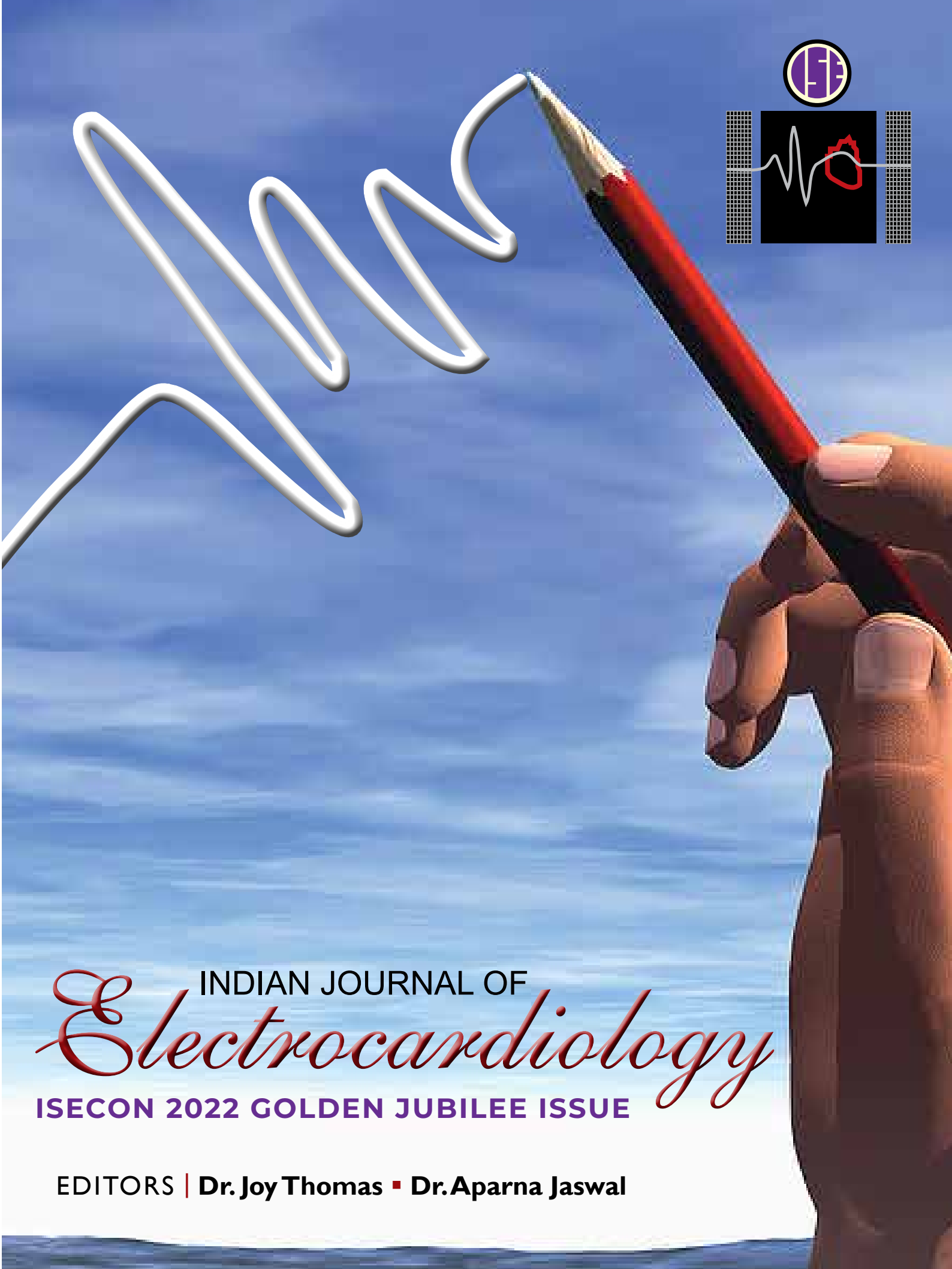

INDIAN JOURNAL OF Electrocardiology

ISECON 2022 GOLDEN JUBILEE ISSUE

EDITORS | **Dr. Joy Thomas** ▪ **Dr. Aparna Jaswal**

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Editorial

The Many Nuances of the ECG

The Electrocardiogram (ECG or EKG) was developed over a period of time and first employed in clinical use more than a century ago. Over the years its utility has only increased and it remains one of the most easily available investigations that is ubiquitous in its availability and done right from the first point of contact of the patient with the health workers.

Today it has become so common that it has even entered the homes of the layman, and more interestingly a part of the everyday personal dressing, take for example the Apple Watch 4. This Apple watch 4 can be used with some training to take pretty accurate limb lead I, II and III ECGs and has made it easier for the research worker following up with various studies on arrhythmias such as the follow-up of Atrial fibrillation patients who underwent RF ablation, studying the follow-up of arrhythmias with drugs etc. Wearables that can record an ECG is a reality and its uses and limitations has to be clearly understood by the astute clinical cardiologist.

The basics of ECG reading such as abnormalities of various segments of the ECG, the significance of the asymptomatic pre-Excitation, and the method of identifying the Ventricular premature beat that is of concern has been dealt with to give a clear understanding of when a particular patient needs more careful follow-up or management.

Heart failure is an important cause of mortality among all sections of society and its prevention can be affected by preventing the heart damage consequent to a heart attack and in this respect early identification of which patient is going in for a myocardial infarction is very essential to correctly target this population in the golden hour and prevent loss of valuable myocardium.

Various arrhythmias and their diagnosis whether it be interruptions of the conduction system or tachyarrhythmias from the ventricles or the atria or both are important for the clinician. A ventricular arrhythmia can be the precursor of a deadly cardiac arrest and tell-tale signs of which patient is at danger of sudden cardiac death is essential to correctly triage these patients to either drugs or ablation or devices. Pacemakers and heart failure devices are in common use in clinical practice and an understanding of how to diagnose heart attacks and other problems through the ECG is essential to troubleshoot problems associated with them.

Systemic hypertension and its effect on the ECG and the ability to identify the various target organ damage caused by hypertension can be carefully identified if one were to look for tell tale signs from the ECG.

The last two years saw the emergence of a new variant of the Corona virus that affected more than 15-20 million people worldwide with effects that spared none of the organs in the human body through the vascular complications and the heart was not without its share of problems. Myocarditis, heart blocks and heart attacks have been reported in the Covid patient and these have been identified through ECGs as also the occasional tachyarrhythmias, with cardiac arrest occurring in some. Here again the ECG has been at the forefront of diagnosis.

Suffice it to say that the ECG will continue to be a very handy and useful tool in our armamentarium to fight diseases and any effort in learning some more of the nuances of ECG diagnosis will not be a waste and will come in handy in difficult situations. Hopefully this anthology of ECG articles will be just that.

Joy M Thomas **Aparna Jaiswal**

From the Desk of Advisor

Dear Members,

It is indeed a great pleasure that after a long gap, Indian Journal of Electrocardiology, the Official Journal of Indian Society of Electrocardiology is being brought on the eve of Golden Jubilee Conference of Indian Society of Electrocardiology being held at Hotel Westin Powai (Renaissance earlier) from 18th to 20th November 2022 for the ISE members and to all the attending delegates.

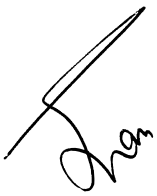
Indian Journal of Electrocardiology was quiet popular amongst physicians, especially because of the interesting ECGs. Somehow it got discontinued because of some internal problems. I am happy that new issue is being launched and hopefully, we will try to make it as regular as in the past.

Current issue has various review articles of physicians' interest and will help them in practicing medicine.

I would like to thank Dr Yash Lokhandwala, President ISE, Dr Ashish Nabar, Treasurer ISE the back bone to bring out this issue and Dr Ketan Mehta, Secretary ISE for his support.

No words to thank the Journal Editors, Dr Joy Thomas and Dr Aparna Jaswal for their untiring efforts to bring back the glory to IJE and bring the issue well in time.

Long Live ISE.



Dr. S.B. Gupta
Advisor
Indian Society of Electrocardiology

From the President's Desk

Dear Members,

The simple ECG continues to be the most widespread tool for cardiac emergencies for more than a century. While for arrhythmias this is obvious, for acute coronary syndrome this also remains true. Now, apart from diagnosing myocardial infarction, the ECG interpretation has evolved to tell us about the site of infarct, its extent and the site of coronary artery occlusion- helping identify the high risk patient and facilitating management strategy. ECG recording techniques have mushroomed to miniature devices, smart watches, patches, lockets and smart phones. Transmissibility of the ECG has become child's play with the omnipresence of social media, improving patient management and facilitating education.

The ISE has been at the forefront in disseminating ECG knowledge over half a century. Periodic updates of the diagnostic criteria and the training material have kept the content vibrant and contemporary. The ISE conferences have always been sought after by students and practising physicians. Previously, the ISE sessions at the API conferences resounded to packed halls. At this Golden Jubilee meeting, excellent international and national faculty blend with innovative topics, to provide a unique perspective into topics, based around the ECG.

This issue of the IJE has a panoramic spread of topics with a different slant than the usual approach of an ECG textbook. Editors Joy and Aparna must be commended for their selection as well as being able to make so many stalwarts put their best foot forward to provide a superb array of chapters. I have no doubt this issue will be a valuable accompaniment in the clinics, libraries and homes of many of our colleagues. I for one cannot wait to go through this collection!

Dr. Yash Lokhandwala
President
Indian Society of Electrocardiology

Pre-Excitation in the Electrocardiogram: When is it of concern?

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1. Introduction

Accessory pathways are residual atrioventricular connections caused by incomplete embryological development of atrioventricular annuli with failure of fibrous separation of the atria and ventricles. These microscopic muscular bundles connect the atrium and the ventricles, thus bypassing the normal conduction system.

2. Terminology

2.1 Types of accessory pathways

There are several different types of accessory pathways, including the atrioventricular (previously called Kent bundle), atrionodal (James fibres), atriohisian, atriofascicular, fasciculoventricular, nodofascicular and nodoventricular pathways.

2.2 Types of accessory pathway conduction

The accessory pathways typically exhibit fast conduction as they are dependent on sodium channels, with the exception of the atypical pathways. The majority of the accessory pathways conduct both anterogradely and retrogradely, but some conduct impulses in only one direction. When the accessory pathway conducts anterogradely, it is referred to as manifest preexcitation. A variable degree of preexcitation occurs depending on the relative contribution from ventricular activation by the normal atrioventricular nodal conduction versus the manifest accessory pathway. Accessory pathways that do not manifest anterograde conduction are called concealed accessory pathways, but they can conduct retrogradely, creating a reentrant circuit. Accessory pathways that conduct only retrogradely are more frequent (≤ 50%), whereas those that conduct anterograde only comprise ≤ 10%. The term latent accessory pathway is used when the accessory pathway conducts antegradely, but preexcitation is barely visible or not visible on the ECG due to the location of the accessory pathway or due to faster conduction through the atrioventricular node.

2.3 Types of accessory pathway syndromes

Wolff-Parkinson-White syndrome

It refers to the combination of ventricular preexcitation and either a documented tachycardia or symptoms of tachyarrhythmia.

Asymptomatic Preexcitation

They are asymptomatic patients with electrocardiographic abnormalities related to the presence of manifest atrioventricular accessory pathway. They are sometimes referred to as isolated preexcitation.

Lown-Ganong-Levine syndrome

The typical ECG findings of a short PR interval, absent delta waves, and normal QRS duration are either due to preexcitation via the atrio-Hisian accessory pathway or due to enhanced atrioventricular nodal conduction in the absence of the accessory pathway. It is only an ECG description and not a recognized syndrome.

3. Sudden death risk

3.1 Incidence

In patients with preexcitation, whether asymptomatic or symptomatic, there is a concern of sudden death in the future. The incidence of sudden cardiac death in patients with WPW syndrome is low, from 0% to 0.39% annually.[1] However, sudden cardiac death can be the first manifestation of WPW syndrome in about 50% of those with SCD. It also accounts for 1% of sudden death in athletes.[2]

3.2 Mechanism

Unlike the atrioventricular node which limits the ventricular rate during atrial fibrillation, accessory pathways may have a short refractory period and allow rapid ventricular rates. The reason for sudden cardiac death in WPW syndrome is the occurrence of atrial fibrillation or atrial flutter with very rapid ventricular rate degenerating into ventricular fibrillation.

The incidence of atrial fibrillation in WPW syndrome varies from 12-39%.[2] Patients with antidromic AVRT, multiple accessory pathways, and accessory pathways with short ERP are more prone to develop atrial fibrillation. There is a very low prevalence of coexisting structural heart disease and other predisposing factors for atrial fibrillation with WPW syndrome. Therefore, the accessory pathway itself could be related to the development of AF, and this is also supported by the fact that catheter ablation can cure atrial fibrillation in about 90% of patients. The mechanisms probably include the rapid atrial rate during atrioventricular reentrant tachycardia (AVRT), fractionation of activation wavefronts due to the complex geometry of networks of accessory pathways and atrial stretch caused by atrial contraction against closed atrioventricular valves during ventricular systole.

4. Risk stratification

4.1 Clinical factors

Clinical factors associated with increased risk for ventricular fibrillation include age < 30 years, male gender, history of atrial fibrillation, prior syncope, familial WPW syndrome, associated congenital heart disease, symptomatic AVRT, multiple accessory pathways, Ebstein's anomaly and septal location of the accessory pathway.

4.2 Electrocardiography (ECG)

The most important factor associated with ventricular fibrillation in a patient with preexcitation is the ability of the accessory pathway to conduct rapidly to the ventricles. This is determined by measuring the shortest pre-excited RR interval (SPERRI) during atrial fibrillation. SPERRI (Figure 1) measured in an ECG recorded during atrial fibrillation appears to be a sensitive clinical marker (sensitivity 88-100%) for identifying the risk of sudden cardiac death. A mean preexcited RR interval of >250 msec and the shortest preexcited RR interval >220 msec predicted low risk for SCD with a negative predictive value of >95%, but the positive predictive value is low (20%).[3] A SPERRI of 220-250 msec and especially ≤ 220 msec is more commonly seen in patients who have experienced cardiac arrest.

Intermittent loss of preexcitation on resting ECG (Figure 2) or ambulatory monitoring is associated with a long refractory period of the accessory pathway. However, this finding should be interpreted with caution because studies have shown that more than one-fifth of patients with intermittent preexcitation have accessory pathway effective refractory period less than 250 msec.

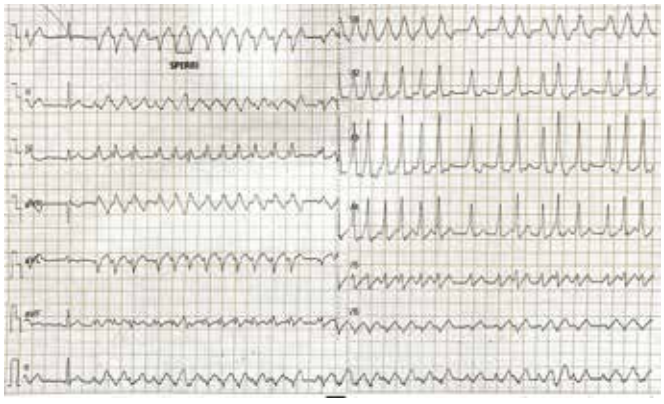

Figure 1: Shortest Pre-excited RR Interval (SPERRI) in pre-excited AF

12 lead ECG shows an onset of a fast irregular wide QRS tachycardia, with a ventricular rate of 240 bpm, with varying QRS morphology. Delta waves are positive in precordial leads and positive in inferior leads suggesting a left lateral pathway. SPERRI measured in this ECG is 240 msec indicating high risk.

4.3 Treadmill test

Abrupt and complete normalization of PR interval with loss of delta waves during exercise testing denotes a long refractory period of the accessory pathway (>300 msec). Typically, atrioventricular nodal conduction becomes faster with exercise and this may mask preexcitation. Therefore, only abrupt and complete loss of preexcitation during exercise is related to long anterograde refractory period of the accessory pathway. But the predictive value of these non-invasive parameters suggesting low risk is low. Interobserver variability on the loss of preexcitation is also a drawback.

4.4 Drug Testing

Drug testing is rarely done nowadays. Complete normalization of PR interval with loss of delta waves following ajmaline, procainamide, propafenone or disopyramide administration denotes a long refractory period of the accessory pathway (>270 msec). The specificity of loss of preexcitation after administration of sodium channel blockers was poor compared to SPERRI during preexcited AF.

4.5 Electrophysiological study

The ability to conduct rapidly to the ventricles can be assessed during rapid atrial pacing or by measuring the anterograde effective refractory period of the accessory pathway during an electrophysiological study. (Figure 3) An anterograde refractory period of the accessory pathway less than 250 msec indicates high risk. SPERRI during rapid atrial pacing and during induced atrial fibrillation also has a similar predictive value with a similar cut-off value of 250 msec. (Figure 4)

Other factors associated with high risk in EP study include multiple accessory pathways and inducible sustained accessory pathway mediated tachycardia with or without isoproterenol, especially when AVRT spontaneously degenerates into AF. Accessory pathways with decremental conduction generally

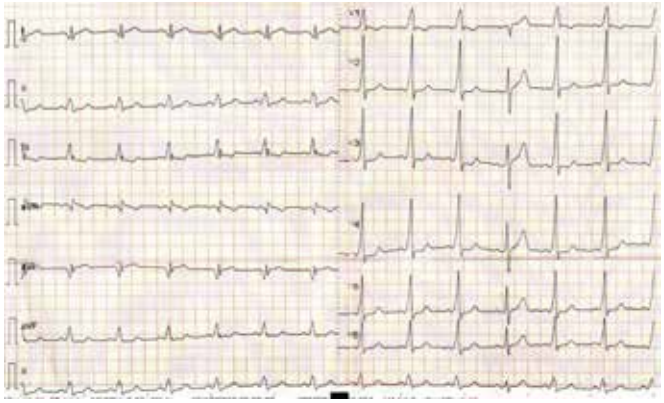

Figure 2: Intermittent preexcitation

12 lead ECG shows intermittent preexcitation with positive delta waves in all the precordial leads and inferior leads and negative delta waves in leads I and aVL suggestive of a left lateral accessory pathway. The eleventh beat is not preexcited.

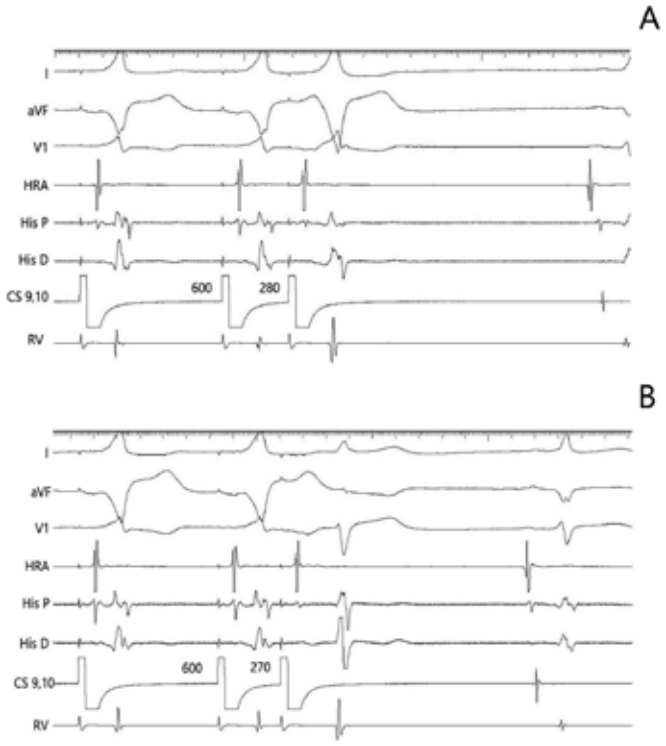

Figure 3: Accessory pathway anterograde effective refractory period

Figure 3A: EP tracing shows atrial extrastimulus from coronary sinus catheter at 600/280 msec. Surface ECG shows positive delta waves in leads V1 and I and negative delta waves in lead aVF with both drive train of 600msec and with extrastimulus 280 msec.

Figure 3B: EP tracing shows atrial extrastimulus from coronary sinus catheter at 600/270 msec. Surface ECG shows positive delta waves in leads V1 and I and negative delta waves in lead aVF with drive train of 600msec and loss of preexcitation with extrastimulus 270 msec. Hence, the accessory pathway's anterograde effective refractory period is 270 msec.

have poor anterograde conduction characteristics and carry a lower risk of SCD. Concealed accessory pathways give rise to only orthodromic AVRT and are not associated with an increased risk of sudden cardiac death.

5. Management

Catheter ablation is the treatment of choice in symptomatic and recurrent AVRT. But there was always a concern in managing asymptomatic preexcitation. A prospective RCT involving asymptomatic preexcitation patients showed that catheter ablation reduced the frequency of arrhythmic events (7 % vs 77% without any treatment, $p<0.001$) over five years.[4]among 224 eligible asymptomatic patients with the Wolff-Parkinson-White syndrome, patients at high risk for arrhythmias were randomly assigned to radio-frequency catheter ablation of accessory pathways (37 patients The rationale behind ablation of asymptomatic preexcitation is to prevent sudden cardiac

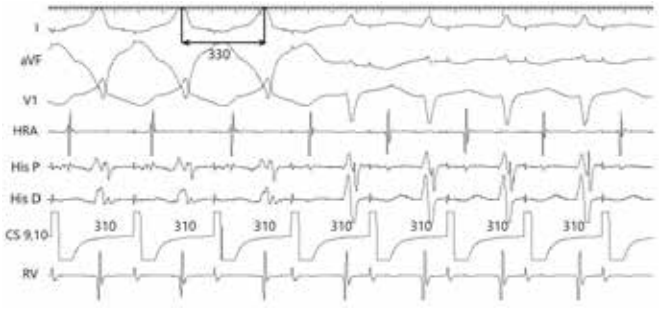

Figure 4: SPERRI with rapid atrial pacing

EP tracing shows atrial incremental pacing from coronary sinus catheter at 310 msec. Surface ECG shows loss of preexcitation after the third beat. The shortest preexcited RR interval with rapid atrial pacing at 310 msec is 330 msec. Hence the SPERRI with rapid atrial pacing is 330msec.

death in high-risk patients. Invasive electrophysiological studies in asymptomatic preexcitation should be performed in patients in high-risk occupations or competitive athletes. Even when patients do not fall into either of these categories, EP study can be used as a risk stratifying tool, or non-invasive tests such as exercise testing, ambulatory monitoring, or drug testing can be done. High-risk patients should undergo catheter ablation of the accessory pathway, and when performed by an experienced operator, the success rate is ≥ 95 %, and the major complication rate is ≤ 0.5%.[5]

There is also evidence that electrical asynchrony in asymptomatic preexcitation can lead to left ventricular dysfunction, especially in children. These patients should undergo catheter ablation if there are no other reasons for left ventricular dysfunction.[6]

Another concern in managing WPW syndrome is when they present with pre-excited AF. Synchronized DC cardioversion is recommended in hemodynamically unstable patients with pre-excited AF. In hemodynamically stable patients with pre-excited AF, intravenous ibutilide or procainamide should be considered. Flecainide or propafenone may also be considered. If drug therapy fails to convert or control the tachycardia, then synchronized, DC cardioversion is recommended. Amiodarone is not safe as previously thought and should not be used because enhanced pathway conduction and ventricular fibrillation have been reported. The treatment of choice for patients with an initial presentation of pre-excited AF is catheter ablation.

6. Conclusion

Although the common presentation in patients with preexcitation is paroxysmal tachycardia, there is a small risk of sudden cardiac death in these patients, even if asymptomatic. While some clinical factors are associated with the occurrence of ventricular fibrillation and sudden cardiac death in this population, ECG findings may help with risk stratification. Finding of intermittent preexcitation and abrupt, complete loss of preexcitation with exercise indicates low risk, while a

short preexcited RR interval during atrial fibrillation indicates high risk. An invasive electrophysiology study should be considered for risk stratification and asymptomatic patients at high risk should undergo catheter ablation.

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The ECG to Decipher SVT Mechanism

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Supraventricular tachycardia (SVT) is a term to describe tachycardia that depends on the involvement of cardiac tissue above the ventricles. Although atrial fibrillation (AF) is, strictly speaking, an SVT, the term SVT generally does not refer to AF. 12 lead ECG recording during tachycardia episodes is the most important diagnostic test for patients with SVT. ECG characteristics during SVT on 12 lead ECG or Holter recording may help differentiate SVT, which in turn helps formulate acute and long-term management. We recommend the following approach in a stepwise manner to reach the diagnosis.

Narrow vs. Wide Complex Tachycardia

Since in the majority of SVT, activation of ventricles occurs through the AV node and normal functioning His-Purkinje system (HPS), the resultant QRS complex is narrow (<120ms). A wide QRS complex (≥120ms) in SVT results from aberrancy of the conduction system or preexisting bundle branch block. Rarely, a wide QRS complex in SVT is because of activation of the ventricles through the antegrade conducting accessory pathway.

In wide complex tachycardia (WCT) with a 1:1 AV relationship, morphology criteria utilizing lead V1 and V6 are beneficial to differentiate SVT from VT (figure 1). During RBBB morphology WCT, positive triphasic QRS complex in lead V1 and R/S ratio >1 in lead V6 suggests a diagnosis of SVT. During LBBB morphology WCT, a narrow initial R wave and a sharp smooth, and rapid descent of the S wave in lead V1 and a monophasic R wave in lead V6 suggest a diagnosis of SVT.[1] In certain cases, QRS complex morphology criteria are of limited value, e.g., a) pre-existing intraventricular conduction defect during sinus rhythm, b) antidromic AV reentrant tachycardia where QRS complexes will depend on the location of accessory pathway along the AV ring and its ventricular insertion site. In these cases, comparing ECG during tachycardia and sinus rhythm is immensely helpful. Combining morphology criteria with single lead aVR criteria with an initial Q wave <40ms for the diagnosis of SVT may increase the sensitivity and specificity of diagnosing wide-complex tachycardia.

Regular Vs Irregular tachycardia

Once the mechanism of tachycardia is ascertained as SVT, the next step in diagnosing narrow complex tachycardia is to define the regularity or irregularity of successive QRS complexes. Differential diagnosis of regular narrow complex tachycardia is sinus tachycardia, atrial tachycardia (AT), atrial flutter, AV nodal reentrant tachycardia (AVNRT), and AV reentrant tachycardia (AVRT). Irregular narrow complex tachycardia includes AF and multifocal AT (MAT). Atrial flutter (AFL) and AT can also be irregular because of variable AV conduction block.

P & QRS ratio

If the number of P waves exceeds that of the QRS complexes, the diagnosis is usually either AFL or AT with regular AV conduction. Rarely AVNRT may show a 2:1 anterograde block at the level of His or lower common pathway, and in these cases, a retrograde P wave is visible in the middle of two QRS complexes. Narrow QRS tachycardia with more QRS complexes than P waves are very rare and comprise 1) junctional tachycardia with retrograde block, 2) Dual AV node-dependent non-reentrant tachycardia which rarely occurs in patients with dual AV nodal physiology with anterograde conduction of sinus beat over a fast and a slow pathway, yielding two QRS complexes for every sinus P wave and 3) upper septal VT.[2]

RP interval

The next step in making the diagnosis of a narrow complex regular tachycardia is to notice the presence of P waves and their correlation with QRS complexes. Depending on the relationship between the P wave and QRS complex, tachycardia can be defined as:

a. short RP tachycardia (RP interval<PR interval) and

b. long RP tachycardia (RP interval>PR interval).

In short RP tachycardia, when P waves are buried in QRS complexes the diagnosis is usually typical AVNRT or very rarely Junctional tachycardia (JT). When the P waves are away from the R waves in short RP tachycardia the most common diagnosis is AVRT or uncommonly atypical AVNRT. Differential diagnosis of long RP tachycardia is sinus tachycardia, atrial tachycardia, atypical AVNRT, or permanent junctional reciprocating tachycardia (PJRT).

In AVNRT, the AV node is thought to be functionally dissociated into two pathways namely a slow pathway and a fast pathway with different electrophysiological properties setting up a re-entrant circuit. In typical AVNRT (slow-fast), the antegrade limb of the circuit is a slow pathway and the retrograde limb is a fast pathway. This leads to near-simultaneous depolarization of the atria and ventricles. The P wave can distort the initial portion of the QRS (mimicking a q wave in the inferior leads), lie just within the QRS (inapparent), or distort the terminal portion of the QRS (mimicking an s wave in the inferior leads or a terminal r wave in lead V1). In atypical AVNRT (fast-slow) because the retrograde limb of the circuit is a slow pathway, it results in long RP tachycardia mimicking AT. In slow-slow AVNRT, the RP interval is usually shorter than, and

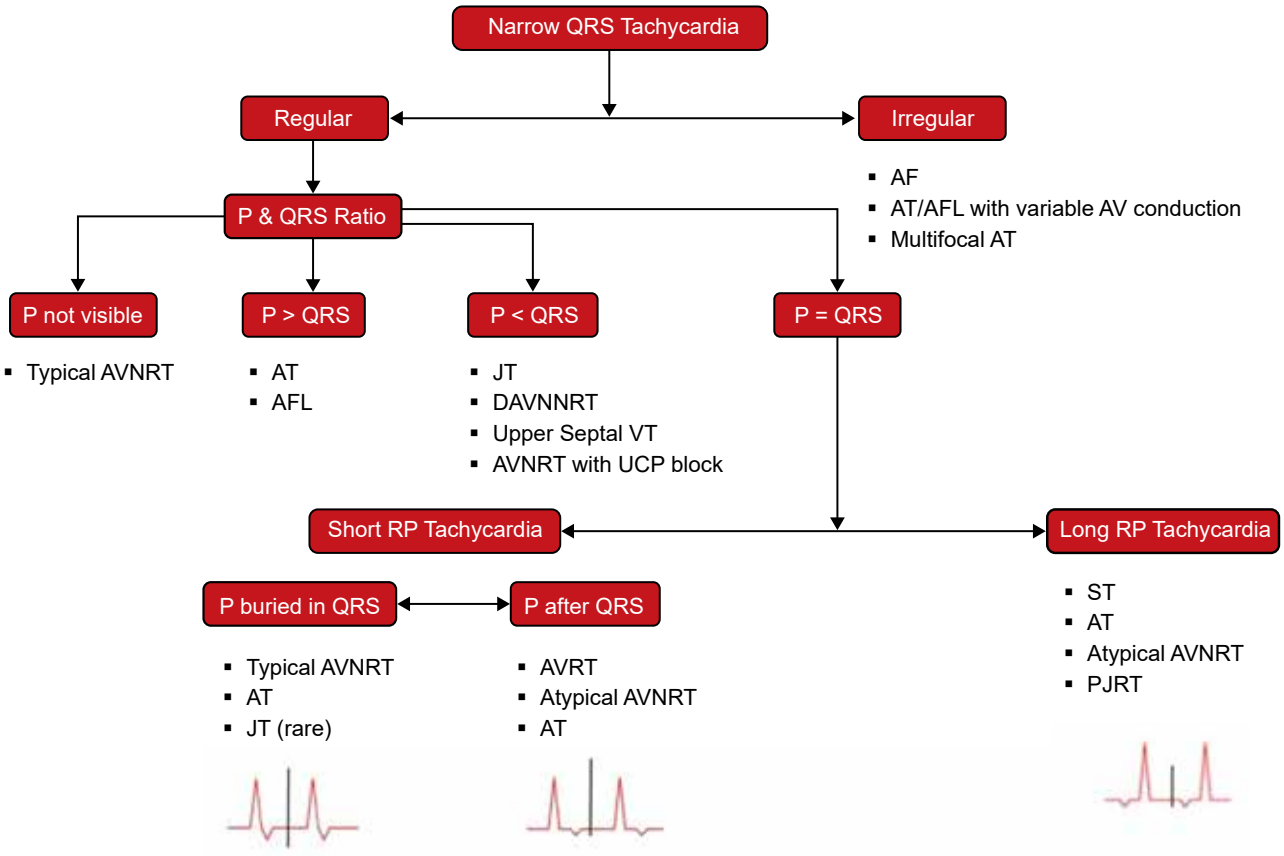


Figure 1: 12 lead ECG approach to narrow QRS tachycardia. (AF=atrial fibrillation, AT=atrial tachycardia, AFL=atrial flutter, AVNRT=Atrioventricular nodal reentrant tachycardia, AVRT=atrioventricular reentrant tachycardia, JT=junctional tachycardia, DAVNNRT=Dual AV nodal nonreentrant tachycardia, VT=ventricular tachycardia, UCP=upper common pathway, ST=sinus tachycardia, PJRT=permanent junctional reciprocating tachycardia)

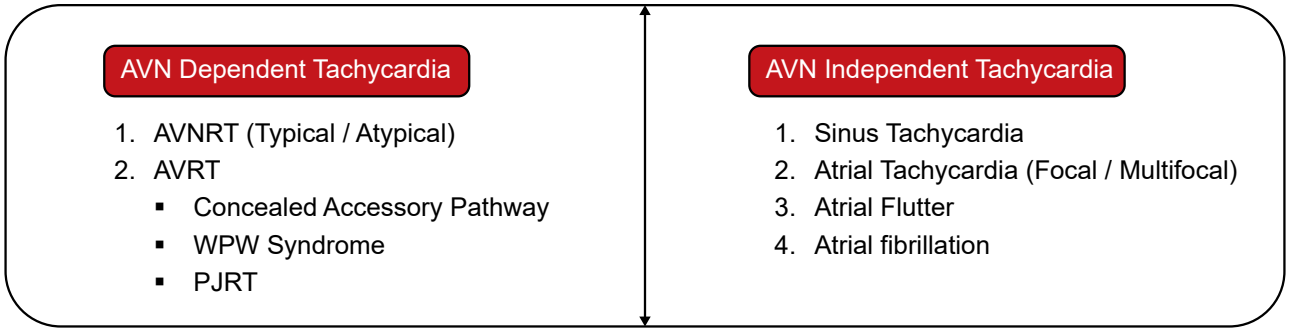


Figure 2: Differential diagnosis of narrow QRS tachycardia based on the response to AV nodal blockade.

sometimes equal to the PR interval mimicking AVRT.

Orthodromic AVRT results from a re-entrant circuit using a normal AV conduction system as an antegrade limb and an accessory pathway located across the valvular annuli as a retrograde limb. Ventricular activation completes before retrograde atrial activation through a rapidly conducting accessory pathway, resulting in short RP tachycardia with P waves inscribed within the ST-T wave segment.

Though AT is classically a long RP tachycardia, associated prolonged PR interval may bring the subsequent P wave closer to the QRS complex and thus can result in short RP tachycardia. In permanent junctional reciprocating tachycardia, the retrograde limb of the re-entrant circuit is an accessory pathway with decremental conduction properties resulting in long RP tachycardia.

P wave Morphology and Polarity

It is difficult to determine P wave morphology and polarity in the case of short RP tachycardia like AVNRT as P waves are

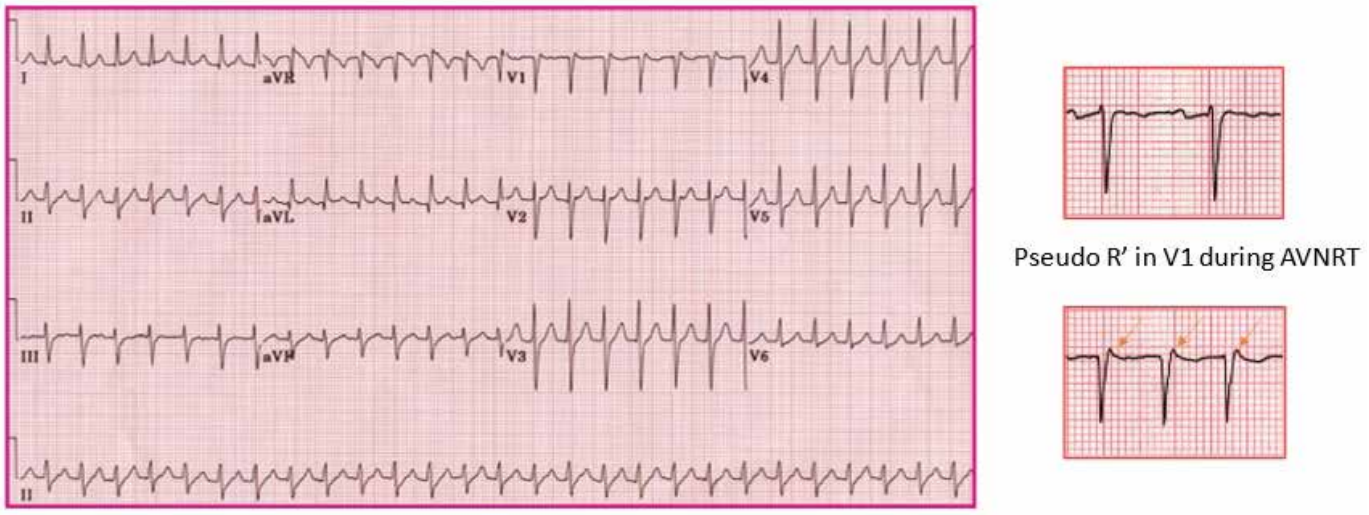


Figure 3: Regular narrow QRS tachycardia with P wave buried in the QRS complex revealed as a sharp deflection just after the QRS complex (R' wave) is classical ECG finding of typical AVNRT.

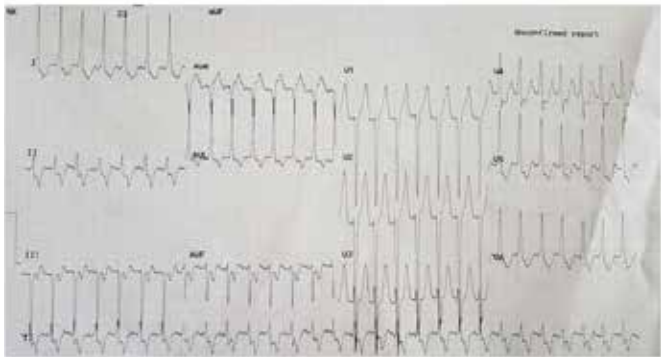


Figure 4: Regular narrow QRS tachycardia with short RP interval. Negative P wave in leads I and aVL is suggestive of AVRT using left sided bypass tract.

inscribed in the QRS complex or within the ST segment. In AVNRT, when P waves are apparent, the P wave is relatively narrow, negative in the inferior leads, and positive in lead V1 consistent with retrograde concentric activation. The polarity of the retrograde P wave during AVRT is dependent on the location of the atrial insertion of BT. P wave morphology is useful for localization of the bypass tract rather than the mechanism of SVT. Generally, P wave morphology in lead I, V1, and inferior leads are looked at. A negative P wave in lead I is highly suggestive of left free-wall BTs, whereas a positive vector is suggestive of right free-wall BTs. On the other hand, a negative P wave in lead V1 predicts right-sided BTs. P wave polarity in the inferior leads, positive or negative suggests the superior or inferior location of the BT, respectively.

In the case of long RP tachycardia mainly AT, P wave morphology depends on the anatomic location of the atrial focus which can be used to approximate the site of origin of the AT.[3] Sinus tachycardia is characterized by P wave morphology suggestive of sinus origin. The sinus origin frontal P wave axis is approximately 60 degrees. Consequently, P waves are upright in lead I, II, and aVF with some variability observed

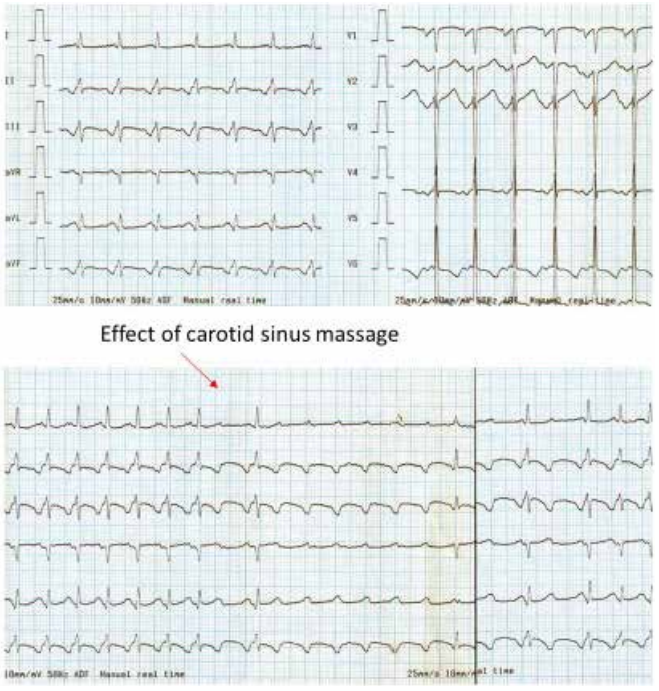
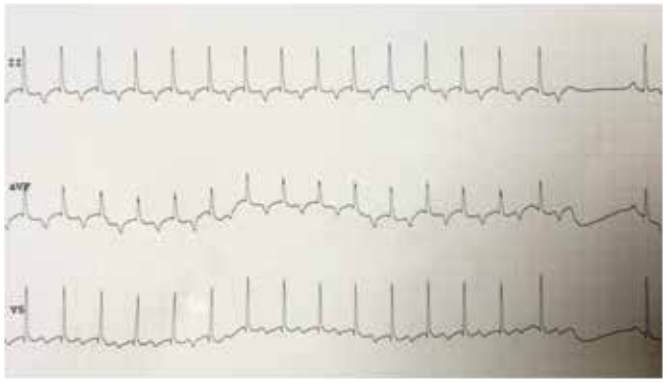


Figure 5: Long RP tachycardia on carotid sinus massage unmasks underlying atrial tachycardia during transient AV block.

in lead III and aVL. In lead V1, the P wave is biphasic with terminal negative deflection. P wave morphology, different from sinus P wave points towards focal AT. However, AT in the vicinity of the sinus node may appear like sinus tachycardia and one has to understand the behavior of tachycardia to differentiate it from sinus tachycardia. In PJRT, P waves are negative in II, III, and aVF, and positive in V1 and have to be differentiated from atypical AVNRT and focal AT.

