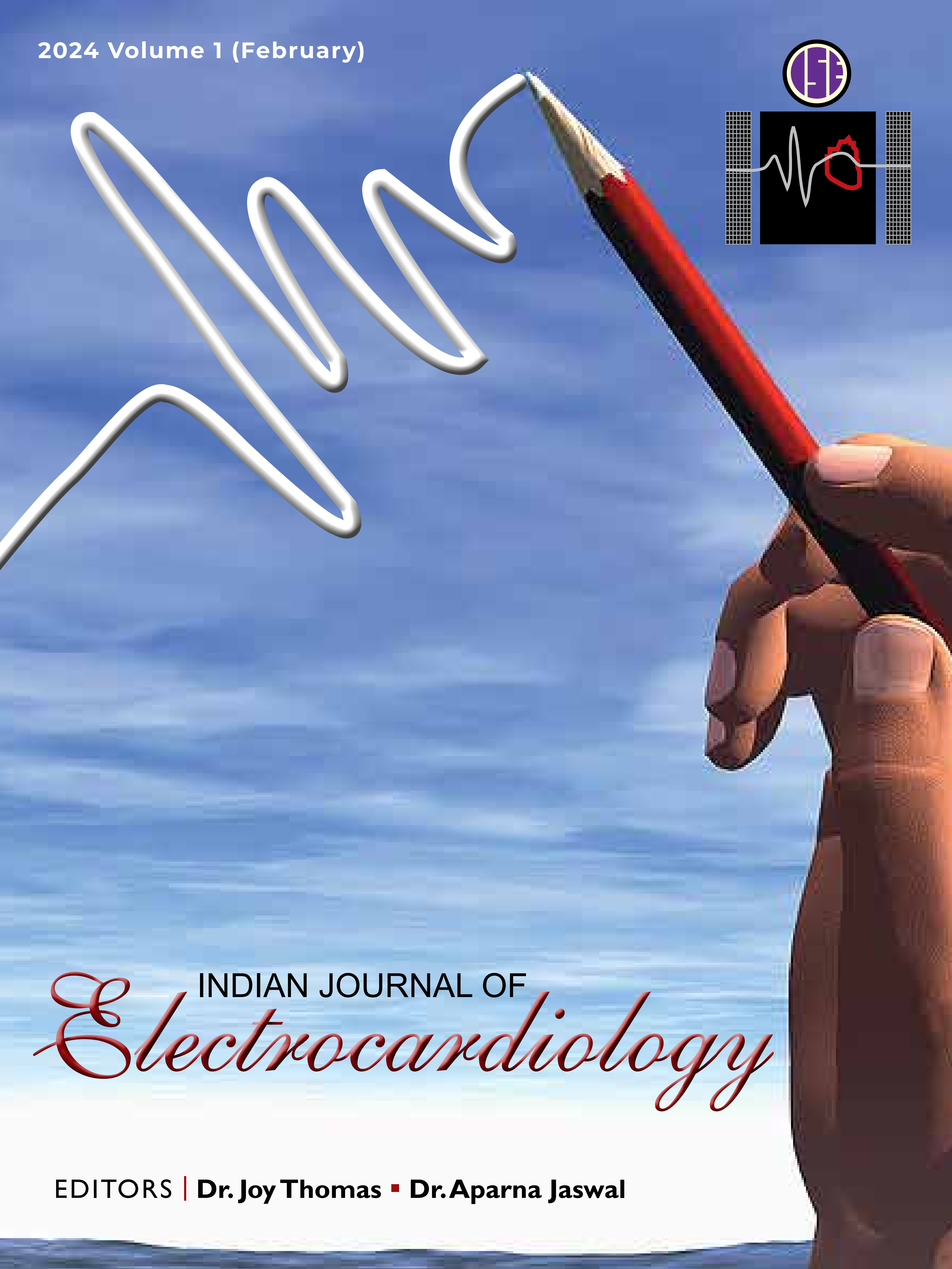
2024 Volume 1 (February)



INDIAN JOURNAL OF Electrocardiology

EDITORS | Dr. Joy Thomas ▪ Dr. Aparna Jaswal

UPCOMING EVENTS

ISECON Mid-Term Conference 2024

September • Amritsar

ISECON 2025

February • Kolkata

ISECON Mid-Term Conference 2025

August • Srinagar

ISECON Mid-Term Conference 2026

September • Bhuvaneshwar

ISECON 2027

February • Lucknow

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SECRETARIAT
Ketan K. Mehta
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Editorial

It has been two years since Dr Aparna and I have been given the honourable task of bringing out the editions of the Indian Journal of Electrocardiology of the Indian Society of Electrocardiology, starting with the Golden Jubilee edition in 2022, the Mid term edition of 2023 coinciding with the meeting at Sholapur, and now the annual meeting edition to time with the annual meeting at Ahmedabad in February 2024. The current edition sought to cover topics that were hitherto untouched.

The ECG in children can sometimes be intimidating to the uninitiated. Drs Patnaik et al have very succinctly with wonderful illustrations given a clear understanding of which ECG in children can be branded as not being normal and worthy of further attention.

The treadmill test is often times included in the Master Health package of many insitutions and the reporting has sometimes baffled clinicians. Dr Jayaprakash Shenthar, a keen teacher, has taken great pains in bringing out the salient features of abnormal versus normal ECG during an exercise test. Hopefully this will help to bring in more correctness to treadmill test reports and interpretation.

The emergency is where errors have to be minimal to provide better care to the suffering. Drs. Ajith Kumar et al have brought out the ECGs that can confront us in the ER and have also covered the unusual presentations that lead to an ER admission.

Premature ventricular complexes (PVC) are worrisome. How to figure out which are the cases that need greater attention and workup and specialized treatment including interventional electrophysiology procedures like Radiofrequency ablation using 3 D Mapping systems have been discussed by Dr. Daljeeth Saggu

Commonest cause of low heart heart rate with symptoms of giddiness and syncope is the sinus node dysfunction in its various avatars and Dr. SB Gupta has brought out the features succinctly in a wonderful article.

The long QT besides its hereditary origins is seen more often these days as a result of drug effects and interaction. Its various features has been discussed very well by Dr. K. Jaishankar with neat diagrams that are self explanatory.

The ECG with a normal rhythm but with a wide QRS has been dealt with by Dr Poppy Bala and sums up the various situations that can present with a wide QRS.

Atrial fibrillation the commonest tachy-arrhythmia keeps recurring in a patient who already has had an episode or episodes like the famous adage- “AF begets AF”. Dr. Deepak Padmanabhan et al. has provided with enough scientific data to guide one in the choice of further management of these vexatious cases.

We are grateful to the leadership and the executive committee of the Indian Society of Electrocardiology for the confidence reposed in us and we hope that we have justified it in at least a small measure.

Dr. Joy M Thomas **Dr. Aparna Jaswal**

From the Desk of Advisor

Dear Members,

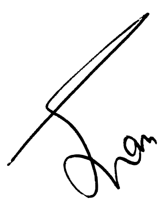
It is indeed a great pleasure that Indian Journal of Electrocardiology, the Official Journal of Indian Society of Electrocardiology is becoming a regular feature. All the credit goes to our esteemed editors, Dr Joy Thomas and Dr Aparna Jaswal, who have taken great pains to bring IJE regularly and having contents worth reading

Current issue of Indian Journal of Electrocardiology has very useful articles for practicing physicians, who will enjoy reading them and be benefitted,

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

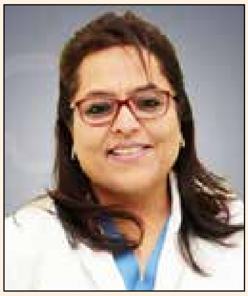
My heartfelt thanks to the Journal Editors, Dr Joy Thomas and Dr Aparna Jaswal for their methodical planning and execution of the work to bring the IJE February 2024 issue in time and an academic treasure to read and keep for future reference.

Long Live ISE.



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

From the President’s Desk



Year 2020 -An unforgettable Year in the Calendar of our Lives because of Covid -19 pandemic. To hate it and to wish it away, would mean that we are letting go of the opportunity to admit the crucial lessons it taught us. To name a few they will be – to know that work will go on with or without you, there is life in stillness too, time moves on, no one’s indispensable, family is more important than work & career and above all 2020 taught us that we have all become a part of "Rat Race" which was halted within minutes by a "filament" of RNA, non-visible to naked eye. It also taught us patience, empathy, sharing, caring, living with minimum- realizing we don’t need soo much.

We are survivor of challenges, we thrived through the worst, we healed from losses and we have adapted like never before. These are the great lessons from the great teacher - Year 2020.

A change of date or year will not make all the problems vanish, it will change when our mind set changes. We lookback at 2020 with utmost respect and honor. Most importantly offer immense gratitude, as it is the gratitude that puts us in place of abundance and shifts our focus on what we have, rather than what we don’t have. We have to accept the fact that we have evolved and matured and grown at another level altogether within just a year.

