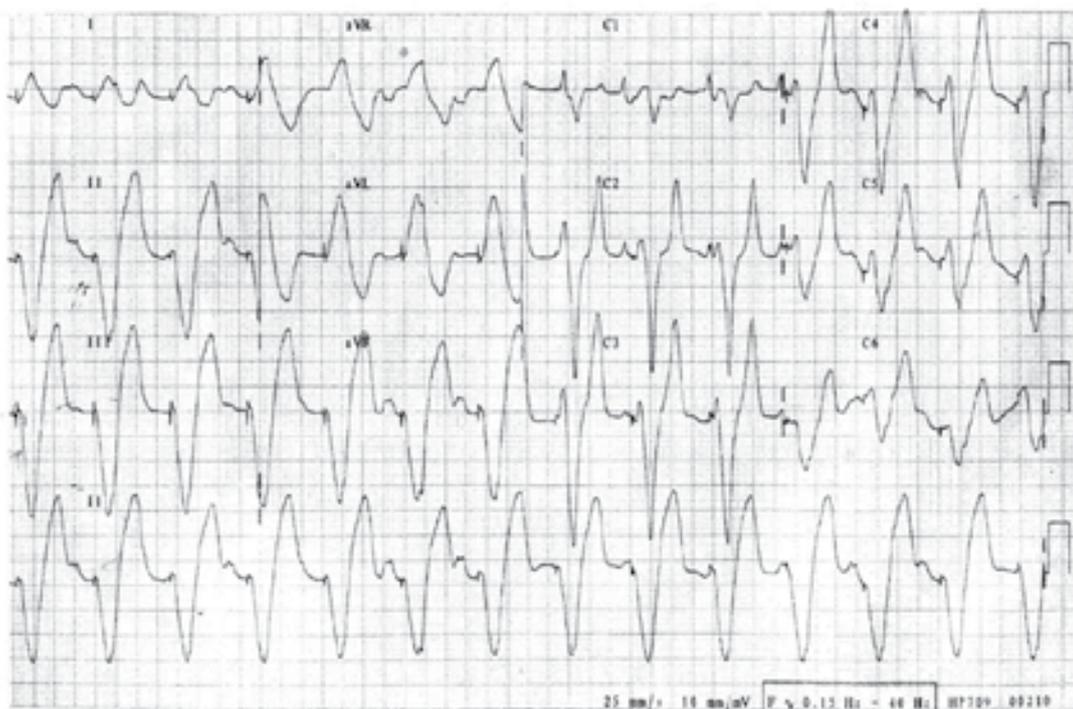
**The correct answer is ‘b’ – Atrial fibrillation**

The basal ECG shows a hint of pre-excitation as evidenced by a borderline short PR interval in lead V4 and a hint of a delta wave. The arrhythmia is fast, broad and irregular. The RR interval varies markedly. There are two types of QRS complexes in the ECG: (1) those which are predominantly negative in leads V2 and V3 showing rS (\*) and (2) those which show a monophasic R wave as shown by a # mark. A polymorphic ventricular tachycardia would have beat-to-beat variation in the QRS complexes. A monomorphic VT would never be so irregular. In this case, the very rapid ventricular rate is because of conduction down two accessory pathways. The WPW pattern during atrial fibrillation becomes much more marked because AV node is refractory during rapid rates. In sinus rhythm, however, the AV nodal conduction masks partly the WPW pattern.



## ECG - 6

Uncontrolled HT. Pacemaker implanted for bradycardia a few years ago.  
Patient now comes with ill-health and dyspnea.



6. Likely cause for symptoms:
- Ischemia
  - Renal failure
  - Pacemaker malfunction

For correct answer see overleaf

**ECG - 6**

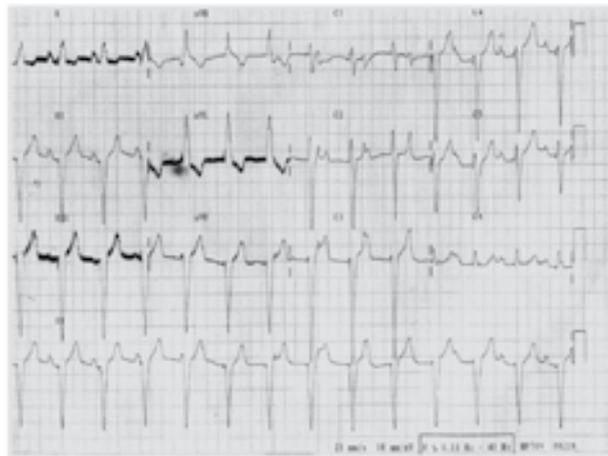
**The correct answer is ‘b’- Renal failure**

The QRS complexes are all paced. They are extremely wide measuring 280 ms.