AV Node Dependence

A review of ECG tracings during AV block which can be spontaneous or induced by vagal maneuvers, carotid massage,



Termination of tachycardia with P wave

Figure 6: Spontaneous termination of tachycardia with P wave rules out AT as a mechanism of tachycardia

or AV node blockade by adenosine provides an insight into the diagnosis. Termination of tachycardia with perturbation of the AV node is indicative of its participation in the tachycardia circuit and indicates the diagnosis of AVRT or AVNRT. In AT or AFL, tachycardia would continue with the unmasking of underlying atrial rhythm. Adenosine sometimes may terminate focal AT with triggered activity as an underlying mechanism. It will not affect the tachycardia despite AV block during AT because of the reentry mechanism. Termination of the tachycardia with a P wave after the last QRS complex is most common in AVRT and typical AVNRT and is rarely seen with AT whereas termination of the tachycardia with a QRS complex is more common with AT, atypical AVNRT, and PJRT.

Additional ECG findings

Beat-to-beat variation in QRS amplitude named 'QRS alternans' during SVT has generally been considered to be the feature of AVRT. However, it is more commonly a function of the heart rate rather than the mechanism of SVT and it can also occur during rapid AVNRT. Beat-to-beat variation in RR interval named 'cycle length alternans' also occurs more commonly with AVRT with alternating antegrade conduction over a slow and fast AV nodal pathway, however, it has been reported with AT and AVNRT as well. Prolongation of cycle length with slowing of heart rate during tachycardia when bundle branch block develops points towards the AVRT as a mechanism of tachycardia.[4] This occurs in AVRT using a bypass tract on the free wall of the same side of the ventricle as the blocked bundle branch because of the prolongation of circuit length. Initiation of SVT after a single atrial premature beat during sinus rhythm after marked PR prolongation suggests the presence of dual AV nodal physiology and AVNRT as the mechanism of SVT. Initiation of SVT with PAC without significant PR prolongation is suggestive of AVRT. Also, initiation and termination of SVT with single premature ventricular beat points toward AVRT.

SVT is a common arrhythmia encountered in clinical practice. The management of SVT may include just a simple Valsalva maneuver or may require AV nodal blockers, antiarrhythmic drugs, or cardioversion. Fundamental knowledge of the mechanism of SVT and a stepwise approach to ECG of SVT helps the clinicians to arrive at the diagnosis of SVT which in turn helps their management and counseling of the patient and timely referral for further therapy. Because of the diagnostic yield of 12 lead ECGs for SVT, there is a limited role of diagnostic electrophysiology study, unless one considers ablation for SVT.

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12 Lead ECG Based Localization of Ventricular Arrhythmias

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Introduction

Ventricular Arrhythmias (VA) originating from Outflow Tracts (OTVAs) are the most common type of idiopathic VA. They typically present in young female patients, between 30-50 years, and have a notably increasing incidence.[1] It is classically a benign, focal arrhythmia but patients can be highly symptomatic and refractory to medical therapy. Moreover, frequent ectopy can progress to a premature ventricular complex (PVC) induced cardiomyopathy. VA arising from other sites are non-OT VA; They are divided into two categories based on whether there is underlying structural heart disease.

Applied Anatomy

The OT are the superior-most extensions of the heart; The two OT are intricately entwined around each other because of the embryonic folding of the cardiac tube. Appreciation of the intricacies of these relationships are important aids to mapping.

The RVOT extends from the base of the RV and traverses anteriorly extending from the right side of the body to the left. The pulmonary valve and the pulmonary artery therefore are more left sided structures than right. The outflow tracts above the pulmonary valve can contain muscle sleeves which can be the source of ectopy and need careful mapping. Like them, the sinuses of Valsalva (SoV) also contain sleeves of muscle. Ventricular muscle is found in the right and left SoV whereas the non-coronary SoV is bereft of any ventricular muscle. The SoV form the base of the aorta and are essentially posterior to the RVOT and to its left to begin with. But as the RVOT and PA cross over to the left, this relationship becomes more antero-posterior than right left with the ascending aorta occupying a more rightward and posterior position to the RVOT. As a result of this, the septum between the two OT becomes antero-posterior after starting out in the right-left direction. The SoV have their own orientation with the right SoV being most anterior and rightward whereas the left SoV being more posterior and left ward as compared to the right. (Ref. Fig. 1) The posterior most SoV remains the non-coronary SoV where there is lack of ventricular muscle but owing to its position anterior to the two atria, atrial signals will be recorded there. The coronary arteries arise higher in the sinuses and hence mapping, and ablation of ventricular arrhythmia can be done from the depths of the sinuses if catheter position can be tracked in real time and 5mm distance from the coronary ostia can be ensured at the site of origin of the focal ventricular arrhythmia.

The junction of the right SoV and the non-coronary SoV in its' inferior aspect is the site where the bundle of His penetrates the interventricular septum in its membranous part. Posterior to this site, owing to the relative apical displacement of the tricuspid valve as compared to the mitral valve and

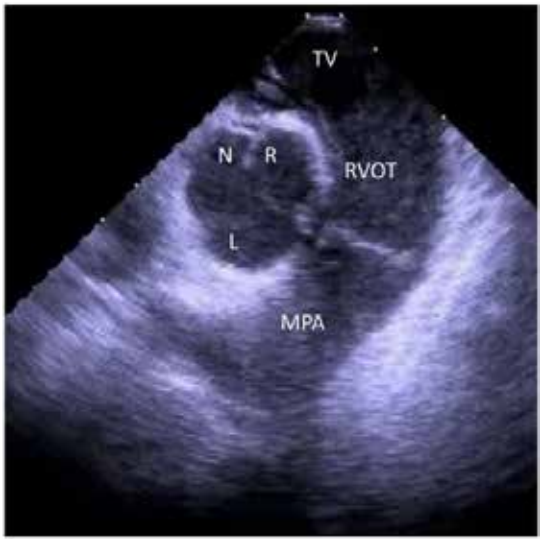


Figure 1: Intracardiac echocardiography image showing the relationship of the RVOT and the aorta. N noncoronary sinus of Valsalva; L left sinus of Valsalva; R right sinus of Valsalva; RVOT right ventricular outflow tract; MPA main pulmonary artery; TV tricuspid valve

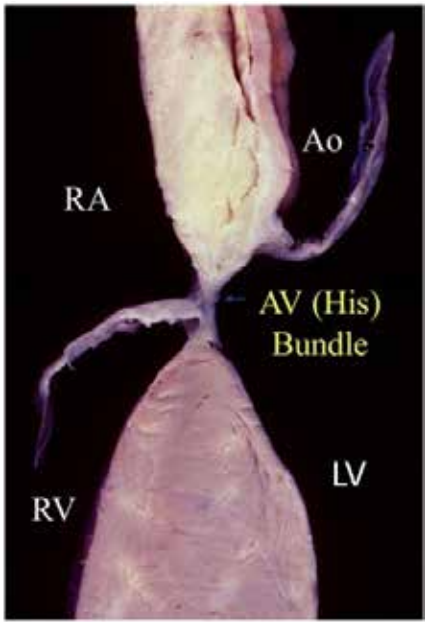


Figure 2: Illustration of the location of the penetrating His bundle at the atrioventricular portion of the membranous septum[33]

the absence of the infundibulum of the LVOT, there is the atrioventricular septum which separates, the right atrium from the left ventricle. (Ref. Fig 2) This is the site for access into difficult entry LV when there are metallic valves in both the aortic and mitral positions.

The left coronary SoV and the anterior mitral valve are connected by a curtain of fibrotic tissue which can rarely provide origin to some VA. The mechanism and tissue involved in the origin of these arrhythmias is unclear.

The left ventricle is unique in that there are intracavitary papillary muscles which can be the sources of arrhythmias. The unique interspersal of conduction tissue within these, make mapping challenging. The right ventricle (RV) is draped around the left ventricle (LV) in a crescent shaped manner and contains the moderator band. This intracavitary structure contains conduction tissue and has heads supporting the anterior RV free wall and the lateral RV free wall. Near simultaneous activation of the free and the septal wall of the RV is owing to this structure. Additionally, the RV remains a trabeculated structure whereas the LV is a non-trabeculated structure.

Left main coronary artery (LMCA) is adjacent and immediately posterior to RVOT below the pulmonary valve. It is lateral in relation to RVOT as it branches into left circumflex artery (LCx) and left anterior descending artery (LAD). The presence of an atrial signal when mapping a ventricular arrhythmia in this region suggests proximity to the LAD whereas proximity to the LMCA is suggested when there is no atrial signal in this region. Because of lateral course of RVOT, the right coronary artery (RCA) is in closest proximity to proximal RVOT near the tricuspid annulus (Fig. 3).

The GCV, anterior interventricular vein, middle cardiac vein are other sites where mapping to secure a vantage point for intra mural or epicardial site of ventricular arrhythmia can be done. AIV-GCV Junction is immediately lateral to the LCC. Proximal to distal, the AIV is adjacent to the posterolateral sub valvular RVOT, or the epicardial lateral RVOT, and the anterior epicardial space. (Ref. Fig 2) OT VTs may also originate from the region of the LV summit, the most superior LV epicardial region at the top of the interventricular septum. The LV summit lies between the LAD and LCx arteries, near the junction of the GCV and the AIV, and superior to the aortic valve cusps. The superior-most region of the LV summit is close to the LAD and LCx arteries, along with prominent overlying epicardial fat, thus is inaccessible to epicardial catheter ablation; the lateral region is accessible via the GCV. (Fig. 3)

General Principles

Bundle Branch Block Pattern

VA with right bundle branch block(RBBB) pattern arise from the left ventricle and LV OT whereas VA with left bundle branch block(LBBB) pattern might arise from either the right or left ventricle or their OTs

Importance of QS in leads AVR and AVL

This is the sine qua non for an OT origin of the VA; Since the OT are the highest structures in the cardiac anatomy and the AVR and AVL leads are positioned high above all the other leads, it is only when the vectors are away from both these leads that it can be said with certainty that the origin or exit is within the OT.

Any changes in this suggests that the VA is a non-OT VA.

Importance of lead Inferior leads (lead II, III, aVF)

The vertical dimension of the electric vectors is best reflected

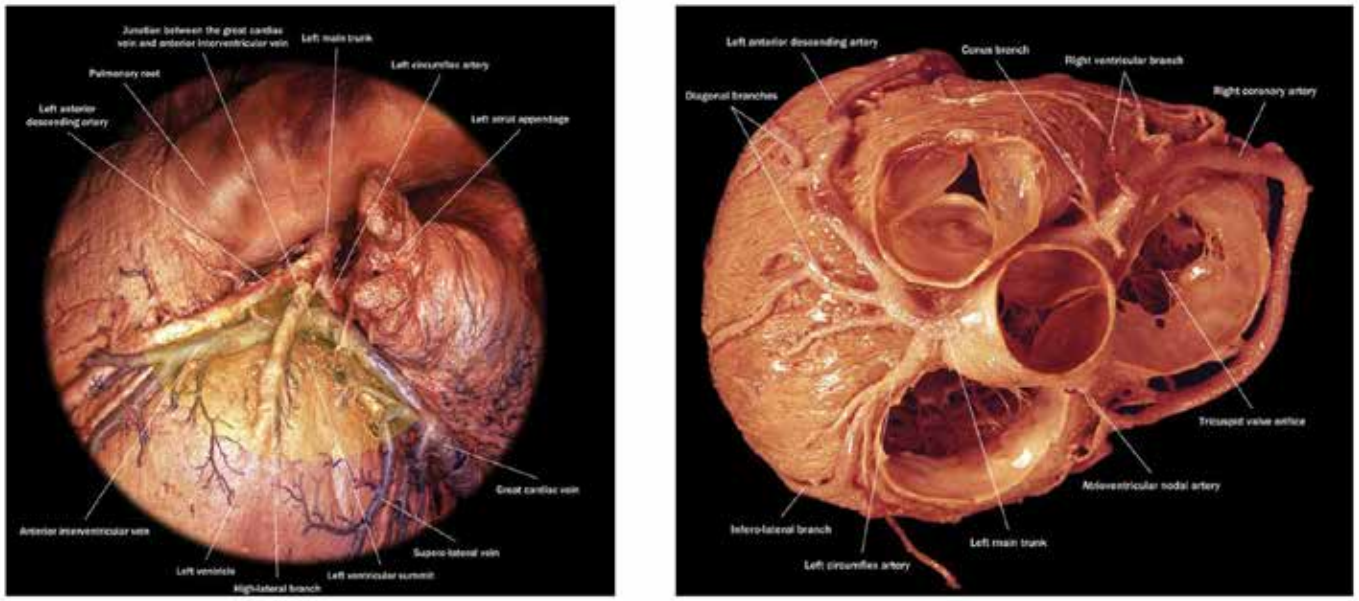
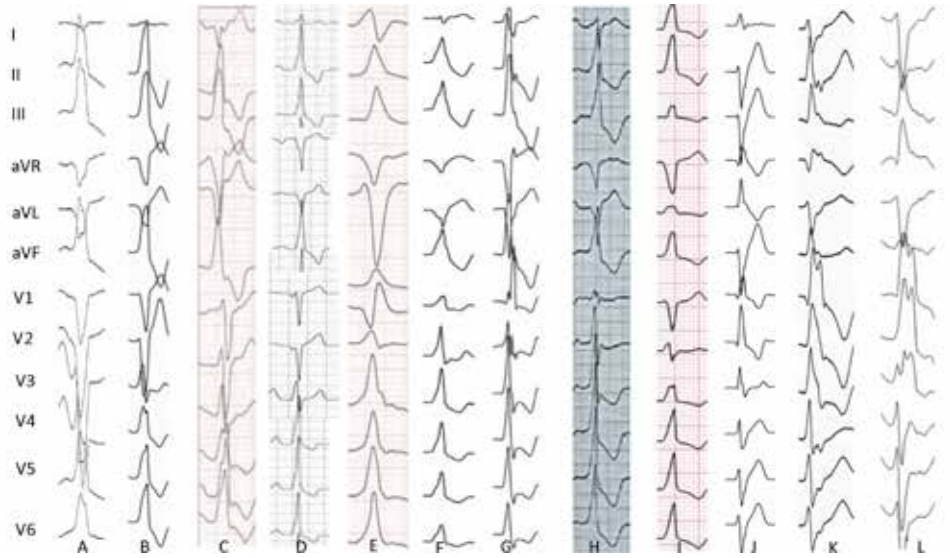


Figure 3: Relationship of the coronary arteries and veins to the ventricular outflow tracts [Courtesy Dr K Shivkumar;Atlas of Cardiac Anatomy]



Central Figure 1: 12 lead ECG with most likely site of origin from A. RVOT Ant Wall, B. RVOT Posterior Wall/RCSoV, C. Supravalvular above the Pulmonary Valve VAs, D. LCSoV, E. AMC, F. LV Summit G. Anterolateral Mitral Valve VAs, H. GCV-AIV VAs, I. Parahisian Region VT, J. ILVT, K. Anterolateral Papillary Muscle VT, L. Posteromedial Papillary Muscle VT.[10,16] Refer abbreviations table for Abbreviations.

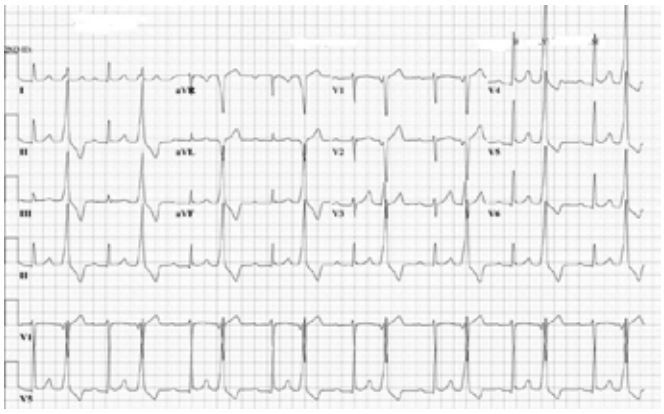


Figure 4: An example of Outflow Tract PVCs originating from the Left coronary sinus of Valsalva

by inferior leads II, III and avF. A positive deflection in inferior leads is indicative of the origin from superior portion of the ventricles including the outflow tracts and superior aspect of mitral and tricuspid valve; whereas a negative deflection in inferior leads is indicative of inferior aspect of either ventricle.

Importance of lead I

Lead I is placed on the left side of the heart. If Lead 1 is negative, the source of VAs is more leftward. If lead I is isoelectric or positive, VAs will have rightward origin from midline. A negative or isoelectric lead I suggests leftward origin from the midline. Lead I along with lead V1 can really help in making a good guess of the site of origin, especially in OT VTs where all the possible sites are in very close proximity to each other.

Importance of lead V1

Lead V1 is a right sided anterior unipolar lead. Wavefronts which are completely negative in lead V1(qs) pattern suggest origin from the right anterior ventricular structures whereas a completely positive V1 lead suggests a left sided or posterior origin for the VA.

Precordial Transition

In PVCs with RBBB pattern, transition becomes later as the site of origin moves from LV apex to base whereas in PVCs with LBBB pattern, transition becomes later as the origin moves from septal to RV free wall

Diagnosis

Outflow Tract Ventricular Arrhythmias (Ref. Fig. 4 And Central Figure)[10,11,16]

If VA is predominantly positive in lead I, the origin is on the right side of the midline. Possible structures of origin include includes infra valvar RVOT and right coronary SoV. If VA is predominantly negative, the origin is on the left side of the midline which includes the supravalvar RVOT and the mitral annular region. RS patterns in this lead suggest structures in between, namely the right and left coronary SoV

If VA is QS in lead V1, then the anterior most structure namely the anterior RVOT is the site of origin. However, if there is a small r in V1 with a predominant S wave, it is likely that the origin is from structures posterior to this. As we proceed more posteriorly in origin, the R wave becomes more prominent at the cost of the S wave. At the mitral annulus, the predominant deflection is positive in this lead (Ref. fig. 5). The precordial transition is early (lead V2 or earlier) at sites which are left ward in location whereas the right sided sites are more later in transition during the VA.

When the VA transition is later than the transition in sinus

rhythm, then the site of origin is right sided but if transition is at or earlier than sinus rhythm, we must measure V2 transition ratio. The ratio of the R wave amplitude during VA and during sinus rhythm in lead V2 is the V2 transition ratio (Ref. fig. 6).[15] A ratio of >=0.6 predicts LVOT origin. Unique ECG features for various site of origins are summarized in Table 1.

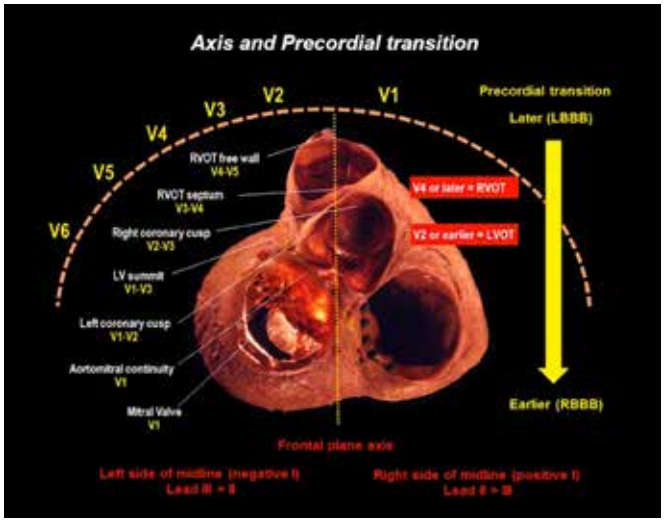


Figure 5: Anatomical schema to understand the electrocardiographic patterns of outflow tract VAs, showing the value of precordial transition and frontal plane axis. The free wall of the RVOT is the most anterior structure, and the precordial transition occurs progressively earlier as we move toward the anterolateral mitral annulus. Lead I polarity allows one to discriminate structures located leftward from the midline from those located on the right side. Note that the anterior aspect of the RVOT is a leftward structure while the right coronary cusp of the aortic valve is a rightward structure. Reproduced from Dr K. Shivkumar. Copyright UCLA Cardiac Arrhythmia Center, McAlpine Collection.

A wide notched appearance of the VA suggests origin from the free wall whereas a narrow VA suggests septal locations of origin (Ref. fig. 7).[13] Epicardial origin of the VA is suggested by an initial slur and a high ratio (>1.5) of the time taken for the intrinsicoid deflection to the rest of the QRS in the VA beat (Ref. Fig. 9)[17]

Non-Outflow Tract Ventricular Arrhythmias

Ventricular Arrhythmias In Structurally Normal Heart

Fascicular VAs[10,16] is a type of a re-entry VA where there is localized peri-conduction system reentry. The site of reentry is usually in the vicinity of the posterior fascicle. Since the VA circuit exits at the posterior fascicle, the VA itself has the morphology of a bi-fascicular block, namely a RBBB and left axis deviation. This is the most common morphology of the arrhythmia, although exits at the left anterior fascicle have also been reported. The morphology then is a RBBB and right axis deviation consistent with left anterior fascicle exit (Refer central figure)

Papillary Muscle VAs[10,16] usually exhibit an RBBB pattern with a wider QRS duration, an R, Rsr', or qR pattern in lead V1. Depending upon which papillary muscle is involved, will give a different activation pattern. Posteromedial papillary muscle VT will have superior axis and anterolateral papillary muscle will have an inferior axis. Precordial transition is usually at V3-V6 (Ref. central figure).

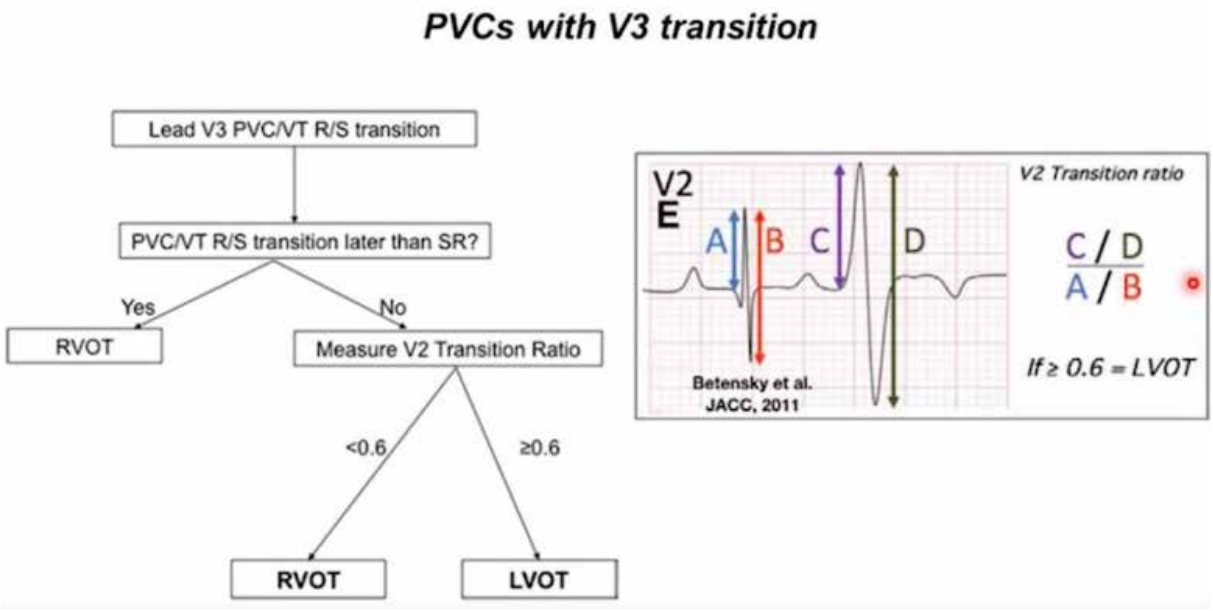


Figure 6: Diagnostic Algorithm for Outflow Tract VT With Lead V3 PVC/VT R/S Transition; If the PVC/ventricular tachycardia (VT) transition to an R>S occurs later than the SR transition (i.e., SR transition lead V1 or V2), then the PVC origin is the RVOT (100% specificity). If the PVC transition occurs at or earlier than the SR transition (i.e., SR transition lead V3 or later), then the V2 transition ratio is measured. If the transition ratio is <0.6, then RVOT origin is likely. If the transition ratio is >=0.6, then LVOT origin is likely (sensitivity 95%, specificity 100%)[15] Betensky et al. JACC Vol. 57, No. 22, 2011; The V2 Transition Ratio[15]

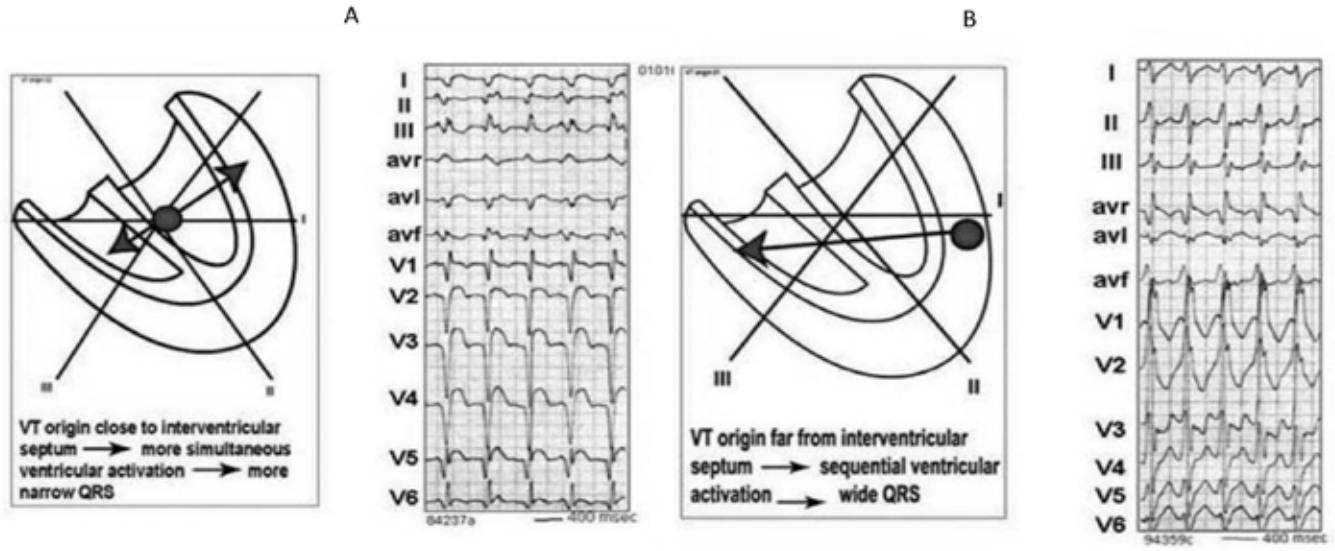


Figure 7: Influence of site of origin of ventricular tachycardia (VT) on QRS width. QRS duration during VT depends on the site of origin. Septal (A) and lateral (B) VTs are shown. Septal VTs produce narrower QRS durations because of earlier access to the His-Purkinje system[13]

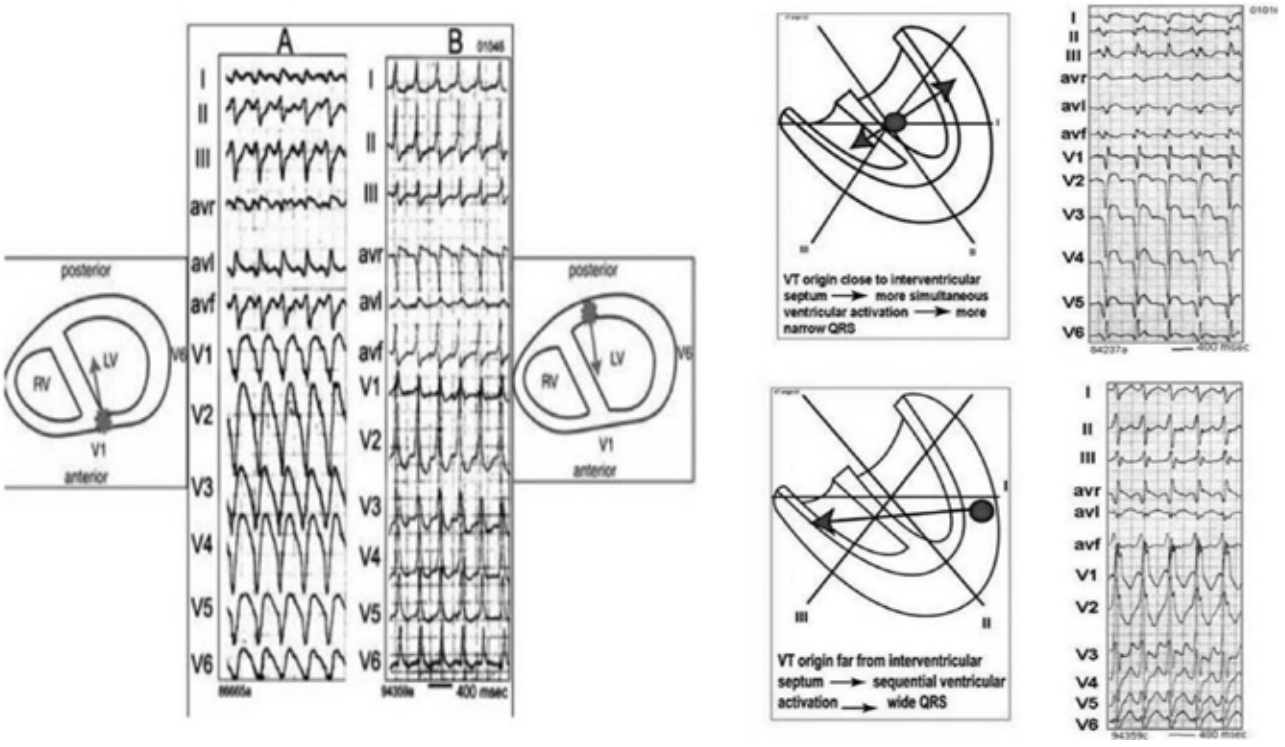


Figure 8: Precordial concordance during ventricular tachycardia (VT). Negative concordance is seen during VT that arises from the anterior most regions of the ventricle, typically in anterior apical infarction. Positive concordance is seen during VT that arises from the posterior most regions, such as the basal inferior wall.[13] Refer abbreviations table for Abbreviations.

Ventricular Arrhythmias In Structural Heart Disease (SHD)

Arrhythmia localization in patients with structurally abnormal hearts based on an ECG suggests only the exit points of the potential circuits. This is not to suggest that only reentrant arrhythmias happen in these hearts but by far, focal arrhythmias remain less common. So, the ECG localization of the arrhythmia exit serves as only a starting point for the arrhythmia management especially if mapping of the arrhythmia is planned.

These arrhythmias follow much of the same principles of localization as above, with the understanding that, there is no QS in aVR and aVL leads. A negative concordance of the precordial leads is always an apical exit of the arrhythmia

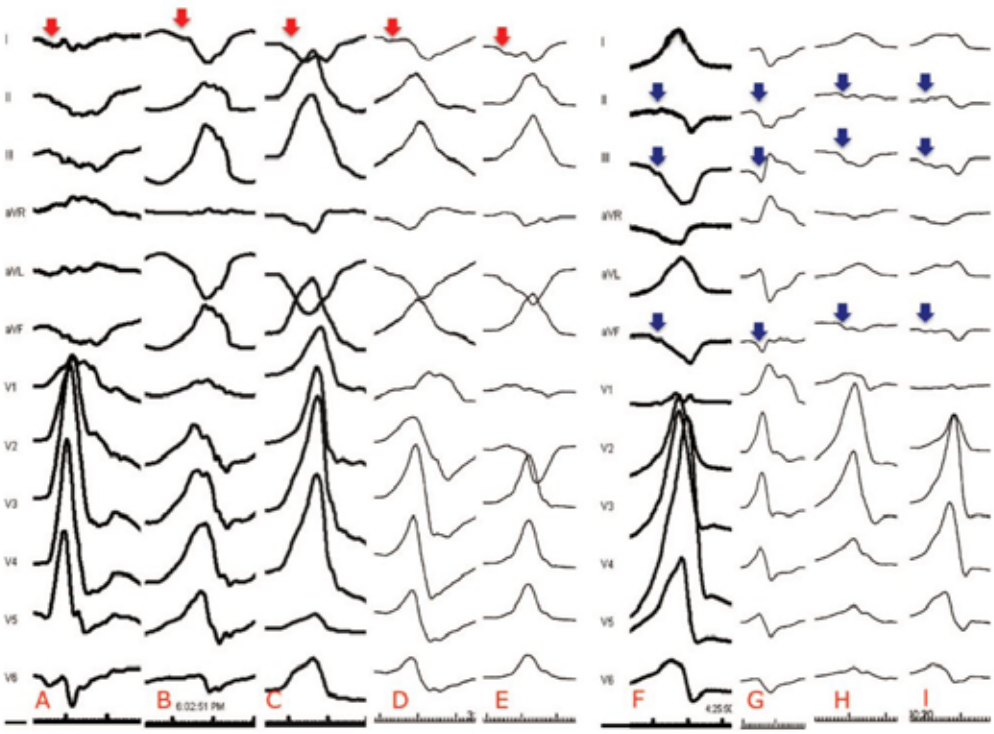


Figure 9: Twelve-lead ECG showing characteristic morphological features of epicardial VAs versus endocardial VAs. A through E, ECG tracings show 5 VAs arising from different EPI superior and lateral basal origins. Red arrows point out q waves in lead I, representing the initial rightward activation from the EPI to the ENDO. F through I, ECG tracings show 4 VTs arising from the opposite ENDO location for comparison. There is no q wave in lead I as the initial activation goes leftward from ENDO to EPI. Blue arrows point out small q waves in inferior leads, representing the initial superiorly directed activation from the ENDO to EPI.[17]

Reproduced from "ECG Criteria to Identify Epicardial Ventricular Tachycardia in Nonischemic Cardiomyopathy" Ermengol Valle`s, MD; Victor Bazan, MD; Francis E. Marchlinski, MD.[17]

whereas positive precordial concordance suggests mitral annular exit (Ref. fig. 8)[13]

In the presence of VA, if QS complex is present in either inferior leads or in lead I, there is high likelihood that the arrhythmia is exiting epicardially in that region. The lack of the small r wave in these complexes indicate that the direction of the vector is predominantly epicardial to endocardial. A small r wave here would indicate that there is endocardium to epicardial conduction (Ref. fig. 9,10).[17]

Early involvement of the conduction system in patients with VA in patients with SHD results in a sharp intrinsicoid deflection or an initial slurring followed by a sharp intrinsicoid deflection. Sometimes in these patients, the conduction limb itself form a part of the reentrant circuit of arrhythmias. These arrhythmias are called bundle branch reentry arrhythmias where the typical morphology is a typical LBBB type VT with an inferior axis. (Ref Fig 11) The arrhythmia usually exits at the RB exit into the myocardium and is perpetuated by the slow transseptal conduction of the diseased heart. Other combinations of fascicular blocks with RBBB morphology exist but are uncommon. The key feature to their recognition remains the presence of the typical BBB and mid cavitary transition, all suggesting fascicular exits of the VA.