As a doctor the era of Covid has made me adapt changes like never before. I understood the importance of use of technology in order to reach out to my patients, to be connected to them, to diagnose and treat them. Instead of in-person clinic, I learned to make a diagnosis with video/tele consult. The diagnostic equipment of pulse oximeter, digital BP apparatus, glucometer made their household presence. The most important diagnostic equipment which found its place in clinical practice are single lead ECG, Sanketlife and Apple watch. These devices have a huge place in clinical cardiac practice, alongwith setting up Remote/Home Monitoring for patients implanted with Cardiac devices. Remote Monitoring is a way for the patients implanted with heart device to communicate with doctor’s clinic using a small monitor, potentially reducing the number of times of travel to doctor’s clinic for a check of the implanted device.

Another Slogan is a campaign for prevention of Sudden Cardiac Arrest (SCA) which is a life-threatening emergency that occurs when the heart suddenly stops beating. As a result, blood no longer pumps throughout the body, including the brain. The person suddenly passes out, loses consciousness and appears lifeless. It strikes people of all ages who may seem to be healthy, even children. SCA leads to death if no action is taken within the first 6 minutes.

We have talked a lot about CAD and heart attack for years but no one has thought to discuss the more fatal, more dangerous condition of SCA.

It’s a common misconception that SCA and Heart Attack are the same thing. While a Heart Attack is described as a “plumbing problem,” SCA is more of an “electrical problem” that stops the heart. >90% patients survive the heart attack, >90% will not survive SCA.

Unfortunately, less than 1/3rd of SCA victims receive Cardio Pulmonary Resuscitation (CPR) from bystanders and fewer than 5% of victims are treated with automated external defibrillators (AEDs) before Emergency Medical Services arrive at the scene. But for every minute that passes without CPR and defibrillation, the chances of survival decrease by 7-10%. As a result, hardly 10% of victims survive. Yet survival rates could triple if more people knew what to do when SCA strikes. In fact, tens of thousands of additional lives could be saved each year if bystanders acted quickly.

Why don’t more people know and use these fundamental lifesaving skills? It is soo wonderful to Save A Life. Well, my aim is to make “SCA awareness” a campaign and spread the learning about CPR and importance of having AED at all places where there are fire extinguishers.

Jai Hind

Dr. Vanita Arora
President
Indian Society of Electrocardiology

ECG in Children - When is it Abnormal ?

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Introduction

Thorough clinical examination, electrogram (ECG) and echocardiogram are the three important pillars of pediatric cardiology. ECG plays a key role in diagnosing congenital heart diseases, cardiac arrhythmias and in evaluating the symptoms like breathlessness, chest pain and syncope. ECG should never be interpreted in isolation; it should always be correlated clinically. There are certain challenges in performing and interpreting ECGs in infants and children. Young children hardly stay motionless, producing artifacts that could make comprehension more difficult. Sometimes we have to use additional leads as detailed below. Even when a good tracing is obtained, deciding whether the ECG falls within acceptable normal limits for the age, sex and physiological status or does it warrant further evaluation is the real challenge.

Lead placement

It is critical to get an artifact-free reading for any ECG, but doing so can be particularly challenging in small children. Hence, steps must be taken to minimize movements by using cartoons, movies, stickers, and bubbles as distractions. The positioning of ECG leads in children may differ from that of adults. Young patients can use a typical 12-lead ECG, but occasionally, additional leads are needed because of anatomical variations. The additional leads consist of two leads on the right side of the chest (V3R midway between V1 and V4R, and V4R at the fifth intercostal space at the midclavicular line) and one on the left side (V7 following V6) (Figure-1).

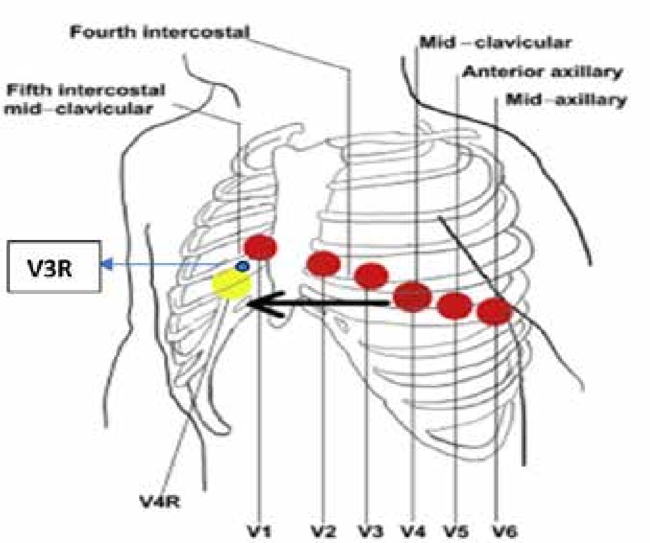

Figure 1: Common positions of lead-placement on the chest

Systematic Approach

Technical aspects**:** One should be aware of the technical aspects of ECG before starting to read the ECG. Correct lead placement is critical to avoid misinterpretation of findings. Knowledge of paper speed is necessary to assess the heart rate and various segment intervals. Standardization and filters are to be adjusted to get a good quality ECG. It is not uncommon to see an ECG where half standardized ECG is used to avoid large voltage complexes overlap each other.

Heart rate: The average heart rate during the neonatal period is about 120 to 180 bpm. It declines gradually with age. In adolescents, the normal range is from 65 to 120 bpm (Table-1).

Rhythm: This is assessed by looking at the relationship of the P wave to the QRS complex, which can be challenging in a pediatric population because children have large P waves and short PR intervals, making it difficult to identify QRS onset. Abnormalities may reveal ectopic atrial rhythm, primary atrial tachycardia, or supraventricular tachycardia. A specialist should be consulted if there is a fast rate, a narrow QRS, and no discernible P wave.

Morphology

P wave morphology is best assessed in lead II and V1. P waves are upright in all limb leads except in aVR. It is upright in all precordial leads and it is biphasic in V1.

QRS morphology: A small q wave is seen in all leads except in V1/V2. Q wave is pathological when it is deep (>2mm) or >25% of the R wave. It is abnormal if it is seen in V1-V3. Normally R waves are smaller in V1 and there is R wave progression till V5/V6.

Axis

P wave axis: The P wave axis is very important in diagnosing some important conditions the situs inversus/dextrocardia, SVC type of sinus venosus type of ASD. P wave axis is also helpful in differentiating atrial tachycardias from other types of supraventricular tachycardias like AVNRT/AVRT.

QRS axis: The normal QRS axis is -30 to 90^0. Determining the QRS axis helps to differentiate TOF-DORV-TGA spectrum. Extreme axis deviation is seen in endocardial cushion defects. Based on the QRS axis focus of ventricular tachycardias can be located.

T wave axis: The T wave axis is usually concordant with the QRS axis. In young children and adolescents right side precordial leads (V1, V2, V3) very often inverted T waves are seen which are considered normal if there is no significant clinical history. This type of pattern is called the juvenile T wave inversion.

Intervals: The PR, QRS, and QT intervals should be measured and compared to normal values based on the patient's age.

Bundle branch block, congenital long QT syndrome, ventricular tachycardia, electrolyte abnormalities, and Wolff-Parkinson-White (WPW) syndrome are possible causes of variations.

The QT interval is the interval from the start of Q to the end of the T wave. QT interval varies with the heart rate. Hence, the corrected QT (QTc) interval is used to standardize the values. The most commonly used formula used to calculate QTc is Bazett's formula (QTc= QT/√RR). In children, values of up to 440 ms and 490 ms in neonates are considered normal. In place of Bazett's formula, the Fridericia formula (cube root of RR) is applied to neonates.

Table 1: Heart rate at different ages

Age	Heart rate
Birth-4wks	130-190
1-3 months	125-185
3-6 months	110-165
6-12 months	100-155
1-3 years	70-120
3-5 years	60-110
5-8 years	55-100
8-12 years	50-100

Long QT syndrome (LQTS) is characterized by abnormal cardiac repolarization resulting in QT interval prolongation which predisposes patients to torsade de pointes.

Chamber hypertrophy enlargement: ECG can be used to assess atrial enlargement only when the patient is in sinus rhythm, whereas ventricular enlargement can only be diagnosed in the absence of bundle branch block, ventricular pre-excitation, paced ventricular rhythm, or ventricular arrhythmia. It can be difficult to differentiate between right bundle branch block and right ventricular hypertrophy in children due to their narrow QRs

Right/left atrial enlargement (Figure-4A): P wave morphology should be assessed in lead II and V1. A tall P wave of more than 3 mm in infants below 6 months and 2.5 mm in older children suggests right atrial enlargement. Left atrial enlargement is suggested by the presence of a notched, widened P wave of more than 2.5 mm or a biphasic P wave in V1.