The P waves bear no relation to the QRS complexes. Hence, we are talking of single chamber ventricular pacing. The P waves is distinctly abnormal as seen in lead C1 (V1). This suggests a biatrial abnormality. The P wave abnormality could be because of ventricular dysfunction and enlargement because of long-standing uncontrolled hypertension.

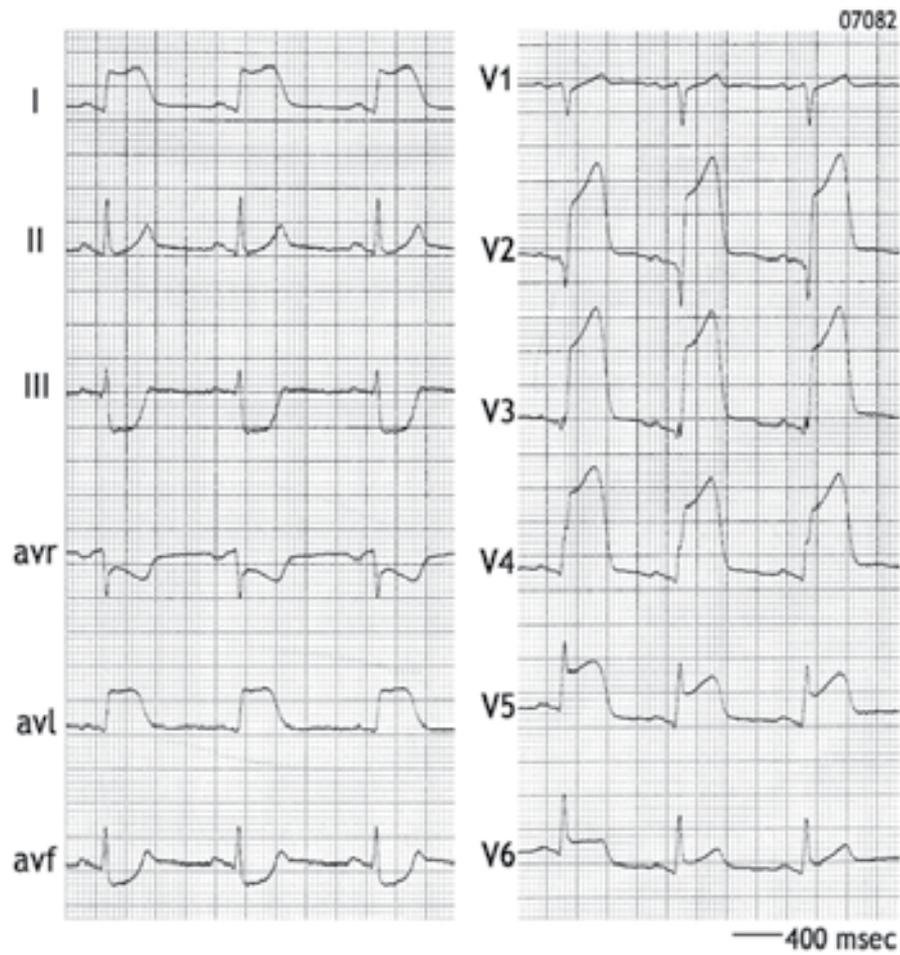
The T waves are extremely tall and peaked. The height measures almost 18 mm in C3 (V3) and the peaked nature is best seen in lead C2 (V2). Ventricular pacing does give wide QRS complexes. However, these are typically in the range of 140-180 ms, depending on the site of pacing and the size of the heart. A QRS complex of 280 ms is distinctly unusual for ventricular pacing. Secondary T wave change opposite to the QRS complex is expected with ventricular pacing. However, the peaked nature is distinctly abnormal. Also, the amplitude is too high for secondary T wave changes. This ECG picture along with the clinical scenario suggests that the patient is having a metabolic problem. Hyperkalemia is the most important cause for this T wave abnormality. If one then evaluates this retrospectively, then the QRS widths suggesting myocardial delay is also a feature of sever hyperkalemia.