Summary

Lead aVR and avL are key differentiating leads for outflow and non-outflow tract VAs. Site of origin of outflow tract VAs can be estimated with fair precision using lead I, V1 and precordial transition. In case of structural heart disease, we can only predict the exit site of the VAs using same localization principles along with precordial concordance. QS in lead I and lead aVR and aVL are also key differentiators between epicardial and endocardial exits of VAs in patients with structural heart disease

Abbreviations

AIV	Anterior Interventricular Vein
AL	Anterolateral
AMC	Aorto Mitral Continuity
AS	Anteroseptal
AV	Aortic Valve
ECG	Electrocardiogram
ENDO	Endocardial
EPI	Epicardial
GCV	Great Cardiac Vein

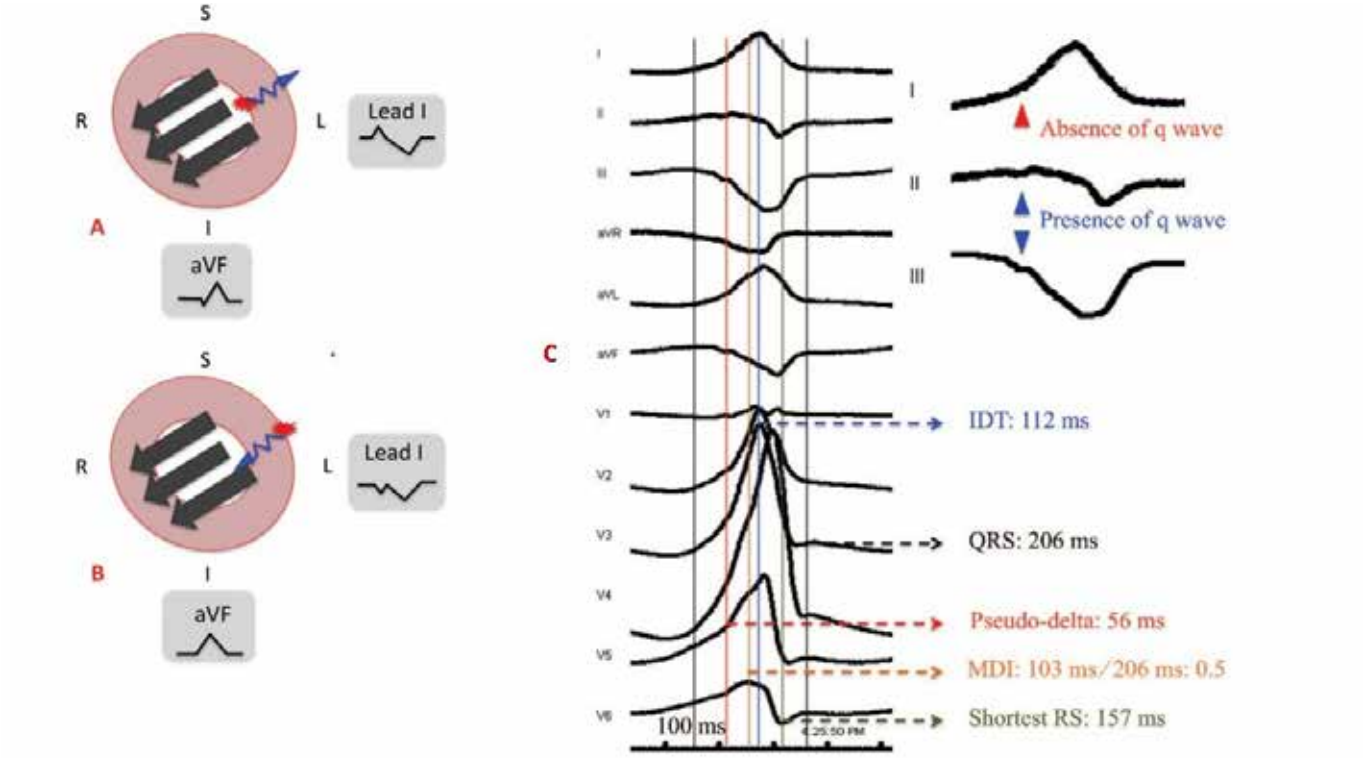


Figure 10: Schematic representation showing suggested basis for morphological differences in QRS in lead I and aVF based on initial regional (small arrow) followed by global LV activation (large arrows) from endocardial versus epicardial VT origin from superior-lateral LV. (A) VT origin from the endocardium shows small q waves in inferior leads and small r wave in lead I represent the small segment of myocardium that depolarizes with an ENDO to EPI activation before the main activation wave front proceeds from a superior to inferior and left to right direction. (B) VT origin from the epicardium will not show an initial q wave in the inferior leads and will consistently show a q wave in lead I as initial activation is more consistent with the net vector of the global activation pattern from left to right and superior to inferior; (C) QRS from ENDO VT showing discrepancy between interval and morphology criteria. This example demonstrates all interval and morphology criteria routinely assessed. The QRS from VT with ENDO site of origin in the example shown demonstrates interval criteria suggesting EPI VT but morphology criteria (absence of a q wave in lead I and presence of small q waves in inferior leads) supporting an ENDO origin.[17]

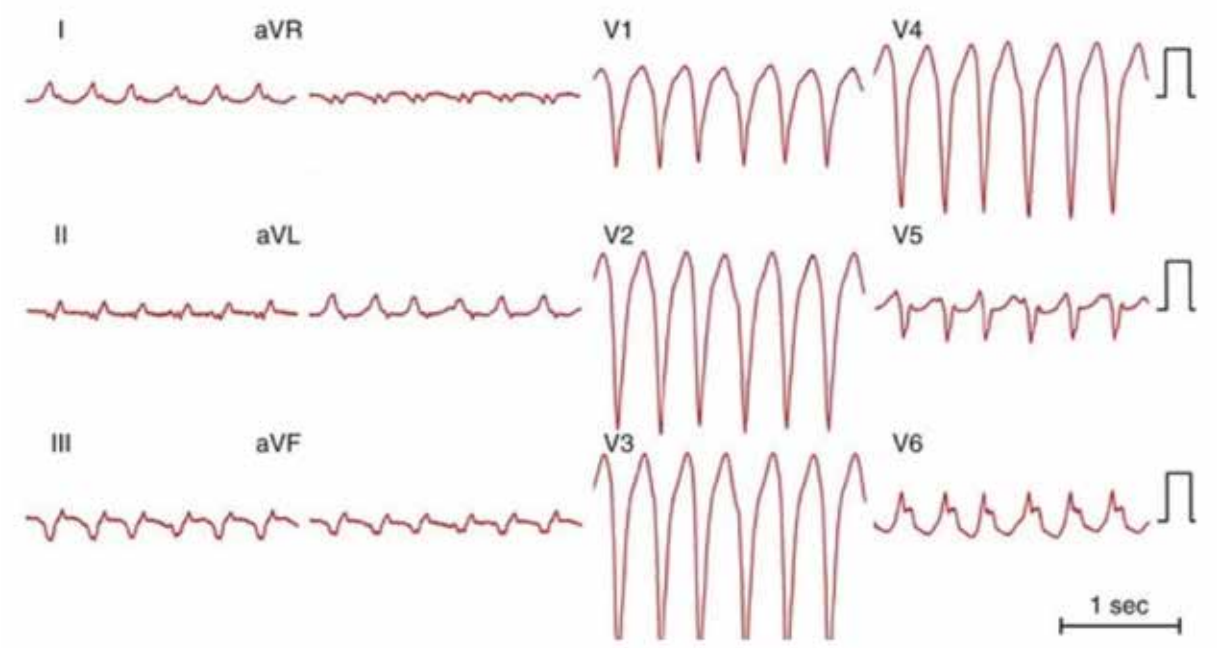


Figure 11: An example of Bundle Branch Reentry Tachycardia with Left Bundle Branch Morphology

Table 1: Features of PVCs arising from various locations

Origin	Ecg Features				
	V1 Morphology	**Lead I**	**Inferior leads**	**Precordial transition**	**Special comments**
Inflow outflow region of RVOT	QS in lead V1	Monophasic R wave	Amplitude of R wave is reduced. R wave in lead III<R wave in Lead II	Early precordial transition[31]	Conus papillary muscle may be present,[30] varying morphology of PVC depending on the exit site of the PVC
RVOT: below the pulmonary valve	No R wave, all negative	Positive	Tall R waves with R wave in lead III<R wave in Lead II	Late precordial transition[32]	-
Right coronary sinus of Valsalva	Initial R wave, then bigger S wave (due to more posterior location than anterior wall of RVOT – which would be most anterior)	Positive/ Biphasic	Tall R waves with R in lead III<R in lead II	Early/late precordial transition	-
Left coronary sinus of Valsalva	R wave > S wave	Biphasic/ negative	Tall R waves in with R in lead III>R in lead II	Early precordial transition	qrS in lead V1 for origin at the commissure between RCSoV and LCSoV [29]
Aorto mitral continuity	Positive (R wave >S wave).	Biphasic/ Negative	Positive for anterior locations.	Positive precordial concordance/early transition	qR pattern, LBBB morphology in V1, transition> V1, varied morphology of PVC may be associated with remnant conduction tissue here[27,28]
LVOT origin below the aortic sinuses of Valsalva	Small R wave (89%)	Biphasic / Negative	Tall R waves with R wave ratio in lead III/II >1.1	Positive precordial concordance/Early transition	aVL/aVR Q-wave amplitude ratio >1.4[26]
Mitral annulus					
Anterolateral(AL)	Monophasic R wave	RS mostly	R wave	Positive precordial concordance in all 3 types	S wave was always present in lead V6 in all the types. Late notching in the inferior leads seen in AL and Pos sites of origin and absent in PS site of origin[25]
Posterior(Pos)	Monophasic R wave	R>s	rS		
Posteroseptal(PS)	Monophasic R wave	R>s	rS		
LV summit	Prominent R wave	Biphasic/ Negative	Tall R waves with R wave ratio in lead III/II >1	Positive precordial concordance/Early transition	Initial slurring in QRS (pseudodelta) wave. Increased maximum deflection index. Pattern break in precordial R wave in lead V2.[19]

HIS	Bundle of His
ILVT	Idiopathic Left Ventricular Tachycardia
LA	Left Atrium
LAD	Left Anterior Descending
LBBB	Left Bundle Branch Block
LCSoV	Left Coronary Sinus of Valsalva
LCx	Left Circumflex
LMCA	Left Main Coronary Artery
LV	Left Ventricle
LVOT	Right Ventricular Outflow Tract
MCV	Middle Cardiac Vein
MPA	Main Pulmonary Artery
MV	Mitral Valve
OT	Outflow Tract
OTVA	Outflow Tract Ventricular Arrhythmia
PA	Pulmonary Artery
PL	Posterolateral
PS	Posteroseptal
PV	Pulmonic Valve
PVC	Premature Ventricular Contraction
RA	Right Atrium
RBBB	Right Bundle Branch Block
RCA	Right Coronary Artery
RCSoV	Right Coronary Sinus of Valsalva
RV	Right Ventricle
RVOT	Left Ventricular Outflow Tract
SoV	Sinus of Valsalva
TV	Tricuspid Valve
VA	Ventricular Arrhythmia
VT	Ventricular Tachycardia

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RBBB: Conduction Abnormality or Brugada Syndrome or ARVC

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Introduction

Right bundle branch block (RBBB) is a common electrocardiographic finding seen in a variety of clinical conditions contrasting in severity from a benign idiopathic variant to those with high risk of malignant arrhythmias. RBBB indicates slow or block in the conduction in the right bundle branch and may be seen in structurally normal heart or may result from trauma during catheterization, ventriculotomy, myocardial disease, congenital heart disease, dyselectrolytemia, lung disease or sometimes due to erroneous electrode placement or inappropriate filter setting among many other causes. Despite the tailor-made criteria used for diagnosing Brugada Syndrome (BrS) and Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC), their ECG features can often masquerade simple RBBB. Hence, it remains a challenge to distinguish those entities from RBBB attributable to conduction disease by surface ECG alone.

Electrocardiographic Representation

1. RBBB in conduction abnormality-

   The characteristic ECG findings in RBBB include-

   i. QRS duration is greater than or equal to 120 milli-seconds

   ii. RSR' in leads V1 and V2

   iii. S wave is of greater duration than the R wave, or the S wave > 40 ms in leads I and V6

   iv. Normal R wave peak time in leads V5 and V6

   v. R wave peak time > 50 ms in lead V1

The T waves tend to be discordant to the terminal QRS vector. This results in inverted T waves in the right precordial leads and upright T waves in the left precordial leads.[1]

2. Brugada Syndrome:

   Brugada syndrome as originally described is character-ized by RBBB with ST segment elevation in V1 to V3 and syncope. Three types have been classically described.

   Type 1: Coved ST segment elevation >2mm in more than one of leads V1-V3 followed by a negative T wave.

   Type 2: Saddleback shaped ST elevation >2mm

   Type 3: Morphology of either type 1 or type 2, but with <2mm of ST segment elevation

3. ARVC: The ECG criteria in the revised task force criteria for diagnosis of ARVC include 2

I. Repolarisation abnormalities

   a. Major: Inverted T waves in right precordial leads (V1, V2, and V3) or beyond in individuals >14 years of age (in the absence of complete RBBB QRS ≥120 ms)

   b. Minor:

      i. Inverted T waves in leads V1 and V2 in individuals>14 years of age (in the absence of complete RBBB) or in V4, V5, or V6.

      ii. Inverted T waves in leads V1, V2, V3, and V4 in individuals >14 years of age in the presence of complete RBBB

II. Depolarisation/conduction abnormalities

   a. Major: Epsilon wave (reproducible low-amplitude signals between end of QRS complex to onset of the T wave) in the right precordial leads (V1 to V3)

   b. Minor:

      i. Late potentials by SAECG in ≥1 of 3 parameters in the absence of a QRS duration of ≥110ms on the standard ECG

      ii. Filtered QRS duration (fQRS) ≥114 ms

      iii. Duration of terminal QRS<40μV (low-amplitude signal duration) ≥38 ms

      iv. Root-mean-square voltage of terminal 40 ms≤20μV

      v. Terminal activation duration of QRS≥55ms measured from the nadir of the S wave to the end of the QRS, including R', in V1, V2, or V3, in the absence of complete RBBB

Is RBBB benign?

Isolated RBBB has generally been considered to be benign,[3] but a recent study indicated that RBBB is associated with higher cardiovascular risk and all-cause mortality.[4] In addition, a higher prevalence of RBBB is seen in patients with idiopathic VF5 and RBBB can conceal the ECG phenotype of BrS6 and ARVC. Hence, complete RBBB does not necessarily indicate a benign ECG finding.

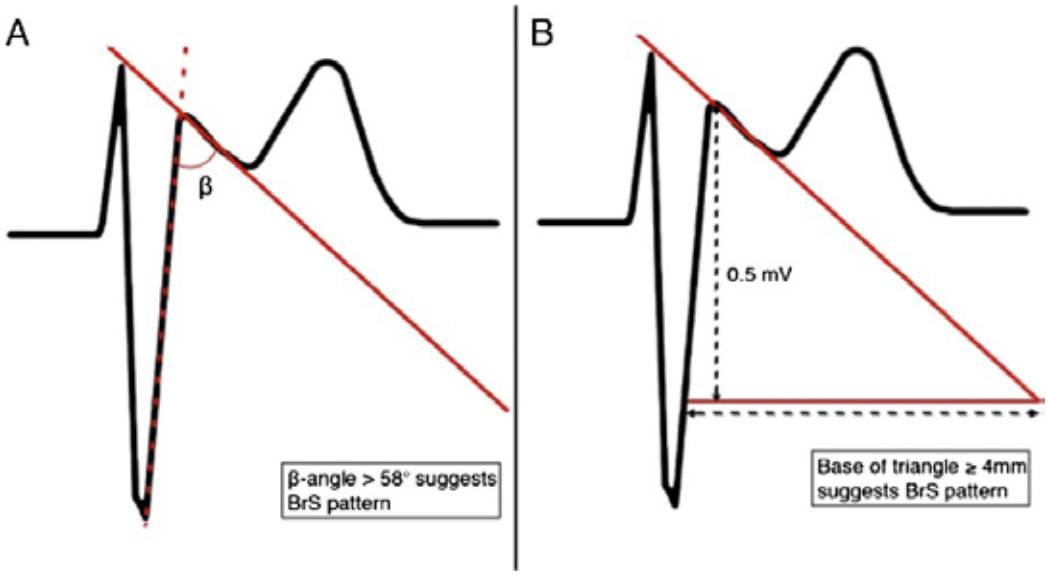

Figure 1A: Distinction between BrS and RBBB by the application of β angle Fig. 1B: Base of triangle as cut off length to distinguish between BrS and RBBB

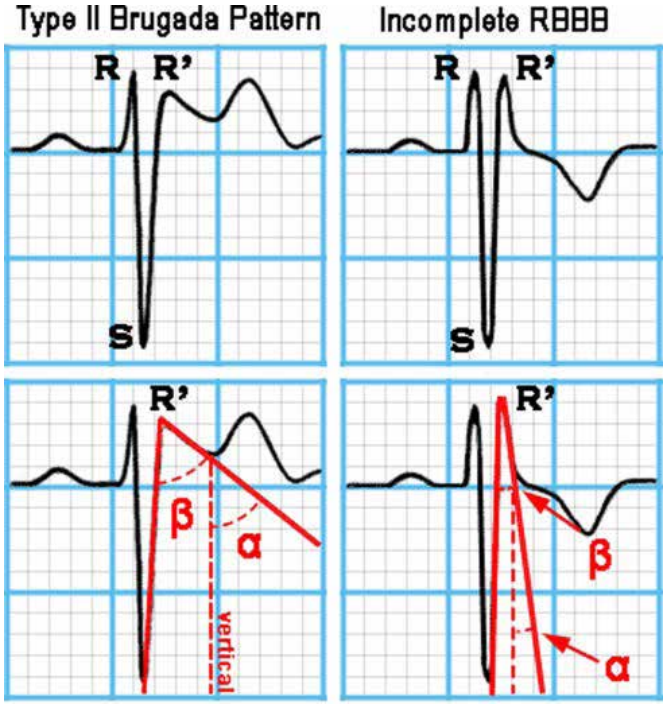

Figure 2: Angles formed between upslope of S wave and downslope of R' to differentiate Brugada syndrome form incomplete RBBB.

How do Brugada syndrome and ARVC simulate RBBB?

Brugada syndrome is a channelopathy characterized by repolarization abnormality representing ST changes and J point elevation in the right precordial leads that often appears to be r'/R' of RBBB in lead V1. On the other hand, ARVC is a genetic fibrofatty infiltrative cardiomyopathy that results in electrocardiographic changes of conduction delay reflected by terminal notch of QRS in V1 i.e., Epsilon wave which often mimics r' of RBBB. Hence it is important to identify and appreciate the nuances of RBBB pattern that can potentially portend much higher risk rather than simple conduction disorder alone.

Discrimination Among Three Entities

BrS Vs RBBB

ECG of Brugada syndrome often mimics RBBB due to J point elevation which results in pseudo RBBB pattern. In type1 Brugada syndrome, the characteristic ST elevation in V1 clearly differentiates it from that due to conduction abnormality whereas ECG of type 2 pattern may be difficult to distinguish from incomplete RBBB. Ohkubo et al[7] and Chevallier et al,[8] both introduced new criteria to discriminate between non-type 1 Brugada from ordinary incomplete RBBB. A **β-angle**, i.e., an angle between the upslope of the S wave and the downslope of the r'-wave (fig1A) of more than 58° indicates BrS with a positive predictive value of 73%. Serra and colleagues showed that the length of the base of the triangle formed by the upslope of the S wave and downslope of the r' wave either at the isoelectric point or at 0.5mV from the r' spike (fig.1B) ≥ 4 mm suggests BrS.[9]

An rSr' pattern in an athlete's heart is characterized by a narrow r' and minimal ST elevation compared to the broader r' in BrS (Fig 2) a distinction that gets easily blurred due to inconsistent ECG electrode placements.10 High pass filtering and high placement of the precordial leads have both been shown to produce rSr' pattern in V1-V2 in normal individuals. Provocation test by drug like ajmaline or sodium channel blocker may help unmask the typical ECG pattern of BrS in non-type 1 variants.11 Brugada phenocopy is a relatively new categorization of etiologies that produce a Type 1 or Type 2 Brugada patterns in precordial leads V1-V3 that normalizes upon resolution of underlying reversible condition.

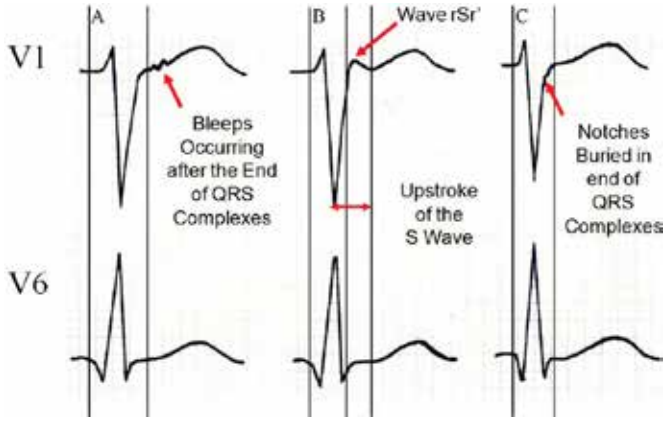

Figure 3: Different types of epsilon waves appearing as incomplete RBBB in ARVC

Sometimes, complete RBBB can mask Brugada phenotype and provocative testing with drugs (ajmaline or Flecainide) to unmask and induce ST elevation may be needed for diagnosis.[11] Febrile illness was also shown to unmask BS in patients with complete RBBB.[12] Aizawa et al studied ECG variables in patients with BrS with complete RBBB and the control subjects with RBBB and found that the patients with BrS had a wider QRS duration (170±13 vs 145±15 ms in V1) and a larger amplitude of R in V1 compared to that in control subjects with RBBB.[13]

ARVC Vs RBBB

ECG in patients with ARVC appears to be RBBB pattern and may simulate conduction system disease (Fig 3). The frequency of complete and incomplete RBBB in patients with ARVC is 6 and 12.5% respectively.[14] As in the Task Force Criteria, T wave inversions in precordial lead beyond V4 in RBBB suggests ARVC.2 In all patients with ARVC, localized right precordial QRS prolongation is seen, i.e., RS duration: (V1+V2+V3)/(V4+V5 +V6) ≥ 1.2 with QRSd ≥ 100 ms in 2 of 3 right precordial leads.[15]

Between ARVD and controls with complete RBBB, the only two parameters which differed were the prevalence of T wave inversion through V4 (59% versus 12% respectively) and an r'/s ratio in V1 < 1 (88% versus 14% respectively).[15]

Overlap between ARVC and BrS

Overlapping ECG characteristics of BrS and ARVC have been reported in patients with varying presentations. Drug provocation by sodium channel blocker can induce ST elevation in subgroup of patients with ARVC and epsilon like waves are seen in some patients with type 1 BrS ECG, especially in drug induced type 1 patten. Of note, in complete RBBB use of ajmaline challenge is contraindicated but carotid sinus massage can sometimes be used to unmask a typical Brugada-like pattern.16

Conclusion

The presence of RBBB in ECG may be attributable to myriad of clinical entities. BrS and ARVC are two important subsets which require critical and comprehensive evaluation of ECG findings to distinguish it from simple RBBB. The diagnostic value of different ECG algorithm and markers should be considered during screening of BrS and ARVC in the presence of a complete or incomplete RBBB.

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Conduction Disorders in Contemporary STEMI Patients

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Introduction

To understand the conduction disorders in AMI setting, knowledge of vascular supply of the conduction system of the heart is very important.

Sinoatrial Node is supplied by the Sinonodal Artery which arises from Right Coronary Artery (RCA) in 2/3rd cases and from Left Circumflex artery (LCx) in rest of the cases. Atrioventricular node again gets its blood supply from the RCA in majority of cases, and rest through LCx. Right Bundle Branch and the Anterior Fascicle of Left Bundle Branch receive their blood supply from LAD, while the post fascicle of left bundle branch has dual blood supply both from RCA and LAD.

In Acute IWMI, usually the culprit vessel is RCA, sometimes LCx and in Acute AWMI, the culprit vessel is LAD.

Following conduction disorders which occur in the setting of AMI will be discussed in detail :

1. At the level of SA Node
    a. Sinus Bradycardia
    b. SA Blocks
    c. Interatrial blocks
2. At the level of AV Node
    a. AV Blocks
3. Below the AV Node
    a. Fascicular blocks
    b. RBBB
    c. LBBB

Sinus Bradycardia

Sinus Bradycardia is quiet common in Acute IWMI setting and is 3 times commoner as compared to Acute AWMI setting.[1] It may be associated with vasodilatation and hypotension – Bezold-Jarisch Reflex. Vagally induced sinus bradycardia disappears with atropine, while bradycardia related to ischemia persists. Patients presenting with RCA occlusion especially proximal occlusion showed bradycardia on admission in 15% of the cases, but none with LCx occlusion.[2]

Sinoatrial Blocks

SA Nodal Artery occlusion (which supplies the SA node) leads to sinus bradycardia, SA Blocks or even asystole. Sinus Bradycardia and SA Blocks can also happen with atrial infarcts.

Interatrial Blocks

Conduction delay between the right and left atrium leads to interatrial block (IAB) which shows on the surface ECG as biphasic P waves in inferior leads (II, III and aVF) with P wave duration of ≥ 120 ms. Partial IAB is said when the duration of P wave is ≥ 120 ms, however the morphology of P wave is normal. In a series, partial or advanced IAB were noted in about half of the patients acute STEMI and decreased to about a quarter after 6 hours of primary percutaneous coronary intervention (PCI). IAB on admission is also an independent predictor of atrial fibrillation at 12-months follow up. In another study of 972 STEMI patients, 21.3% had partial and 5.9% advanced IAB and patients with IAB had higher all-cause mortality. But on multivariate analysis, IAB did not show any independent association with prognosis in such patients.

Atrioventricular Blocks

AV node, in majority of cases is supplied by RCA. However in left dominant circulation, it is supplied by LCx. Hence, AV blocks are quiet a common finding in acute IWMI settings. 20 Mobitz type 2 and 30 high grade AV blocks (HDAVB) are seen when RCA is the culprit vessel with proximal occlusion. There is increased acetylcholine release from inferior wall myocardium because of RCA occlusion, contributing to AV block. Ischemic AV blocks have a faster heart rate as compared to vagally induced AV blocks.

Mobitz type 1 (Wenchebach) AV Block is also quiet common in acute IWMI caused by RCA occlusion and is usually supra-

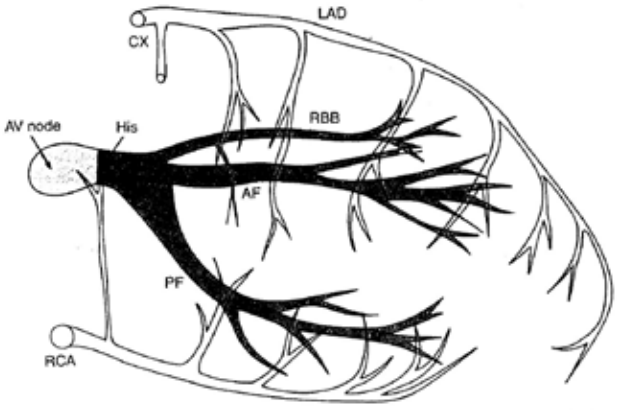

Figure 1: Blood supply of the Conduction System of the Heart

hisian and transient while Mobitz type 2 AV blocks are usually infra-hisian and carry a worse prognosis.

In acute IWMI, third-degree AV block usually evolves from first-degree AV block and gradually progresses and is usually supra-hisian and is usually temporary, benign and nearly always resolves with reperfusion therapy. While, HDAVB in acute AWMI is usually infra-hisian with wide QRS complex and carries worse prognosis and if unstable with hemodynamic deterioration, will require pacemaker implantation.

Earlier studies have reported 19% incidence of second- and third-degree AV block in acute IWMI, however recent studies have reported an incidence of HDAVB in STEMI patients of 1.5% to 2.1%. A recent large registry of 16536 STEMI patients reported the incidence of HDAVB in IWMI as 6.6% and only 0.3% in AWMI.3 Patients presenting with HDAVB in STEMI carries poor prognosis. 30-day mortality in STEMI patients with HDAVB has been reported as 8.8% as compared to 2.3% in patients without HDAVB (p=0.005). HDAVB with AWMI always carried worse prognosis.

Fascicular Blocks

AWMI patients presenting with new-onset left anterior fascicular block (LAFB) indicates proximal LAD occlusion as the first major septal branch of LAD supplies the left anterior fascicle. New-onset LAFB in IWMI indicates significant concomitant stenosis of LAD.

Left posterior fascicular block (LPFB) is quiet rare in STEMI because it is thick and has dual blood supply from RCA and LAD both and development of LPFB in STEMI suggests multi-vessel involvement.

An entity, left septal fascicular block (LSFB) though not universally accepted, has been mentioned. There are few anecdotal reports in the literature of the presence of LFSB in patients with acute MI. ECG criteria for LSFB is as following

- Normal or slightly increased (up to 110 ms) QRS duration
- R wave voltage of $V_1 \geq 5$ mm
- R wave voltage of $V_2 > 15$ mm
- Absence of small q wave in V_5, V_6 and in Lead I.

Right Bundle Branch Block (RBBB)

Proximal LAD occlusion may result in new-onset RBBB and may be associated LAFB in acute AWMI setting as both right bundle branch and left anterior fascicle of the conduction system receive the blood supply from the anterior septal branch of LAD or jointly with AV nodal artery.

RBBB in acute IWMI is rare.

Diagnosis of STEMI is not difficult in the presence of RBBB.

Patients presenting with STEMI and RBBB have poor prognosis.

Challenge remains when patient present with typical ischemic symptoms with new-onset RBBB without associated ST-segment or T wave changes. Emergent coronary angiography and primary coronary intervention in such situations are indicated.[4]

Left Bundle Branch Block

Left bundle branch of the conduction system has dual blood supply from LAD through its anterior septal branches and AV nodal artery of RCA, therefore it is less vulnerable as compared to right bundle branch. New-onset LBBB in STEMI when noted, will happen in the presence of multi-vessel disease. New or presumably new-onset LBBB in the Minneapolis Heart Institute STEMI study in the cohort of 3903 patients was 3.3% and these patients were older, women more commonly affected, had low ejection fraction with more

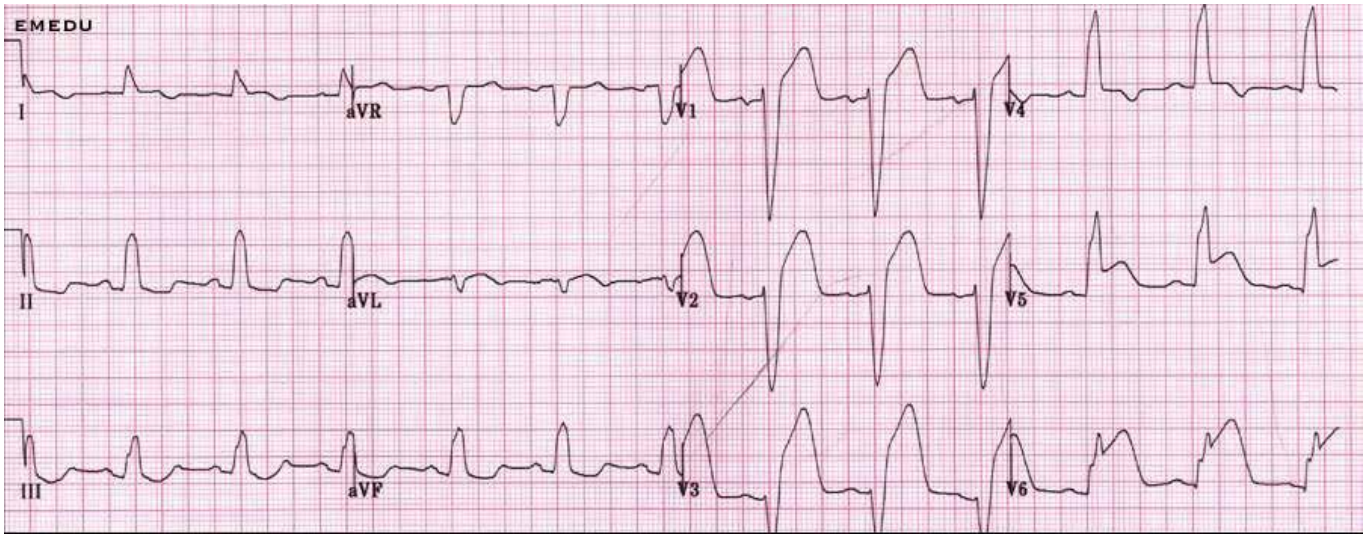

Figure 2: Acute AWMI in the presence of LBBB. Concordant ST-segment elevation in V_5 and V_6

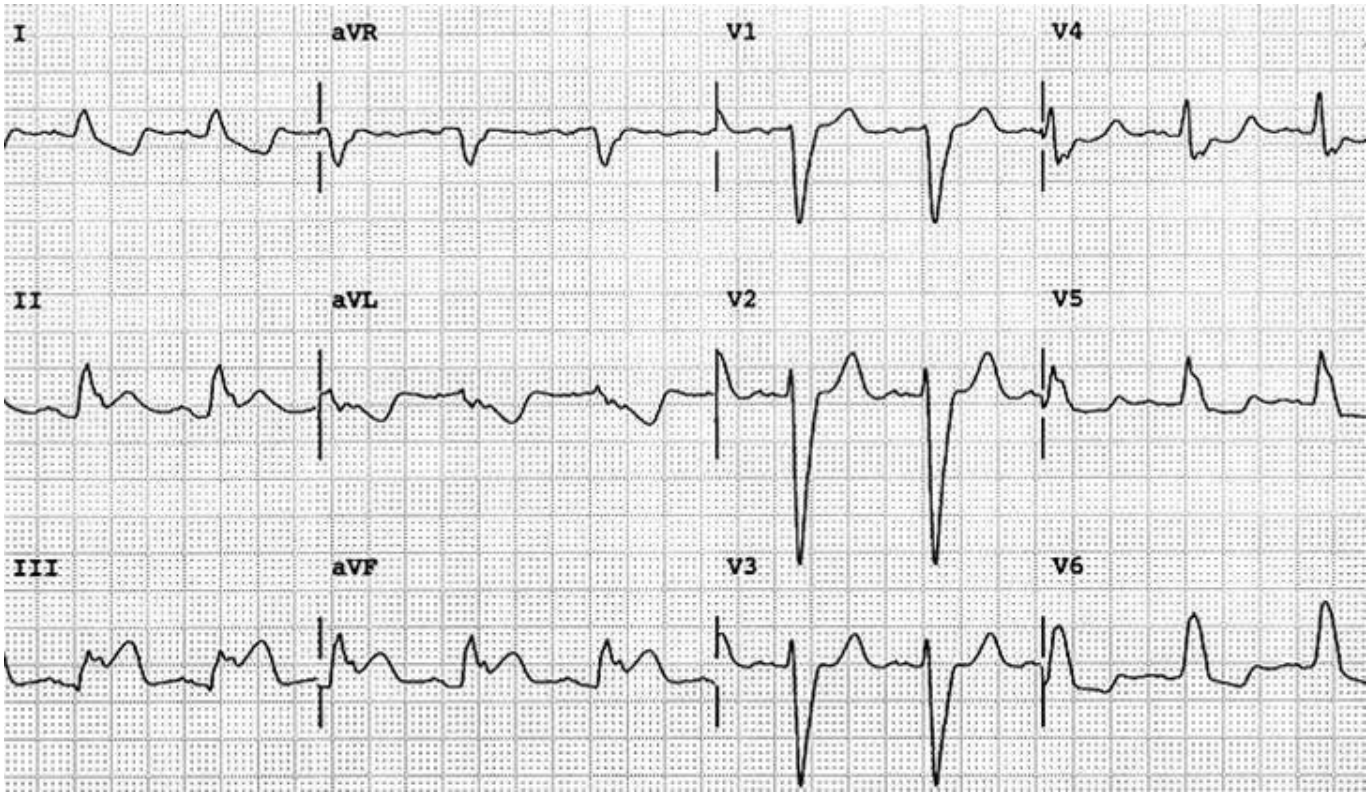

Figure 3: Acute IWMI in the presence of LBBB. Concordant ST-segment elevation in LII, LIII and aVF.

cardiac arrest and heart failure. Higher all-cause mortality was reported at 1-year follow up.

Another study reported 16.2% in-hospital mortality in STEMI patients with LBBB as compared to 6.5% in patients without LBBB.5

Recognition of AMI in the presence of LBBB always remains a challenge. Sgarbossa et al. described to identify AMI in the presence of LBBB6

1.	Concordant ST-segment elevation with QRS complex	≥ 1 mm	5 points
2.	ST-segment depression in leads V_1, V_2, and V_3	≥ 1 mm	3 points
3.	Discordant ST-segment elevation with QRS complex	≥ 5 mm	2 points

3 or more points were considered diagnostic of AMI and 2 points suggest AMI. However, the Sgarbossa criteria was quiet specific ((98%), but less sensitive ((20%).

Emergent coronary angiography in patients with suspected AMI with LBBB until unless ECGs as shown below where the AMI diagnosis can be made with certainty, the decision cannot be taken based on 12 lead ECG. Clinical presentation and highly-sensitive troponin testing are important tools for better decision.

2013 ACCF/AHA STEMI guidelines7 have mentioned that new or presumably new-onset LBBB shall be considered as AMI equivalent. Most of the time old ECG is not available, so it is difficult to ascertain whether LBBB noted in these settings is old or new. 2017 ESC STEMI guidelines[8] remarked that presence of a presumed new LBBB does not predict an MI per se.

Temporary Pacing

In acute IWMI setting, the blocks are usually temporary and benign, and get resolved over a period of time with reperfusion therapy. In most of the situations, temporary pacing is not indicated, however if there is hemodynamic compromise or bradycardia-related arrhythmias, temporary pacing to be done to tide over the crisis. In acute AWMI with HDAVB, prophylactic temporary pacing is indicated.

Conclusions

Conduction disorders are frequently associated in acute MI setting. SA blocks and AV blocks are more often associated with acute IWMI, while fascicular blocks and RBBB are commonly associated with acute AWMI in relation to blood supply of the conduction system affected with the type of MI. Usually, the conduction disorders associated with STEMI are transient and benign and are reversible with reperfusion therapy. However, the prognosis is affected if the conduction disorders persist. New-onset or presumably new LBBB with typical history of ACS may be treated as MI equivalent as diagnosis of MI in the presence of LBBB is difficult

and it denotes severe coronary artery disease and carries worse prognosis. Temporary pacing is indicated if there is hemodynamic compromise and/or HDAVB in acute AWMI setting.

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COVID-19 Infection and its Electrocardiographic Manifestations

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Introduction

The COVID 19 infection has been one of the worst pandemics in the recent times infecting over 5.1 million people and causing more than 330,000 deaths.[1] Though predominantly the disease affected the lower respiratory tract, COVID 19 also caused a variety of cardiac involvement and electrocardiographic manifestations.[2–4] It is therefore essential for the emergency clinicians to recognize the electrocardiographic (ECG) manifestations of COVID 19.[5,6] SARS-CoV 2, RNA based virus, utilizes the ACE-2 receptor to enter cells by receptor-mediated endocytosis.[7,8] Respiratory symptoms associated with COVID-19 infection primarily coming from ACE2 expression in the type 2 alveolar cells; additionally, 7.5% of myocardial cells express the ACE2 receptor.[9–11] The virus can cause an hyperinflammatory state, leading to vascular inflammation, cardiac injury, plaque instability, hypercoagulability, and myocardial depression. Myocardial abnormalities with electrocardiogram (ECG) changes in COVID 19 may be due to this cytokine storm, hypoxic injury, electrolyte abnormalities, plaque rupture, coronary spasm, and microthrombi as well as direct endothelial and myocardial injury.[2–4]

Types of Arrythmias

A variety of arrhythmia may occur with COVID 19 infection.[2,3,5,6] ECG abnormalities are common in COVID 19 infected patients and present up to 93% of the hospitalized critically ill patients. Palpitations likely reflective of dysrhythmia may be present in approximately 7% patients and incidence of dysrhythmias can be noted in 17% of the general cohort and 44% patients in the IntensiveCare unit (ICU) setting experience dysrhythmias.[2,3,5,6,12] ECG abnormalities are also associated with increased risk of in-hospital mortality.[12,13]

Supraventricular Tachycardia

Sinus tachycardia is the most common supraventricular tachycardia encountered in the ill COVID-19 patient, resulting from the usual causes, including hypovolemia, hypoperfusion, hypoxia, elevated body temperature, pain, and anxiety. Atrial fibrillation (Fig. 1) is the next most common SVT noted in patients with COVID 19 infection.[14] Atrial fibrillation can present in varied ways including new- onset, recurrence of a pre-existing dysrhythmia, and persistence of permanent atrial fibrillation with new rapid ventricular response. Both sinus tachycardia and atrial fibrillation are independent predictors of illness severity, myocardial injury, and poor outcomes in COVID-19.[14] One study conducted in New York hospitals found atrial fibrillation/flutter was present in 14.3% of patients at admission and occurred in 10.1% of patients during hospitalization.[15] Another study found atrial fibrillation/flutter to be present in 22% of critically ill patients requiring mechanical ventilation.[16] Atrial fibrillation is more common in patients following an inflammatory insult such as cardiomyopathy from COVID-19 and occurs in up to half of the patients admitted to an ICU.[12,17] Two case reports demonstrated the highly variable impact that COVID-19 has on the heart;[18,19] in one case,[18] the patient presented in atrial flutter and then converted the atrial fibrillation, eventually reverting to sinus rhythm 48 hours later.

Other SVT, like AV nodal re-entrant tachycardia (Fig. 2) and AV re-entrant tachycardia have not been frequently described in patients with COVID19 patients. COVID-19 patients admitted for these arrythmias were found to have asymptomatic or mildly symptomatic infections.

Malignant ventricular arrhythmias are a known complication of viral myocarditis and cardiomyopathy, with ventricular tachycardia (VT) and/or ventricular fibrillation (VF) occurring in 1-6% of patients.[5,13,20,21] In patients with COVID-19, these arrhythmias may be due to a combination of QT interval prolonging medications, metabolic abnormalities, and myocardial inflammation. COVID-19 patients with elevated troponin have been shown to have a higher incidence of VT than those with normal troponins.[22,23]

VT with a pulse is seen with both monomorphic and polymorphic presentations. Monomorphic ventricular

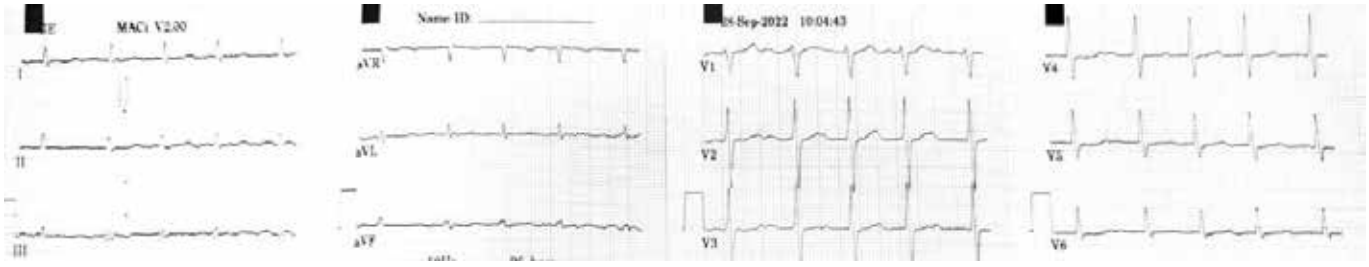

Figure 1: Atrial fibrillation with fast ventricular response in a patient admitted with severe COVID-19 infection

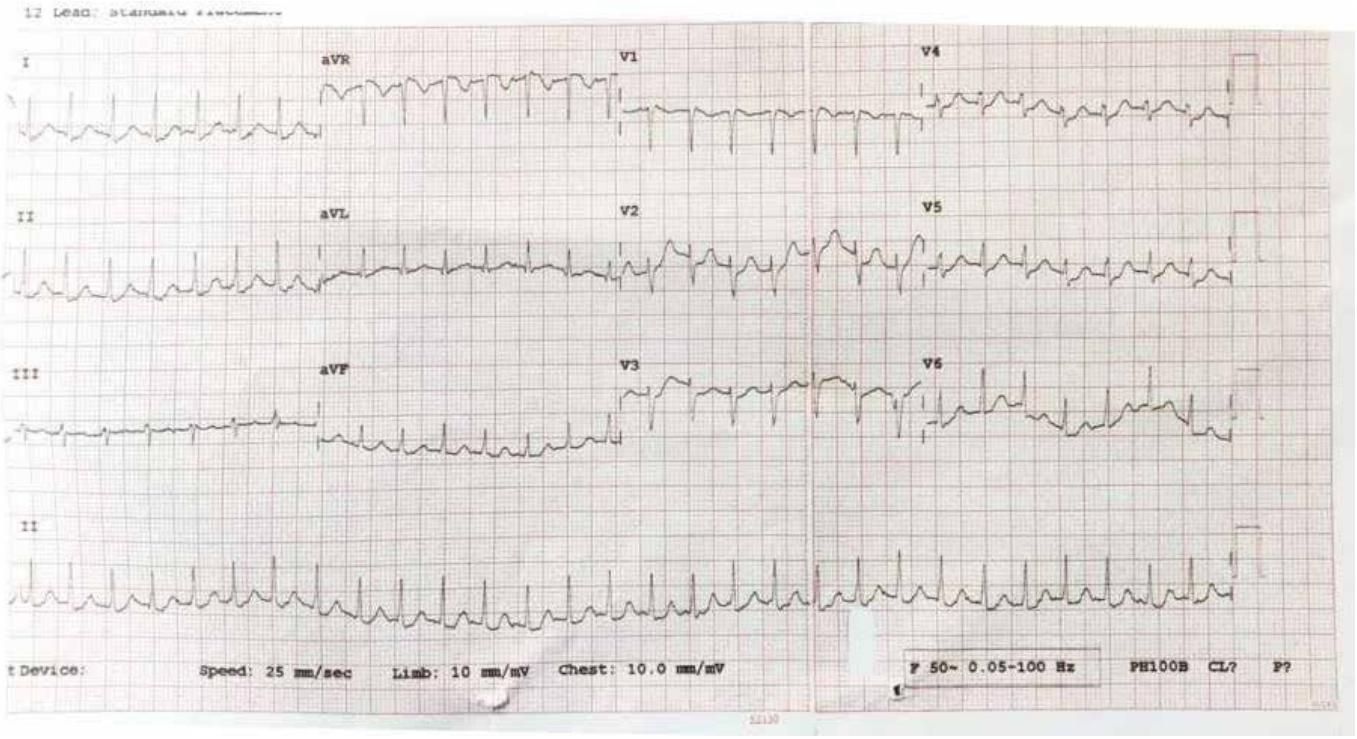

Figure 2: AVNRT in a patient admitted with COVID 19 infectionVentricular Tachycardia and Ventricular Fibrillation

tachycardia (MVT) is the most frequent form of VT seen in the COVID-19 patient, frequently resulting from structural heart disease such as acute coronary syndrome with STEMI, myocardial injury, or myocarditis; patients with pre-existing structural heart disease of many types can provide the substrate for MVT during periods of extreme physiologic stress due to the range of issues encountered in the COVID-19-infected patient. Polymorphic VT (PVT), including the PVT subtype torsade de pointes, results from functional (i.e., non-structural) heart disease and is likely less common; it most often occurs in situations involving medication toxicities, electrolyte abnormality, and various pro-arrhythmic states (e.g. Brugada syndrome, long QT syndrome).