Right ventricle hypertrophy (RVH) (Figure-4B): A specific finding for RVH is an R-wave amplitude in lead V1 larger

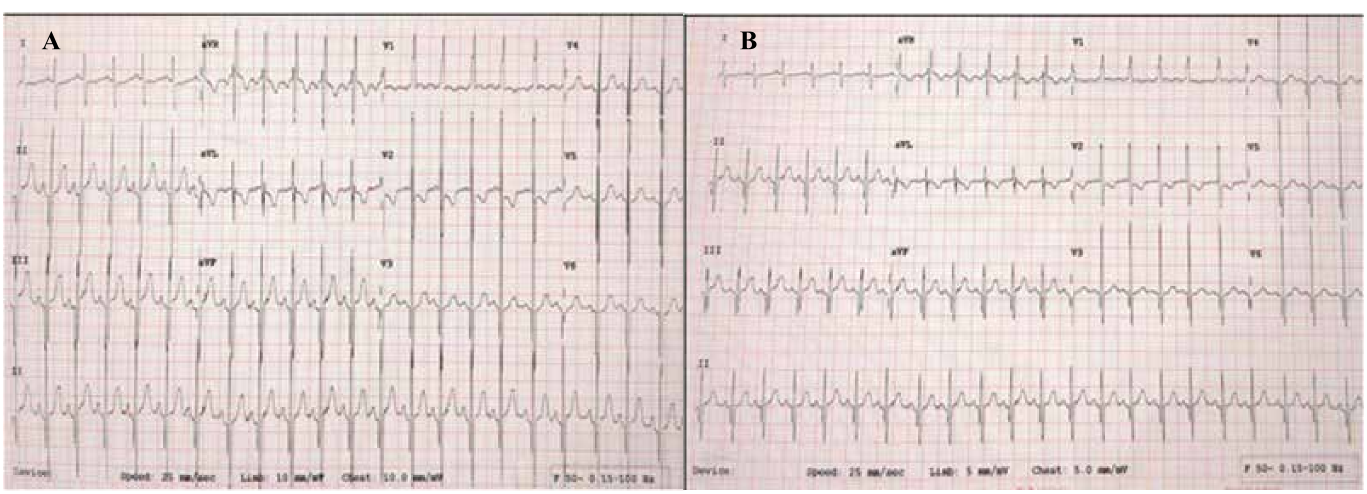

Figure 2 A: ECG of a VSD patient with normal standardization(1mv/10mm) showing overlapping QRS complexes. B) ECG with half standardization (1mv/5mm)

Table 2: ECG parameters at different ages

Age	PR interval (msec)	Qrs Duration (msec)	R wave (S wave) amplitude in mm	
			Lead V1	Lead V6
Birth	80-160	<75	5-26 (1-23)	0-12 (0-10)
6 months	70-150	<75	3-20 (1-17)	6-22 (0-10)
1 year	70-150	<75	2-20 (1-20)	6-23 (0-7)
5 years	80-160	<85	1-16 (2-22)	8-25 (0-5)
10 years	90-170	<85	1-12 (3-25)	9-26 (0-4)

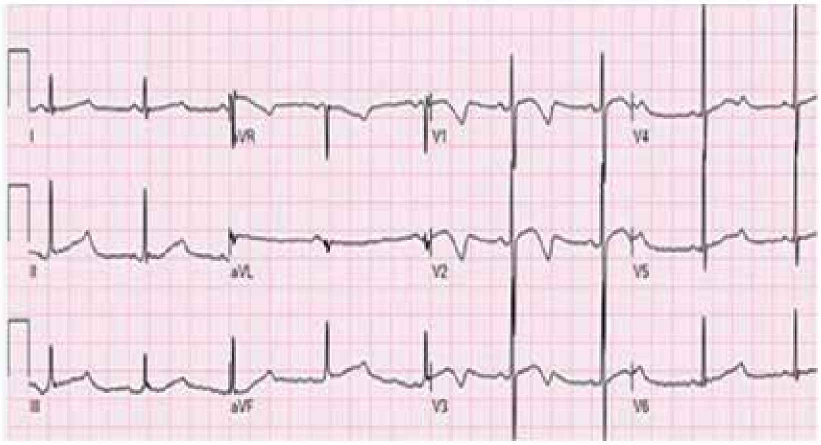

Figure 3: ECG of a long QT syndrome patient with QTc 530 msec

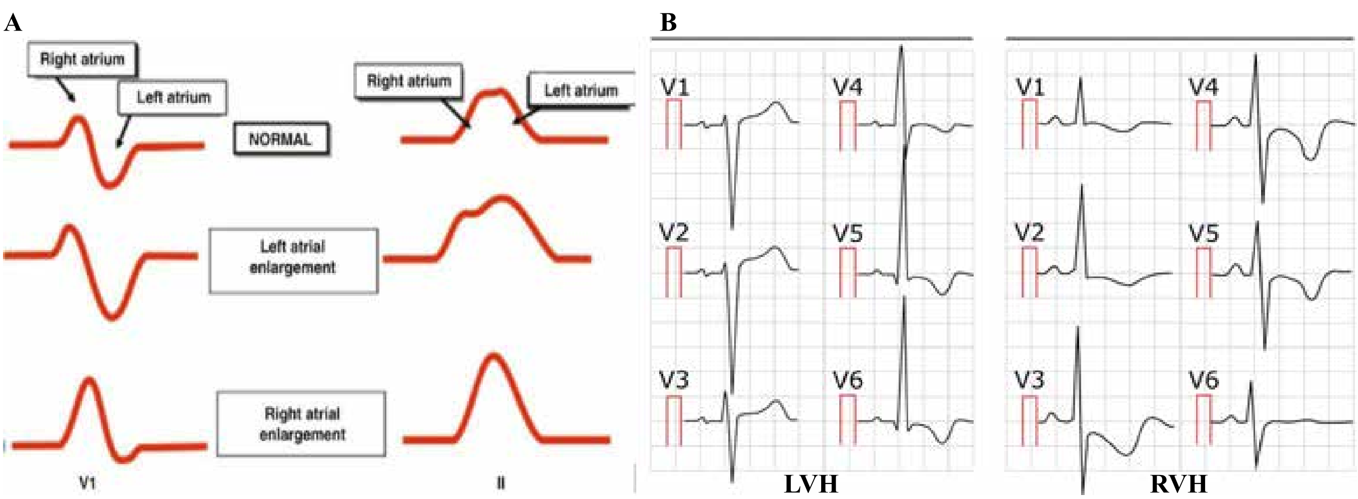

Figure 4 A: P wave morphology in RAE and LAE in leads V1 and II; **4 B:** Left ventricular (LVH) and right hypertrophy (RVH)

than the 98th percentile for age. RVH is also indicated by an R/S ratio in V1>1. When there is no ventricular inversion, a qR pattern suggests RVH. In the case of a right bundle branch block R'>11mm denotes RVH. Because of normal right ventricular predominance in the neonate and the rapid changes in T-wave vectors in the first 2 weeks of life, the ECG interpretation of RVH in infants may be difficult.

Left ventricular hypertrophy (LVH) (Figure-4B): In adults many criteria for LVH based on voltage and morphology have been proposed. In children R-wave amplitude greater than the 98th percentile for age in lead V6 and S-wave amplitude greater than the 98th percentile in lead V1 predict LVH. Occasionally normal children can have R waves in lead V6 that are above the 98th percentile because of a thinly built chest. In that case, a better criterion is the large S wave in lead V1 owing to increased posterior forces.

In newborns, if an adult pattern of R-wave progression i.e. when a newborn manifests small R waves and deep S waves over the right precordium progressing to tall R waves and small S waves in the left lateral precordium, is evident it suggests that there is left ventricular dominance.

Transition of ECG pattern at birth, infancy and later

In the intrauterine period, the right ventricle is the dominant ventricle. Hence in a newborn, the principal QRS potentials result from the right ventricle. There is a gradual transition from RV dominance to LV dominance. Loss of right ventricular dominance starts at about 1 month of age and left ventricular dominance is well established by 1 year. These changes can be appreciated by R wave progression in precordial leads. In early infancy, right precordial leads show tall R waves and small S waves whereas left side leads show a small R wave and a deep S wave. By the end of 1st year adult pattern of R wave progression is seen i.e., a small R wave on right precordial leads and a tall R wave on left precordial leads. Hence the age of the child has to be noted before opining on any given ECG.