**Narrowing of QRS after correction of hyperkalemia**



The next ECG after correction of hyperkalemia shows marked reduction in the QRS widths to about 120 ms. The T waves also become much lower measuring only 8 mm in lead V3. The P wave abnormality is still seen suggesting residual ventricular abnormality.

## ECG - 7



7. **The ECG shows:**
- Left main occlusion
  - LAD occlusion proximal to S1 and D1
  - LAD occlusion distal to S1, prox. to D1
  - Distal LAD occlusion

For correct answer see overleaf

**ECG - 7**

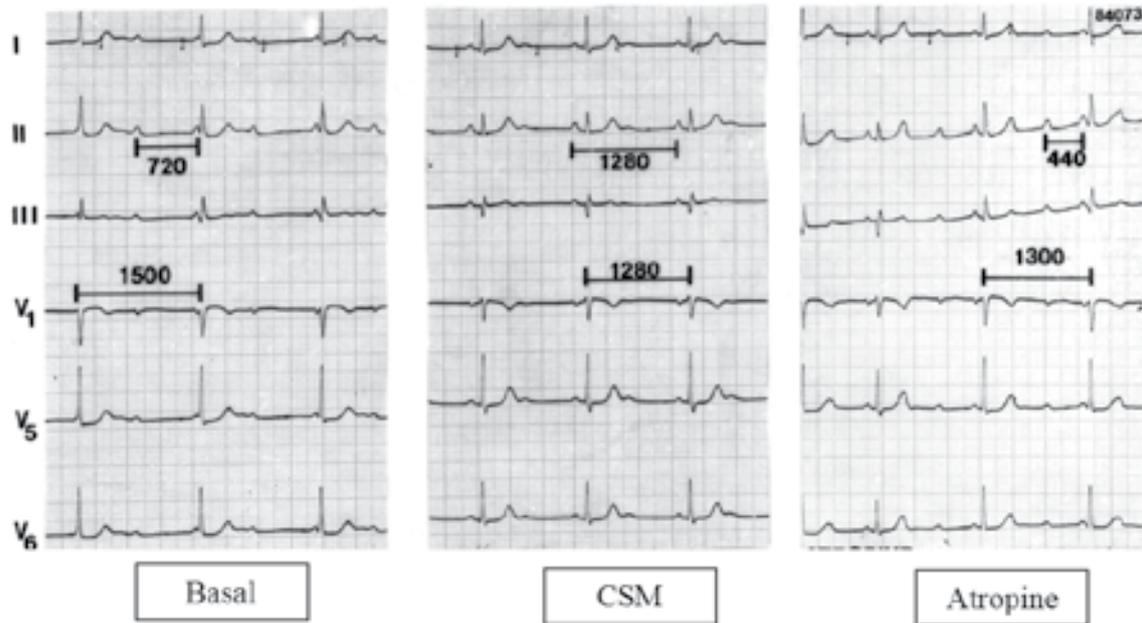
**The correct answer is 'c' - LAD occlusion distal to S1, prox. to D1**

The limb leads show gross ST elevation in leads I and aVL. In fact, there is Grade 3 ischemia (Sclarovsky et al). Grade 3 ischemia indicates that the ST elevation is more than 50% of the R wave height and may even lead to distortion of the descending limb of the QRS complex. This indicates a large infarction and a grave prognosis and a propensity to ventricular fibrillation. The precordial leads similarly show grade 3 ischemia from leads V2 to V5 and also ST elevation in lead V6. There is importantly no ST elevation in lead V1. Leads III and aVF show remarkable ST depression and so does lead aVR.

So, we are talking of infarction of the anterior wall, as evidenced from the precordial leads. The limb leads suggest that the area proximal to the diagonal artery is infarcted as seen by ST elevation in leads I and aVF. The proximal septum seems to be spared. Signs of proximal septal infarction are ST elevation in V1, RBBB and ST elevation in aVR. None of these are present. In fact there is ST depression in lead aVR confirming that the proximal septum is spared.