Cardiac arrest dysrhythmias include the traditional 4 rhythm scenarios: the "shockable" (pulseless VT and VF) and "non-shockable" (pulseless electrical activity [PEA] and asystole) dysrhythmias. Specific dysrhythmia diagnosis in the cardiac arrest patient, whether out-of hospital or hospital-based, is unchanged in the COVID-19 pandemic. During the pandemic, the occurrence of both out-of-hospital cardiac arrest (OHCA) and hospital-based cardiac arrest were increased.[24,25] A study conducted in Italy found close to a 60% increase in OHCA in 2020 compared to 2019.[24] A second study in France found a 52% increase in OHCA between February and April 2020, compared to 2019.[25] This unfortunate increased rate of OHCA has also been seen in the United States. For example, in New York City there was a three-fold higher rate of patients undergoing resuscitation in the out-of-hospital setting when compared to a similar 2019 pre-pandemic period; the odds ratio (OR) of encountering "non shockable" dysrhythmias increased significantly with an OR for PEA of 3.50 and an OR for asystole of 1.99.[26] The mortality rate of patients in cardiac arrest increased in all three studies.[24,25] There are several factors that may contribute to this increased occurrence with more frequent poor outcome, including the COVID-19 infection itself as well as the delays in seeking care related to the pandemic, and lower rates of important bystander care in the pre-arrival period of cardiac arrest management.[27]

Patients hospitalized with COVID-19 are at increased risk of cardiac arrest, but the rhythms associated with cardiac arrest vary in the inpatient population. A study focusing on hospitalized patients in New York City noted that asystole occurred most often, followed by PEA and then pulseless VT/VF.[15] Another study found 9 patients out of 700 experienced cardiac arrest, with only one having a shockable rhythm.[57] A study of 136 Chinese inpatients with COVID-19 complicated by cardiac arrest during hospitalization found most arrests were respiratory in origin with non-shockable rhythms (90% asystole and 4% PEA); only 3% survived to 30 days, with just 1% having intact neurologic function.[28]

Bradycardia and AV Block

Bradycardias and AV Blocks (AVB) are less commonly encountered as compared to tachydysrhythmias, though they may account for up to 11.8% of cardiac dysrhythmias.[29] A case report details a patient with COVID-19 presenting with first-degree atrioventricular block (AVB); during hospitalization, the rhythm transitioned to Mobitz type 1 second-degree AVB with further evolution ultimately to third- degree AVB.[29] Other cases describe older patients with multiple cardiac risk factors experiencing progression to high-grade AVB (second-

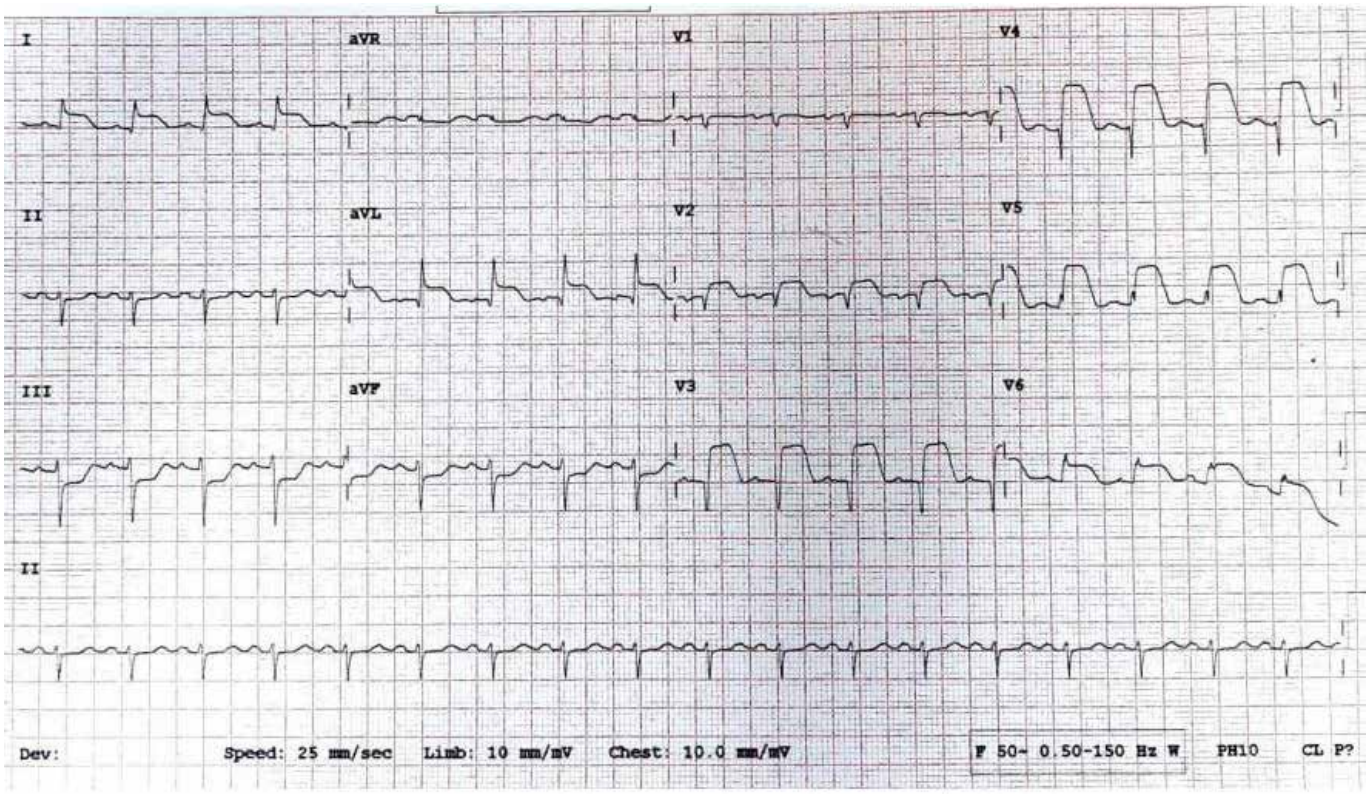

Figure 3: Anterior wall myocardial infarction pattern in a patient with COVID 19 infection. The coronary angiogram done revealed normal coronaries

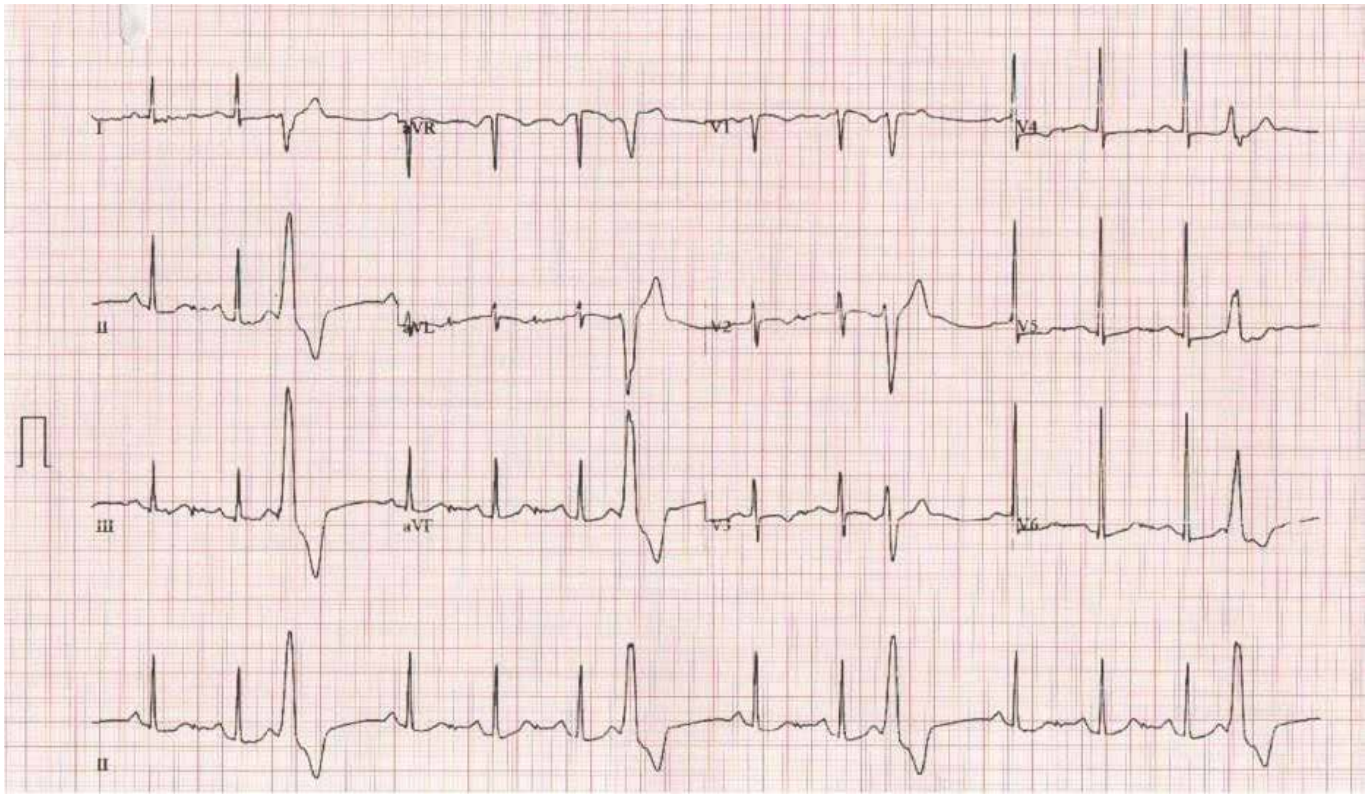

Figure 4: Increased VPC from right ventricular outflow during perioperative COVID infection.

degree type II and third-degree AVB) and/or intra-ventricular conduction block; many of these patients developing these more concerning conduction abnormalities progressed rapidly to cardiac arrest.[26] Three of these dysrhythmias (sinus

bradycardia, junctional rhythm, idioventricular rhythm) occurred immediately prior to cardiac arrest onset – thus, in these cases, 184 the development of significant bradycardia in the critically ill COVID-19 patient is a major marker of risk of impending cardiovascular collapse.[30]

ECG manifestations secondary to systemic effects of COVID 19

COVID-19 infections are associated with higher risk of myocardial infarction due to the endothelial injury, hypercoagulable state and increased inflammatory markers. Patients can manifest with STEMI ECG with apparently normal coronaries.[31] Right ventricular strain secondary to pulmonary embolism is another common manifestation in patients with COVID 19 infection.[32] Fever associated with COVID 19 infection can bring out Brugada pattern of ECG in patients with Brugada Syndrome.[33]

Conclusion

COVID-19 infection though predominantly is a respiratory tract infection, has more multisystem manifestations secondary to the acute inflammatory state. The cardiac manifestations producesignificant ECG abnormalities that are often associated with increased mortality and hence has to be carefully evaluated and managed.

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Early Repolarization Syndrome: A Contemporary Review

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Introduction

Early repolarization syndrome (ERS) is defined as sudden cardiac death (SCD) or documented Ventricular Tachycardia (VT) or Ventricular Fibrillation (VF) in a person with an early repolarization pattern in standard 12 lead ECG. Early repolarization (ER) pattern is an electrocardiographic entity characterized by J-point elevation manifested as either QRS slurring at the transition from the QRS segment to the ST-segment or a positive deflection on terminal S wave, ST segment elevation with prominent T-waves in at least two contiguous leads in standard 12 lead ECG. Grant et al described ST segment deviations and T wave inversion as an early repolarization pattern (ERP). ER was indisputably regarded as a normal variant or a benign early repolarization in healthy population until 2000. Thereafter, numerous reports have suggested a relationship between ER and an increased risk of SCD from life-threatening cardiac arrhythmias especially idiopathic ventricular tachycardia or ventricular fibrillation.[1,2] Early repolarization detected in inferior ECG leads was specially found to be associated with idiopathic VT/VF.

The J-wave deflection at QRS–ST junction also known as the Osborn wave which was first described in 1953 and is seen in many clinical conditions such as acute myocardial infarction, early repolarization syndrome, hypothermia, hypercalcemia and brain injury. ERS is commonly encounter in athletes, cocaine users, hypertrophic obstructive cardiomyopathy, those predisposed to vagotonia, male gender and African Americans. Prevalence of ERS varies from 3-24% in the general population depending on age (predominant in young adults), race (highest among black population), sex (predominant in males) and the criteria used to measure J-point elevation (0.05 vs 0.1 mV). Tikkanen et al demonstrated that the location (inferior vs lateral leads) and J-point elevation of > 0.2 mV are linked to a high risk of sudden cardiac death from life threatening cardiac arrhythmias.[2] The overall prevalence of ERS is approximately 31% in a structurally normal heart and the prevalence of the ERS having J-wave elevation ≥0.2 mV was found to be 16%.[3,4] Majority of asymptomatic young adults with early repolarization pattern regressed by middle age but in black race, lower body mass index, lower serum triglyceride levels and longer QRS duration, it persists over time.[5]

Various ion channel mutations have been associated with ERS. Mutation of SCN10A gene produce patterns of Brugada and early repolarization. Other associated gene mutation involve KCNJ8, CACNA1C, CACNB2, CACNA2D1 and the SCN5A gene which enhance the underlying inward – outward current imbalance responsible for accelerated epicardial repolarization.[6,7] The most frequent association between KCNJ8 gene and ERS has been reported. An increase in net repolarizing current due to either a decrease of inward Na+ or Ca2+ currents (INa, and ICa,L) or augmentation of outward currents (Ito, IK–ATP and IK–Ach) lead to augmentation of the J wave or appearance of ST-segment elevation which becomes more prominent during slow heart rate.

Antzelevitch et al described three subtypes of ER and a pattern of risk profile:[8]

Type 1: ER pattern in lateral precordial leads, seen in healthy male athletes and has the lowest risk of life threatening arrhythmias.

Type 2: ER pattern in the inferior and infero-lateral leads and is associated with a greater risk of life threatening arrhythmias.

Type 3: Global ER pattern (inferior, lateral and right precordial leads) and has the highest risk of life threatening arrhythmias and electrical storms.

Historical Perspective

The J point presenting as QRS slurring or notching was first described in 1936 by Shilpey et al and has been considered as a normal ECG variant. In 1953, Osborn demonstrated a current of injury, later named as Osborn wave in acidotic and hypothermic dogs at rectal temperatures < 25 °C. In 1961, Wasserburger et al defined ER as 1-4mm ST-segment elevation at the end of the QRS complex with a distinct notch or slur on the downslope of the R wave in the mid to left precordial leads.

In recent years, ER has been suggested to be a non-benign condition and was described as an abnormal finding in patients diagnosed with idiopathic VT/VF. In 1999, Gussak et al suggested that ER may be malignant in some cases with polymorphic ventricular tachycardia. In 2000, Kalla et al and Takagi et al reported VF in patients with prominent J wave and ST segment elevation in inferior leads with structurally normal heart. In 2008, Haïssaguerre et al and Nam et al showed a strong relationship between J point and many different forms of ventricular arrhythmias in the absence of structural heart disease.

Clinical Manifestation

The clinical presentation of ERS can be divided into two main groups-

The first group includes patients having recognized symptoms as syncope or cardiac arrest survivors. Abe et al demonstrated that the ER was noticed in 18.5% in patients with syncope compared to 2% in healthy individuals in control group. Haissaguerre et al demonstrated 41% recurrence of cardiac arrhythmia when he followed up for 51 months.[1] Life-threatening arrhythmias are often the first clinical manifestation of ERS.

The second group includes asymptomatic patients who are incidentally noted to have an ER pattern on their ECG. In general, this group is less likely to have adverse cardiac events although difficult to distinguish between those who are at high risk of developing SCD and those who are likely to have a benign course.

Diagnosis

The universally accepted criteria for the diagnosis of early repolarization pattern involves the presence of an elevated junction between the end of the QRS complex and the beginning of the ST segment called J-point. This condition is

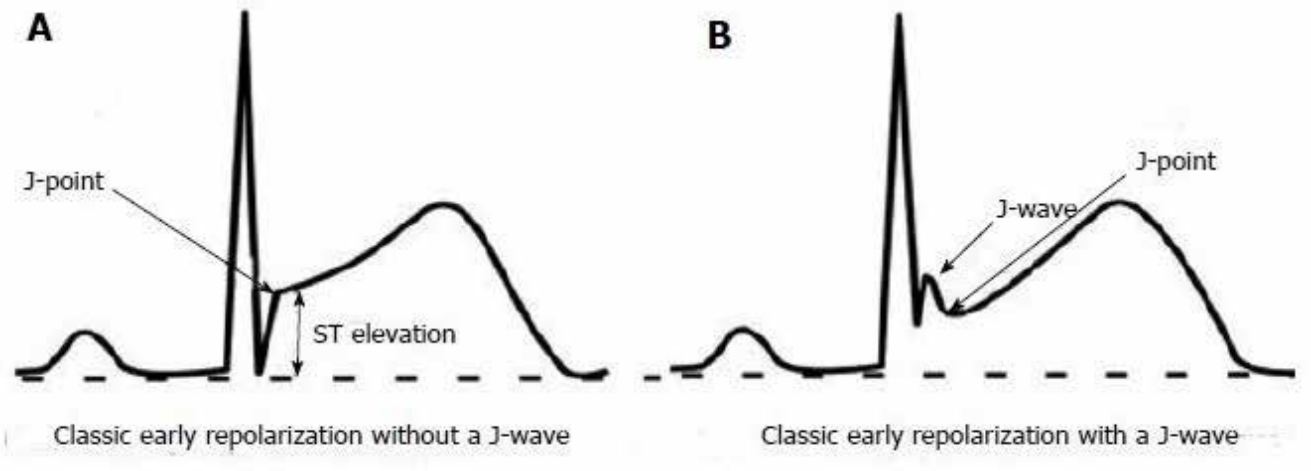

Figure 1: Classic definition of early repolarization: ST Elevation

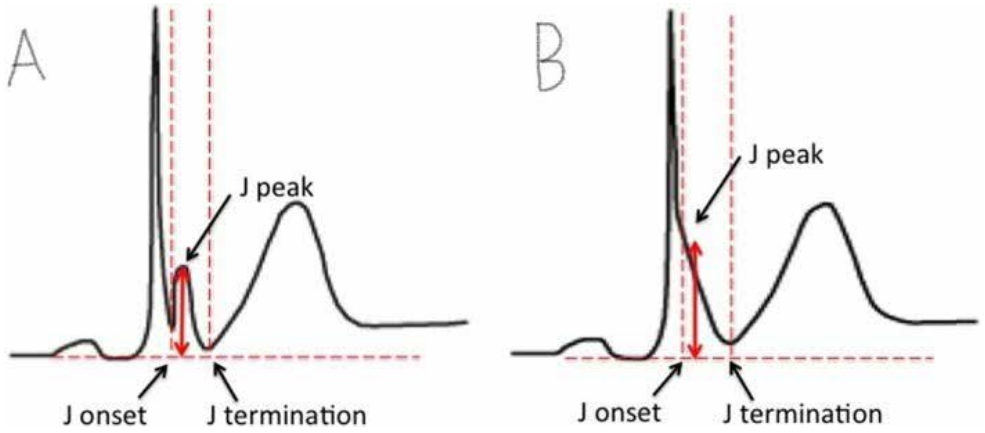

Figure 2: New definition of early repolarization

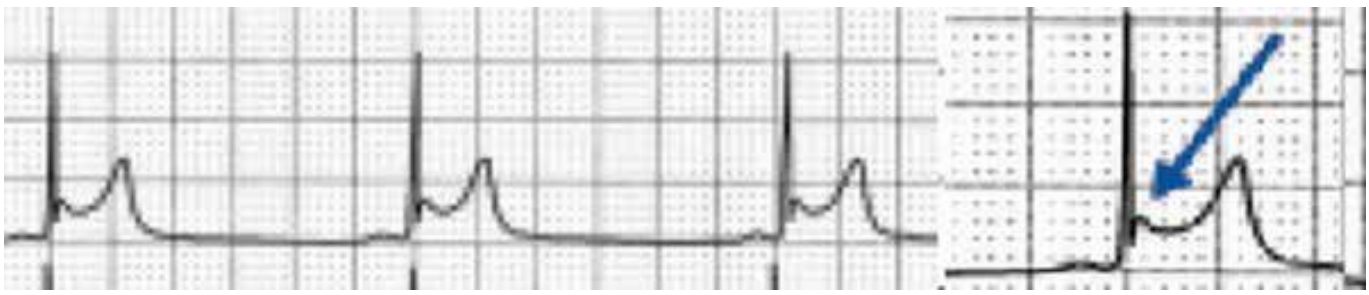

Figure 3: Electrocardiographic pattern of J-point elevation in early repolarization.

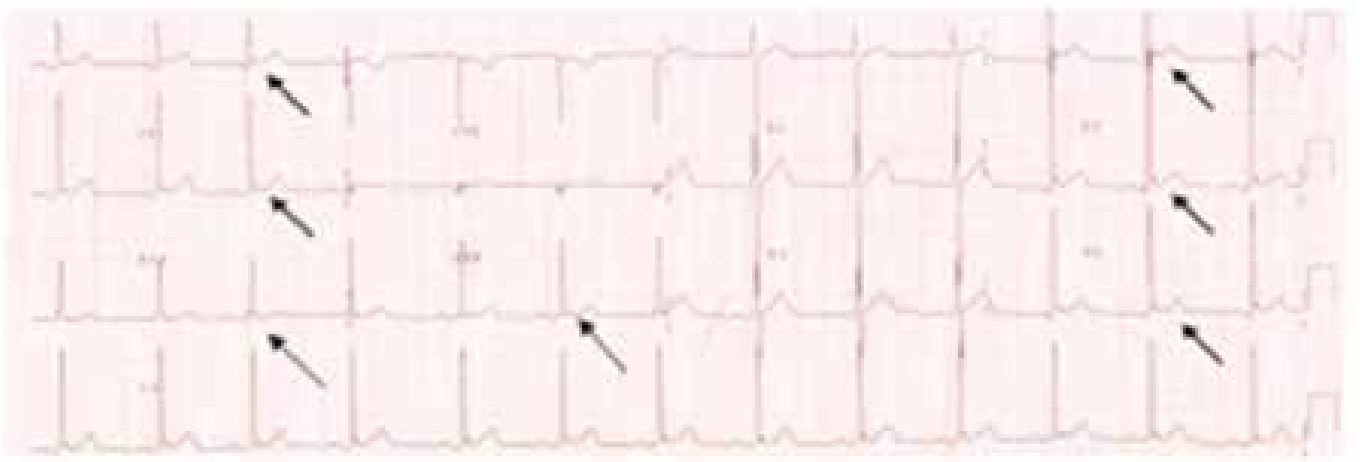

Figure 4: Standard 12 lead ECG showing early repolarization pattern.

characterized by an elevation of ST segment (≥1 mm) from baseline in at least two adjoining leads on standard 12-lead ECG. (Figures 1,2,3 and 4).

ER expert consensus (HRS/EHRA/APHRS) recommendations on early repolarization diagnosis are[9]

1. ERS is diagnosed in the presence of J point elevation ≥ 1 mm in ≥ 2 contiguous inferior and/or lateral leads in standard 12-lead ECG in a patient resuscitated from otherwise unexplained VF/polymorphic VT.
2. ERS can be diagnosed in an SCD victim with a negative autopsy and medical chart review with a previous ECG demonstrating J point elevation ≥ 1 mm in ≥ 2 contiguous inferior and/or lateral leads in standard 12-lead ECG.
3. ER pattern can be diagnosed in the presence of J point elevation ≥ 1 mm in ≥ 2 contiguous inferior and/or lateral leads in standard 12-lead ECG.

J point elevation without STE Slurred QRS downstroke without STE

In the setting of SCD survivor, a familial screening is recommended but in isolated, asymptomatic early repolarization pattern, there is no recommendation for familial screening.

Differential Diagnosis

There are numbers of causes of J point elevation. Some of the conditions with J-point elevation on the electrocardiogram are-

- Early repolarization syndrome
- Brugada syndrome
- ST segment elevation MI
- Acute pericarditis
- Hypothermia
- Hypercalcemia
- Hyperkalemia
- Short QT syndrome
- Pulmonary embolism
- Arrhythmogenic right ventricular cardiomyopathy
- Subarachnoid hemorrhage
- Idiopathic VF

ER Benign or Malignant

The identification of high-risk patient with ER remains challenging. A horizontal or descending ST-segment elevation has been linked to adverse outcome compared with a rapidly ascending ST-segment elevation following J-point elevation. A slurred or notched J-point elevation ≥ 2 mm is also associated with a higher risk of sudden cardiac death from cardiac arrhythmia and so called malignant form of ER. Other abnormalities such as localization of the ER pattern in inferior or inferolateral leads compared to lateral leads or extension of ER into a Brugada syndrome may also represent a worse prognosis. The benign type of ER is commonly associated with young age group, left ventricular hypertrophy on ECG, lower blood pressure and lower heart rate which are the features of younger, healthy and physically active individual. Large debates about malignant form and its value for risk stratification in early repolarization are ongoing.

Treatment

Early repolarization pattern (ERP) is relatively frequent ECG finding in the general population but the incidence of idiopathic ventricular fibrillation or polymorphic ventricular tachycardia is relatively low. The majority of individuals presenting as an ERP will remain asymptomatic and does not require further intervention however implantable cardioverter defibrillator (ICD) is indicated in those who survived SCD from idiopathic VT or VF. The reported rate of recurrent VT/VF among SCD

Table 1: Expert consensus 2013 HRS/EHRA/APHRS recommendations on early repolarization therapeutic interventions[9]

Features	Recommendation
Class I	1. ICD implantation is recommended in patients with a diagnosis of ERS who have survived a cardiac arrest.
Class IIa	1. Isoproterenol infusion can be useful in suppressing electrical storms in patients with a diagnosis of ERS. 2. Quinidine in addition to an ICD can be useful for secondary prevention of VF in patients with a diagnosis of ER syndrome.
Class IIb	1. ICD implantation may be considered in symptomatic family members of ERS patients with a history of syncope in the presence of ST-segment elevation > 1 mm in 2 or more inferior or lateral leads. 2. ICD implantation may be considered in asymptomatic individuals who demonstrate a high-risk ER pattern in the presence of a strong family history of juvenile unexplained sudden death with or without a pathogenic mutation.
Class III	1. ICD implantation is not recommended in asymptomatic patients with an isolated ER pattern.

survivor from idiopathic VF ranges between 22% to 37% at two to four years,[10] but have an excellent prognosis for long-term survival if VT/VF is treated with an ICD.[11] There is no current risk stratification strategy for asymptomatic patients with ER pattern in general population and within families with ER pattern that would allow for identification of higher risk individual and a candidate for treatment.

Implantable Cardioverter Defibrillator (ICD) Therapy

In patients with an early repolarization pattern who survived SCD from idiopathic VT/VF, the implantation of an ICD is indicated. The current guideline suggest that ICD implantation may be considered in high-risk individuals with unexplained syncope.[12] (Table 1).

Drug Therapy

The incidence of VF episodes and electrical storm is relatively common in ERS person even after ICD implantation. In such situation, there is an evidence that isoproterenol infusion acutely suppresses recurrent VF. The dose can be initiated with 1.0 μg/min and should target an increase in 20% of baseline heart rate (HR) or an absolute HR > 90 bpm adapted to hemodynamic conditions and suppression of arrhythmia. In long term therapy, quinidine has demonstrated effective suppression of VF recurrence.[13]

Discussion

Understanding of the ERS has evolved rapidly in recent years but significant questions and controversies still remain. ERS has emerged as a marker of risk for idiopathic VT/VF and SCD. However, the incidental finding of a J-point on routine screening should not be interpreted as a marker of high risk for SCD but close follow-up should be offered to patient who has ER pattern along with a personal history of unexplained syncope or a family history of unexplained sudden death. In the recent years, ERS has been associated with a significant risk of developing life threatening cardiac arrhythmias and sudden cardiac death but it is not possible to identify asymptomatic ER individuals to develop life threatening cardiac arrhythmias. Unless we have a better understanding, we are left with observing the patients with inferolateral ER to have life-threatening ventricular arrhythmias or SCD. Since there are a large number of patients who fit such criteria but do not appear to have life threatening cardiac arrhythmias therefore further data is needed to reveal how to identify the group of people who would be at a significant risk and what measures can be taken to prevent it. More research is needed for better understanding of the electrophysiological basis and clinical significance, prognosis prevention and better way of treating the persons with ERS.

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ECG in Hypertension: Is It Still There?

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Introduction

In India, one in 5 has hypertension and 50% of the population is in high normal range. Mortality and morbidity are quite high in hypertension if not detected early and treated properly. The complication in hypertension depends upon the presence of Target Organ Disease (TOD), Secondary causes, co morbidities and complications due to hypertension. Most often we rely upon Echocardiography to diagnose these issues. The Electrocardiogram (ECG) when interpreted skilfully gives valuable information regarding these problems, even when they are not detected by Echocardiography. ECG is underutilised in hypertension. The purpose of the article is to explore, the utilities of ECG in hypertension in diagnosis and treatment. To our knowledge, the management of hypertension has not been looked from this angle so far. ECG aids in ECG (**E**xcellent **C**are **G**iving) in patients with hypertension.

ECG in hypertension is better studied in following headings: **I Target Organ Disease** II **Secondary causes III Co morbidities** IV **Complications**

I. ECG-TARGET ORGAN DISEASE (TOD):

a. LVH: One of the important TODs is LVH. The presence of LVH for the same level of Blood pressure (BP) enhances the risk of Coronary Artery Disease (CAD), Heart failure and Arrhythmias. Although Echo is ideal to detect LVH, the ECG remains a cost-effective method of detecting LVH.

ECG Criteria to detect LVH: *Limb lead:* R in lead avL more than 11 *Chest leads:* R in V5, V6 +S in V1 > 35mm R in V5, V6 more than 26mm

In children and patients with COPD limb leads criteria are preferable as chest lead criteria may not be reliable. The ECG has high specificity and low sensitivity.

b. CAD

Although LVH can produce secondary ST T changes in hypertension, one should suspect associated CAD if the T wave inversion is symmetrical with well-formed ST segment (Fig 2)

c. LV Dysfunction (LVD): Although Echo is a gold standard in diagnosing LVD, ECG gives clue regarding LV Dysfunction (systolic and diastolic) (Fig.3)

II. ECG AND SECONDARY CAUSES:

a. Chronic Kidney Disease (CKD): One of the most important secondary causes of hypertension is CKD. CKD can be suspected when ECG shows signs of hyperkalaemia. (Fig.4)

b. Hyperaldosteronism: ECG changes of Hypokalaemia with LVH is a valuable clue regarding hyperaldosteronism (Fig 5)

a. Cushing and hypothyroidism: Both of these conditions which are known to have hypertension produce low voltage QRS in limb and chest leads. One usually expects high voltage QRS in hypertension. Low voltage QRS is defined as QRS voltage less than 5mm in limb leads and 10mm in chest leads (Fig 6)

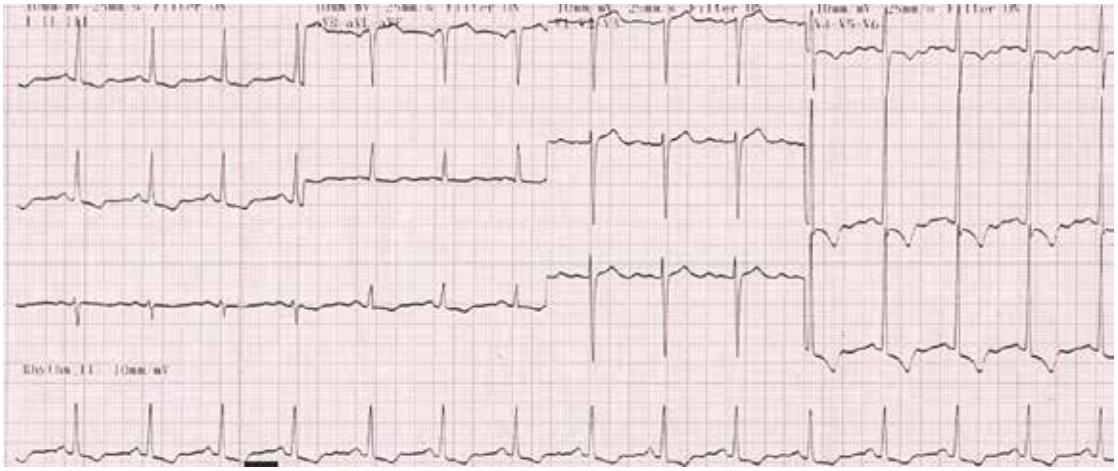


Figure 1: ECG showing LVH- QRS in V5 or V6 added to QRS of V1 is >35 (here more than 50).

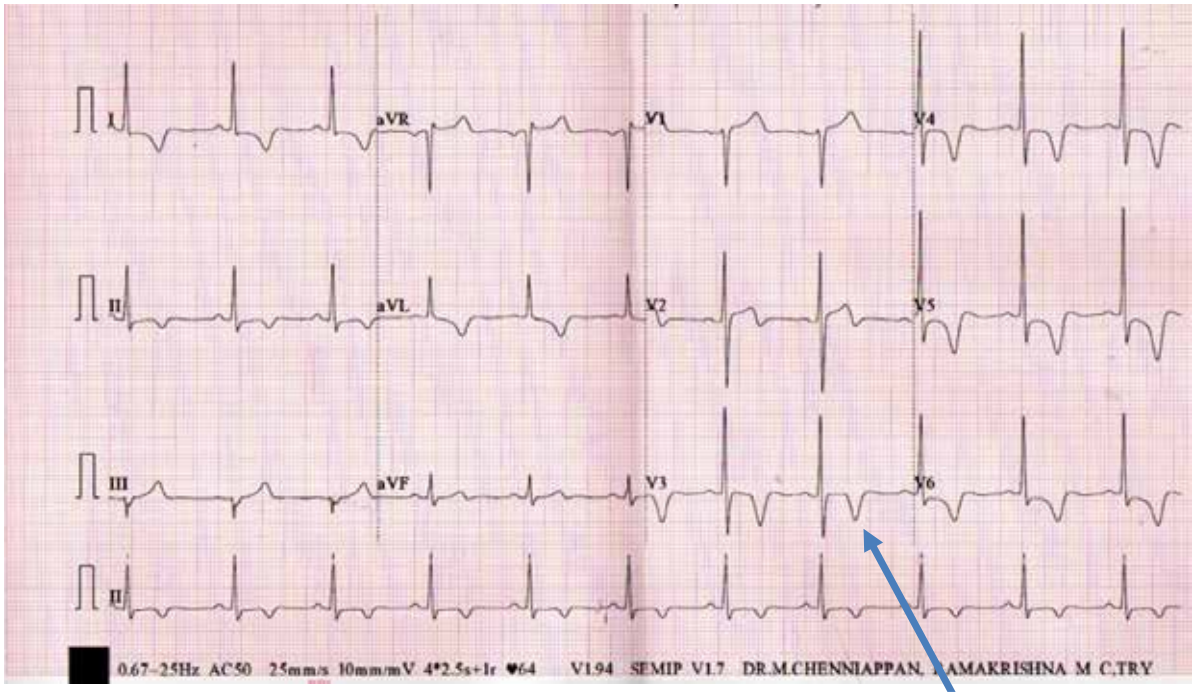


Figure 2: ECG showing LVH and primary ST T changes (Well-formed ST segment, symmetrical T inversion)

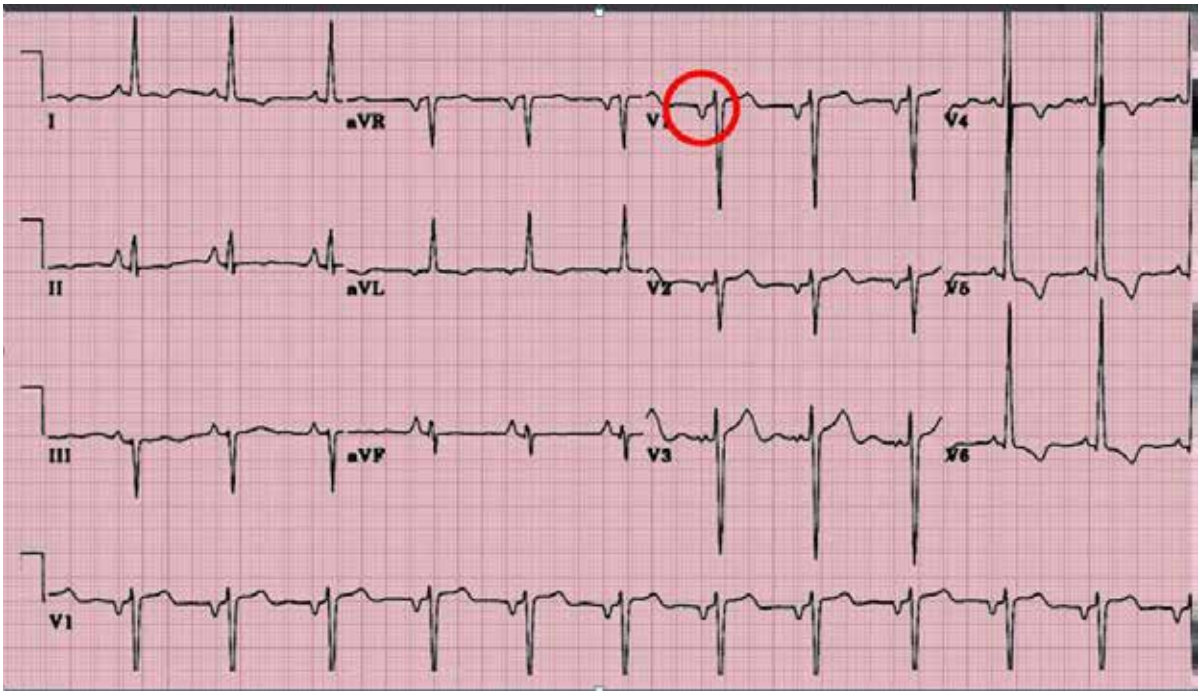


Figure 3: ECG Showing LVH and deep and wide negative component of P in V1 suggestive of LV Dysfunction

a. Pheochromocytoma: When high BP is associated with unprovoked sinus tachycardia, one should suspect pheochromocytoma (Fig 7)

III. ECG in comorbidities:

a. Pulmonary hypertension (PHT): Usually LVH is expected in systemic hypertension. If there is associated Right Ventricular Hypertrophy (RVH) in ECG, one should suspect associated PHT. (Fig 8)

Whenever RVH is present with LVH in hypertension, following conditions should be suspected: Pulmonary hypertension (PHT), LV Dysfunction and PHT, COPD and cor pulmonale and Sleep apnoea (PHT)

b. CAD: Acute Coronary Syndrome (ACS) When a patient with hypertension comes with ACS, ECG plays

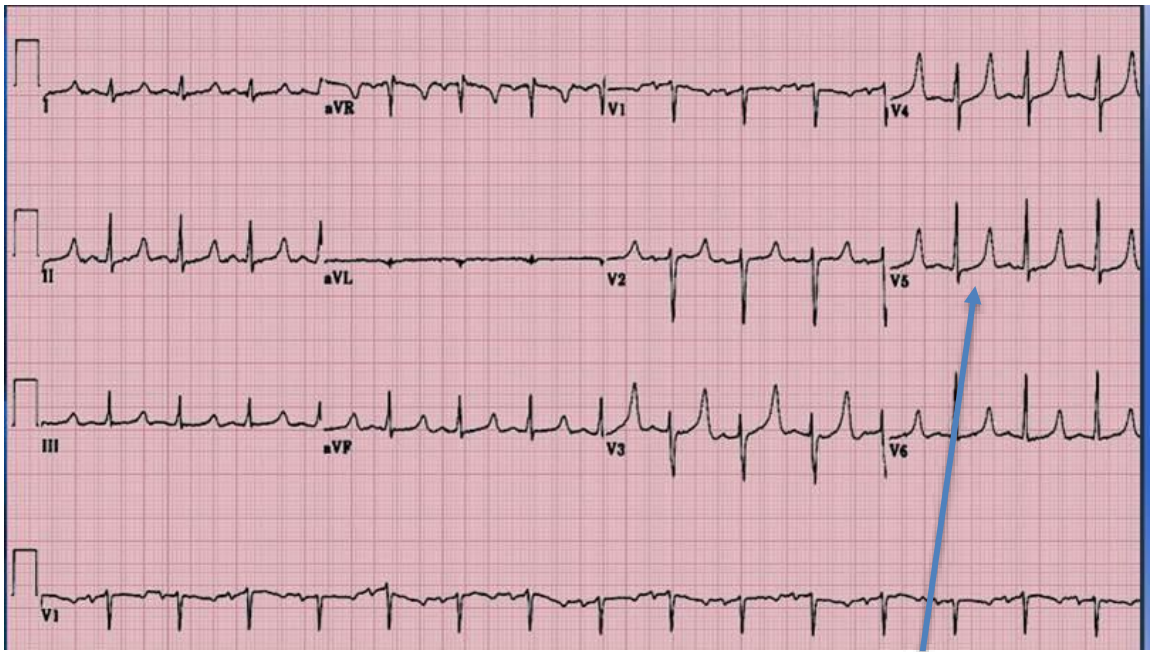


Figure 4: ECG showing features of hyperkalaemia (Tall T with sharp apex and narrow base) Serial ECGs in patient with CKD on angiotensin inhibitors will help to identify early hyperkalaemia.