Some-typical ECGs in common cardiac disease of children

Atrial Septal defect (ASD), ventricular septal defect (VSD) and patent ductus arteriosus (PDA) are the most important lesions with left to right shunt and ASD being pre-tricuspid lesion there is significant right chamber enlargement. In ASD, characteristic R wave notching in inferior leads (Crochetage

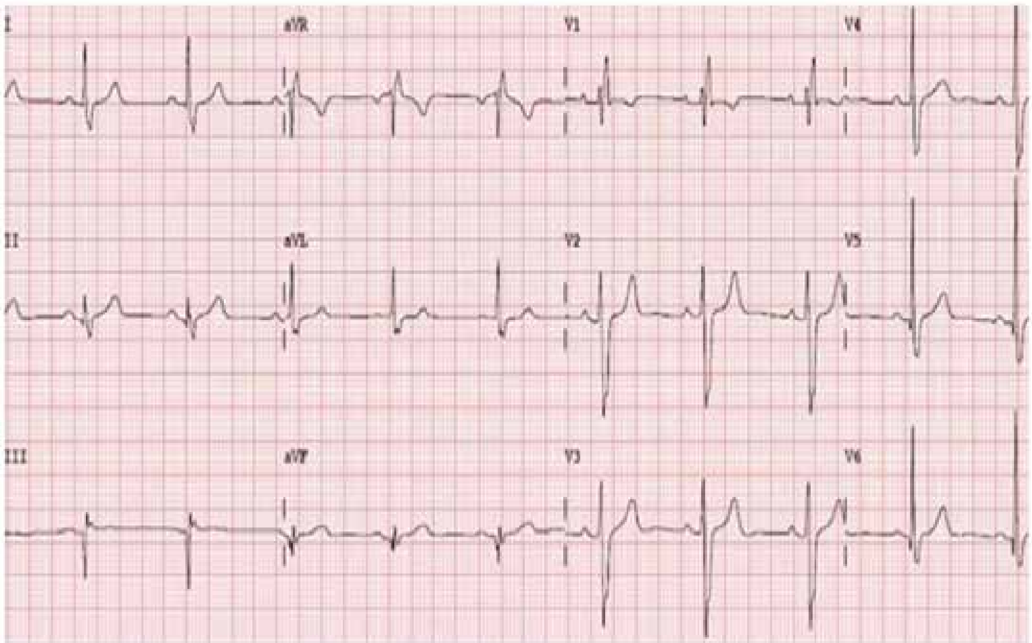

Figure 5: ECG of an ASD patient demonstrating RBBB and crochetage sign in lead II

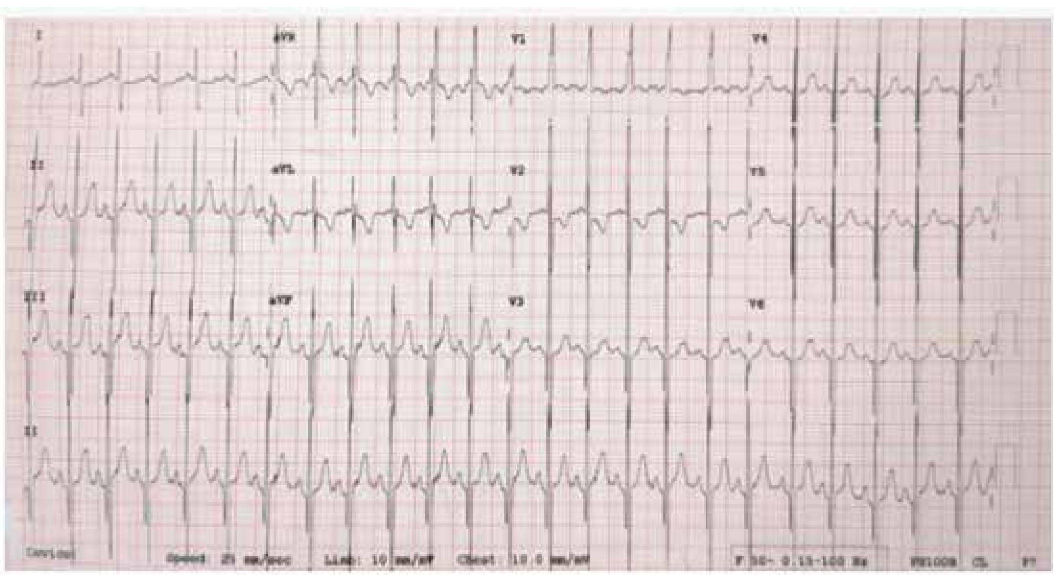

Figure 6: ECG of a VSD patient demonstrating Katz-Wachtel phenomenon in V2-V4

sign), slight right axis deviation (RAD), Voltage evidence of right ventricular hypertrophy (RVH), often in the form of "incomplete" right bundle branch block (RBBB)

Large VSD with significant shunt can show tall diphasic QRS >50 mm in height) in mid-precordial leads (leads V2, V3 or V4) typically associated with biventricular hypertrophy (BVH).

The ECG in PDA also shows left ventricular and left atrial volume over-load and may be mistaken for VSD.

Endocardial cushion defect or atrioventricular canal defects: The characteristic ECG findings in AV canal defects are the presence of counterclockwise looping (q waves in Leads I and aVL) with a superior axis directed more towards the left. Atrioventricular blocks varying from first degree to complete heart block are common in AVCD.

Pulmonary Stenosis: ECG findings are usually normal in mild pulmonary stenosis. Right-axis deviation and right ventricular hypertrophy occur in moderate and severe valvar pulmonary stenosis. The degree of right ventricular hypertrophy is well correlated with the severity of pulmonary stenosis.

Aortic stenosis: The ECG in patients with aortic stenosis frequently shows left ventricular hypertrophy with strain and left atrial enlargement; however, these findings are non-specific for aortic stenosis.

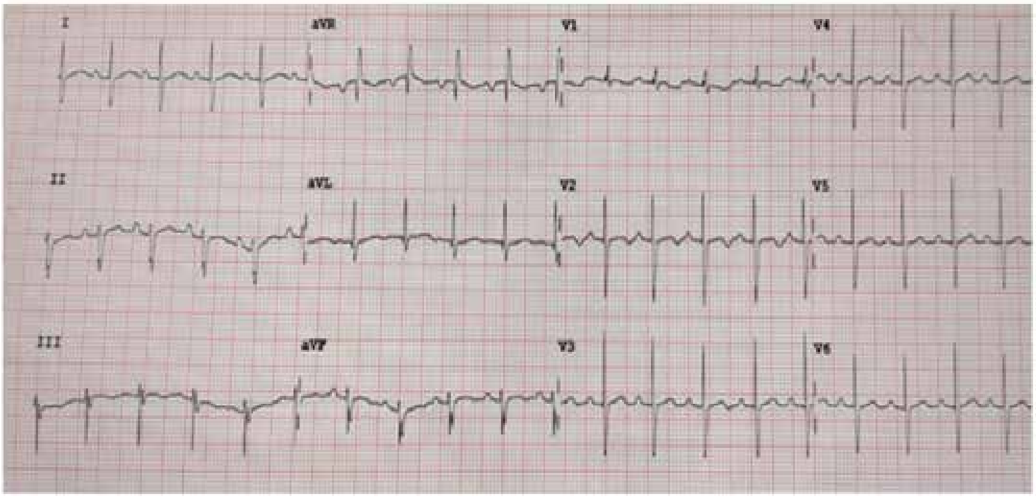

Figure 7: ECG of a AVCD patient showing counterclockwise loop and extreme axis deviation

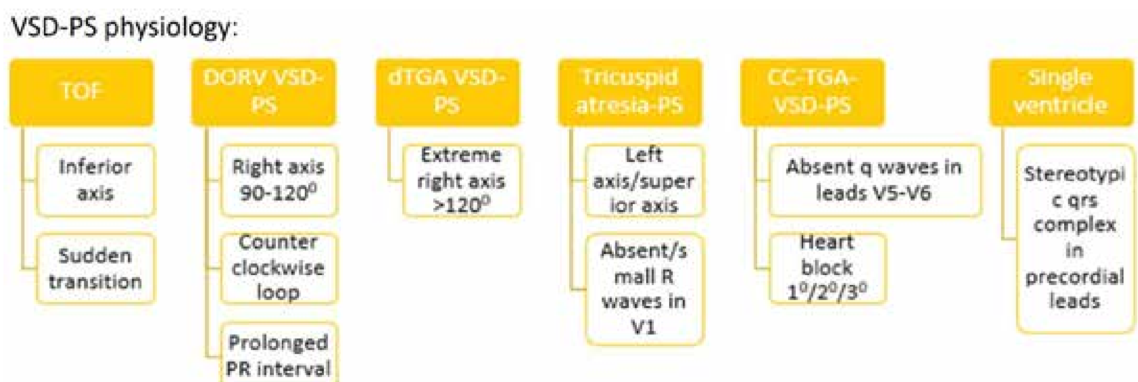

Figure 8: Schematic representation of ECG features in different cyanotic heart diseases with VSD-PS physiology