In a distal LAD occlusion one would in fact have ST elevation in inferior leads and ST depression in aVR and aVL. So clearly we are talking of an occlusion which is distal to the first septal artery but proximal to the first diagonal artery. A left main occlusion would in fact show an elevation in lead aVR and not much ST elevation in precordial leads because of cancelling out of influences of infarction of the anterior and posterior walls.

## ECG - 8



8. Where is the site of AV block?
- AV node
  - Bundle of His
  - Bundle branch system
  - Can't say

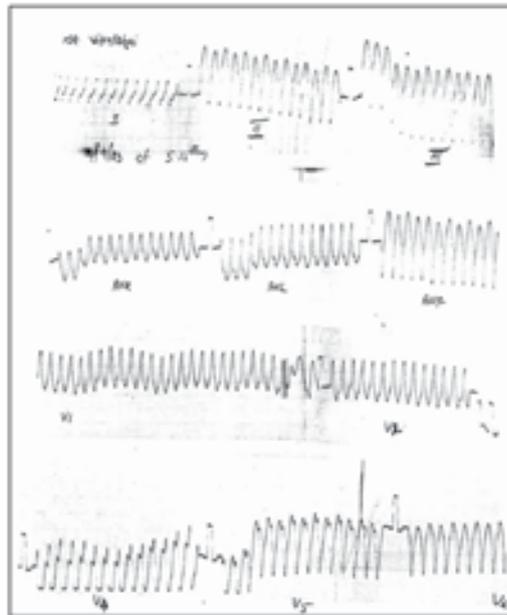
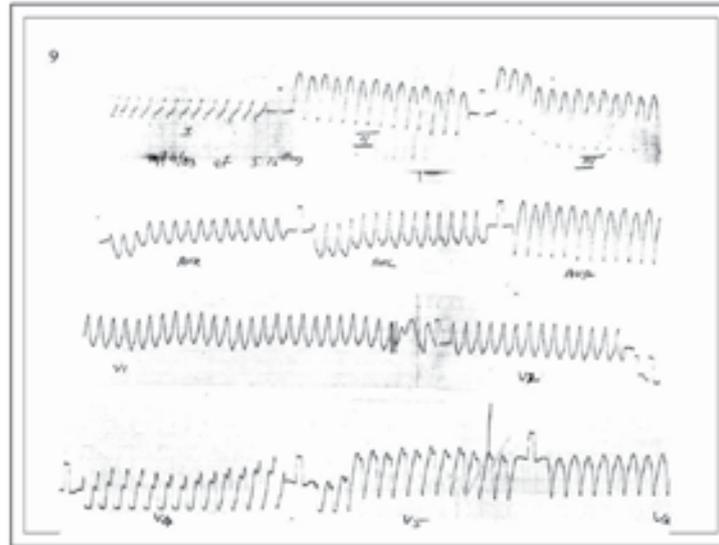
For correct answer see overleaf

**ECG - 8**

**The correct answer is ‘b’ – Bundle of His**

The left panel shows a sinus cycle length of 720 ms (approximately 80 bpm). There is complete AV block with an escape ventricular rhythm at the rate of 40 bpm. The QRS complexes are normal. The middle panel following carotid massage shows slowing of the sinus rate as expected. Interestingly, there is 1:1 AV conduction with normal PR interval. Improvement of AV conduction with carotid sinus massage rules out the AV node as the site of block. This is because both sinus nodes and AV node get inhibited by CSM. The panel on the right after atropine shows acceleration of the sinus rate of 125 bpm (440 ms). However, the AV conduction is absent and there is again complete AV block. The escape ventricular rhythm is at 1300 ms corresponding to a rate of 46 bpm. Again, atropine would improve the sinus node automaticity as it does here. However, it is also expected to improve the AV nodal conduction. So, the AV block again confirms that the AV node is not the site of the block. Clearly, the bundle branch systems are not involved because all three ECGs do not show any evidence of bundle branch block.