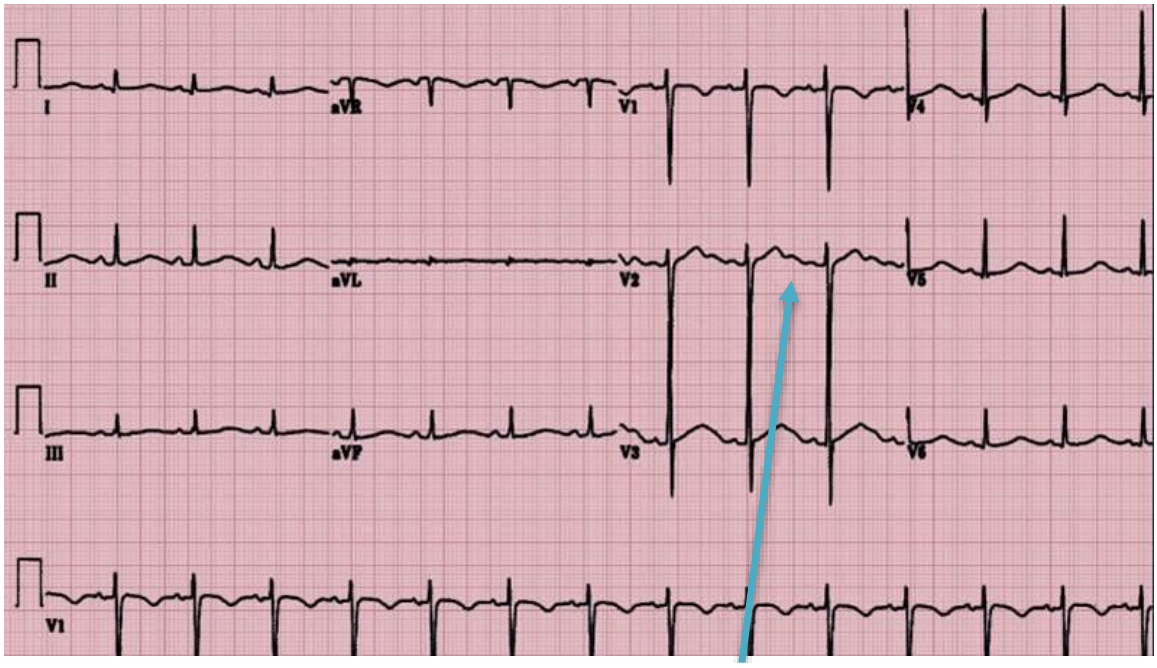


Figure 5: ECG shows Hypokalaemia (low voltage T wave with prominent U wave)

a crucial role in deciding the mode of management. If the ECG shows ST elevation, acute total occlusion with red thrombus (rich in fibrin) is the cause (fig.9)

If the ECG shows ST depression, it means that it is a critical occlusion with white thrombus (rich in platelets) and so, the treatment of choice is heparin and oral antiplatelets. (Fig.10)

c. Hypertension, CVA

When the patient has CVA, the ECG may show deep broad T inversion with prolonged QT. This has to be differentiated from CAD which shows deep symmetrical T inversion with normal or shortened QT (Fig 11)

The T wave in Hypertension may give a lot of information about etiology, organ damage, complications as well as co morbid conditions.(Fig. 12)

VI. Complications (Due to disease or drugs)

a. Malignant VPDs: show features such as Couplets, Runs, Multiform, R on T. With signs of LVD, Short,

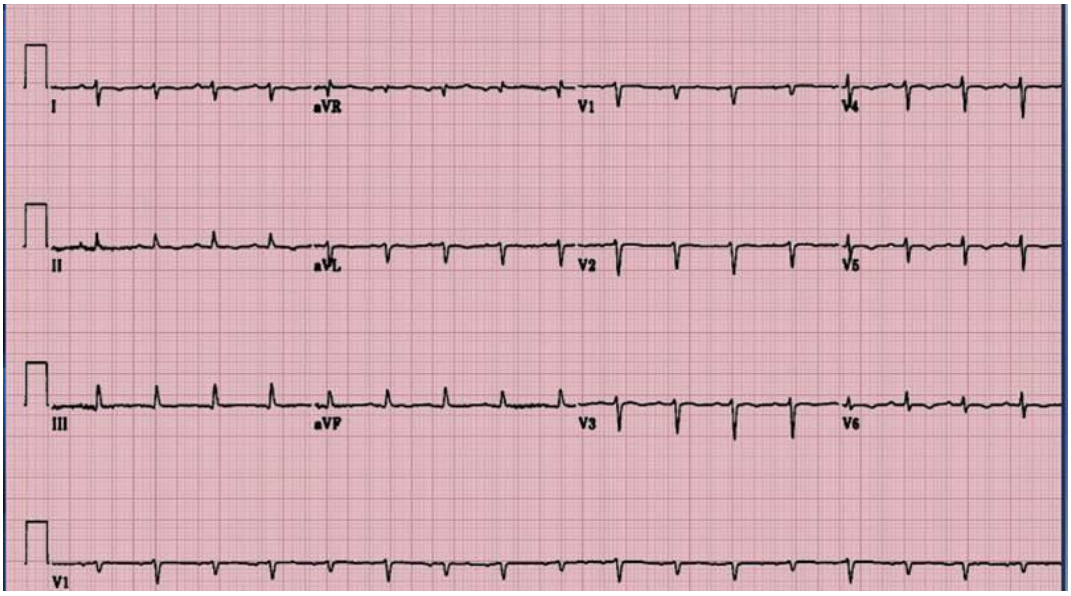


Figure 6: ECG showing low voltage QRS complexes in limb and chest leads

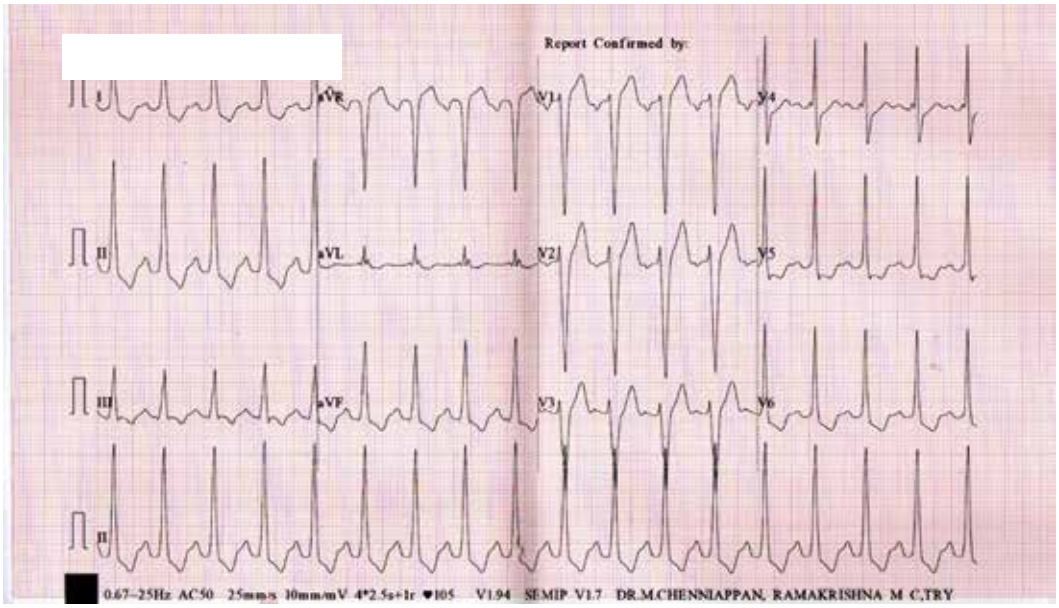


Figure 7: ECG Showing LVH and sinus tachycardia

broad with notches. During ACS (Fig 13)

b. Hypertension with Atrial Fibrillation (AF):

One of the commonest causes of AF is hypertension. It worsens systolic and diastolic function and precipitates heart failure. It also complicates CAD. As the patient is prone for embolism, the anticoagulants and their problems are added. (Fig 14)

c. Hypertension and bradycardia:

The bradycardia in hypertension may be due to sinus node or AV node (FIG 15, 16, 17)

d. Hypertension, LBBB:

LVH is difficult to diagnose in the presence of LBBB. High voltage LBBB may be a clue for underlying LVH. The presence of LBBB with hypertension has poor clinical outcome (CAD, heart failure). In elderly presence of LBBB indicates, advanced and advancing disease (Fig.18)

e. Hypertension, Bifascicular block

Bifascicular block indicates, complete block of right bundle and one of the fascicles on left side. Only one fascicle is conducting the impulse to the ventricles. (Fig 19)

f. Hypertension and QT interval:

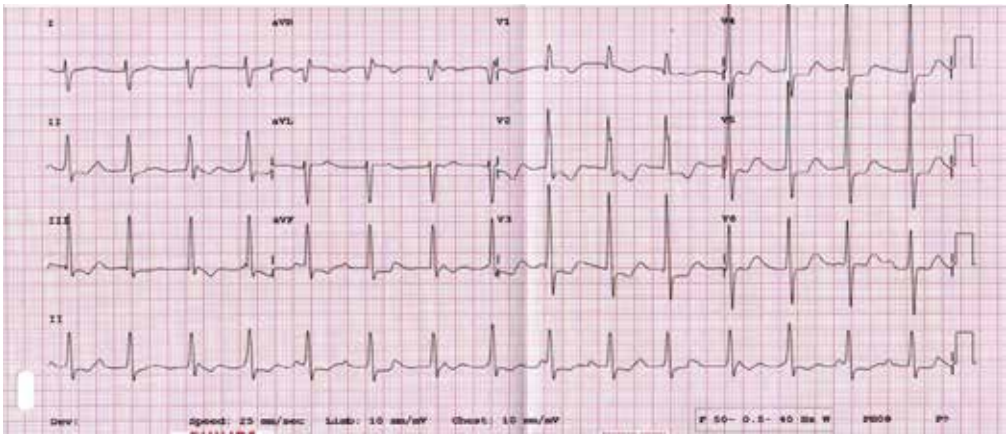


Figure 8: ECG showing LVH and RVH (Tall R in V1 with Right Axis deviation)

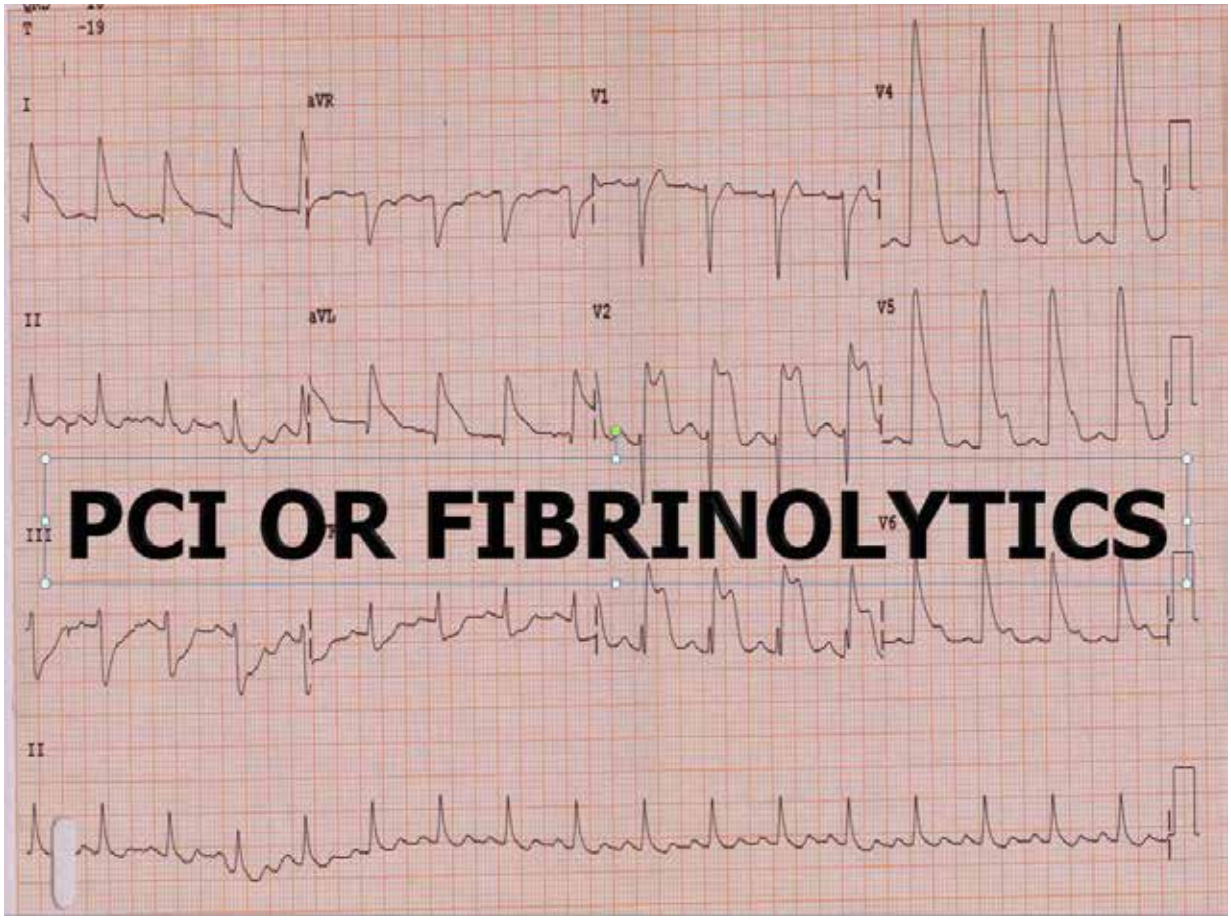


Figure 9: ECG shows ST elevation (acute total occlusion, red thrombus)

Hypertension maybe associated long or short QT intervals which may lead on to dangerous ventricular arrhythmias. Although majority of anti hypertensives don't affect QT directly, one should always look at QT interval when seeing the ECG in hypertension. The long QT may be due to abnormalities in QRS, ST or Wave. (Fig.20,21) The summary of QT interval in hypertension is given in Fig 22 .

Conclusion

As shown above, ECG in hypertension gives valuable clues regarding TOD, Secondary causes, comorbidities and complications. It also aids in proper selection of drugs in various situations. It also provides much vital information, not shown by Echo. Being, a simple and cost-effective investigation, ECG should be done as the first investigation in all hypertensive patients.

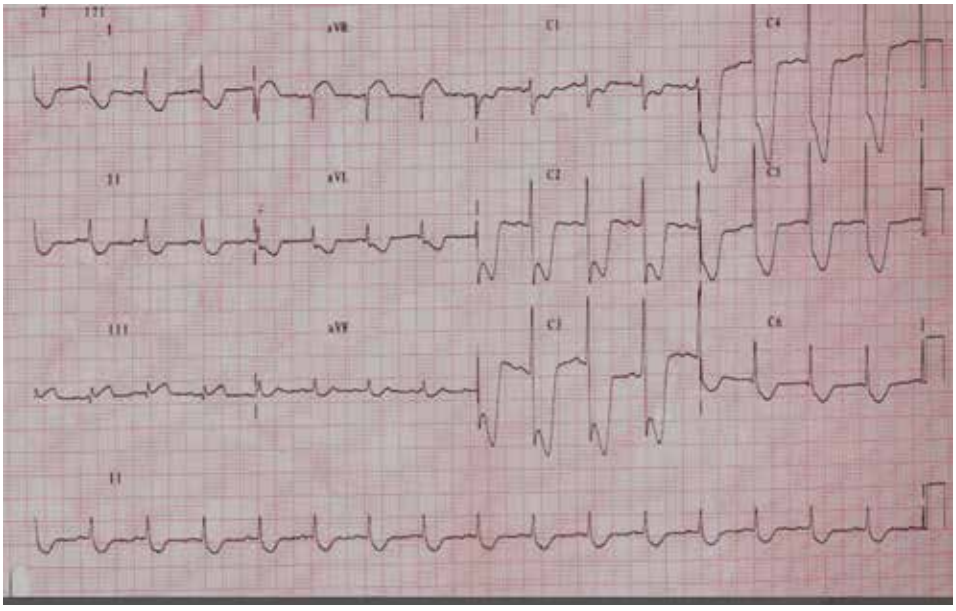


Figure 10: ECG showing ST depression (Critical occlusion, white thrombus)

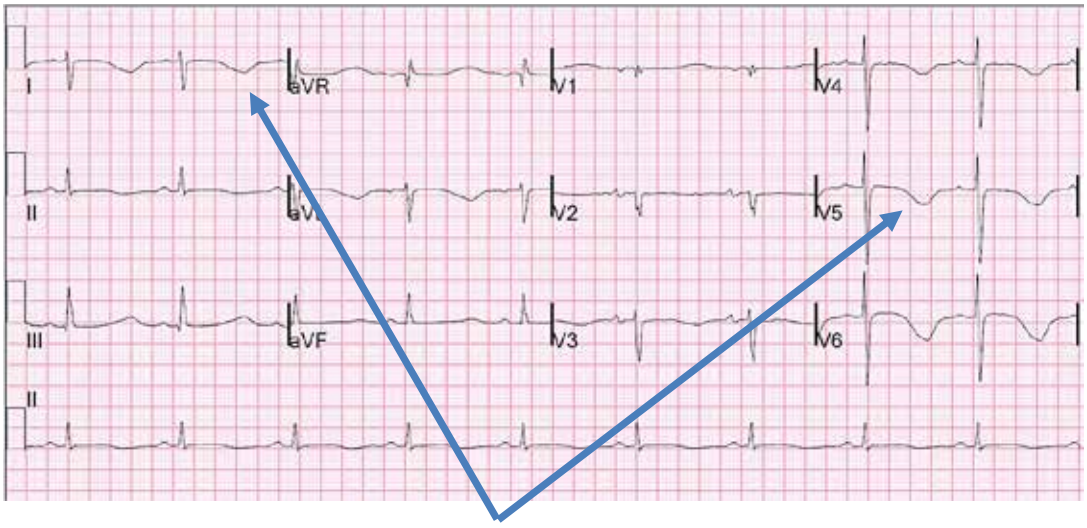


Figure 11: The ECG shows deep broad T inversion with prolonged QT (CVA)

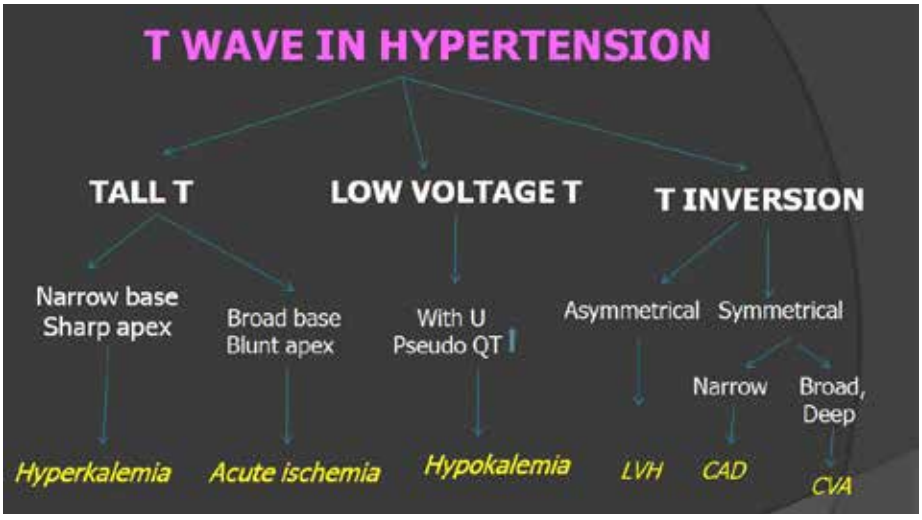


Figure 12: T wave in hypertension

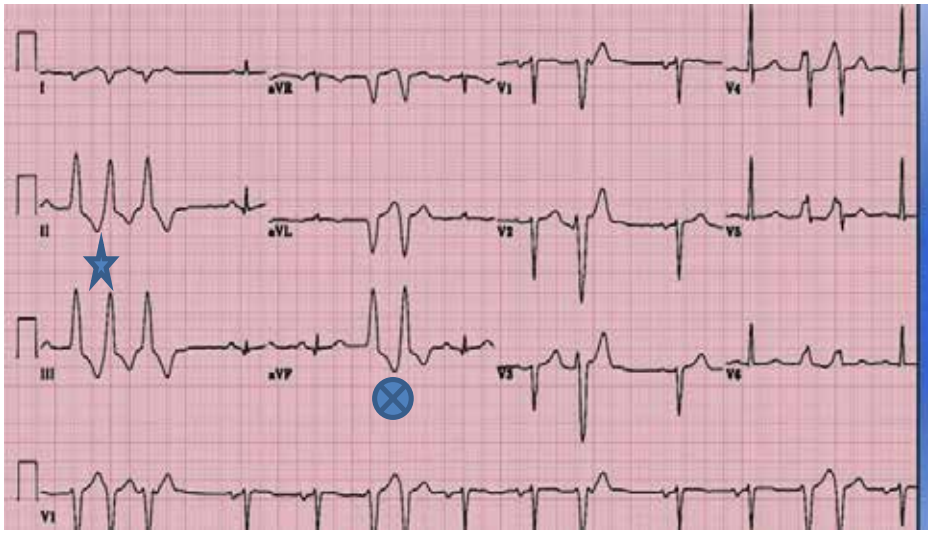


Figure 13: The ECG showing malignant VPDS (Couplets ● + Runs ★)

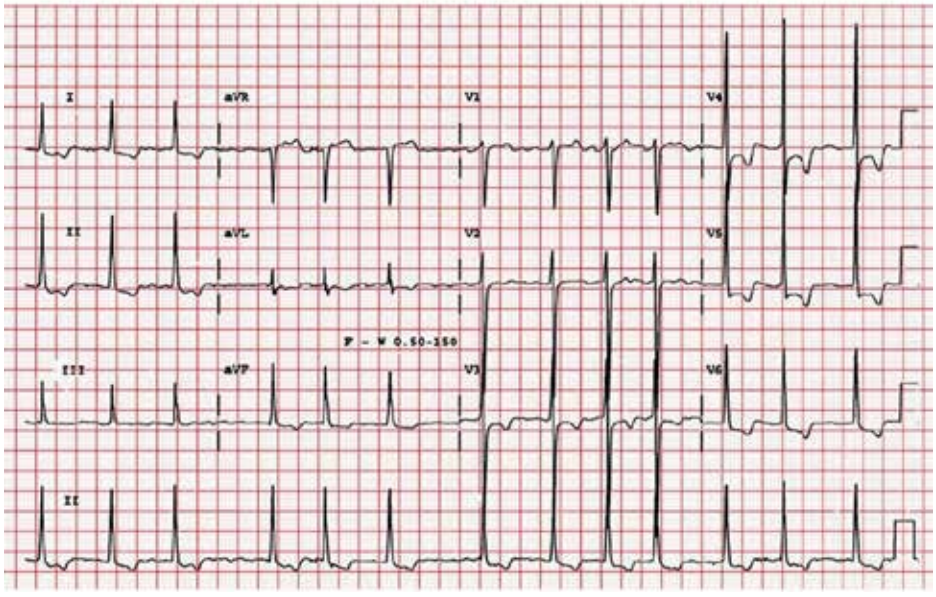


Figure 14: Atrial Fibrillation (absent P, irregular QRS, presence of fibrillary waves)

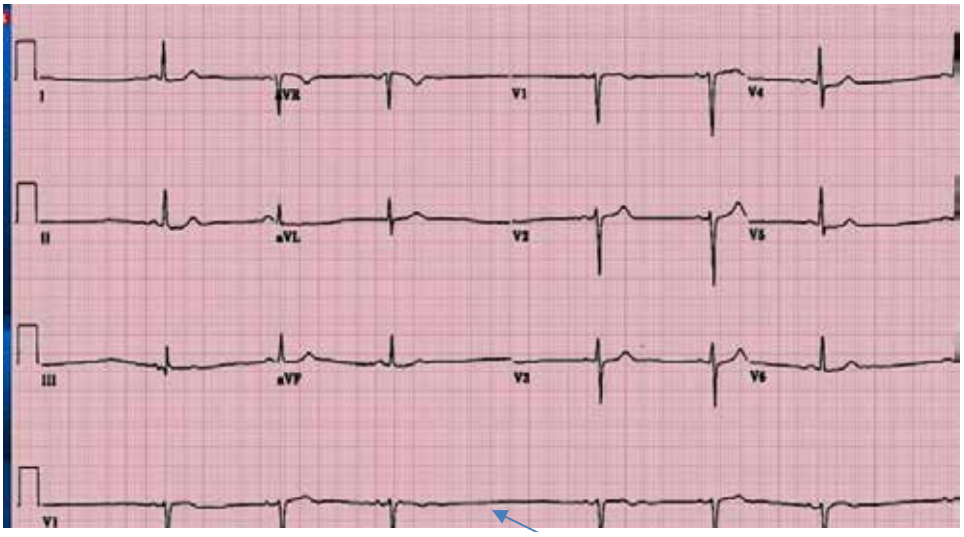
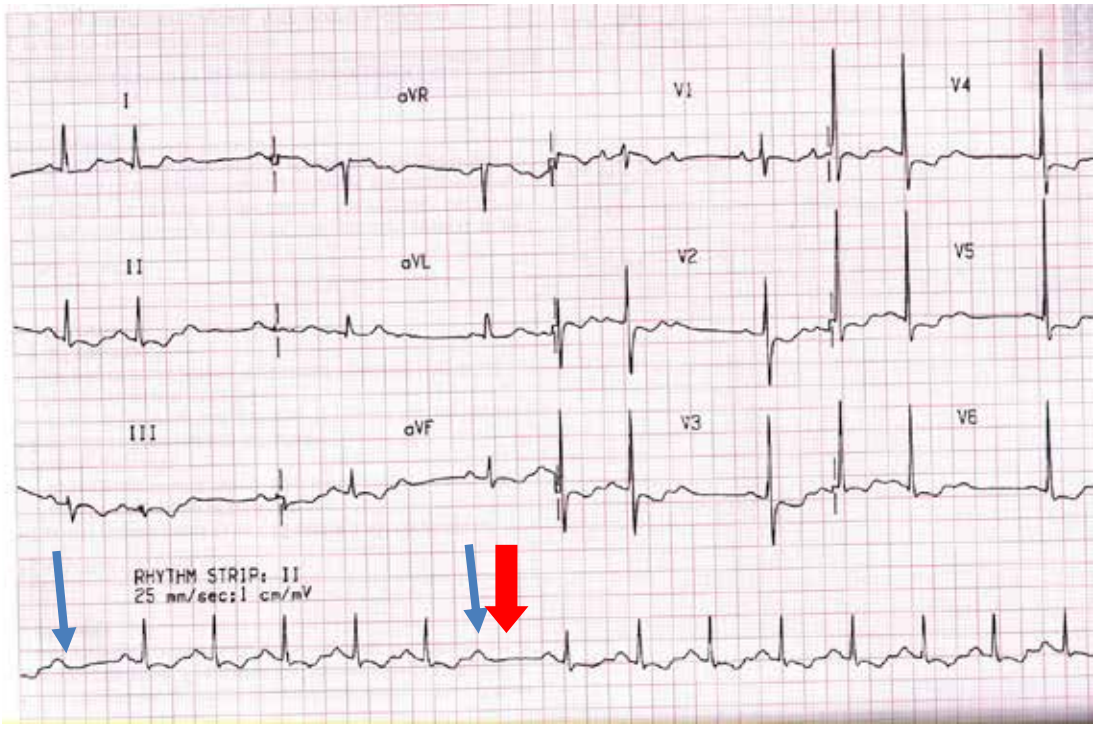


Figure 15: ECG Showing Sinus Bradycardia and Sinus Pauses



Figue 16: Mobitz type II 2nd degree AV Block-P wave ↓ is not followed by a QRS ⬇

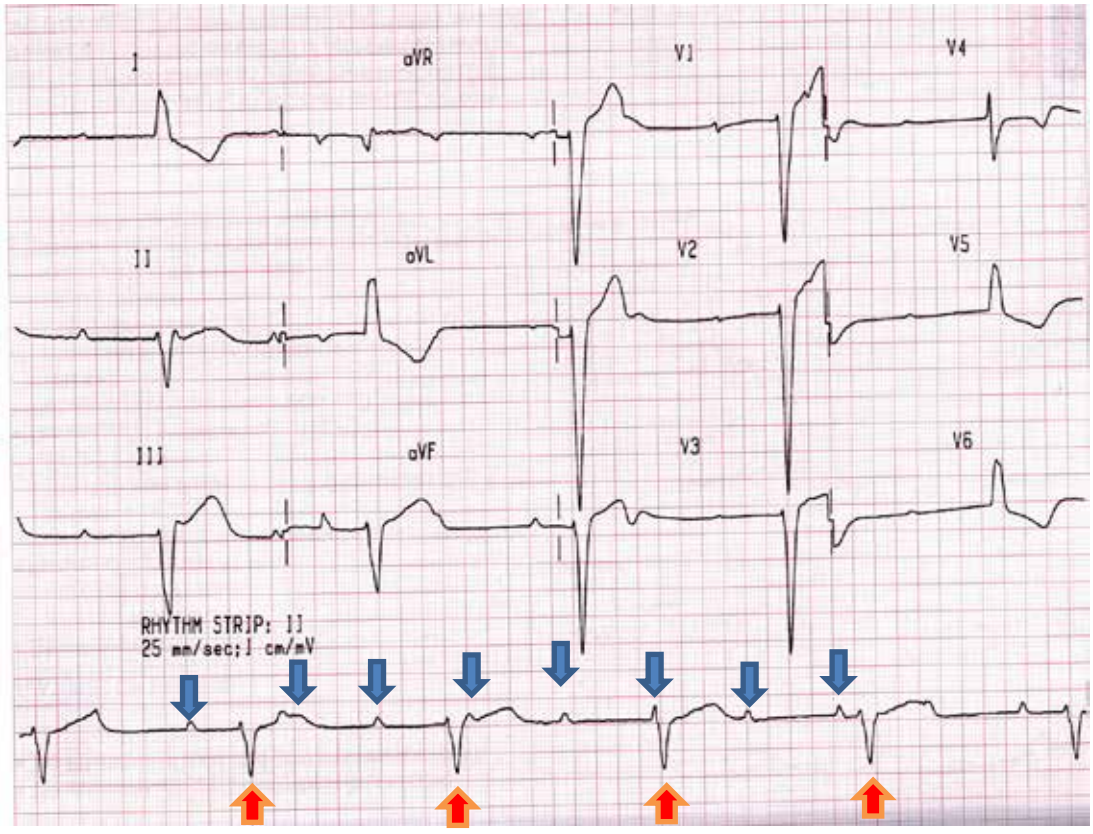


Figure 17: ECG Showing Infra His Complete Heart Block (CHB)- evidenced by wide QRS – QRS > 120 ms, varying P (⬇) wave to QRS (⬆) duration, regular P-P and QRS-QRS intervals as though the P waves were marching through the QRS a sine qua non of CHB

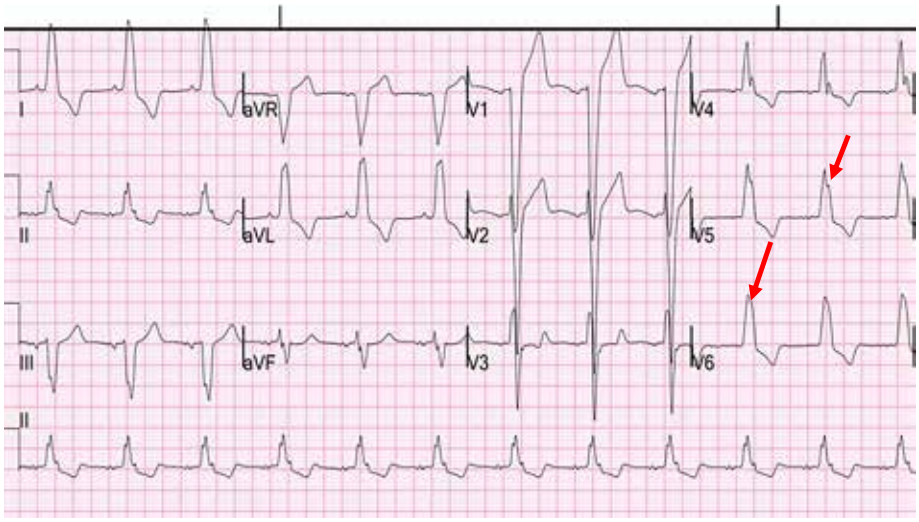


Figure 18: ECG shows High Voltage Left bundle branch block (LBBB). LBBB is characterised by a small "r" (positive) wave in V1 followed by a deep "S" (negative wave) and a wide QRS in V6 with a bifid (two peaks ↖) peak.

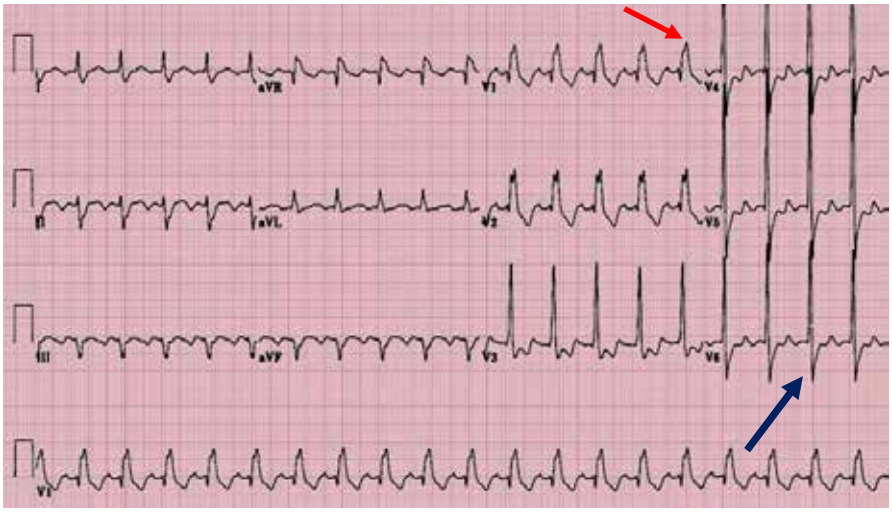


Figure 19: ECG Shows bifascicular blocks (Right bundle branch block -RBBB- characterised by a terminal positive wave in V1- a double peak in this case ↘ and a negative terminal wave in V6 ↗, and Left anterior fascicular block LAFB, characterised by a small q in lead AVL and a left ward axis, which is not found in pure RBBB.

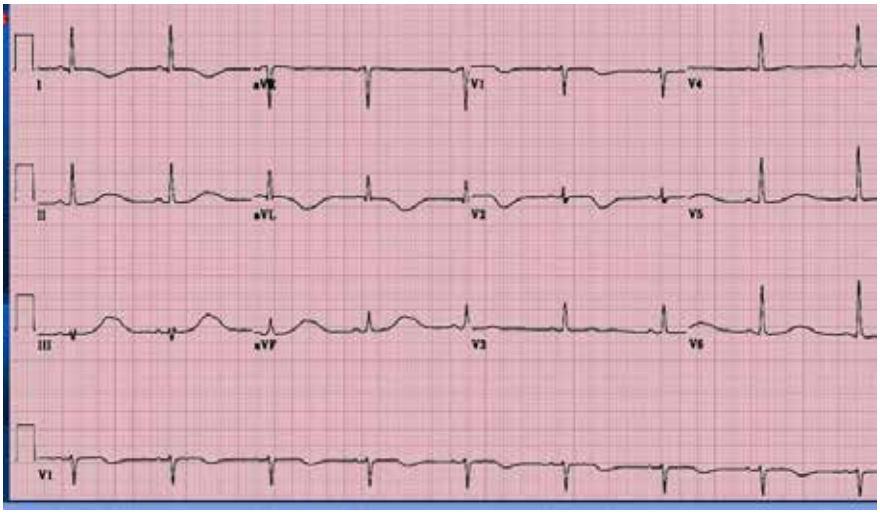


Figure 20: ECG shows prolonged QT(the interval from the beginning of the QRS wave to the end of the T wave which is technically the point where the descending slope of the T wave meets the baseline, a point which may be difficult to discern in some cases) QT is said to be prolonged when the corrected QTc (by Bazzett's formula) is more than 425 msecs.