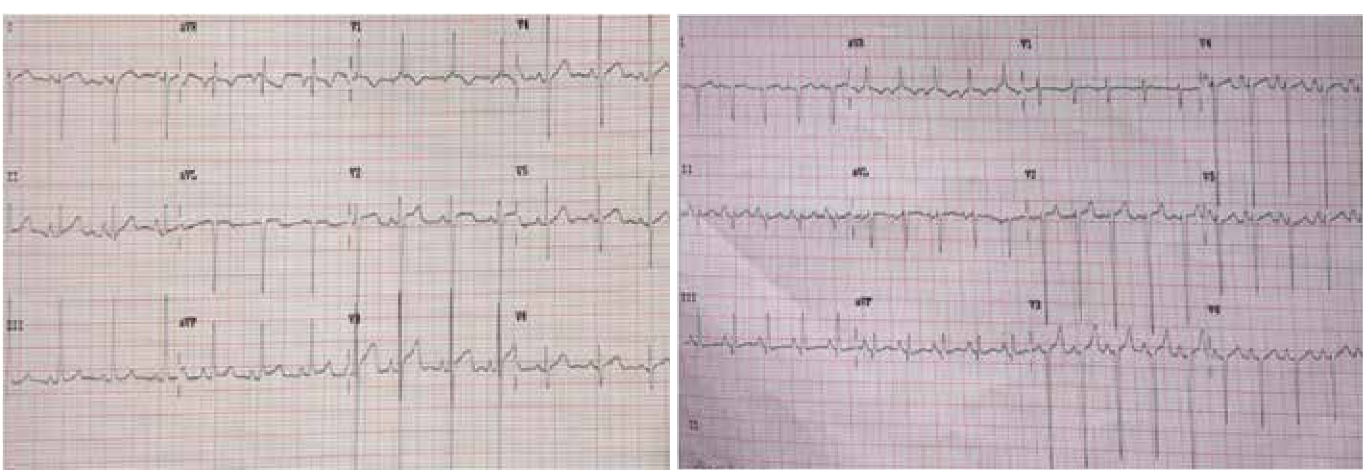

Figure 9: (a) ECG of a patient with Tetralogy of Fallot showing RVH(V1) and sudden transition from V1 to V2 (b) ECG of a single ventricle patient showing stereotypic QRS complex in precordial leads (V1-V6)

Complex congenital heart disease: Evaluation by ECG alone is not easy. The Pediatricians/ physicians need the help of experts in this field for full evaluation and management. However, the figures 8,9 and 10 depicted below show a simplified overview of ECG approach to a complex congenital heart disease especially when there is cyanosis. Grossly the cyanotic heart diseases are categorized into those with RVOT narrowing and decreased pulmonary flow or those without such obstruction to pulmonary blood flow. Further refined diagnosis is done base on ventricular dominance and other connections.

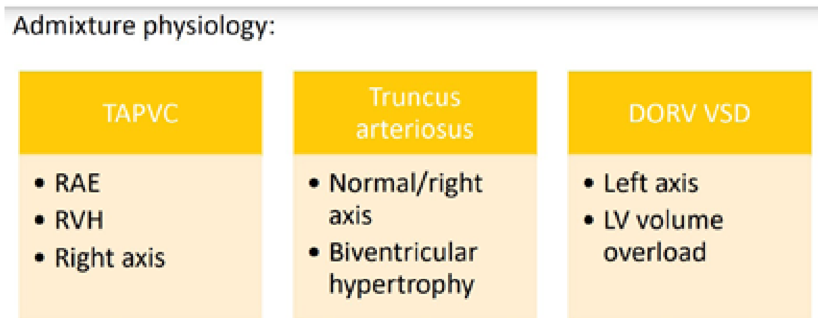

Figure 10: ECG features in different cyanotic heart diseases with admixture physiology

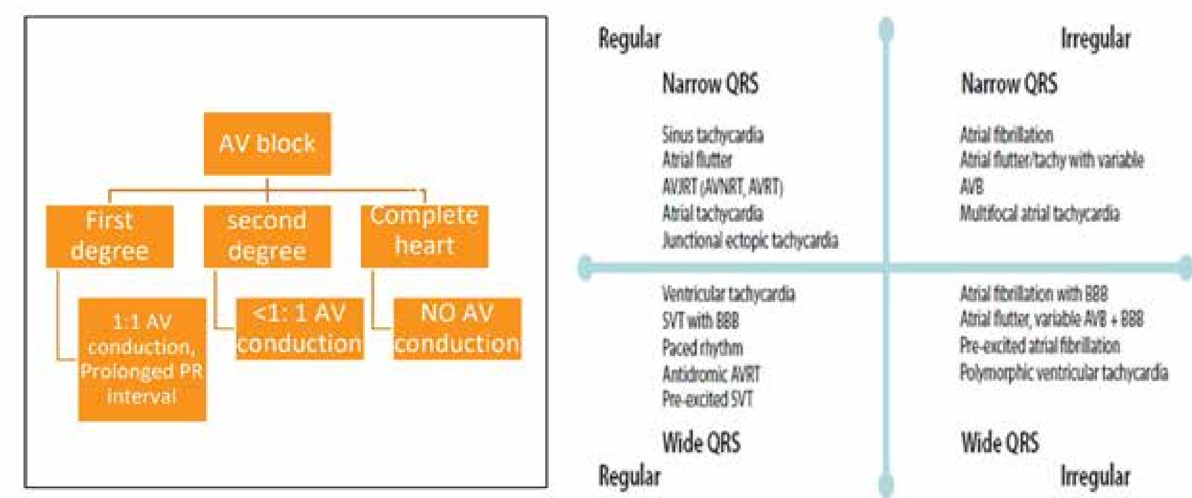

Figure 11: (a) Schematic representation of ECG features in heart block (b) classification of tachyarrhythmias.

Tetralogy of Fallot's (TOF)

It the most common cyanotic heart disease with its ECG being diagnostic in most cases. Right axis deviation right atrial abnormalities and right ventricular hypertrophy are common findings. Only a few subtle changes may differentiate TOF from other mimics of the VSD-PS group of cyanosis with decreased pulmonary flow group. Sudden transition of QRS voltage from V1 to V2. DORV-VSD-PS is a close mimic and it can be suspected on ECG if the PR is prolonged, biatrial enlargement, Left axis deviation and some LV volume overload is present. Left QRS axis suggests Tricuspid atresia -VSD with PS or single ventricle of LV morphology. While any pattern of ECG may be feasible in single ventricle physiologies, uniform QRS morphology in all chest leads increases the chance of single ventricle as a diagnosis. Presence of septal Q waves in V1 and V2 rather than in V5 and V6 may indicate corrected transposition of ventricles.

Cardiac Arrhythmias

Arrhythmias are divided into brady and tachyarrhythmias base on heart rate less than 60 per minute or more than 100 per minute. A systematic approach is needed to diagnose the arrhythmias as depicted in figures-11 and 12. Bradycardia can occur due to sinus node dysfunction, sinoatrial exit blocks or AV nodal blocks. AV nodal blocks are more common.

AV Blocks:

i. First-degree AV block: 1;1 AV conduction with a prolonged PR interval.

ii. A. Second-degree AV block Type I (Wenckebach): there is a gradual progression of the PR interval leading to a P wave that is not followed by a QRS complex.

B. Second-degree AV block Type II: PR interval is constant with occasional non-conducted P waves.

iii. Third-degree AV block/complete heart block: Here, the atria and ventricles work independently of each other with no atrial impulses being conducted to the ventricles. On the ECG, there are more number of P waves than QRS complexes with regular RR and PP intervals, respectively but with varying PR intervals. The cause can be congenital or acquired. In a neonate with a complete heart block, the mother should be tested for anti-Ro and anti-La antibodies to rule out neonatal. Some congenital heart diseases like AV canal defects and CC-TGA have a high risk of heart blocks. CHB is more common in opera ted cardiac patients.