28 yr-old, first episode of palpitations, syncope



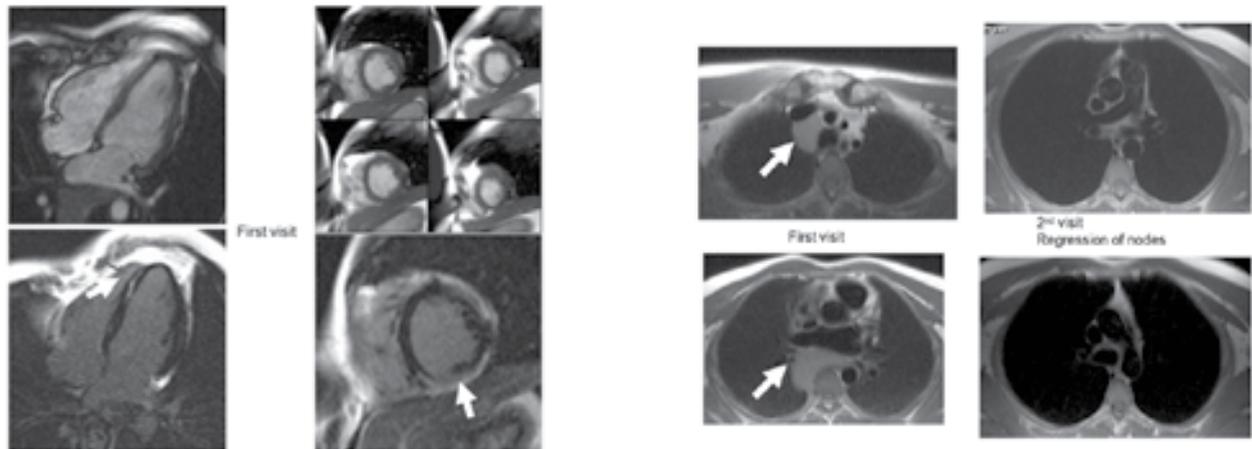
9. This arrhythmia is:
- SVT with RBBB
  - Antidromic tachycardia
  - VT from RV
  - VT from LV

For correct answer see overleaf

## ECG - 9

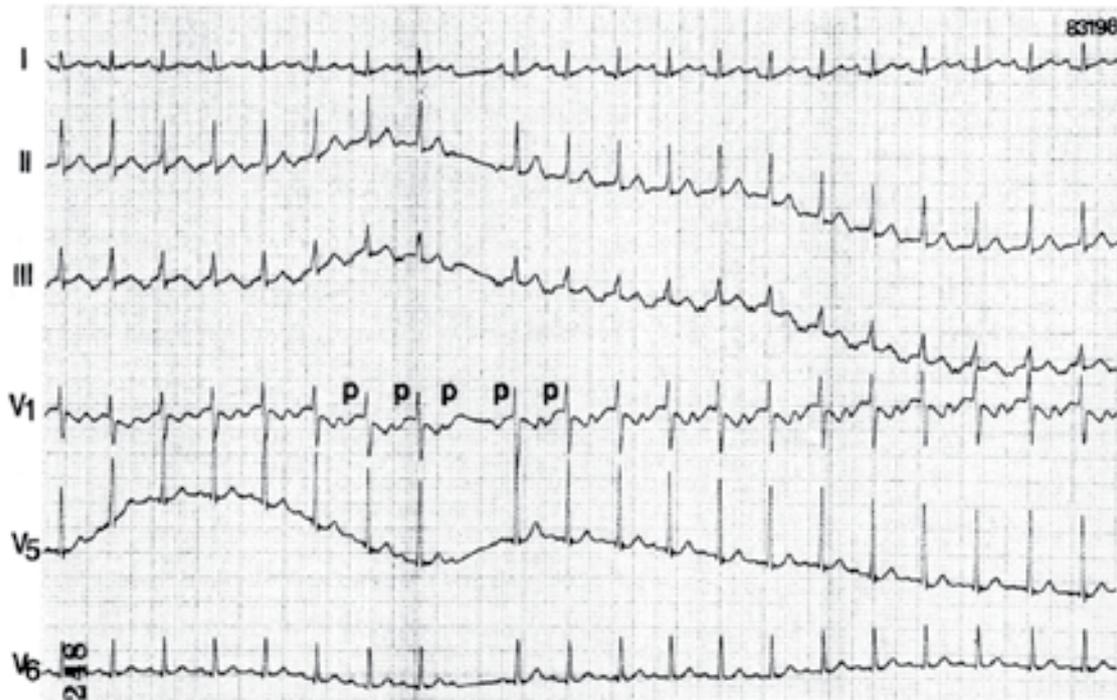
**The correct answer is ‘d’ – VT from LV**