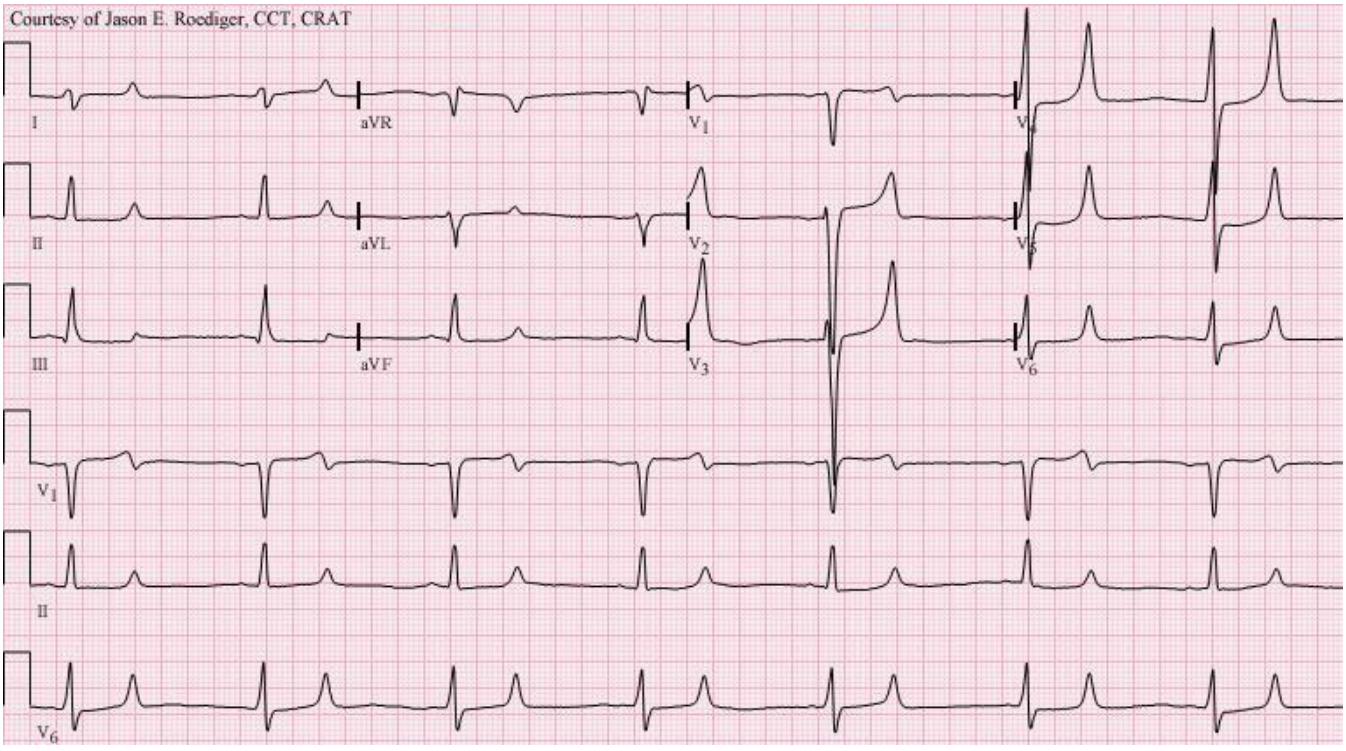


Figure 21: ECG Shows prolonged QT due to ST segment. This ECG suggests CKD due to hypocalcemic and hyperkalemic (sharp apex T wave) changes.

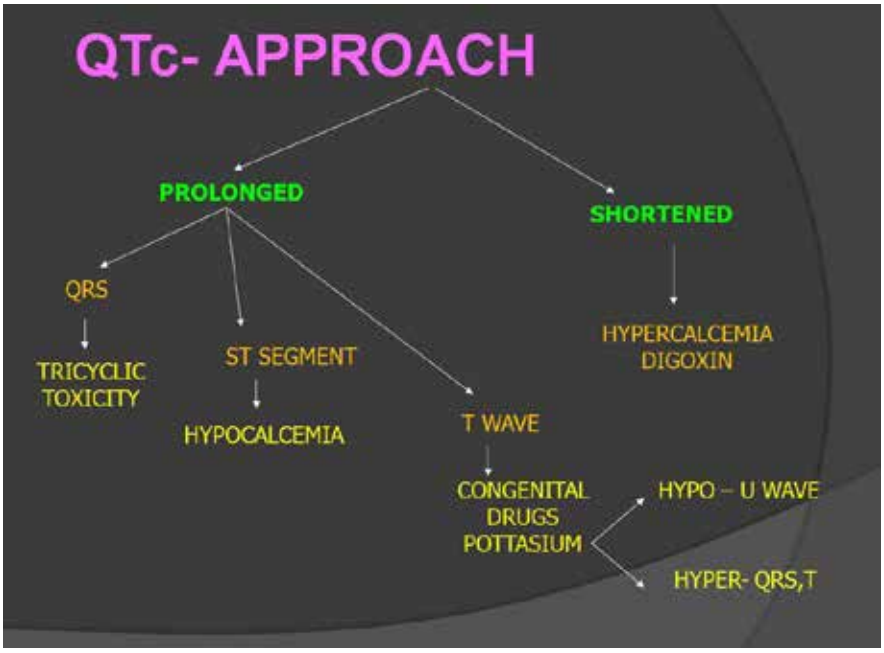


Figure 22: QTc and hypertension

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ST-T Changes: Ischemia, Strain, Secondary or Memory

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ST-T Wave Complex

ST segment represents the phase 2 (plateau) of cardiac action potential which gradually merges with the T wave. The TP segment is the true isoelectric line, as it represents the phase 4 of action potential. Since the entire myocardium is depolarized during phase 2 with no active vector, ST segment is also represented by isoelectric line electrocardiographically.

QRS complex depicts the depolarization, and the ST-T complex represents the repolarization of the myocardium. The depolarization wave front starts in the endocardium and spreads towards the epicardium. Epicardium has a shorter action potential with faster recovery and hence repolarizes earlier than the endocardium. Thus, the resultant vector during repolarization is also directed towards the epicardium. This explains the concordant deflection of QRS complex and T wave on electrocardiogram (ECG).

ST-T changes can be broadly divided into primary ST-T changes and secondary ST-T changes. ST-T wave in ECG is formed during the repolarization phase of the myocardium. Hence, any ST-T changes that occur due to abnormal repolarization is referred to as primary ST-T changes. These include ST-T changes due to myocardial ischemia, dyselectrolytemia, drug toxicity etc.

An altered ventricular activation or abnormal depolarization in-turn causes abnormal repolarization. The resultant ST-T changes are termed secondary ST-T changes.

ST-T changes due to myocardial ischemia are most frequently encountered in clinical practice. Needless to say, they require early diagnosis and treatment. These changes occurring in various other physiological and pathological states, mimic those seen in myocardial ischemia and pose a diagnostic conundrum. It thus becomes imperative to understand and differentiate between the various causes of ST segment deviation. The morphology and extent of ST deviation and its association with QRS and T wave abnormalities along with clinical signs and symptoms helps to decode the cause of ST-T deviation.

1. ST -T changes due to Ischemia:

Myocardial injury is one of the most important cause of ST segment deviation. ST-T deviation depends on the site (ST segment vectors are directed toward the injured tissue), duration and extent of injury and presence of prior abnormalities (like LBBB or paced rhythms).

ST-T changes due to myocardial ischemia can be broadly categorized into 4 variables:[1]

	ST elevation	ST depression
Ischemia	Non infarction transmural ischemia: Vasospastic angina	Non infarction subendocardial ischemia
Infarction	ST elevation / Q wave MI	Non-ST elevation/ non-Q wave MI

- **ST segment elevation (STE) due to acute myocardial infarction (MI):**

  New ST segment elevation measured at J point should be ≥ 1mm in 2 contiguous leads. Except in lead V2 and V3 where it should be ≥ 2mm in men ≥ 40 years, ≥2.5mm in men <40 years and ≥ 1.5 mm in females regardless of their age. Leads oriented away from the injured myocardium show reciprocal changes in from of ST segment depression **(figure 1).** These reciprocal changes help to

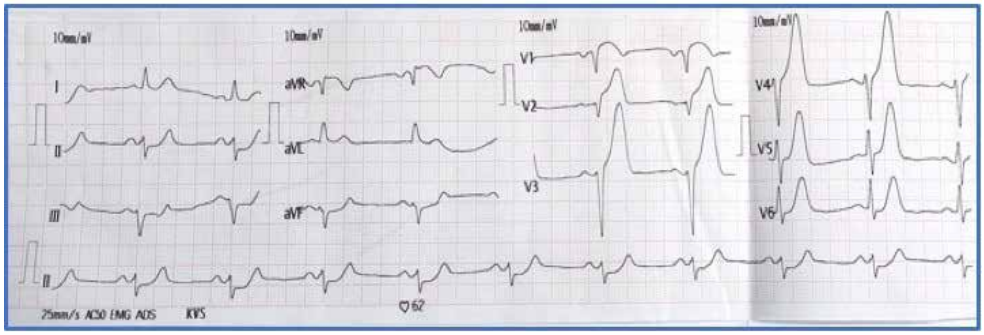

Figure 1: J point elevation with tall T waves in anterior leads associated with reciprocal depression in inferior leads indicating hyper acute phase of anterior wall MI

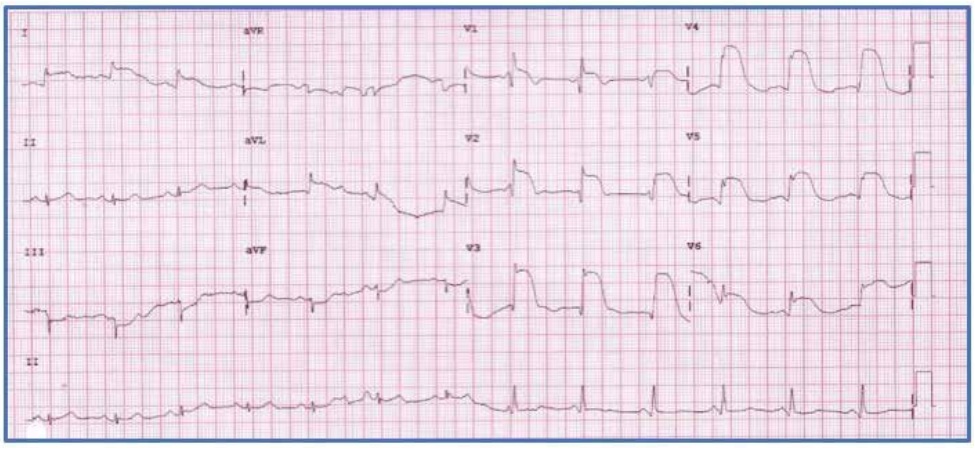

Figure 2: ECG showing extensive anterior wall STEMI. "Tombstone" STE can been seen in V2 to V5 with reciprocal ST depression in inferior leads

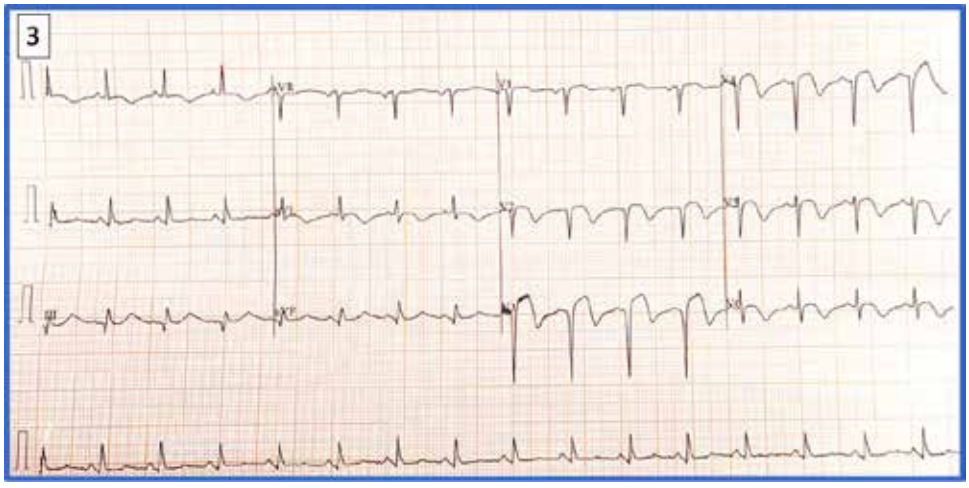

Figure 3: ECG showing evolving anterior wall MI. QS complexes associated with upcoving STE and T wave inversions

differentiate STEMI from pericarditis or early repolarisation changes.

As the MI progresses, the concavity of ST segment is lost and it becomes convex upwards. ST segment merges imperceptibly with the T wave. This monophasic QRS-T complex is called 'tomb stone pattern' **(figure 2)**. Gradually, ST elevations settle and T wave inversion can be

seen with or without associated Q waves in the same leads. The changes typically occur within hours to days of MI and may gradually disappear or even persist. These T wave inversion which suggest evolving MI or chronic ischemia have characteristic deep, inverted, symmetrical limbs and pointed appearance **(figure 3)**.

Wellens' sign or LAD-T wave pattern: Biphasic T waves (type A) or deep symmetrical T wave inversions (type B) in anterior precordial leads. These changes are seen when chest pain has subsided with or without cardiac enzyme elevation. These changes indicate critical proximal left anterior descending (LAD) artery stenosis and impending anterior wall MI **(figure 4)**.

STE related to aneurysm: ECG changes in aneurysm mimic evolving MI- persistent STE after acute MI (usually 3 weeks or more) which are convex upwards and are associated with q waves, fragmented QRS and even reciprocal changes. Wall motion abnormalities like akinesia, dyskinesia or aneurysm are seen on 2D-echocardiography.

- **Non-infarction transmural ischemia :**

  Transient ST Segment elevations with concavity upwards can occurs during episode of coronary vasospasm. This is the hallmark of Prinzmetal or vasospastic angina. Usually troponin are not elevated and ST segments return to baseline without associated q waves in most.

- **Non ST elevation myocardial infarction (NSTEMI)**

  New ST-depression ≥0.5 mm in 2 contiguous leads and/or T inversion >1 mm in 2 contiguous leads with prominent R wave or R/S ratio >1 is pathological. These ECG changes in combination with elevated cardiac biomarkers help to diagnose NSTEMI.

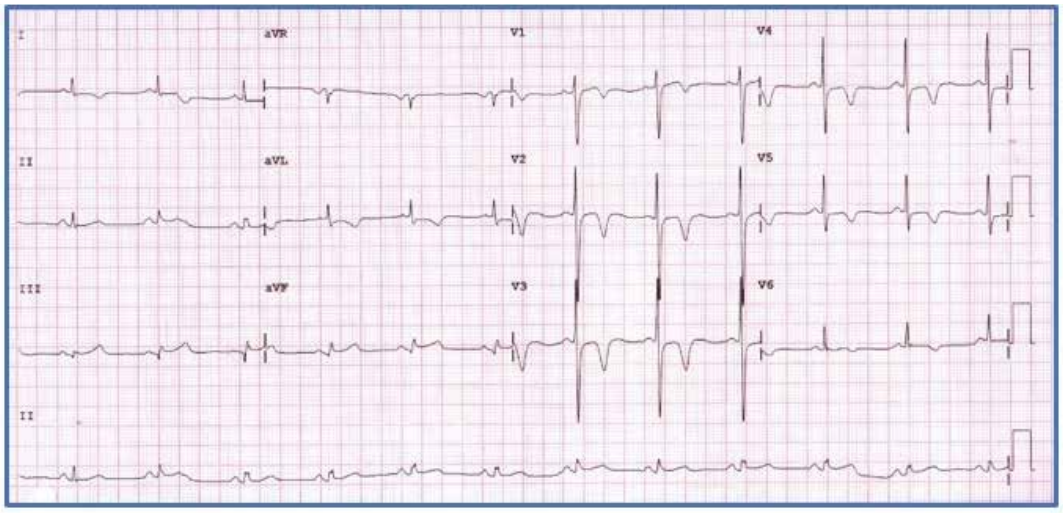

Figure 4: LAD-T wave pattern: Deep symmetric pointed T wave inversions in anterior precordial leads indicate Wellens' pattern type B (75% of cases).

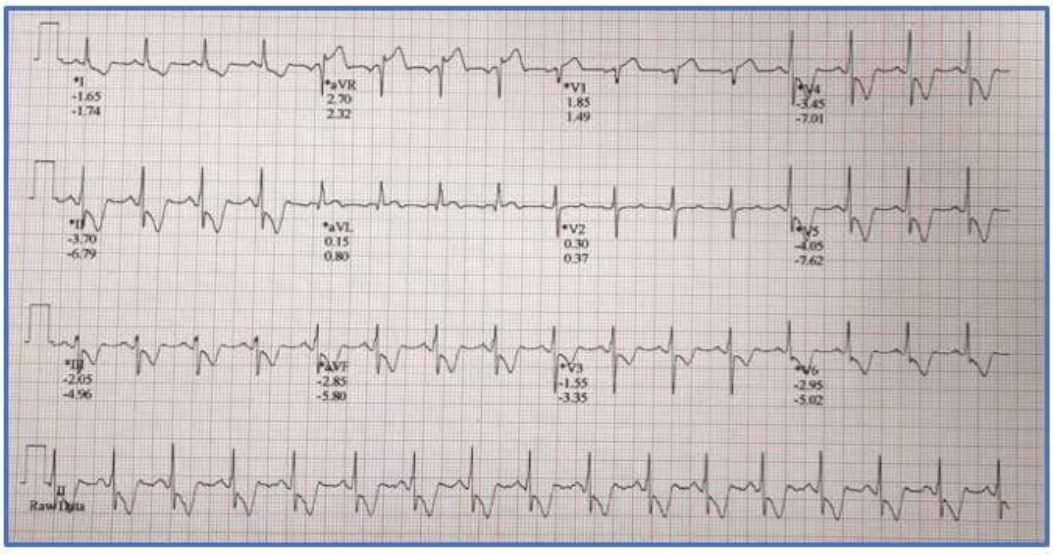

Figure 5: Strongly positive treadmill test: Downsloping ST segment depression in inferior and lateral leads with STE in avR. Patient had severe triple vessel disease on subsequent coronary angiogram

- **Non infarction subendocardial ischemia:**

  During sub endocardial injury, the ST vector is directed away from the epicardium towards the ventricular cavity. Hence leads oriented towards the epicardial surface (V5, V6) will show ST depression as opposed to lead facing the ventricular cavity (aVr) which shows ST elevation. This forms the basis of positive stress test in patients with coronary artery disease **(figure 5)**.

  However an important caveat is that patients with severe left main stenosis can have a similar ECG pattern.

  Horizontal or down sloping ST segment depression have strongest association with ischemia **(figure 5, 6)**. Upsloping ST depressions are physiological and can be seen in sinus tachycardia, hyperventilation etc. Upsloping ST depression when associated with upright T waves should almost always be considered normal.

  There is a notable exception to this rule. Upsloping ST segment depression (>1mm) in anterior precordial leads associated with tall prominent and symmetrical T waves with reciprocal ST segment elevation in avR (>1mm) in most cases, is suggestive of occlusive myocardial infarction. This is called de Winter's sign and is an anterior STEMI equivalent.

2. ST-T changes due to strain:

ST-T changes occurring due to pressure overload of ventricle is called strain. These changes occur as a result of relative myocardial ischemia or repolarisation abnormalities consistent with hypertrophied ventricle. ST-T changes are discordant with QRS polarity and are seen in in anterolateral leads (I, avL, V5, V6) in left ventricular hypertrophy (LVH) and right

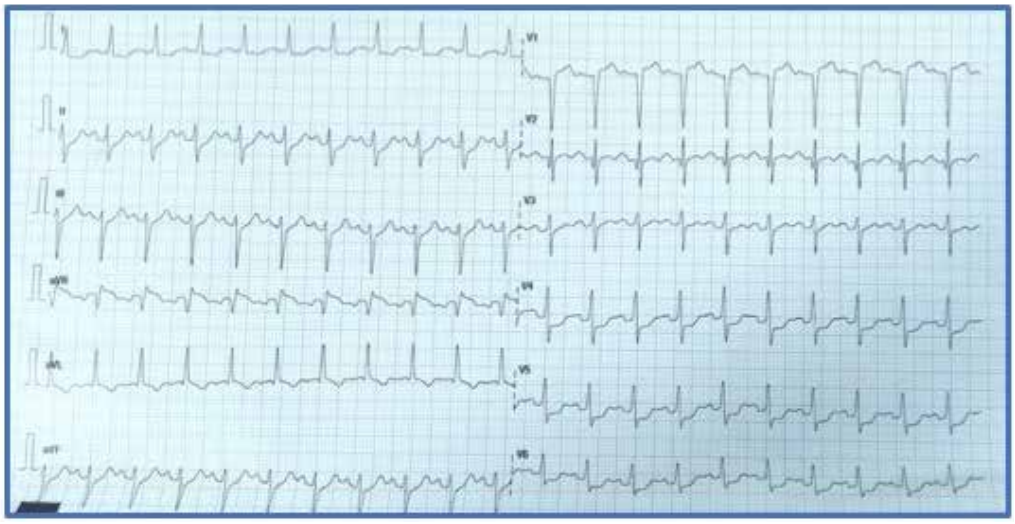

Figure 6: 55 year old patient presenting with angina. Baseline rest ECG shows Horizontal ST segment depression lateral leads with STE in avR, suggestive of myocardial ischemia

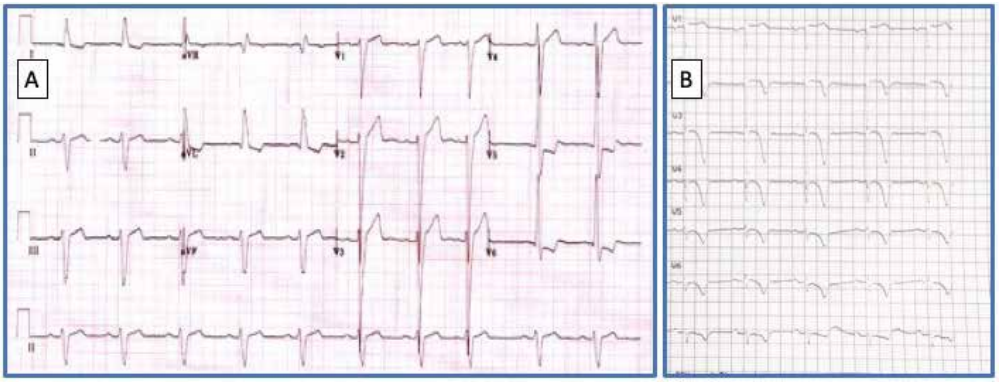

Figure 7: (A) ECG show left ventricular hypertrophy with strain pattern. Voltage criteria for LVH is satisfied. Typical ST depression in lateral leads with asymmetric, inverted, blunt and shallow T waves suggestive of strain pattern. (B) ECG depicts classical pattern of deep, symmetrical and pointed T waves suggestive of evolved MI or chronic ischemia. Compare the morphology of these ischemic T waves with that of strain pattern.

precordial leads (V1, V2, V3) in right ventricular hypertrophy (RVH).

Morphology of ST-T changes in strain: ST segments are usually depressed, down sloping with slight upwards convexity. T waves are inverted, blunt, asymmetric with a shallow proximal limb and not particularly deep. These typical morphological changes in association with QRS voltage criteria for LVH/RVH helps to differentiate them from ischemic ST-T changes **(figure 7).**

3. ST-T changes due to cardiac memory (CM):

Transiently alerted ventricular activation with QRS prolongation can cause T wave abnormalities (mostly T wave inversion) even after normal ventricular depolarisation has resumed. These changes can persists from hours to days and are called as cardiac memory.

The three principles of cardiac memory (CM) are (a) The direction of T wave in sinus rhythm tracks (remembers) the direction of QRS complex/vector during abnormal ventricular activation. (b) Longer the altered ventricular activation continues, larger is the change in amplitude of memory T waves. (c) Repeat episodes of abnormal activation after complete normalization of T waves result in more rapid and prominent accumulation of T-wave changes (hence the term memory).[2]

Altered ventricular activation can be seen in ventricular pacing, premature ventricular contractions, ventricular tachycardia, transient bundle branch blocks and ventricular preexcitation **(Figure 8).**

Differentiating CM from ischemic "T" Wave Inversion (TWI):

Right ventricular (RV) pacing leading to precordial T wave inversions during sinus rhythm is the most common CM encountered in clinical practice. QRS complex morphology mimics left bundle branch block with QS complexes in precordial leads. On resumption of normal conduction, T

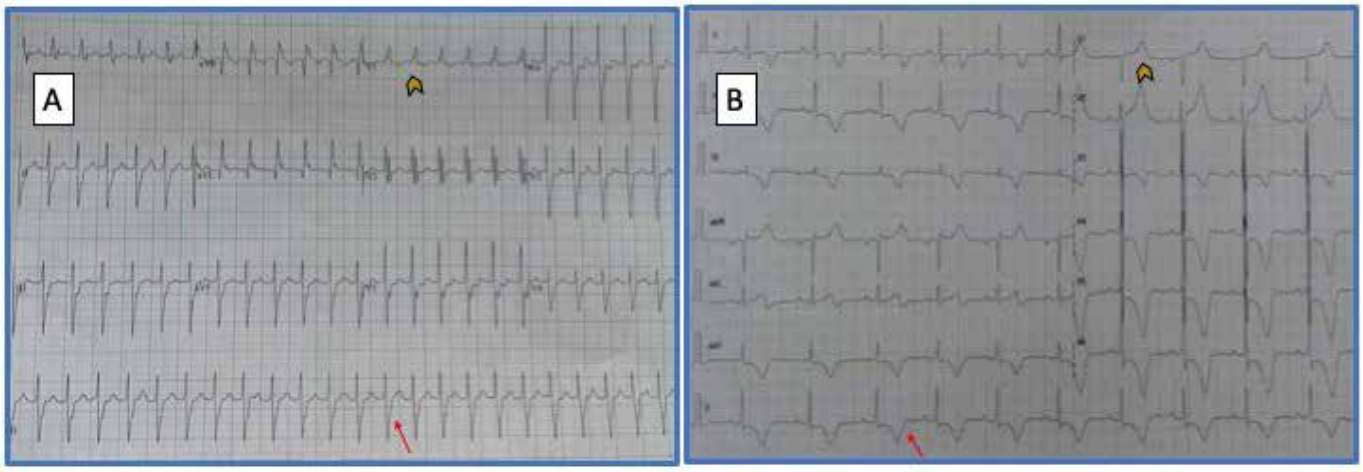

Figure 8: Example of cardiac memory. Figure 8A is an ECG of 31 year old with structural normal heart presenting with recurrent palpitations. ECg shows left posterior fascicular ventricular tachycardia (VT). After intravenous Diltiazem, patient reverted to sinus rhythm as shown figure 8B. The direction of T wave in sinus rhythm tracks (8B) the direction of QRS complex/vector during abnormal ventricular activation (8A). Leads with predominant negative QRS complexes during VT, have negative T waves in sinus rhythm (red arrows). QRS polarity in V1 during VT is positive and hence memory T waves in sinus rhythm are positive in V1 (yellow arrown heads)

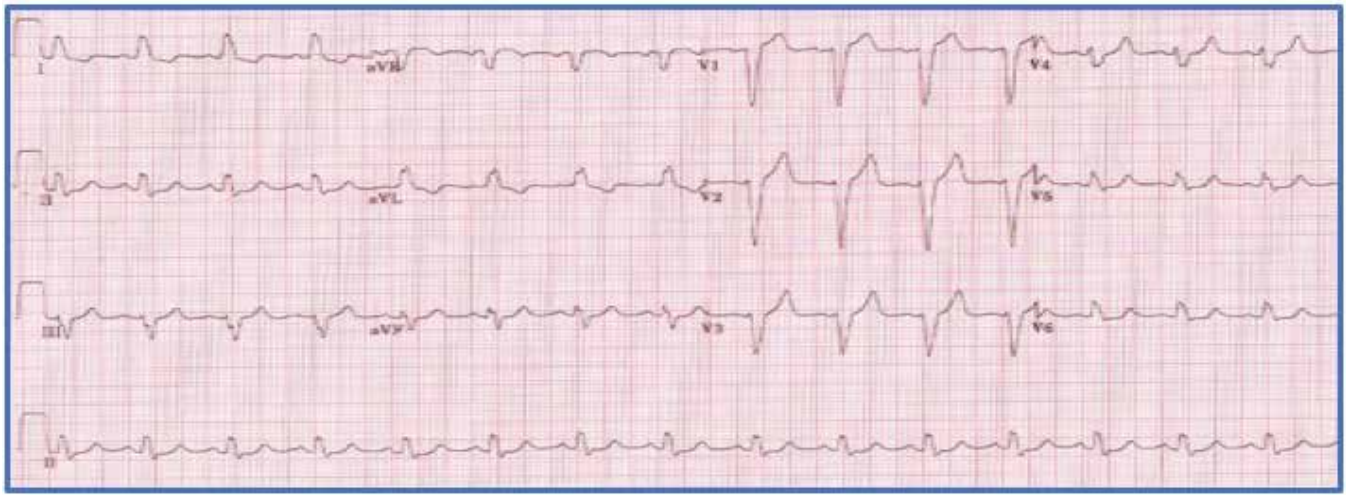

Figure 9: ECG shows left bundle branch block. ST-T changes seen are due to abnormal ventricular activation and hence are termed as secondary ST-T changes. ST-T deviation is opposite is opposite in direction to the terminal QRS defection.

waves in precordial leads become inverted. This is consistent with CM principle- T waves during normal conduction, track the negative QRS polarity which was seen during pacing.

This pattern of TWI in precordial leads can be difficult to differentiate from ischemic TWI seen in Wellens' syndrome. One important differentiator is T wave pattern in leads 1 and aVL. During RV pacing leads I and aVL have positive QRS polarity and hence during CM, T waves remain positive in these leads. In presence of ischemia in the LAD territory, TWI are commonly seen in leads I and aVL along with other precordial leads.

RV apical pacing leads to TWI in inferior leads on resumption of sinus rhythm. These can mimic TWI that occur during right coronary artery (RCA) ischemia. The amplitude of TWI in inferior leads is more than the amplitude of TWI in precordial leads in dominant RCA ischemia.

Thus, combination of positive T wave in lead I, aVL and precordial TWI > inferior TWI produces a unique pacing induce CM signature that is 92% sensitive and 100% specific in differentiating pacing-induced TWI from ischemia.[3]

4. Secondary ST-T changes

Secondary ST-T changes, as mentioned above, are due to abnormal depolarisation which leads to abnormal repolarisation. These changes are seen in LVH, RVH, bundle branch blocks **(figure 9),** preexcitation or during RV pacing. QRS and ST-T segments are discordant , and therefore leads with positive QRS polarity have depressed ST-T segments, and those with negative QRS polarity have ST segment elevation.

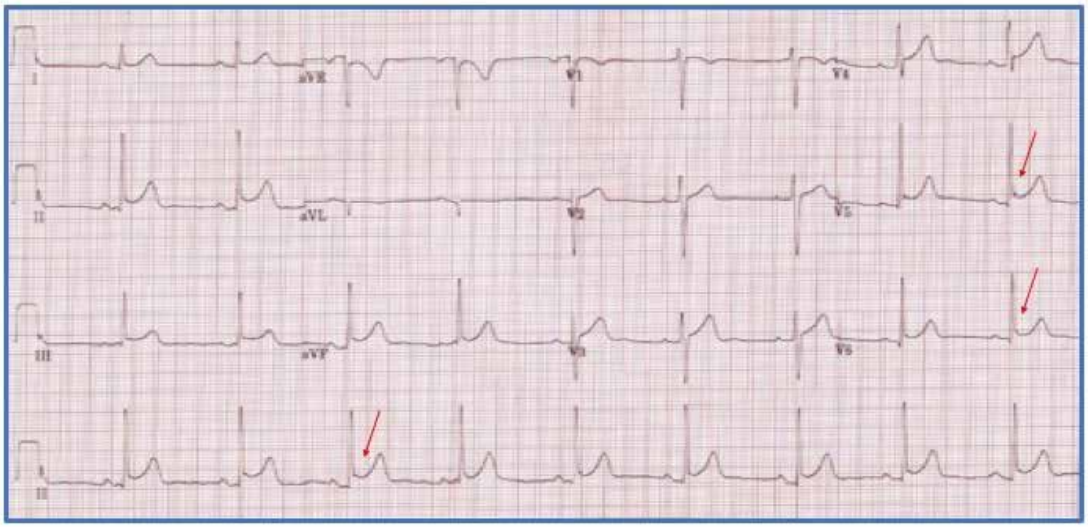

Figure 10: ECG shows type 2 ER pattern. J point elevation in inferior an lateral leads with QRS notching (red arrow)

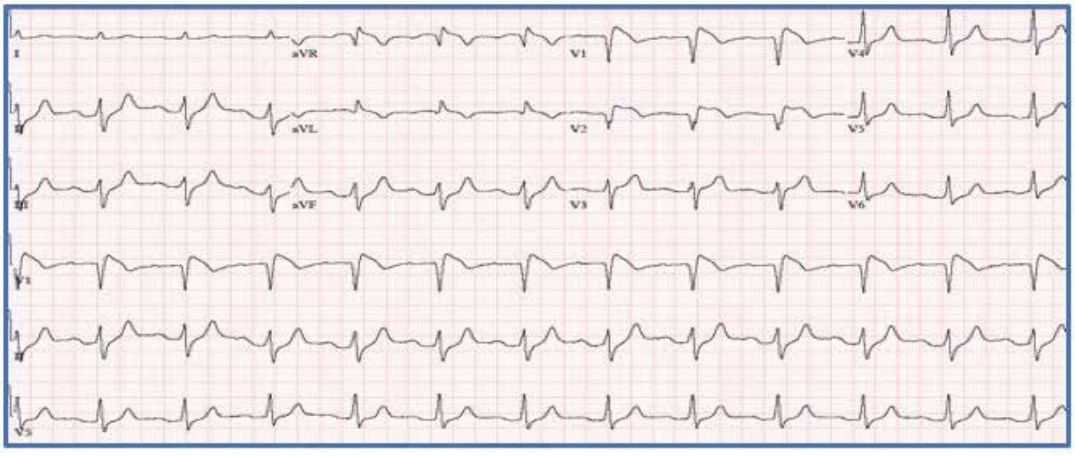

Figure 11: ECG demonstrates Type 1 Brugada pattern. Coved type ST segment elevation > 2mm in V1 with convexity upwards and descending as inverted T wave.

5. ST-T changes associated with other pathophysiological states:

Several other conditions can causes specific ST-T changes which may be confused with myocardial ischemia.

- **Pericarditis:** Widespread concave ST segment elevation with reciprocal depression in avR. Unlike MI, ST segment elevation is not limited to single coronary artery territory and is associated with PR segment depression. PR segment depression reflects atrial current of injury and is the most specific sign of pericarditis on ECG. Deviation of PR and ST segments are discordant.
- **Early repolarisation(ER) pattern:** J point elevation in 2 contiguous leads >1mm with QRS slurring or notching **(figure 10).** Risk of ventricular arrhythmias is least when ER is present in lateral leads **(type1),** moderate when present in inferior / inferolateral leads **(type 2)** and highest when seen globally-inferolateral and right precordial leads **(type3).** Absence of reciprocal changes on ECG, normal cardiac biomarkers and no regional wall motion abnormalities on 2D-echocardiography are few differentiators which favour ER pattern over myocardial ischemia or MI.
- **Brugada pattern:** These patients have pseudo right bundle branch block with persistent ST segment elevation in V1, V2.

  Type 1 (coved type): ST segment elevation ≥ 2mm with convexity upwards and descending as inverted T wave. **(Figure 11)**

  Type 2: ST-T segment has a saddle bag like configuration.

  Stress or Takotsubo cardiomyopathy: Transient ST elevations, depressions, T wave changes with modest elevations in troponin and wall motion abnormalities not limited to single epicardial coronary vascular distribution can be seen in stress or takotsubo cardiomyopathy.

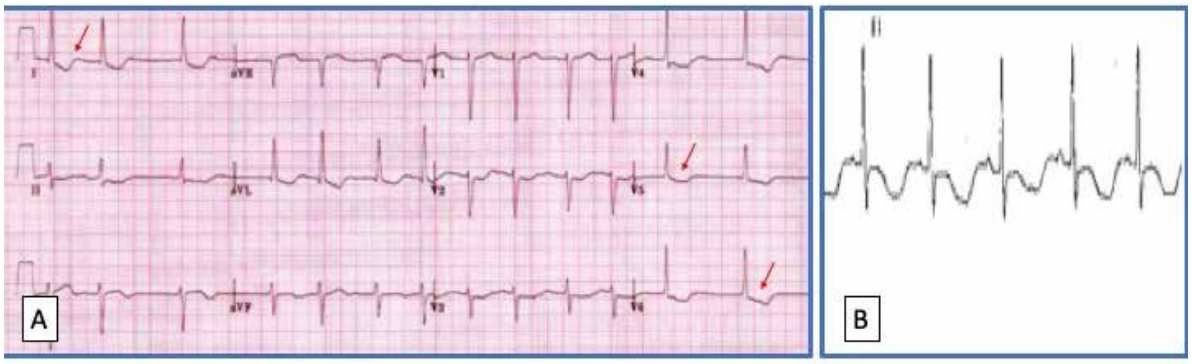

Figure 12: (A) ECG depicts atrial fibrillation with controlled ventricular rate with classical scooped ST-T segment suggestive of digitalis effect (red arrow).(B) Rhythm strip of patient presenting with CVA - subarachhnoid hemorrhage. ECG shows giant T wave inversion with prolong QT.

- **Digitalis effect:** Classical ECG change described is reverse tick sign. J point is depressed and sagging ST-T segment which gives 'scooped' or 'reverse tick' appearance is associated with shortened of QT interval **(Figure 12 A).**
- **Cerebrovascular accident (CVA) - T wave pattern:** Deep inverted T waves in multiple leads usually associated with prolong QT interval are characteristically described in CVA, especially in subarachnoid haemorrhage **(figure 12 B).** Similarly massive T wave inversions can be seen after Stokes-Adams syncope. These are attributed to neurocardiogenic stimulation and can simulate myocardial ischemia or evolved MI.
- **Apical Hypertrophic cardiomyopathy (HCM):** Another important differential of giant T wave inversions especially in left precordial lead is apical variant of HCM.

Conclusion

Not all ST-T changes on ECG should be interpreted as myocardial ischemia or infarction. Specific patterns of ST-T changes give us a good insight into the actual cause of these ST-T segment deviation. However ECG should always be interpreted keeping the clinical context in mind along with the other associated investigations.

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It may be Enjoyable to Hide, But it would be a Disaster not to be Found: Pacemaker ECGS: What Can They Hide!

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Ventricular paced rhythm can often mask various underlying conditions since it produces a depolarization pattern that does not follow the usual pattern of depolarization through the conduction system of the heart. The paced impulse produces an activation front that begins at the tip of the pacemaker lead which then spreads through the myocardium from one cardiomyocyte to another rather than through the His bundle and the bundle branches. This causes widening of the QRS complex and a T wave that is usually of opposite polarity to the QRS complex. The QRS complex during transvenous right ventricular apical pacing therefore resembles that of a spontaneously occurring left bundle-branch block (LBBB), and the ST-T–segment changes are usually discordant (ie opposite in polarity) from the QRS complex. Hence ECG changes associated with several conditions may be masked due to the abnormal depolarization and repolarization patterns produced during a ventricular paced rhythm.

It is very important to be able to diagnose these underlying conditions to avoid missing them. Three major categories of heart diseases that can be masked by paced rhythms are discussed in the following sections:

(A) Myocardial Infarction

(B) Exercise stress test

(C) Arrhythmias

(A) Myocardial Infarction: Pacing-induced ECG changes can often mask the ECG findings of acute myocardial injury. However, it needs to be emphasized that the ***established criteria of diagnosing acute MI in a paced ECG rhythm have been with pacing leads placed at the RV apex.*** There is lack of data on the ECG diagnosis of acute MI in cases of alternative pacing sites like interventricular septum, outflow tract, biventricular pacing and conduction system pacing. As mentioned above, RV apical pacing produces a LBBB pattern and can hence complicate ECG interpretation of acute MI. A careful analysis is important, as it can be easily of great help. Various ECG criteria have been proposed as indicators for myocardial infarction during ventricular pacing.[1] The more useful ones can be divided into (1) analysis of ST segment and (2) analysis of the QRS complex.

1. ***Analysis of ST segment changes is generally the most useful step in diagnosing acute MI in cases with paced rhythms.*** During RV apical pacing most ECG leads show a predominantly negative QRS complex accompanied by ST-segment elevation and positive T waves. Since these changes often resemble those seen in an acute coronary syndrome, it is important to ***identify a threshold level of ST-segment elevation*** that can correctly help discriminate between the two conditions.

   The original Sgarbossa criteria (OSC, Figure 1a) have traditionally been used to assist with the diagnosis of ST-elevation myocardial infarction (STEMI) in patients with LBBB and right ventricular paced rhythm (RVPR).[2] The

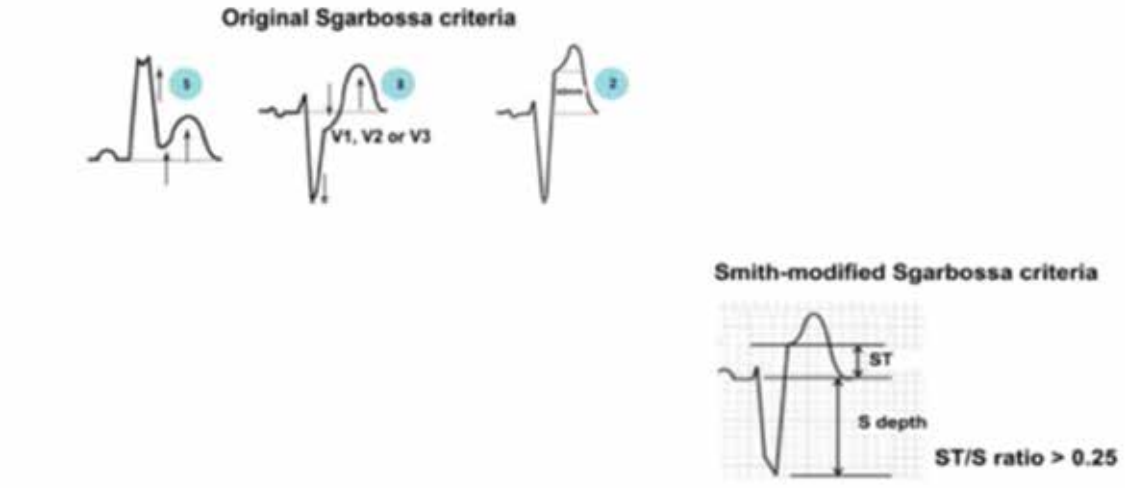

Figure 1: (a) The original and the Figure (b) Smith-modified Sgarbossa criteria for diagnostics of STEMI in presence of LBBB or RVPR.