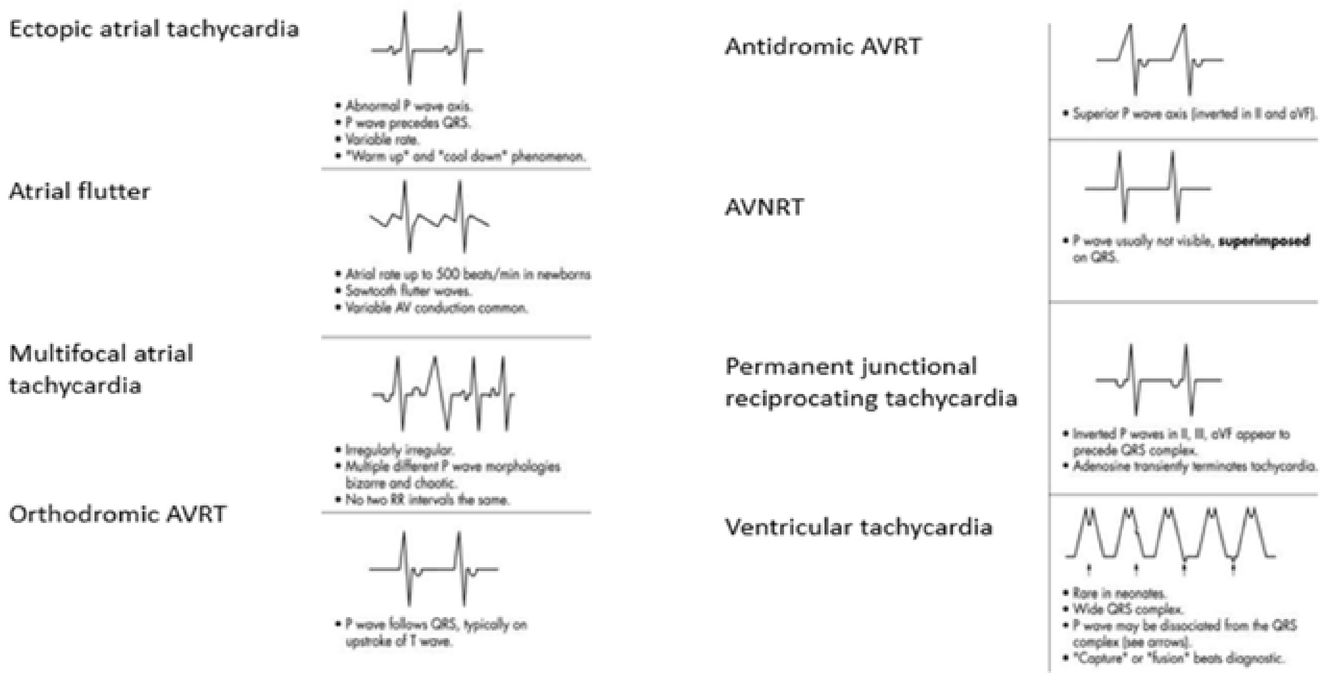

Figure 12: Different types of supraventricular and ventricular arrhythmias.

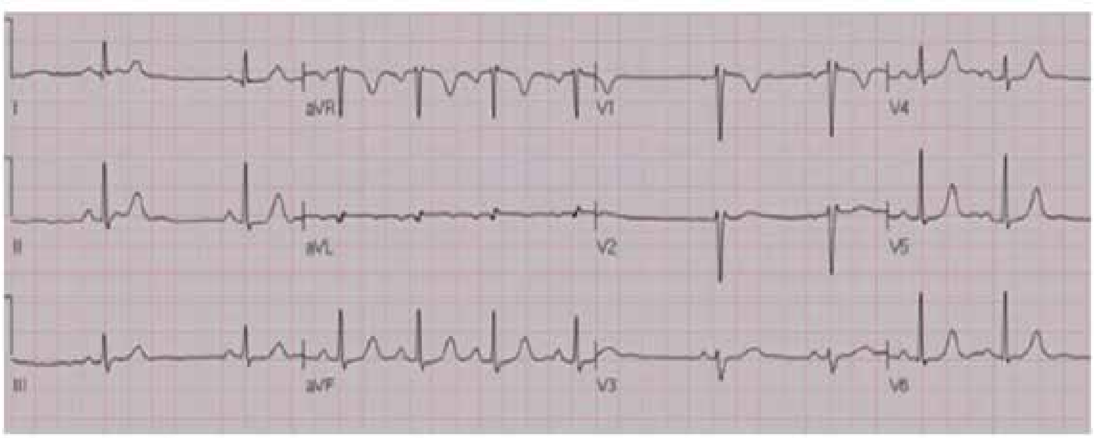

Figure 13: ECG of a patient with sinus arrhythmia showing phasic variation of heart

Tachyarrhythmias:

The salient features of ECG to diagnose tachyarrhythmias is given below.

Sinus arrhythmia is a common ECG finding in the pediatric age group (Figure-13). It is caused by a beat-to-beat change in the PP interval, which results in an irregular ventricular variation in heart rate. Inspiration lowers the vagal tone, which raises the heart rate. The restoration of vagal tone during expiration causes the heart rate to drop. Non-respiratory" sinus arrhythmia is less common and is more likely to be pathological e.g. Digoxin toxicity.

Electrolyte abnormalities

Potassium and calcium play an important role in the cardiac conduction system. So, fluctuations in their blood levels can cause serious conduction abnormalities.

1. Hypokalemia: ECG features are prominent P waves, PR interval prolongation, ST depression and T wave flattening/inversion, and Prominent U waves. Very rarely life-threatening arrhythmias like VT/VF are seen.
2. Hyperkalemia: The earliest feature of hyperkalemia is Tall T waves followed by flattened p waves, widening QRS complexes and bundle branch blocks. Sinusoidal wave pattern, ventricular fibrillation and asystole are manifested when serum K^+ >9mmol/lit.
3. Hypocalcemia: Prolongation of the QT interval is the characteristic ECG feature of the hypocalcemia.
4. Hypercalcemia: The QT interval becomes shorter. Osborn waves may appear in cases of severe hypercalcemia. Extreme hypercalcemia has been associated with ventricular irritability and VF arrest.

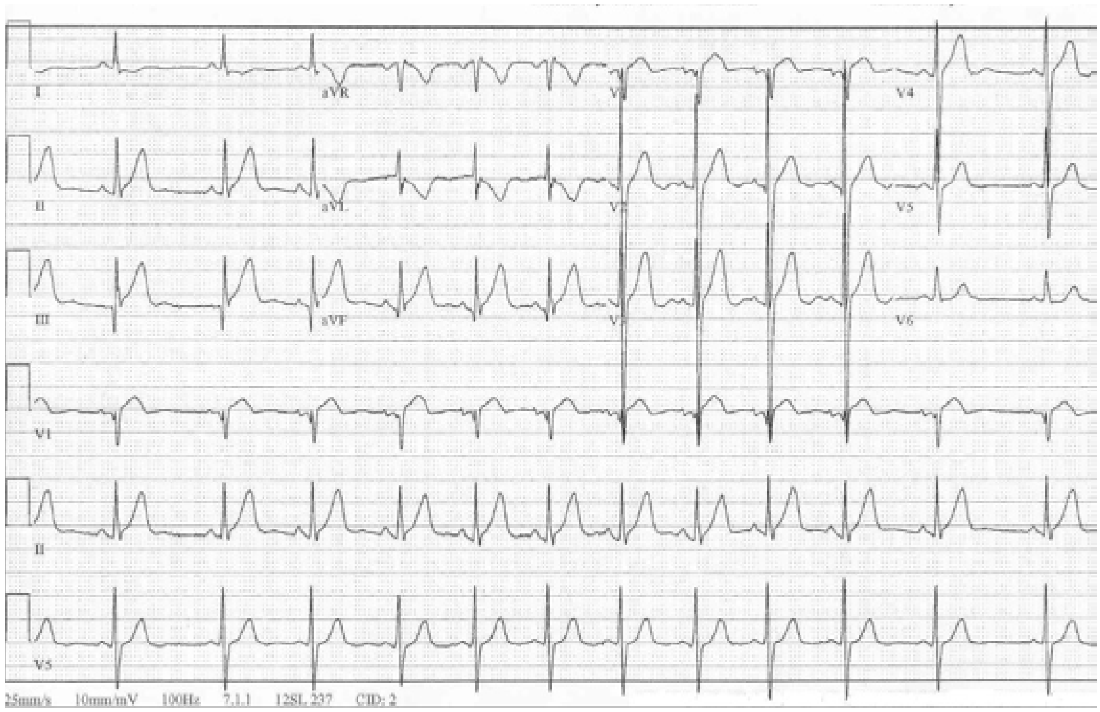

Figure 14: ECG showing situs inversus with dextrocardia ECG of a situs inversus: patient showing inverted P waves in I, aVL and upright P waves in aVR and absent R wave progression from V1 to V6.

5. Hypomagnesemia: Prolonged PR interval, prolonged QT interval, atrial and ventricular ectopy, predisposition to ventricular tachycardia and Torsades de pointes

Pericarditis

Pericardium inflammation can result from infection, localized damage, or systemic conditions, resulting in chest discomfort, dyspnea, and serial ECG changes. ECG is characterized by widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6). Reciprocal ST depression and PR elevation in lead aVR (± V1). Sinus tachycardia is also common in acute pericarditis due to pain.

Myocarditis

The most common feature of myocarditis is sinus tachycardia. Other findings are non-specific ST-T changes, low voltage QRS complexes and atrioventricular blocks.

Early repolarization syndrome (ERS)

ERS is characterized by J-point elevation manifested either as a positive deflection on terminal S wave or ST-segment elevation with upper concavity and prominent T waves in at least two contiguous leads. The prevalence of ERS ranges between 3% to 24% in the general population. Previously, ERS was thought to be a normal or benign variation. Nevertheless, several recent studies have indicated a connection between ER and a higher risk of cardiac arrhythmia-related death. Different clinical studies have found that J point abnormalities in the lateral ECG leads have the lowest risk of VF, but J point abnormalities in the inferior leads and global J point abnormalities have a progressively higher risk of VF.