There is an extremely rapid wide QRS tachycardia at the rate of almost 300 bpm. The QRS width is approximately 180 ms. The QRS axis is  $-60^\circ$ . There are monophasic R waves in V1 and a QS complex in leads V6. Clearly, this does not correspond to a typical RBBB. It also does not correspond to any pre-excitation pattern. A QS complex in V5 and V6 is enough to make a concrete diagnosis of VT. In this young man, it is very unusual to have a VT of this nature. In view of a positive QRS in lead V1 (RBBB like) the VT is arising from LV. The QS complex in V6 additionally tells us that it is arising close to the apex of the LV.



The cardiac MRI showed scattered areas of infiltration in the LV near the apex and posterior walls (arrows). This patient was initially treated with steroids with no response (with a presumed diagnosis of cardiac sarcoidosis). The CT scan had also shown markedly enlarged lymph nodes in the para-hilar and para-tracheal region as shown in the next figure. He was later given empirical anti-tuberculous treatment which showed marked regression of lymph node after 3 months. So the presumed retrospective diagnosis was tuberculosis with thoracic lymphadenopathy and myocardial infiltration, manifesting as VT. It is important to be aware of these unusual entities especially in our country when the younger age groups present with bizarre ventricular arrhythmias.

## ECG - 10

**10. What kind of tachycardia?**

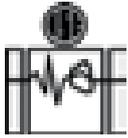
- Atrial tachycardia
- AV Node reentrant tachycardia (AVNRT)
- Atrial flutter
- Tachycardia using an accessory pathway for VA conduction

For correct answer see overleaf

**ECG - 10**

**The correct answer is ‘a’ – Atrial tachycardia**

There is a regular tachycardia except for an isolated pause. The P waves are marked out in lead V1. The P waves are negative in leads II and III. The atrial rate is approximately 140 bpm. This effectively rules out atrial flutter, as also the isoelectric baseline doing pause. An accessory pathway will not continue with AV block. Hence, the 2:1 block despite continuation of tachycardia rules out choice (d). AVNRT can have AV block. This is seen in typical AVNRT. However, in this case, the blocked P wave where the P wave is exactly between two QRS complexes during the block. Also during 1:1 conduction in typical AVNRT, the P wave is within the QRS complex. Hence, clearly, we are dealing with atrial tachycardia arising from low atrium.



**INDIAN SOCIETY OF ELECTROCARDIOLOGY**  
**APPLICATION FORM FOR**  
**LIFE MEMBERSHIP/FELLOWSHIP**

SECRETARIAT

**S. B. GUPTA**

**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.

My particulars are as follows :

Name in full (Surname first) \_\_\_\_\_

Qualifications \_\_\_\_\_

University (Post-Graduation obtained) \_\_\_\_\_

Year of obtaining first Post-Graduation \_\_\_\_\_

Mailing Address \_\_\_\_\_

Tel. No. Hospital \_\_\_\_\_ Clinic \_\_\_\_\_ Residence \_\_\_\_\_

Fax \_\_\_\_\_ E-Mail \_\_\_\_\_

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

No. \_\_\_\_\_ Dated \_\_\_\_\_ of \_\_\_\_\_

\_\_\_\_\_ (Bank), drawn in favour of

“Indian Society of Electrocardiology”, payable at Mumbai.

Thanking you,

Yours sincerely,

Signature of the Applicant

Proposed by (the Member of the Society)

Name \_\_\_\_\_

Address \_\_\_\_\_

Signature \_\_\_\_\_

**FOR OFFICE USE ONLY**

**Recommendations of the  
Executive Body /  
Credential Committee**

Accepted / Not Accepted

**Life Membership No.**

**Hon. Secretary, ISE**

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

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

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

\*Subject to approval of the Executive Body of the Society

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