Table 1: Odds ratio and Score of the original Sgarbossa criteria

Criterion	Odds Ratio	Score
ST-segment elevation > 1 mm and concordant with QRS complex	25.2 (11.6–54.7)	5
ST-segment depression > 1 mm in lead V_1, V_2, or V_3	6.0 (1.9–19.3) 3	3
ST-segment elevation > 5 mm and discordant with QRS complex	4.3 (1.8–10.6) 2	2

criteria were developed from the GUSTO-1 trial experience of 131 patients with acute MI in the presence of LBBB. ***The investigators reported that S***T-segment deviation was the only ECG finding that was useful in the diagnosis of acute MI and the previously proposed ECG signs involving the QRS complex were not useful. The following three ECG criteria were found to have independent predictive value in diagnosing acute MI:

i. Concordant ST-segment elevation ≥1 mm for leads with a predominantly positive QRS complex— score of 5.

ii. Concordant ST-segment depression ≥1 mm in leads V1, V2 or V3—score of 3.

iii. Excessively discordant ST-segment elevation ≥5 mm in leads with negative QRS complexes—score of 2.

A total score of ≥3 suggests that the patient can be diagnosed as having an AMI. ***(Table 1).*** However, although the ***specificity of the original Sgarbossa criteria is good, their sensitivity is low*** and therefore the criteria cannot be ***applied to rule out an acute coronary event with certainty***. Since criteria 3 (ST-segment elevation > 5 mm) is only assigned a score of 2, such patients should probably undergo further testing. In a comparison of ventricular-paced ECG controls with ventricular-paced ECGs with AMI, confirmed by cardiac biomarkers, Sgarbosssa et al[3] reported that

- of the three OSC, ***ST elevation ≥ 5 mm in leads with negative QRS complexes was the only criterion with both relatively high specificity and statistical significance for the diagnosis of acute MI*** in paced rhythms (positive likelihood ratio 4.41).
- The other 2 Sgarbossa criteria had acceptable specificity for the diagnosis of AMI: ST elevation > 1 mm in leads with concordant QRS polarity (positive likelihood ratio 3.1) and ST depression > 1 mm in leads V1, V2, V3 (positive likelihood ratio 1.64), although this was not statistically significant.
- No criteria involving the QRS complex or isolated T waves reached either statistical significance or a specificity > 80%.

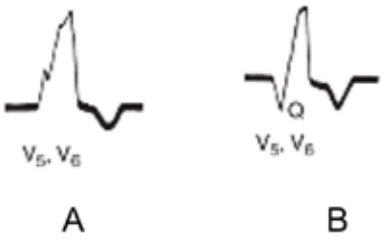

Figure 2: (A) With uncomplicated LBBB, no Q waves will be seen in V5 and V6. (B) With LBBB complicated by anteroseptal MI, prominent Q waves seen in leads V5 and V6

- Hence ***amongst patients with paced rhythm, ST segment elevation > 5 mm in leads with a negative QRS complex was the best discriminator*** to diagnose acute MI while in the ***setting of LBBB, the best performing criteria was ST elevation > 1 mm in leads with concordant QRS polarity.***

Smith modified Sgarbossa Criteria (SMSC)

Smith et al[4] described the Smith-modified Sgarbossa criteria (SMSC), proposing a modification to the original Sgarbossa criteria no. 3 (Figure 1b). Due to the excessively discordant ST elevation that can occasionally be seen in paced rhythms, rather than use an absolute value (≥5 mm, which may overdiagnose AMI in such cases), the authors utilized the ST/S ratio (Total ST elevation measured in proportion to the preceding S-wave depth). ***An ST/S ratio ≥ 0.25 is considered significant.***

- A recent multicentre retrospective study showed that the ***SMSC are far more sensitive than the OSC*** for the diagnosis of AMI in the presence of RVPR (sensitivity of 81% vs 56%) and ***have a high specificity*** (84% vs 90%).[5] The sensitivity of the SMSC is even higher if criterion 2 from the OSC is extended to V1–V6, instead of only V1–V3.

1. Analysis of the QRS complex:

Septal q waves: In LBBB, since initial septal depolarization forces are altered and directed from right to left, there is an initial R wave in the mid precordial to lateral precordial leads and septal q waves are absent. ***Q waves in leads V5 and V6 along with increased r in lead V1 is specific for anterior wall MI*** in the setting of LBBB or a paced rhythm. The presence of ***QR complexes in leads I, V5 or V6, or in II, III, and aVF with LBBB strongly suggests underlying an acute or chronic MI*** involving both the free wall and septum (or the septum itself) (Figure 2).

- A well positioned lead at the RV apex rarely if ever produces a qR pattern in lead I, V5 and V6 in the absence of an underlying MI. ***Thus presence of Q waves in these leads, in a patient with RV apical pacing lead in situ indicates an underlying MI.***
- It however needs to be remembered that in cases

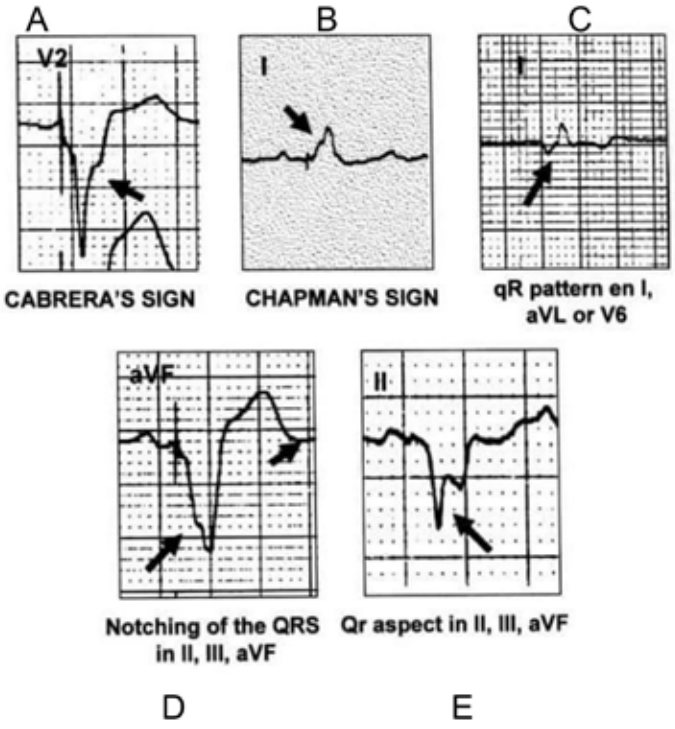

Figure 3: (A) Cabrera's sign (notching 0.04 s in duration in the ascending limb of the S wave in lead V3, V4 or V5); (B) Chapman's sign (notching of the upstake of the R wave in lead I, aVL, or V6); (C) Presence of a qR aspect with a q wave exceeding 0.03 s in lead I, aVL, or V6); (D) notching of the first 0.04 s of the QRS complex in lead II, III, or aVF; (E) presence of a Qr aspect with a q wave exceeding 0.03 s in lead II, III, or aVF.

with RV apical pacing, these Q waves may sometimes simply reflect differences in the lead tip position rather than underlying MI. On occasion, if the lead tip is not at the right ventricular apex, then Q waves may also be normally seen in these leads.

- An important point is to **differentiate a qR/QR from a QS complex**. This differentiation is important because a QS complex carries no diagnostic value during RV pacing in any of the leads (QS complexes can be normal in leads I, II, III, aVF, V5, and V6) whereas qR/ QR complex can have diagnostic value.[6]

Cabrera's and Chapman's sign[1,6]: Cabrera's sign (Figure 3) is the notching (≥ 0.4 secs) of the ascending limb of the S wave in the mid precordial leads, usually in leads V3 and V4, (sometimes also in leads V2 and V5). Chapman's sign (Figure 3) is the notching of the upstroke of the R wave in leads I, aVL, or V6. It has a low sensitivity but is very specific sign.

- In a study of 45 patients with known MI (anterior 23, inferior 22), Kocchiadakis et al[7] reported that the ***most sensitive criteria were Cabrera's and Chapman's*** (91.1 and 86.6%, respectively), ***while the specificity was low*** (range 42.3-69.2%). ***A combination of Cabrera's and Chapman's sign reduced the sensitivity to 77.7%, and increased the specificity to 82.2%.***
- In 107 patients with permanent pacemakers (group 1: controls without a history of MI, group 2: with documented previous MI and group 3: with biventricular pacing for severe heart failure), a 12-lead ECG with full ventricular capture was used for analysis.[8] The sensitivity, specificity, positive predictive value, negative predictive value, and overall accuracy of the ***following 5 criteria to detect a prior MI was calculated*** (Figure 3): 1. Cabrera's sign, 2. Chapman's sign, 3. Presence of a qR in lead I, aVL, or V6 , 4. Notching of QRS in lead II, III, or Avf, and 5. Presence of a qR in lead II, III, or aVF.

The sensitivity for Cabrera's sign was 63.6% while it was poor for all other criteria (9.1-40.9%. ***The specificity was relatively high for all the ECG criteria*** (81.6-100%). ***Combining all 5 ECG signs increased sensitivity to 86.4%, with a specificity of 65.8% and an overall accuracy of 76.8% for the diagnosis of previous MI.*** None of the 5 criteria was particularly useful to assess the site of prior MI. ***In patients with biventricular pacing,*** the accuracy of all 5 ECG criteria was poor and the presence of a qR wave in lead I, aVL, or V6 appeared nonspecific and related to pacing site.

Summary

In paced patients, the diagnosis of acute MI is often challenging. Although comparison with previous ECG's can be done, these are rarely available. Hence it is important to understand the various described ECG criteria that are useful in such situations. Meticulous application of the Sgarbossa criteria, Smith's modification, looking for Cabrera and Chapman signs, among others can be useful in recognizing an underlying MI in paced patients.

(B) Exercise stress test*:* Since RV pacing can produce major QRS and ST-T changes that can alter the resting ECG, ischemic changes during an exercise stress test may be difficult to detect. An exercise ECG is not recommended for diagnosis of obstructive coronary artery disease in patients with chest pain and baseline ECG abnormalities like a paced ventricular rhythm or complete LBBB. Ideally, evaluation for CAD in patients with a pacemaker should include an imaging modality (eg, stress radionuclide myocardial perfusion imaging or stress echocardiography, rather than an exercise ECG) to improve the diagnostic yield.

(C) Arrhythmias*:*

The presence of AF in the background of a continuously paced rhythm may be difficult to recognize especially in a single chamber ventricular pacemaker and small low voltage fibrillatory waves. This potential pitfall to recognise AF may lead to sub-optimal rates of anticoagulation and consequently higher incidence of strokes and other complications. Since the risk of thromboembolic events in AF is comparable among

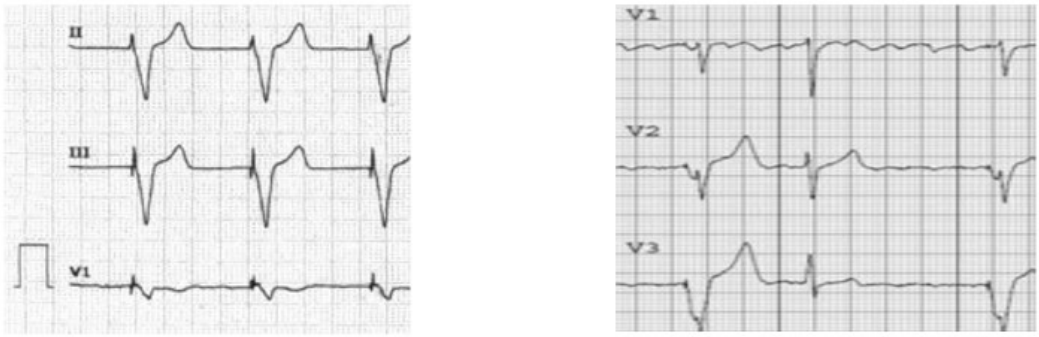

Figure 4: (A) Atrial fibrillation underlying a paced rhythm, (B) Atrial Fibrillation and Ventricular demand pacing.

paced and non-paced patients, it is very important to diagnose underlying AF in patients with a paced rhythm. ***The diagnosis of AF in a paced rhythm is based on the following:***

- detection of the presence of irregularly irregular RR intervals with fibrillatory waves during pacer inhibition or
- presence of clear fibrillatory waves and no discernible P waves between pacemaker spikes on 12 lead ECG (Figure 4).

Conclusion

Paced rhythms can mask various underlying cardiac abnormalities especially an acute MI. Comparison with previous ECG's if available can help in cases with a diagnostic dilemma. However, this may not be possible in all situations. If the patient with a pacemaker has an intrinsic rhythm, a 12 lead ECG with magnet can be taken to reveal the underlying abnormality. Detailed analysis and an orderly approach as described above, would be useful in all cases.

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ECG Markers of Sudden Cardiac Death

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Sudden cardiac death is a major public health burden. Although numerical estimates outside America and Europe are sparse and ascertainment of its true magnitude is highly complicated due to wide variety of methodological issues, some generalizations commonly made include SCD contributing to around 50% of all cardiovascular deaths with 50% being first symptomatic cardiac event. The premature death and associated severe neurological disability upon surviving is responsible for the disproportionate loss of potential years of peak productive life to the society. Hence, prediction and prevention is of utmost importance. The current best marker, left ventricular ejection fraction, is woefully inadequate as majority of the events happen in people with normal EF while on the other hand current ICD appropriate discharge rates in primary prevention are abysmally low in patients with poor EF. This article reviews ECG markers which reflect arrhythmic substrates underlying VT/VF which is the primary treatable rhythm associated with better survival in sudden cardiac arrest.

ECG markers can be broadly classified into 2 categories, those pertaining to the depolarization or the repolarization phase.

QT interval

Multiplicity of heart rate correction formulae exist which can be broadly categorized into those utilizing proportional scaling (such as Bazette's and Fridericia's) and linear scaling such as Framingham's. Bazette's is most widely used because of its simplicity, however it leaves strong positive residual correlation with heart rate and QT intervals are significantly overestimated at high heart rates.

While heart rate correction is well known, QT/RR hysteresis is rarely considered. When the heart rate changes change in QT interval is not instantaneous. Studies of abrupt heart rate changes via pacing have revealed that it takes around 2 to 3 minutes for the change in QT interval to happen. So, for example when a QT interval is corrected for current ongoing RR interval which is shorter than the previous steady state RR interval, prolonged QT interval is obtained. Hence heart rate needs to stable prior to calculating corrected QT intervals which is harder to ensure in usual 10 second ECG recordings obtained in stable supine position considering the psychological and mental stimuli component.

Amongst the leads, many a times suggestion is made to measure the interval in lead II → lead V_5 along with tangent method to better ascertain the end of T wave. This is not based on any sound reasoning or data, as conceivable, inter-lead differences exist due to variable projections of the 3-dimensional T wave vector-cardiogram loop, which does not stay constant even when considering single subject due to variation induced by respirations and other factors, making serial assessment imprecise despite using the same lead in a particular person. Most current digital ECG acquisitions machines record all the leads simultaneously which permits temporal alignment and superimposition of the template permitting accurate assessment of end of T wave. However, most systems do not display tracings or onset offset points used by algorithms to derive automated QT intervals. Tangent method was proposed to identify true end of T wave in cases of partial merging of T and U wave and to increase accuracy and reproducibility. This method gives significantly underestimated readings and hence should not be used routinely.

QT interval has obvious limitations in cases of prolonged QRS durations. This can be overcome by using JT interval corrected for heart rate. Subtracting QRS duration from corrected QT interval (Bazette's) cannot be used because of the retention of strong residual correlation with rate, even larger than seen corrected QT interval. Moreover, JT interval cannot be corrected for heart rate by Bazette's method. Both the situations are problematic because of the underlying use of logarithmic transformation leads to different values of the coefficient and hence cannot be applied to any interval of different length than the physiological range of QT interval durations. However linear regression correction formula such as Framingham's is equally applicable.

Upper cut-off defined around the 99th percentile in consensus document have been set at 470ms for males and 480ms for females, with persistently elevated values specially above 500ms requiring additional evaluation. Similarly derived cut-off for short QT is 320ms however in absence of data suggesting long term morbidity in asymptomatic person, it needs to be further evaluated only in context of concerning clinical markers. Although relationship between QT interval and cardiovascular risk has been demonstrated in population-based studies as well as in post MI subjects, its utility in individuals who do not have long QT either due to congenital syndrome as well as drug related, for sudden cardiac death risk prediction is weak and not clinically useful.

QT Dispersion

The variability in QT interval between different leads arises due to orientation of leads axis to the axis of terminal part of the T wave vectorcardiogram loop. Leads whose axis is perpendicular to the terminal T wave have shortest QT interval whereas those parallel have the longest QT interval duration. Furthermore, decreased spatial amplitude of T wave loop plays a part too by increasing uncertainty in determining the end of low amplitudes T waves along with having an

influence on number of leads with measurable QT interval. Thus, T wave vectorcardiogram morphology is the major determinant of QT dispersion with smallest values for narrow high amplitudes loops and largest for small, wide loops. Wide small loops being considered a sign of various pathological condition hence associate with QT dispersion indirectly and not the least because of action potential durations in the heart getting mapped onto discrete areas on the body. In addition to absence of any direct mechanistic link with repolarization heterogeneity, QT dispersion measure is also marred by high inter and intra-observer variability, with errors reaching order of difference between normal and cardiac patients show in some of the trials, leading to inability of establishing reference values.

QT interval subcomponents: QRS duration, J – T peak & T peak – T end

QRS prolongation is more prevalent in patients with advancing heart disease and could simply be a surrogate for severe myocardial disease. Analysis of MADIT-II by Centers for Medicare and Medicaid services surmised it to be an important indicator of patients likely benefiting from ICD therapy. Similarly, subgroup analysis from MUSTT trial showed patients with LBBB and IVCD had increased risk of sudden cardiac arrest independent of EF.

Further investigations into the components of QT interval responsible for its association with sudden cardiac death lead to defining two sub-entities: J - Tpeak and T peak – T end. Rationale being division of QT interval into early repolarization mainly reflecting the plateau of the action potential and late repolarization reflecting terminal part of repolarization. In fact, research has shown that 25% of ventricular electrograms having underwent repolarization by the time of T wave achieves peak and so even J – T peak interval may be influenced by changes in later part of the action potential.

Interest in the interval came into being from studies in canine heart wedge preparations. As such preparations allowed to collect signal reminiscent of surface ECG, it was proposed that the interval approximated the spread of action potential across myocardium, leading to simplification of it representing complete hearts and interval reflecting repolarization heterogeneity.

However, well conducted studies by FDA showed drugs affecting J – T peak to be at higher risk of inducing torsades than those only altering T peak – T end.

Projection of T wave loop leads to substantial differences in T wave peak timing in different leads. Also, beat to beat changes frequently leads to variations exceeding the mean differences provided in previous reports between VT/VF patients and normal people. This is due to T wave morphology being affected by physiological processes such as respirations and meal intake because of change in position as well as axis of heart.

Moreover, there has been an uneven practice of reporting T peak – T end with some reporting it after correcting the interval with Bazette's while others not. Previous analyses have reported that T peak – T end interval is practically rate independent with most of the rate related variability lying in J – T peak segment.

The current practice to overcome these shortcomings as done in seminal FDA QT studies has been to calculate the interval with tangent method in vector magnitude lead computed using the Guldenring transform of 12 lead ECG or Mason-Likar leads to orthogonal X,Y,Z configuration. Another modification proposed has been to be done away with peak instead using point signifying 50% of area under single lead representation of T wave as peak is sensitive to the generally low voltages present at the end of T wave while area is little influenced by imprecision in discerning J point or end of the T wave.

QRS – T angle

This parameter can be calculated in 2 ways either considering the difference in direction of QRS axis and T wave axis in frontal plane or considering the same in 3 dimensions. Although benefit of 2 dimensional value is the ease with which value can be arrived by with the use of commonly provided values of the QRS and T wave axis in most automated ECG systems however has shown to be of low in sensitivity for predicting cardiac events when compared to the 3 dimensional calculations which require 3 dimensional vectorcardiogram loops to be generated with the conversion of 12 lead ECG into orthogonal lead system.

Values in frontal plane system above 90 degrees are considered abnormal whereas 3 dimensional values are generated with use of total cosine R to T method which are more abstract to be conceptualized. In this method the T wave vector is defined by its maximal vector magnitude. Cosines of the angles between this T wave vector and all vectors within the QRS complex exceeding a predefined proportion of the maximum vector magnitude are averaged. This was designed to deal with the problem that while T wave can be considered to have an underlying spatial orientation, the QRS loops are much wider and spatially curved so that no single vector can represent their orientation.

Microvolt T Wave Alternans

TWA has been thought to reflect repolarization heterogeneity. It is strongly influenced by heart rate likely by the way of having an impact on intracellular calcium cycling. However, heart rate is not the sole determinant, with autonomic neurotransmitters having the ability to increase TWA during fixed rate pacing. Similarly, this capacity of sarcoplasmic reticulum to reuptake calcium gets hampered in myocardial ischemia and heart failure.

Finnish Cardiovascular study, the largest study till date, involving > 3500 low risk patients referred for routine symptom limited exercise testing showed ability of TWA to

predict sudden cardiac death and cardiovascular and total mortality. However, in post MI MADIT II type patients although it predicted total mortality but was not the case for ventricular tachyarrhythmic events. Till date only one trial has tested its capacity in guiding prophylactic ICD implantation, the ABCD trial. It showed that predictive value being time dependent as Kaplan-Meier curve converged and hazard ratio decreased particularly after 1 year, which is understandable as most markers of VT are dynamic with risk prediction changing with change in parameters over time as shown by the upsurge in alternans preceded in spontaneous ventricular tachyarrhythmias in ESVEM trial. Another shortcoming has been of treating a continuous variable as binary which would have reduced its predictive power significantly as studies reveal that higher magnitude increased risk for ventricular tachyarrhythmia. Because of this association with heart rate and autonomic association method of assessing TWA usually involve some kind of exercise or ambulatory testing.

Fragmented QRS

Concept arose initially to improve the ability to detect a prior MI as compared to by Q waves alone because of q wave regression over time along with ability to include to non-Q wave MI. Location of fQRS in ECG correlated well with location of myocardial scar on imaging studies. However, fQRS is not specific for CAD and was also subsequently reported in myocardial disease such as cardiomyopathy. This was followed by studies reporting it to be an independent predictor of both SCD.

Criteria originally provided by initial investigators included the presence of an additional R wave (r') or notching in the nadir of the R wave or the S wave in 2 contiguous leads. Bundle branch blocks were excluded, however later on wide QRS morphologies were included and an extended definition of fQRS for the same were published. New criteria added presence of > 2 notches in R or S wave, similarly for the premature ventricular complexes. Afterwards a proposal for morphology-based classification advocated for removal of requirement for 2 contiguous leads in some morphology so as to increase sensitivity of the marker, in addition to improve the ability to differentiate between normal vs truly pathologic patterns, as a large population based study in Finland involving over 10,000 middle-aged subjects with or without cardiac disease showed the pattern to be present in a significant proportion of person without cardiac disease. Although it may be possible that some morphologies could be an early marker of subclinical cardiac disease in those without a known cardiac disease. Further the group showed fragmentation not to be associated with increased mortality in subjects without cardiac disease.

Furthermore, an issue of contention has been the subjective nature of ascertainment of fQRS pattern leading to weak repeatability between different clinics and investigators in addition to being dependent on quality of ECG acquired. Similar to presence of epsilon wave in right precordial leads whose value has been questioned because of its identification and interpretation are influence to a large degree by ECG filtering and sampling rate along with its subjective nature giving rise to significant interobserver variability as shown by Epsilon Wave initiative by the international society for holter and noninvasive electrophysiology. Hence in 2020 international diagnostic criteria this ECG marker has been downgraded to minor ECG criteria.

Signal-averaged ECG

The concept of SAECG is based on prolonged activation of small portions of ventricle being common in regions of scar especially in patients with VT. Late potentials identified in signal average ECG by reducing noise and thus allowing gain amplification along with filtering to remove T wave signals, refer to low amplitude signals that occur after the end of the QRS complex. These have been correlated to electrograms recorded directly from heart and are thought to represent a substrate for reentry. However, this technique is not useful in patients with bundle branch blocks.

Prolonged filtered QRS duration is the most robust measure correlated with outcome including mortality and increased risk of arrhythmic events.

The presence of late potentials which was minor criteria in 2010 TF criteria is no longer included among 2020 international criteria since use of signal averaged ECG has been abandoned by majority of cardiological centers worldwide because of its limited diagnostic accuracy.

Early Repolarization Pattern

Traditionally viewed as benign and often encountered particularly in healthy young males, seminal study by Haissaguerre et al first provided evidence for an increased risk for development of life-threatening arrhythmic events and sudden cardiac death in a case control study. Features identified with worse prognosis included early repolarization pattern of J point elevation in multiple especially in inferior leads with high voltage (at least 0.2mV) and with horizontal / down-sloping ST segments instead of a rapidly ascending one found associated with J point elevation in anterior leads in healthy individuals. Although the risk has been confirmed in multiple population-based studies but risk associated is still quite low (1:10,000). Because of which asymptomatic individuals with early repolarization even with those higher risk patterns do not require further evaluation except when there is strong family history of SCD or when associated with Type 1 Brugada pattern or short QT. Although ERS patter has been most associated with VF, increased risk seen in population studies cannot be possibly related to idiopathic VF which is a very rare disease. One hypothesis presented has been of slightly increased VF risk in the event of a common disease such as acute myocardial ischemia.

Brugada Type 1 Pattern

This distinct pattern consists of coved rSr' pattern, a ST

segment elevation >2 mm and inversion of terminal portion of the T wave in leads V1, V2, V3, needs to be investigated in absence of symptoms even.

Ventricular Preexcitation

Pattern of short PR and delta wave has been associated with SCD and alarmingly around half of the patients SCD is the first clinical manifestations of the syndrome. This pattern warrants further evaluation of the refractory period of the accessory pathway. Intermittent pre-excitation during sinus rhythm on resting ECG is consistent with low risk pathway. Furthermore, loss of preexcitation at high heart rates also points towards a low-risk accessory pathway. Spontaneous triggering of pre-excited AF in such patients also a harbinger of SCD.

Ventricular Premature Complexes

In addition to burden, certain features have been associated with sudden cardiac death, related to morphology and coupling interval. Wide (QRS > 150ms), multiforme PVC's have been associated with risk for adverse cardiac outcomes along with short coupling interval. Based on limited studies which has been currently identified to be <450ms. Although short coupling interval VPC have been associated with risk of VF, predominant mechanism is through reduction of cardiac function.

Although in general population RVOT ventricular premature complexes (left bundle branch block with inferior axis) are usually idiopathic and benign they can early and only manifestation of ARVC. Combination of QS morphology in lead V1, predominantly negative QRS complex in lead 1 (typical features of arrhythmias arising from the anterior RVOT wall near the pulmonary valve rather than the septal/ postero-septal RVOT region) and intrinsicoid deflection time > 80 ms (because of segmental fibrofatty replacement and epicardial origin) showed a high specificity for an underlying arrhythmogenic cardiomyopathy.

Conclusion

Despite having some predictive values in population-based studies, the same results have not been borne out in individual patients which is an altogether a different ball game due to low absolute risk. Major hopes generated from T wave alternans and Signal averaged ECG have slowly withered due to the inability to find practical ways to incorporate the information into clinically actionable preventive approaches. Problem listed with various parameters have been enumerated in the current review, including the dynamic nature of most ECG based markers hence the value of assessing them over time instead of being one-time analysis.

Maybe, answer of SCD risk prediction may not lie in simplified one time ECG parameters assessment which can be gleaned off superficially from tracing but might be involving specialized ECG analytical techniques including machine learning and artificial intelligence.

Apart from finding abnormal QT interval leading to diagnosis of congenital long QT syndrome or Type 1 Brugada pattern leading to its diagnosis or making the diagnosis of arrhythmogenic cardiomyopathy i.e. specific disorders, currently there are no practical ECG based approaches to be brought into clinical practice to better predict sudden cardiac death.

Ventricular Ectopy: Harmless or Life Threatening

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Definition

A premature ventricular complex (PVC) refers to an ectopic cardiac impulse originating from the ventricle. The Heart Rhythm Society consensus recommends the term 'premature ventricular complex' and not 'ventricular premature depolarization' or 'premature ventricular contraction' to standardize the literature and acknowledge that electric activity may not lead to mechanical contraction.[1]

Prevalence

Although PVCs were found in a healthy military population in only 0.6% of those <20 years of age and 2.7% of those >50 years of age[2] on 12-lead ECGs, longer term monitoring shows PVCs in about 50% of all people with or without heart disease.[3] PVCs are common and increase in frequency with age. An analysis using the ARIC study[4] cohort demonstrated that older age, male sex, black race, a history of hypertension, evidence of other heart disease, a higher heart rate, lower educational attainment, and hypomagnesemia were each associated with the presence of at least 1 PVC during a 2-minute ECG recording.[5] In addition, performing less regular physical activity, and smoking have been identified as potentially modifiable risk factors associated with increasing PVCs.

Pathophysiology

Frequent PVCs are defined as the presence of at least 1 PVC on a 12-lead ECG or >30 PVCs per hour. Generally, frequent PVCs have a good prognosis in the absence of structural heart disease.[6] In some studies, they have been associated with an increased risk for all-cause and cardiovascular mortality.[7] Negative hemodynamic effects of ventricular premature beats include shorter ventricular filling time, absent atrial contribution to ventricular filling, asynchronous ventricular contraction, AV valve incompetence when VPB starts before atrial contraction, an increase in pulmonary capillary wedge and caval venous pressures and atrial size due to atrial contraction against closed AV valves. Very frequent PVCs, >10,000 to 20,000 a day, can be associated with depressed LV function in some patients that is reversible with control of the PVCs, and has been referred to as PVC-induced cardiomyopathy.[8]

PVCs could be seen with or without underlying heart disease. In the absence of structural heart disease, reversible causes include electrolyte abnormalities (hypokalemia, hypomagnesemia, hypercalcemia, sleep apnea, hyperthyroidism, alcohol, amphetamines, caffeine, cocaine, tobacco, medications that may precipitate ectopy such as digoxin, sympathomimetics, and tricyclic antidepressants, and anxiety. Left ventricular hypertrophy, ischemic heart disease, heart failure are the common underlying heart diseases. PVCs in this setting is associated with increased mortality.

Clinical Evaluation

PVCs are commonly an incidental finding. However, patients might present with symptoms of palpitations, skipped or irregular heartbeats. The symptoms are often due to the stronger beat following a PVC. Many patients do not feel them and some patients feel some PVCs and not all of them. If ventricular arrhythmia is precipitated by PVC, syncope could be the presentation. A family history should include any instance of sudden death in first degree relatives, any heritable cardiac conditions, or coronary artery disease at an early age. Patients should be screened for alpha, beta or dopamine receptor agonist drugs, sympathomimetic medications, and usage of illicit substances such as cocaine and amphetamine. Physical examination is aimed to look for evidence of structural heart disease, especially the dilated heart.

Diagnostic Investigations

Electrocardiography

ECG should include a long 12 lead rhythm strip of 30 to 50 seconds. One should look for QT interval, pathological Q waves that reflects a scar, an early precordial transition accompanied by a prominent S wave in V6 indicative of non-ischemic cardiomyopathy, ST segment elevation in leads V1 and V2 consistent with Brugada pattern, Epsilon waves seen in right ventricular arrhythmogenic cardiomyopathy, and any conduction disease that could be a manifestation of cardiac sarcoidosis. About 60-80% of idiopathic PVCs arise from right ventricular outflow tract . They typically occur in the middle age group, between 30-50 years but range from adolescents to elders. More women than men are affected. ECG based localization of the site of PVC is given below.[9]

V1 showing predominantly negative QRS complex (LBBB pattern) (Fig. 1)

V1 showing predominantly positive QRS complex (RBBB pattern) (Fig. 2)

Other rarer sites of origin for PVCs in the absence of structural heart disease include the tricuspid annulus, mitral annulus, papillary muscles, ventricular tissue adjacent to aortomitral continuity, and other Purkinje adjacent structures such as left ventricular false tendons.

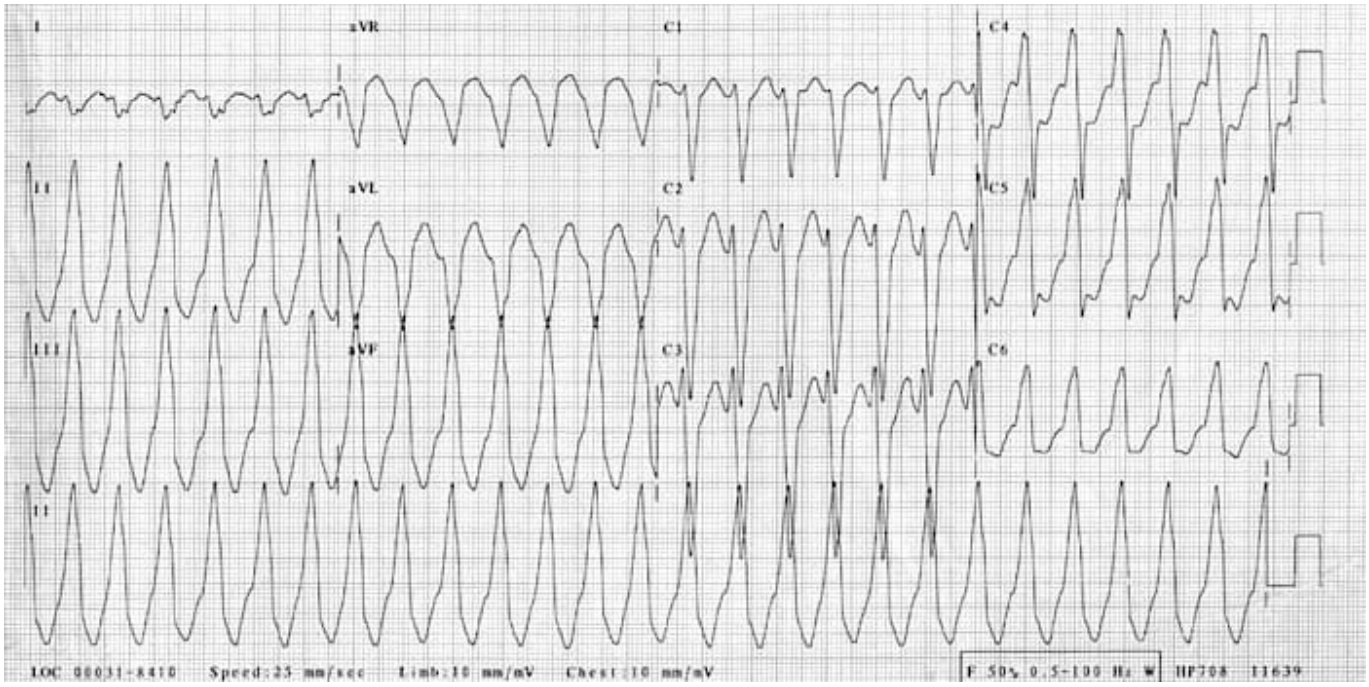

Figure 1: Wide QRS Tachycardia, LBBB morphology (rQS in V1 and RR' in V6) and Right Axis (Positive QRS in III and aVF) suggest Right Ventricular Outflow Tract VT (RVOT VT) (*Courtesy Internet ECG Library)*

Table 1: ECG Localization of Outflow Tract Ventricular Tachycardia

Inferior leads	aVR , aVL	Precordial transition	Site of PVC
Positive	Negative	At or beyond V4	RVOT
		At V3	RVOT or LVOT
		At V1-V2	Left sided, often from right or left coronary cusps
Negative	Both or either one positive		Rule out arrhythmogenic right ventricular dysplasia, sarcoidosis or infiltrative diseases

Ambulatory monitoring

Holter monitoring is used to determine the burden of PVCs and to detect any tachyarrhythmia. It also would help to assess the morphology of PVC – unifocal or multifocal and the relationship of VPCs to the symptoms. If a single Holter monitoring fails to give the needed information, a 30-day ambulatory event recorder, also called as wearable loop recorder could be ordered. In general, if the PVCs are more than 15 to 25% of total cardiac beats, it is considered as high PVC burden. Even if they have no symptoms or only minimal symptoms, they have to be followed up because of the possibility of developing cardiomyopathy later.[10]

Echocardiography

Echocardiography has to be ordered for all as it is imperative to rule out structural heart disease in every patient with PVCs. Left and right ventricular dilatation or dysfunction, evidence of myocardial scar, valvular heart disease or any other structural disease need to be ruled out.

Exercise testing

If the patient's symptoms occur with activity, exercise electrocardiography could be ordered. It is important that it is done under supervision with the availability of defibrillator at the bedside. Exercise testing can be combined with either echocardiographic or nuclear perfusion imaging to evaluate the possibility of myocardial ischemia. Suppression of PVCs with light exercise was associated with benign prognosis in a pediatric population.[11] However, in patients with known or suspected cardiovascular

disease, PVCs during exercise testing or the recovery phase of exercise testing may be associated with an increased risk of cardiac events and mortality .

Cardiac magnetic resonance imaging

Magnetic resonance imaging should be considered if the PVCs are not arising from a common location such as right ventricular outflow tract or if there is history suggestive of sustained ventricular tachycardia or if the left ventricular

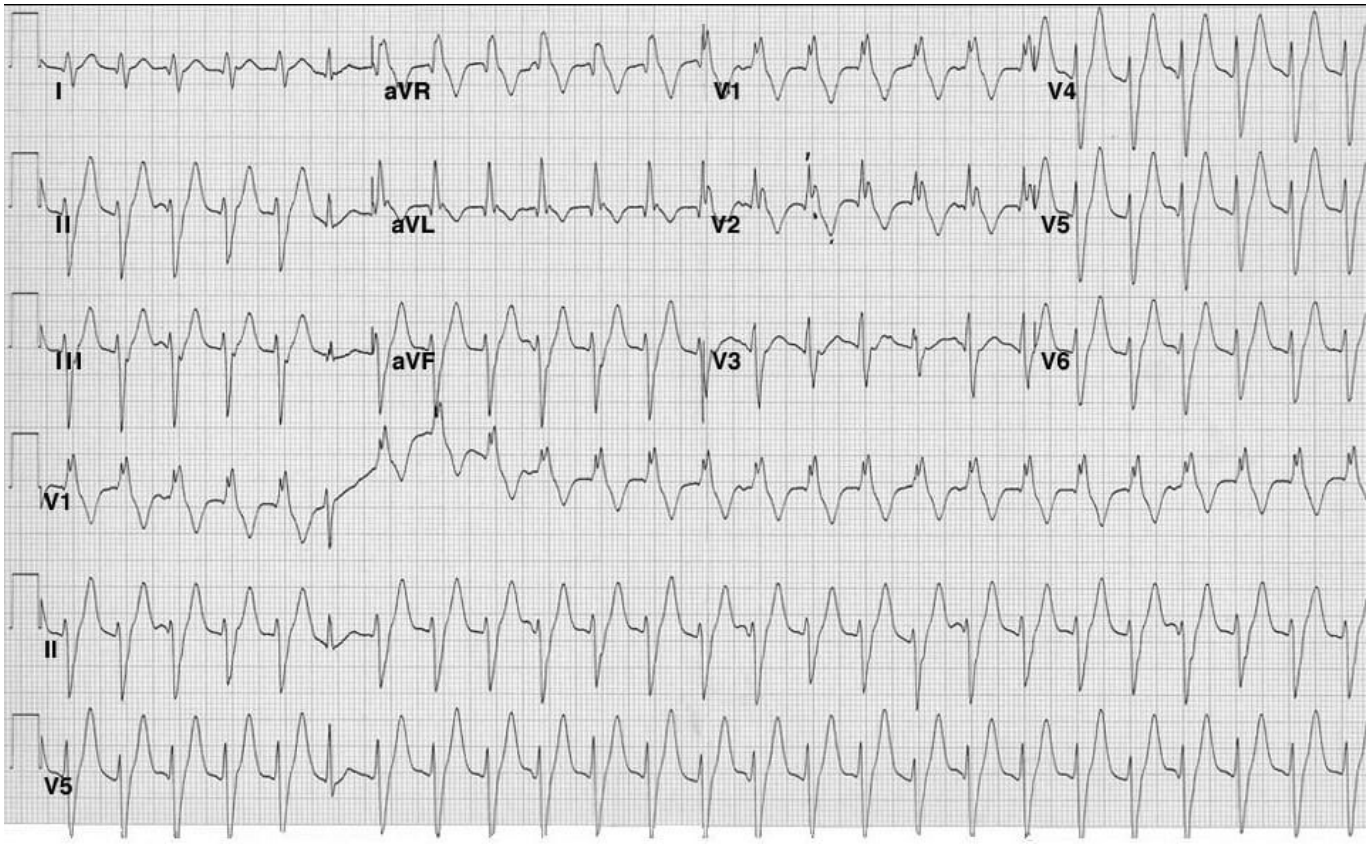

Figure 2: Wide QRS Tachycardia with RBBB morphology (V1 showing an RR' pattern and RS pattern in V6) and left axis (Positive QRS in aVL) indicates an origin from the left posterior fascicle. A sinus capture beat in the sixth QRS in the rhythm strip confirms the VT. *(Courtesy Internet ECG Library)*

Table 2: ECG Recognition of Idiopathic Ventricular Tachycardia

Inferior leads are positive	LVOT origin, from right or left coronary cusps. A small subset arises deeply in a triangle of epicardial tissue underneath the left main coronary artery called as left ventricular summit.
Anterior hemiblock pattern	Posterior fascicle, relatively narrow QRS
Posterior hemiblock pattern	Anterior fascicle, relatively narrow QRS

systolic function is reduced. Late gadolinium enhancement MRI is useful in identifying any form of structural heart disease, microvascular obstruction, myocardial edema or fibrosis. It can distinguish between areas of fibrosis caused by ischemic and non-ischemic cardiomyopathy. It would provide information regarding cardiac sarcoidosis or amyloidosis. In selected patients, positron emission tomography scan would be helpful in assessing infiltrative and inflammatory processes.