Situs inversus/Dextrocardia

Situs inversus can be recognized in the ECG (Figure-14) by (a) right axis deviation, (b) Positive P waves, QRS complexes and upright T waves in aVR. (c) In Lead I, inversion of all complexes i.e. inverted P wave, negative QRS, inverted T wave and (d) Absence of R-wave progression in the chest leads. These modifications can be corrected by mirroring the precordial leads on the right side of the chest and reversing the left arm and right arm leads.Accidental reversal of the leads and right arm electrodes may produce a similar picture to dextrocardia in the limb leads. However, the typical R wave progression is evident

Anomalous origin of the left coronary artery from pulmonary artery (ALCAPA)

In this rare condition as left coronary arises from PA, ECG shows infarction of the left ventricle. This causes severe LV dysfunction and dilated left ventricle. Classically, because there is an anterolateral infarct by the time the infant presents for diagnosis, there will be pathological Q waves in leads I, aVL, and precordial leads V4 to V6 (Figure-15).

Ebstein's Anomaly:

In an Ebstein's anomaly there is an abnormal apical displacement of the tricuspid valve, atrialization of RV with or without Tricuspid regurgitation. There are a few characteristic ECG features (Figure-16) like tall Himalayan P waves (>5mm),

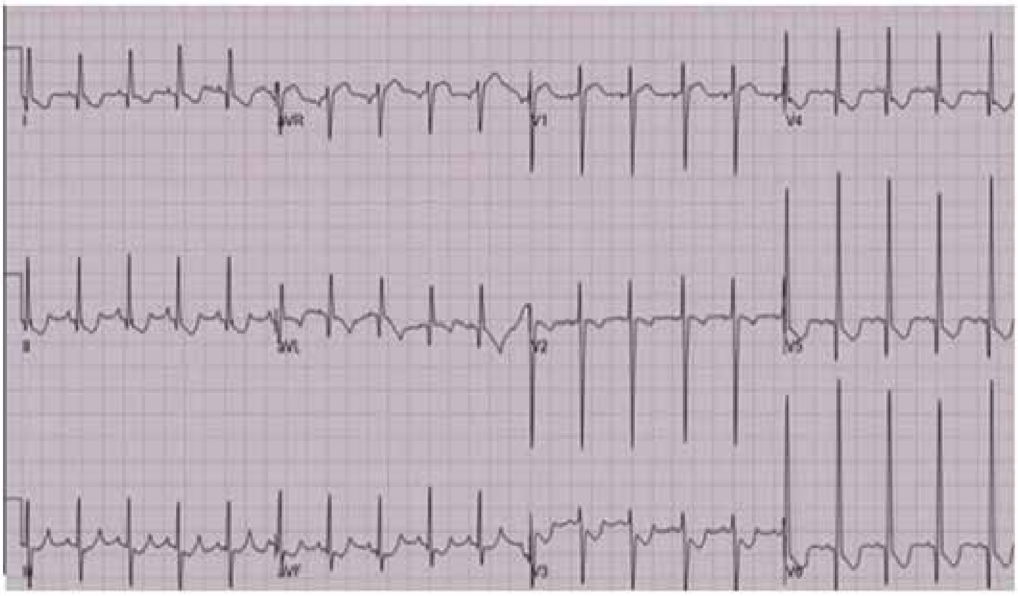

Figure 15: ECG of a ALCAPA patient demonstrating pathological deep q waves in I, aVL and T wave inversions in V5, V6

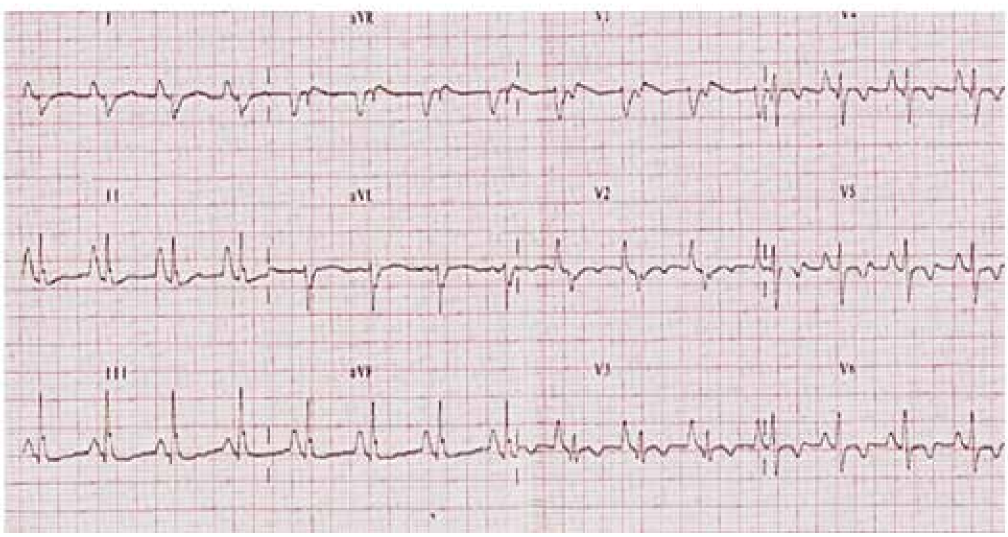

Figure 16: ECG of a Ebstein anomaly patient showing Himalayan (>5mm) P waves in lead II with evidence of bundle branch block (wide QRS)

splintered QRS complexes, right bundle branch block, and prolonged PR interval (1° block). Approximately half of those with Ebstein's abnormality exhibit signs of Wolff-Parkinson-White Syndrome. Supraventricular tachycardia and other rhythm abnormalities such as atrial flutter or atrial fibrillation might occur.

Conclusion

To obtain an artefact-free ECG, the clinician needs to be familiar with the key technological aspects of the ECG machine and lead placement. It is always best to assess ECG alterations in light of the clinical history and age of the patient. Certain ECG abnormalities, such as sinus arrythmia, juvenile T wave inversions or early repolarization in kids, might not be pathological. The clinician should be conversant with age-specific ECG parameters to pronounce an ECG as abnormal in children.

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ECG During Exercise- The Treadmill Test - Separating The Grain From The Chaff

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Introduction

The exercise electrocardiogram (ECG) is a validated procedure for diagnosis and prognosis of coronary artery disease. It also helps in ascertaining functional capacity. It is widely available and relatively low cost. Exercise ECG indirectly detects myocardial ischemia which results from oxygen supply and demand mismatch. The ECG identifies silent ischemia that often occurs at a lower ischemic threshold than that needed for the symptomatic expression of ischemia. The exercise ECG variables beyond ST segment along with clinical variables yield a lot of information in predicting outcomes and guiding therapy in a broad range of individuals.

Exercise testing is generally a safe procedure. However, both myocardial infarction and death have been reported and can occur up to 1 per 2500 tests. Good clinical judgment should therefore be used in deciding which patients should undergo and which patients have a clear contraindication for exercise testing.

Some important Indications of exercise testing are listed out here:[1]

1. For diagnosis of obstructive coronary artery disease
2. Risk assessment and prognosis in patients with past history of coronary artery disease
3. After Myocardial infarction for prognostic assessment, activity prescription, evaluation of medical therapy, and cardiac rehabilitation.
4. Screening of Asymptomatic patients
5. Patients with Valvular heart disease, for evaluation of exercise capacity and diagnosis of coronary artery disease.
6. Exercise using ventilatory gases for evaluation of exercise capacity in heart failure patients.
7. Exercise testing before and after revascularization
8. Investigation and diagnosis of heart rhythm disorders.

Some important absolute and relative contraindications for exercise ECG are:

1. Acute myocardial infarction (within two days)
2. Ongoing unstable angina
3. Uncontrolled arrhythmias with hemodynamic compromise
4. Symptomatic severe valvular stenosis
5. Decompensated heart failure
6. Active endocarditis
7. Acute myocarditis or pericarditis
8. Acute aortic dissection
9. Acute pulmonary embolism, pulmonary infarction, or deep venous thrombosis
10. Physical disability that precludes safe and adequate testing

Limitations of Exercise ECG

Exercise ECG may not be appropriate in few individuals. These Include

1. Patients who are unable to exercise sufficiently due to leg claudication, arthritis, deconditioning, pulmonary disease, or other conditions. An exercise ECG test that does not achieve 85% of the patient's predicted maximal heart rate may reduce sensitivity to detect ischemic heart disease.
2. Patients with Resting ECG changes interfere with interpretation of test. Some Baseline ECG changes that interfere with interpretation are
   a. Ventricular preexcitation (Wolff-Parkinson-White pattern)
   b. Ventricular paced rhythm
   c. Left bundle branch block
   d. Greater than 1 mm ST depression at rest
   e. Digoxin use with associated ST-T abnormalities
   f. Left ventricular hypertrophy with ST-T abnormalities
   g. Hypokalaemia with ST-T abnormalities

Analysis of Exercise Test

Various parameters are assessed during exercise testing. Among those are exercise induced symptoms, functional capacity, heart rate responses, blood pressure responses, and electrocardiogram (ECG) changes.