Treatment

In the absence of symptoms and structural heart disease, no treatment is needed, and reassurance is recommended. There is no evidence that suppression of PVCs with medications in this situation improves mortality.[12,13] No drug has been approved by US FDA for treating PVCs or non-sustained ventricular tachycardia.

In the presence of symptoms, treatable causes should be addressed initially. Avoidance of tobacco, alcohol, caffeine, and anxiety may reduce the burden of PVCs. Daily magnesium supplementation with potassium supplementation has also been shown in a randomized clinical trial to decrease the occurrence of PVCs.[5] Enhancing physical activity also can mitigate PVC frequency . A Beta blocker or non-dihydropyridine calcium channel blockers could be started at a low dose and titrated until symptoms are alleviated. If unsuccessful, amiodarone can also be considered. In the presence of frequent symptomatic, predominantly monomorphic PVCs that are drug resistant or drug intolerant, catheter ablation is recommended (Class II A, level of evidence C).[14,15]

Summary

PVCs are frequently encountered clinically. The can occur with or without underlying structural heart disease. Without symptoms or structural heart disease, echocardiography and a 24-hour Holter monitoring would suffice. Underlying structural, electrical, or ischemic diseases; very frequent or complex PVCs, such as couplets, triplets, and non-sustained

VT; multifocal PVCs; an increasing number of PVCs during exercise; non-outflow tract PVCs and non-left ventricular (LV) fascicular PVCs; short coupling intervals; and wider PVCs indicate a potentially non-benign character in a PVC and need further evaluation. Medical management includes betablockers and non-dihydropyridine calcium antagonists, rarely amiodarone and other antiarrhythmic drugs are used. When medical treatment is ineffective or not tolerated, catheter ablation of the PVC is the final option.

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When is ST Elevation not an MI

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Introduction

In today's era of early reperfusion therapy for acute myocardial infarction (AMI), the decision to start thrombolysis or activate cath lab for primary PCI in a patient with chest pain rests on the recognition of ST elevation on a 12 lead ECG. However, approximately a significant percentage of patients with ST segment elevation on the presenting ECG, ultimately are found to not be associated with STEMI.[1] In certain conditions, a patient's ECG can resemble STEMI, yet manifest ST segment elevation from a non–acute coronary syndrome (ACS) entity, the so-called STEMI mimics. In other situations, the patient's ECG makes it difficult or impossible to determine whether STEMI is present, the so-called STEMI confounders; these confounders to STEMI diagnosis are also mimickers of AMI. Failure to recognize these mimics can lead to inappropriate use of resources, exposure of patients to unnecessary risk, and increased rather than decreased morbidity and mortality. The purpose of this review is to describe other conditions that mimic infarction and emphasize the electrocardiographic clues that can be used to differentiate them from true infarction.[2]

Stemi Mimicking Patterns

1. Normal ST segment elevation

   In a study of normal electrocardiograms from 529 men, the prevalence of ST-segment elevation of at least 1 mm in one or more of leads V1 through V4 was 93 percent in the men who were 17 to 24 years old and this prevalence declined gradually with increasing age.[3] Since the majority of men have ST elevation of 1 mm or more in precordial leads, it is a normal finding, not a normal variant, and is designated as a male pattern; ST elevation of less than 1 mm is designated as a female pattern. In these patterns, the ST segment is concave, and directly proportional to depth of S wave.[4]

2. Normal variant ST elevation

   Two entities have been described as normal variants:

   a. Early Repolarization (Also Known as Benign Early Repolarization)

      Benign early repolarization (BER) is a normal variant electrocardiographic pattern; it manifests as ST segment elevation at the J point. The electrocardiographic description of BER focuses on ST segment elevation with the following associated features: ST segment elevation with an upward concavity of its initial portion, notching or slurring of the terminal QRS complex (the J point), prominent T waves that are symmetric and concordant with the QRS complex (except in leads V1-V2) widespread distribution of ST segment elevation, and relative temporal stability of the pattern. The ST segment and T-wave abnormalities of BER are most often seen in leads V1 to V4. At times, coexistent changes are also seen in leads II, III, AVF, V5, and V6. BER-related changes noted only in the limb leads are unusual and likely result from some other pathologic process, such as STEMI.

   b. In some young black men, the ST segment is elevated in the midprecordial leads in combination with a T-wave inversion11,12 as a normal variant. This entity has been surmised as a combination of an early-repolarization pattern and a persistent juvenile T-wave pattern. Often, it is difficult to differentiate it from acute myocardial infarction and an echocardiogram is necessary to make a distinction. In most cases of this normal variant, the QT interval is short, whereas it is not short in acute infarction or pericarditis. This normal variant differs from the early-repolarization pattern in that the T waves are inverted and the ST segment tends to be coved.[5]

3. Myocarditis and Myopericarditis

   Seventy-three percent of patients diagnosed with acute myopericarditis will have ST segment elevation on initial ECG. The evolution of ST/T wave changes occurs less rapidly than that seen with STEMI; thus, serial ECGs, which demonstrate changes in the ST segment and T-wave morphologies over minutes, are frequently useful in making the distinction. ST segment elevation in multiple coronary distributions, especially when seen in patients who are clinically stable, favours inflammation over STEMI. The presence of PR segment depression n leads with ST segment elevation as well as PR segment elevation in lead AVR favours inflammation over STEMI.

4. Previous Infarction with Ventricular Aneurysm

   Approximately one-quarter of patients with persistent ST segment elevation following STEMI will have an LV aneurysm as determined by echocardiography. The most frequent electrocardiographic manifestation of ventricular aneurysm is ST segment elevation, most often in the anterior distribution; inferior and lateral aneurysms are also encountered. Pathologic Q waves are usually observed in leads with ST segment elevation. Inverted T

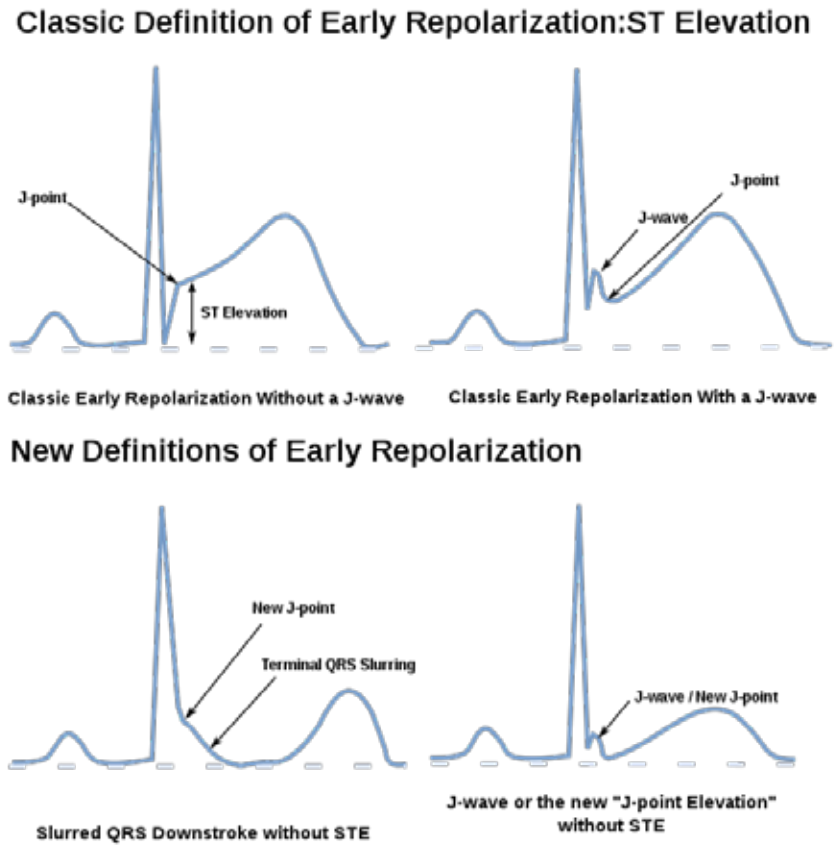

Figure 1: Diagram of Benign Early Repolarisation. Adapted from Froelicher et al, Wikipedia

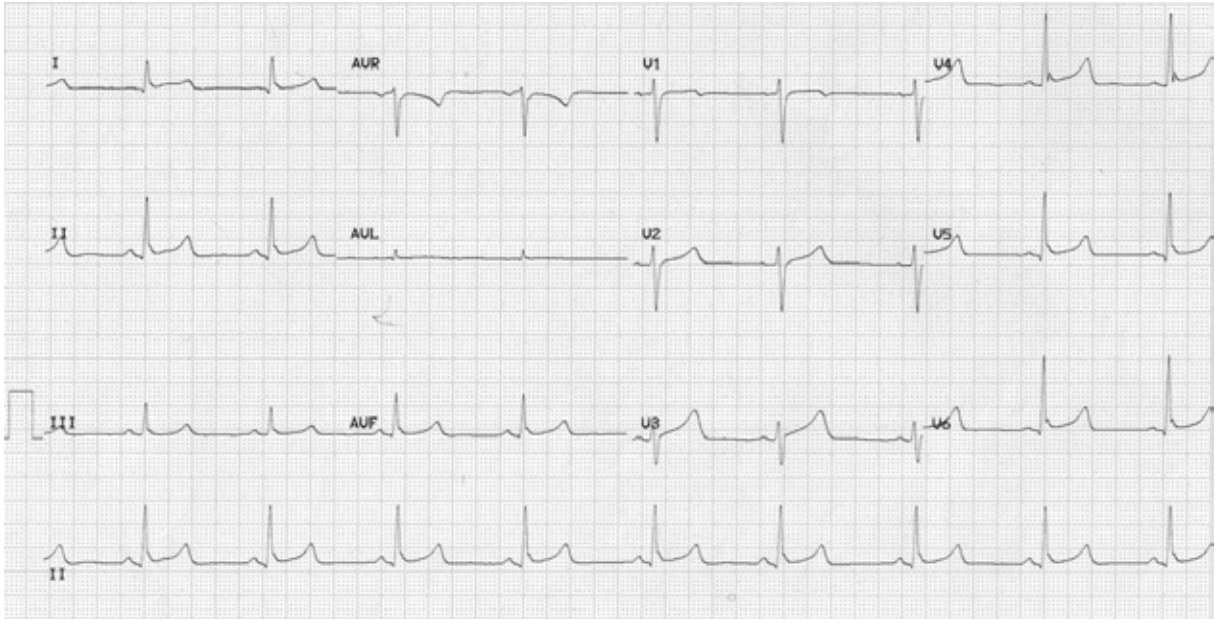

Figure 2: ECG of Benign Early Repolarisation. Picture courtesy of ECG Library

waves of minimal magnitude are also seen in these same leads. Reciprocal ST segment depression is usually not present and this is helpful in distinguishing aneurysm from true STEMI.

5. Coronary Vasospasm

   Because acute coronary vasospasm can lead to near complete cessation of blood flow in the affected coronary artery, its presentation and pathophysiology parallels

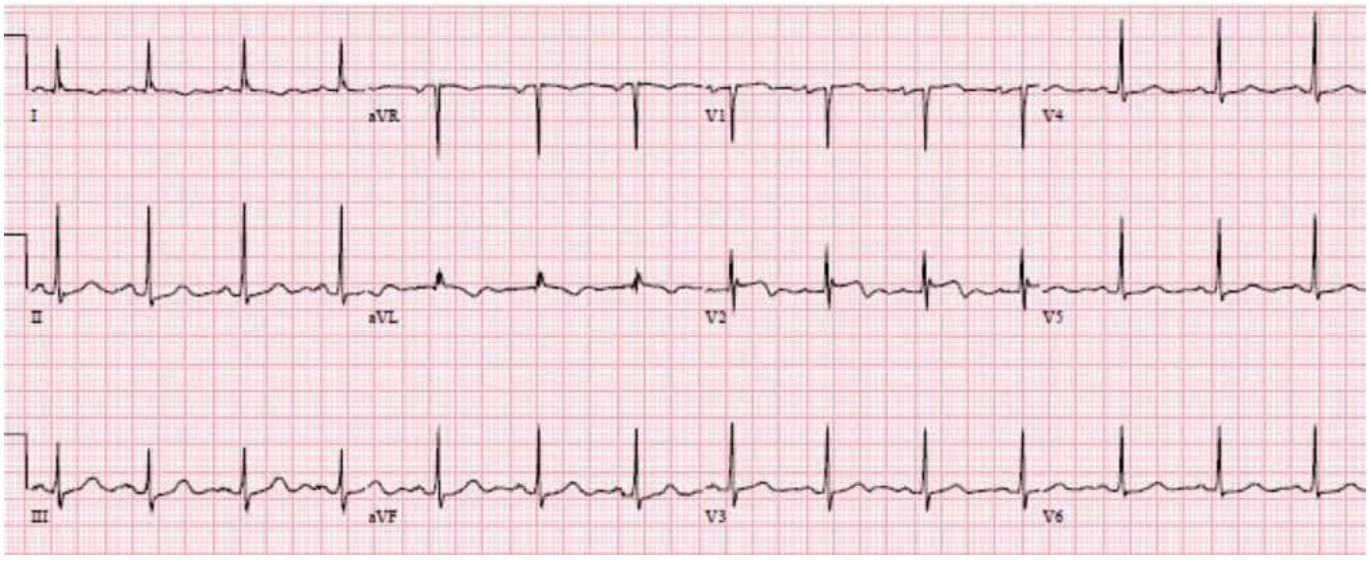

Figure 3: Early repolarisation with T inversion in mid precordial leads. Courtesy of EM Cases Summit

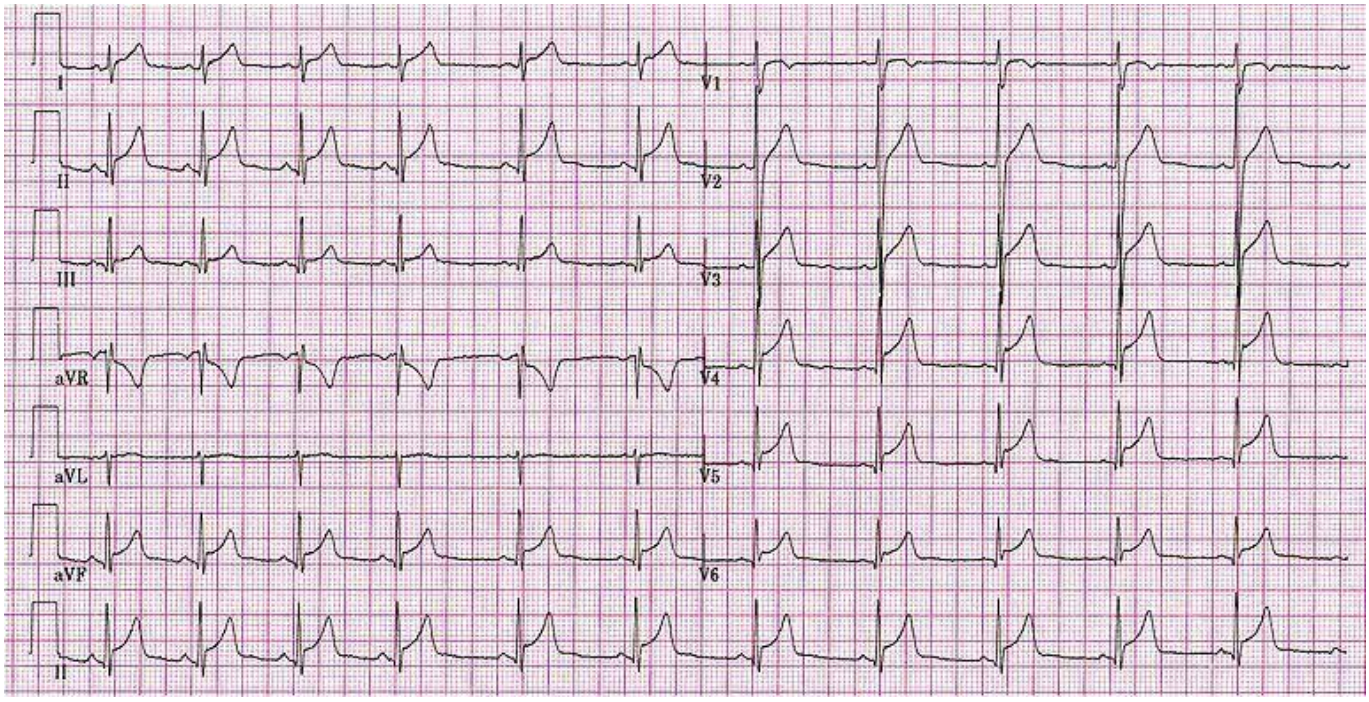

Figure 4: Myocarditis with PR sement depression in L II. Courtesy Wikidoc

STEMI with the important distinction of absent vessel thrombosis. This form of ST segment elevation is indistinguishable from that seen in STEMI on ECG.

6. Takotsubo cardiomyopathy

   The name and classic presentation involve dyskinesis at the LV apex, multiple variations, including basal and mid-cavitary dyskinesis, have also been reported. The clinical presentation of ABS is frequently indistinguishable from ACS and is well known to mimic STEMI. The spectrum of ECG findings is likewise varied, from minimal abnormality to profound anterior ST segment elevation. In its most common apical form, the ECG changes are limited to the anterior precordial leads, reflecting apical location of injury. The inferior leads can also be involved, reflecting inferoapical injury.

7. Brugada syndrome

   Brugada syndrome was first described as a clinical entity in 1992 to explain the observation that a particular cohort of patients prone to ventricular fibrillation despite structurally normal hearts had a distinct ECG pattern. The Brugada pattern includes right bundle branch block (RBBB) (both incomplete and complete) with ST seg-

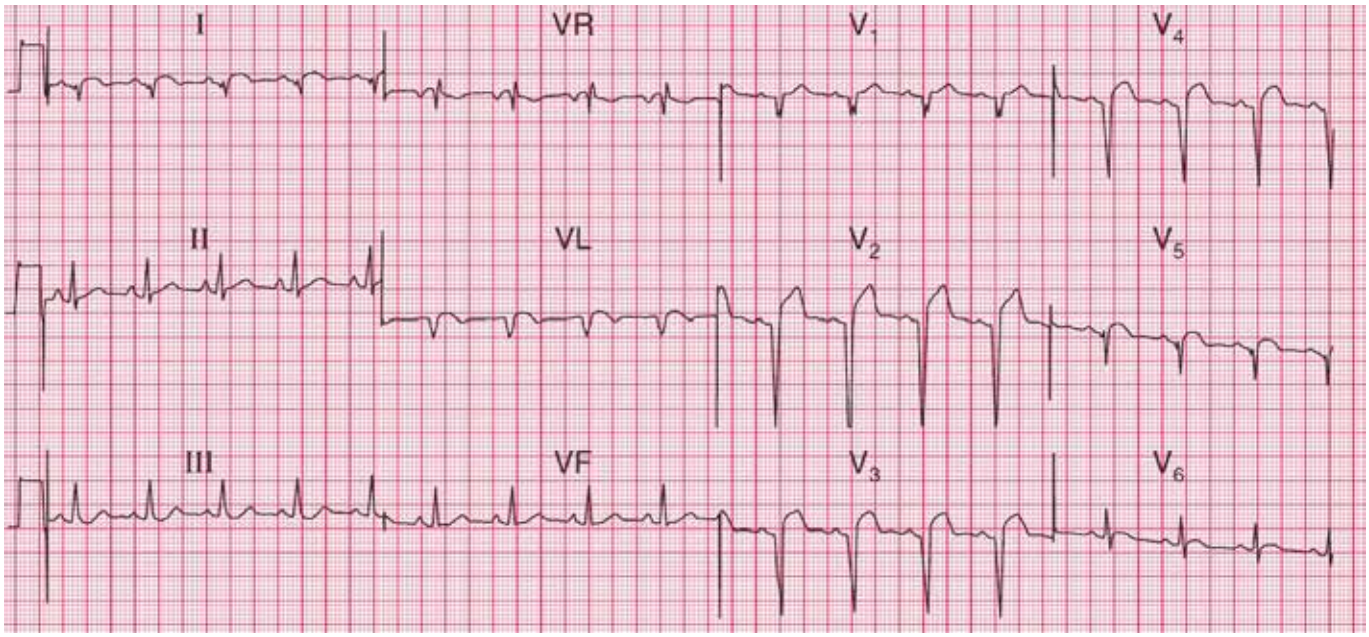

Figure 5: Anterolateral MI with Aneurysm. Courtesy Manual of Medicine

ment elevation in leads V1 to V3.

8. Hyperkalemia

   The elevation of the ST segment occurs following peaking of the T wave and widening of the QRS complex, suggesting high serum potassium concentrations. Although the accompanying ST segment elevation may be most impressive in a single region, the ECG changes of hyperkalemia are present throughout the limb and precordial leads.

9. Postelectrical cardioversion/defibrillation

   ST segment elevation has been noted following both cardioversion for atrial as well as ventricular tachyarrhythmias in up to 20% of patients. The ST segment elevation is transient and resolves within minutes but can be profound, as much as 5 mm. Although impressive and alarming, this finding has not been associated with evidence of ongoing myocardial injury or additional adverse sequelae.

10. Hypothermia and Osborn waves

    Prominent J-point elevations, also known as Osborn waves, are a common finding in patients who are hypothermic. When profound, the ST segment can be elevated, mimicking STEMI. Other findings include bradycardia and motion artifact. The mechanism and clinical implications of the Osborn wave remain unclear, but it is a transient finding that resolves with normothermia.

Stemi Confounding Patterns

These patterns include entities that markedly reduce the electrocardiogram's ability to detect changes related to ACS, because these conditions obscure the detection of ST segment elevation. These are briefly described below:

1. LBBB

   The LBBB pattern on the ECG includes an elevated ST segment at baseline making it impossible to use the standard STEMI criteria for ST segment elevation. An understanding of the ECG in the LBBB presentation is important. Such an understanding allows the clinician to recognize the expected findings of LBBB and, thus, the inappropriate findings that are associated with AMI. In particular, loss of the normal QRS complex–T-wave axes discordance in patients with LBBB may imply an acute process, such as AMI. Patients with known LBBB presenting with a clinical presentation potentially suggestive of AMI is the most appropriate application of the criteria Sgarbossa and colleagues derived from the GUSTO trial.[6]

   The ECG criteria suggesting a diagnosis of AMI, ranked with a scoring system based on the probability of such a diagnosis, include (1) ST segment elevation greater than 1 mm, which was concordant with the QRS complex (score of 5); (2) ST segment depression greater than 1 mm in leads V1, V2, or V3 (score of 3); and (3) ST segment elevation greater than 5 mm, which is discordant with the QRS complex (score of 2). A total score of 3 or more suggests that patients are likely experiencing an AMI based on the ECG criteria. The Sgarbossa criteria suffer from low sensitivity of 36% while retaining a relatively robust specificity of 96% in the validation cohort.

2. LV Hypertrophy

   Approximately 80% of patients with the LVH by voltage pattern demonstrate the strain pattern; the strain pat-

tern includes significant ST segment changes (elevation and depression) and T-wave abnormalities (prominent T waves and T-wave inversion). ST segment elevation in the setting of LVH is almost exclusively seen in the anterior distribution (leads V1 to V4), along with prominent T waves; the ST segment elevation can reach up to 5 mm in height in the anterior leads. The lateral leads (leads I, aVL, V5, and V6) demonstrate large, prominent, positively oriented QRS complexes with marked ST segment depression and T-wave inversion. Although the strain pattern can also be seen inferiorly, inferior ST segment elevation should not quickly be ascribed to LVH. The LVH with pattern is associated with poor R-wave progression, most commonly producing a QS pattern; these complexes are located in leads V1, V2, and V3; furthermore, a leftward axis and left atrial abnormality add credence to ST segment elevation stemming from LVH. The ST segment elevation associated with LVH is generally unchanging over time, making a previous ECG for comparison particularly useful.

3. Ventricular Paced Pattern

Because a paced ventricular rhythm causes bundle-branch block morphology, it produces many of the same diagnostic limitations as LBBB described above. Ventricular pacing leads can be placed in the apex of the right ventricle causing a left bundle pattern or on the surface of the LV either in a coronary vein or in an epicardial location causing more of a RBBB type of pattern. Applying the Sgarbossa criteria to patients with an LBBB caused by paced rhythm yields poor sensitivity and modest specificity; in a very basic sense, similar findings are similar to those noted in the ECG diagnosis of AMI in the setting of LBBB. the longstanding maxim has been the presence of RBBB does not alter the detection of ST elevation. However, this notion has been challenged by those who point out T-wave inversions in the anterior leads of patients with RBBB can impair the detection of more subtle ST segment changes; also, severe proximal coronary occlusions can be associated with new RBBB with hemiblock and, thus, do not demonstrate typical ST segment elevation.[7]

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ECG from Wearables

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Introduction

Chest discomfort and cardiac related symptoms are the second most cause for an emergency room visit with figures in the USA being around 4.9% to 5.3% over a period of 2013 to 2019. Hence the ECG is an important tool to diagnose the condition. The American Heart Association and the American College of Cardiology have recommended that an ECG should be taken within 10 minutes of a patient arriving at the emergency room (ER).[1] However this has been possible only in 34-40.9% in a study across eight centres across USA and Canada, although an improvement was seen from 16-64% when an ER study used the ECG to triage the patients soon after presentation.[2] Wearable devices seek to reduce this time interval as the patient has ready access to the ECG and with mobile technology can communicate the ECG to the health care worker instantaneously.

Approved Devices

1. **Alive Cor**, is an FDA and CE approved device made by Alive Cor Kardia Mobile (KM). It uses a smarphone app that records and send to the health care worker a single lead ECG when the subject presses the device buttons with fingers. The KM has been found in studies to have twice more sensitivity in recognising arrhythmias than conventional cardiac monitoring care in patients with palpitations.[3] In the acute care setting the KM device has a five fold to eleven fold increase in the diagnostic capability and reduced the mean time to rhythm detection in symptomatic patients.[4]

2. **Apple Watch** uses a photoplethysmography sensor to identify irregular heart beats to detect atrial fibrillation and flutter and generate a single lead ECG through its application. Apple watch 4 can be adjusted to the six positions and provide an ECG with leads I, II and III and the precordial chest leads with comparable quality.[5] This application can identify STEMI in select patients where it can be identified by the limb leads. (Fig 1 and 2)

3. **Zio Patch** is a water resistant adhesive patch that can be worn over the left pectoral region and can send information to the Zio Patch for 14 days that is comparable to standard Holter monitoring. The diagnostic yield was much better with this compared to the Holter in unexplained syncope.[6]

4. **ECG Check** device is a single lead ECG monitoring device available over the counter and is useful for follow-up of atrial fibrillation ablation patients to detect recurrences with a sensitivity of 100% and specificity of 96% in detecting Atrial flutter and fibrillation, reduces OP visits and is useful in the prevention of stroke in AF patients[7].

Non-Approved Devices

Cardio Secur is a four electrode device CE approved that can transmit a 22 lead ECG that can identify STEMI with comparable sensitivity and specificity to a conventional 12 lead ECG and has significantly reduced the time to diagnosis of an infarction[8]

Some other devices are the Zenicor EKG 2 which transmits the ECGs when the thumbs press on two sensors for 15 seconds, the My Diagnostick that registers and analyses single lead ECGs after holding with both hangs for a minute and the wearble ECG west are all used for arrhythmia detection particularly atrial fibrillation detection[9]

Drawbacks of the Wearable ECG Recordings

The accuracy of arrhythmia detection depends on the patient wearing it continuously as the program application is done to detect the average of a certain number of recordings and this might fail if not worn as recommended. Failure of elderly to correctly apply the electrodes and to correctly wear the device is a point of concern as well the exposure to unnecessary marketing by device companies that may be faced by the at concern population.

Conclusions

The wearable ECG devices are the promise of the future and show promise in making available to patients an easily available tool in their hands for the detection of arrhythmias and cautioning them when to attend to the ER of the hospital at an earlier time than might be possible with standard monitoring and ECG recognition devices. With the smartphones that are available at present the possibility of an ECG being taken as soon as a person experiences a symptom and its transmission to a health care worker or center will ensure earlier care for the patient.

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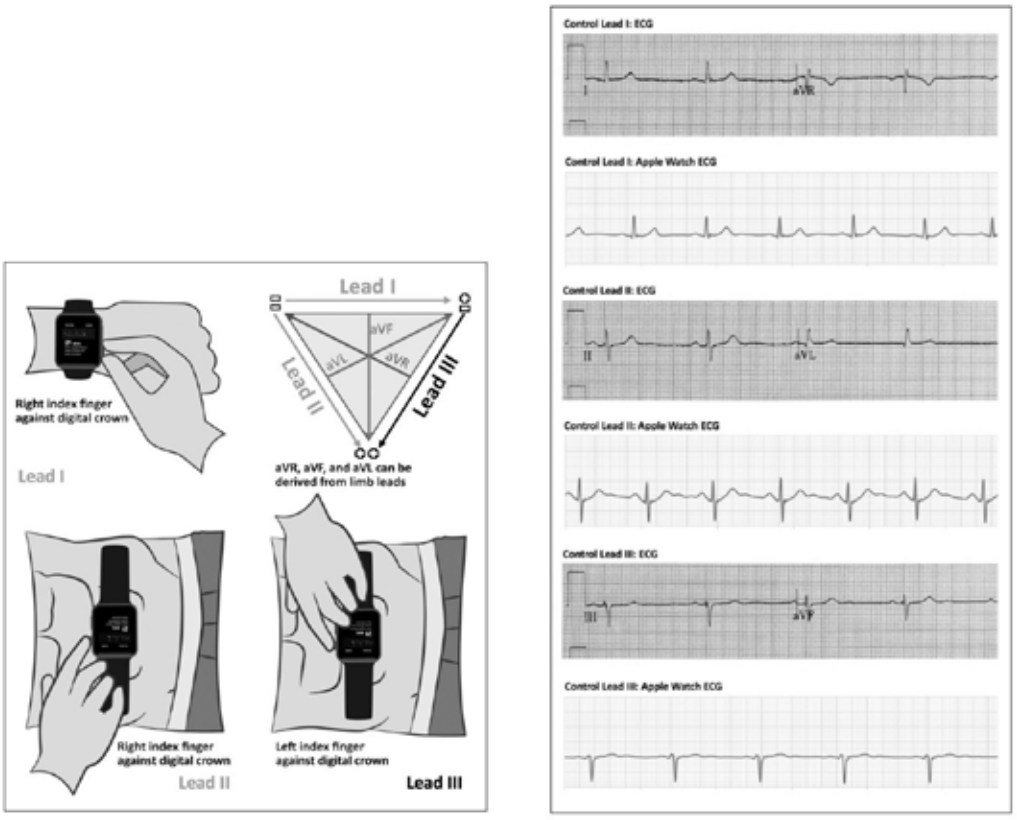

Figure 1: Left Panel Method of taking Lead I, II and III ECGs by an Apple 4 Watch. Right panel showing ECG leads I, II and III with those taken from a standard ECG machine

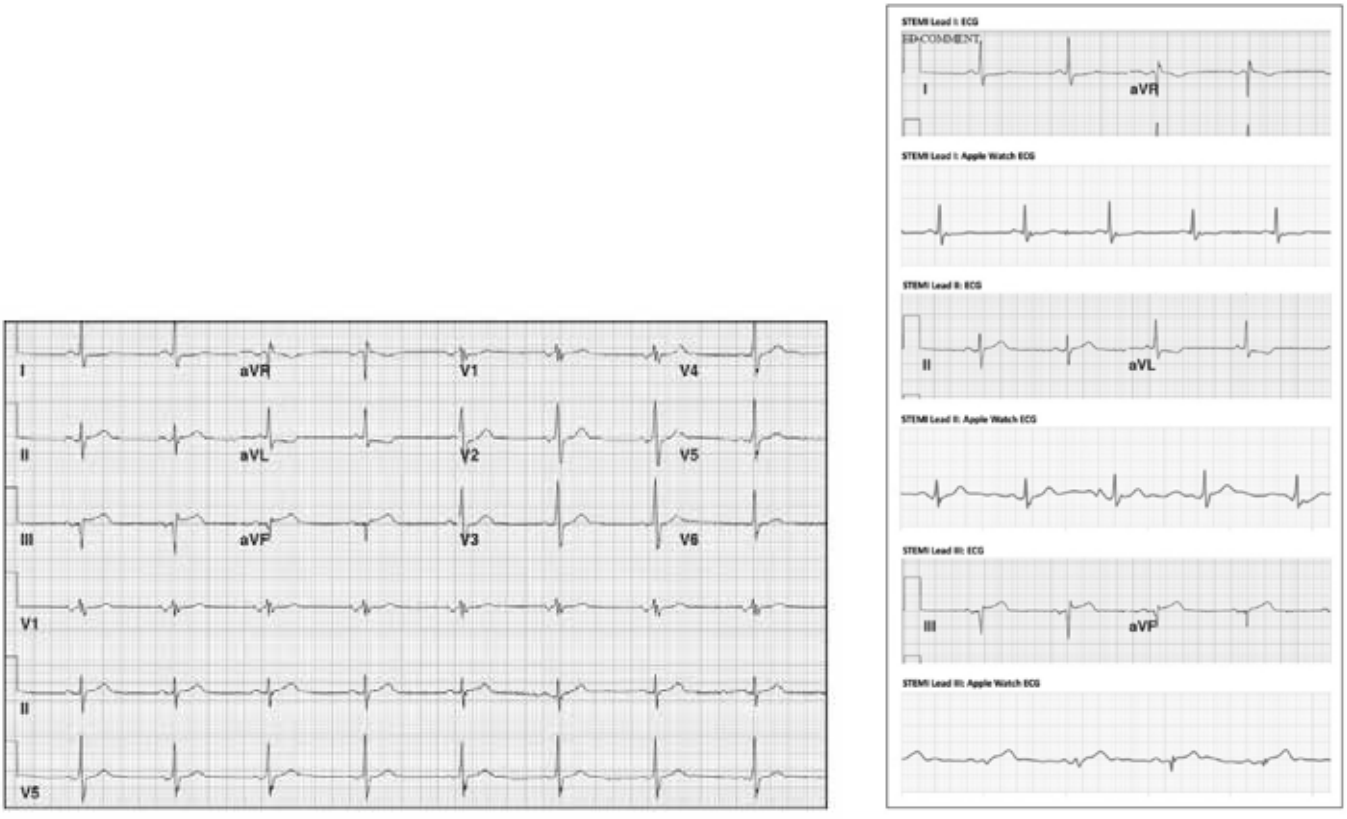

Figure 2: Top panel Standard 12 lead ECG of a 52 year old person with Myocardial Infarction and Bottom Panel showing the Apple 4 Watch ECG with the corresponding standard ECG

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NOTES

INDIAN SOCIETY OF ELECTROCARDIOLOGY

APPLICATION FORM FOR LIFE MEMBERSHIP/FELLOWSHIP

SECRETARIAT
Prof. Dr. Ketan K. Mehta
Indian Society of Electrocardiology
Health Harmony, 2-Dattani Chambers, S V Road, Malad (W), Mumbai 400064
Mobile : 91-98200 51849 • e-mail : drketanmehta@yahoo.com • www.iseindia.org

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. 2360/- (Rs. 2000/- Membership Fees + 18% GST Rs. 360) 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 _____

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Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No.

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Hon. Secretary, ISE

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

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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 self-attested **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/- + 18% GST Rs.360/-. Tota Rs. 2360/- 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
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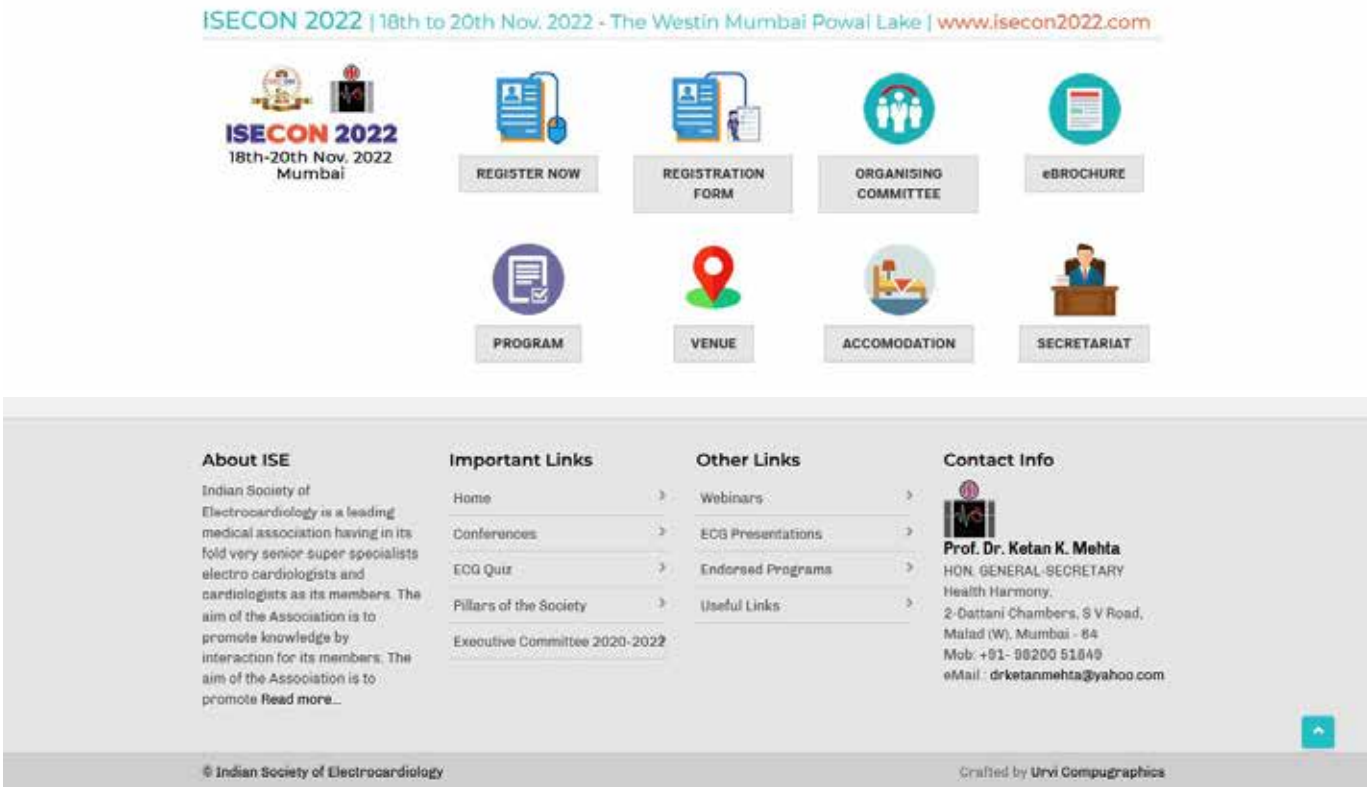

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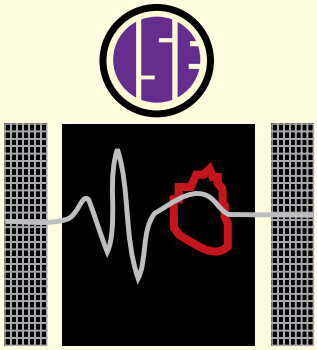

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