Electrocardiogram: The interpretation of the exercise ECG focuses on the presence or absence of ischemic ECG changes along with any induced arrhythmias. These abnormalities are discussed in greater detail below.

The most important ECG changes during exercise are related to Changes in the ST segment. However, the full spectrum of the ischemic ECG involves changes in the ST segment, the T wave, and the U wave. These changes can be interpreted accurately in patients who do not have baseline ECG abnormalities.

Electrophysiology of Myocardial Ischemia

The effects of myocardial ischemia are responsible for the ST-T wave abnormalities seen on the ECG during exercise testing in patients with coronary heart disease. Brief review, the QRS complex reflects the time sequence of ventricular depolarization as the activation front spreads through the myocardium (phase 0 of the action potential). The ST segment reflects the plateau or phase 2 of ventricular repolarization, and the T wave reflects the time sequence of phase 3 repolarization. Phase 4, the resting membrane potential, is responsible for the TQ segment.

Some Key concepts:

Cardiac ischemia causes

- Increased extracellular K and a less negative EK
- Myocyte depolarization
- Fast Na channel inactivation in non pacemaker cells
- Depressed phase 0 slope of action potentials
- Reduced action potential conduction velocity.

Ischemia leads to cellular depolarization by altering ion chemical gradients and membrane conductance to ions. These changes alter action potential depolarization and repolarization and depress their conduction within the heart, leading to altered ECG wave morphology, intervals, and segments.

There are quite a significant number of confounding factors that may lead to alteration of sensitivity and specificity of exercise ECG. These confounders include the presence of vulnerable, non-obstructive lesions in the coronary arteries, subthreshold severity of ischemia with inadequate effort tolerance or blunted heart rate response to exercise secondary to beta blockers or digitalis, causes of high endocardial wall stress that can limit subendocardial flow reserve, and electrolyte and drug effects on the action potentials.

Electrocardiographic Changes During Exercise

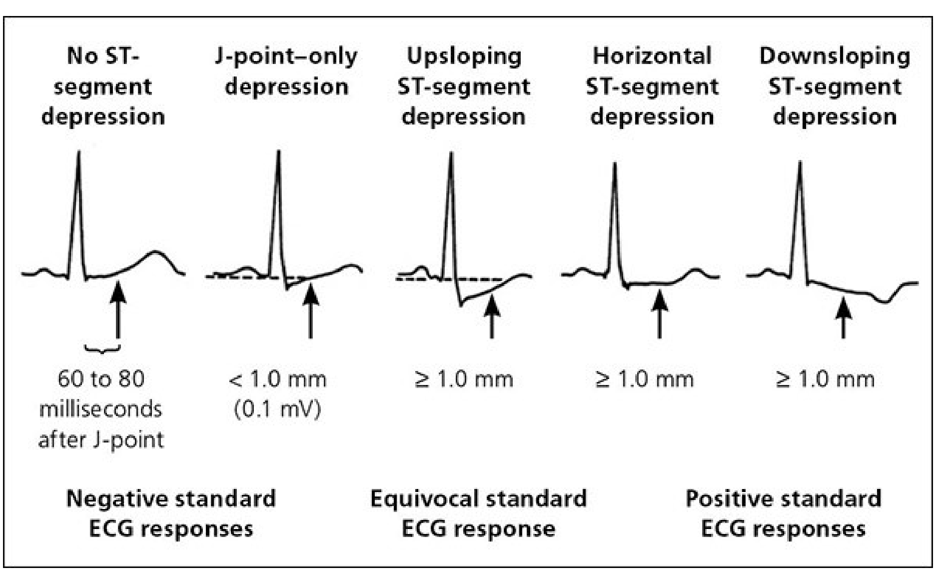
ST Segment Changes

ST-segment depression is the characteristic finding associated with exercise-induced, demand-driven ischemia in patients with significant coronary obstruction but no flow limitation at rest. Exercise related ischemia, unlike obstructive Acute coronary situations, is generally limited to the sub endocardium and is proportional to increase in oxygen demand of myocardium. Progressive Ischemia during exercise results in constant changes of action potentials in sub endocardium during both diastole and systole. Combined effects of systole and diastole leads to drifts in ST segments. The greater the ST deviation, larger is the territory involved.

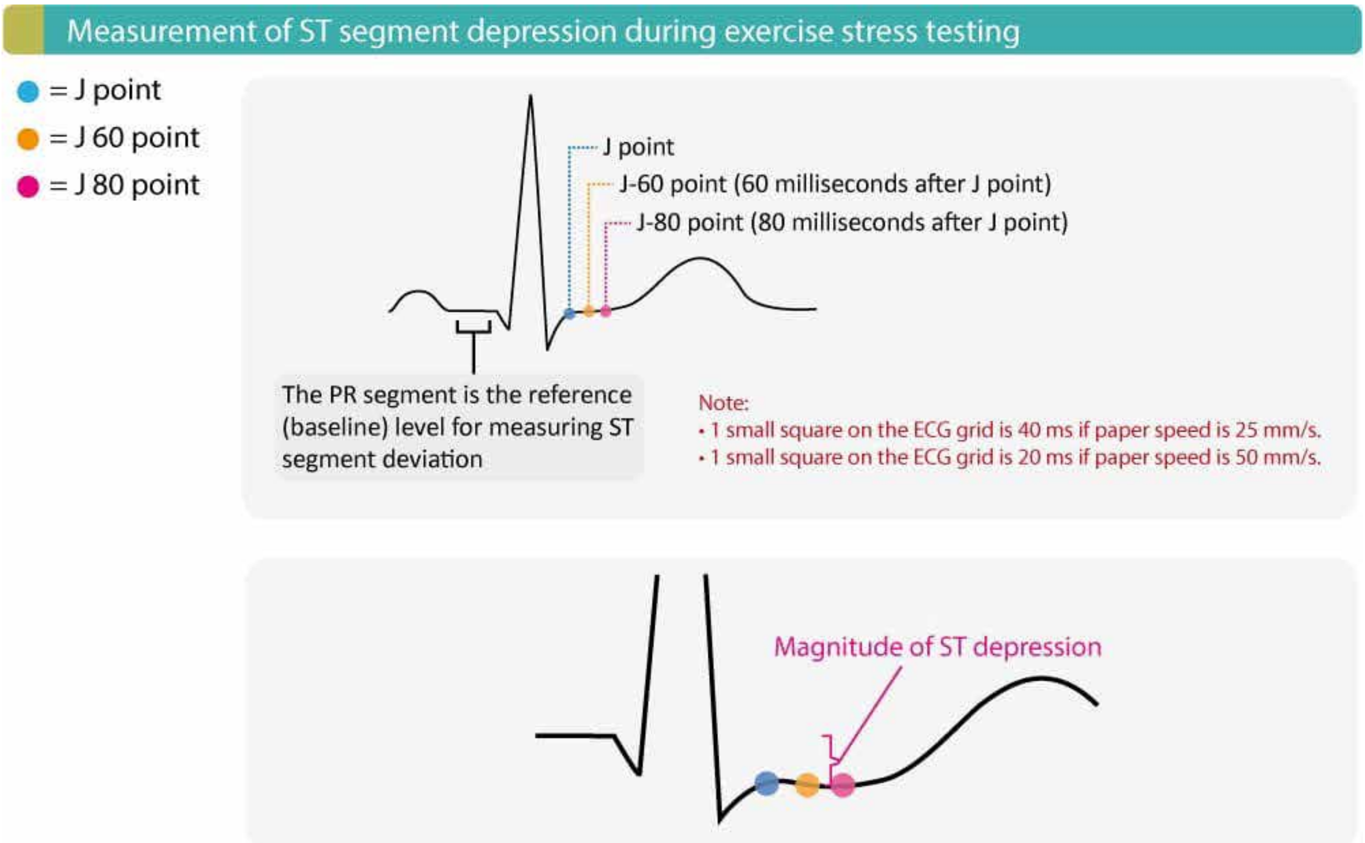
The value of ST segment depression is only moderately useful with a sensitivity and specificity of 60%-70% and 70%-80%, respectively. Overall, prognostic value of ST segment changes is not superior to functional capacity and heart rate responses.

The various ST Segment changes that can occur are

- HORIZONTAL OR DOWNSLOPING ST SEGMENT DEPRESSION: It is generally considered abnormal or positive for ischemia when there is ≥1 mm or greater or 0.1mV or greater of horizontal or down sloping ST segment depression in three consecutive beats in one



Courtesy: AAFP- from Google search



Courtesy: ECG Waves from Google Search

or more leads that persists at 80 milliseconds after the J point. This criterion should be an add on to existing resting ECG. In early repolarisation, ST segment changes should be measured from the baseline isoelectric line.

Leads V4, V5, and V6 are the most sensitive leads for detecting the ST depression of subendocardial ischemia; However, ST depressions does not localise the territory of ischemia. In addition, ST segment depression confined to the inferior leads II and aVF is most often a false positive response, and is of little value in diagnosing coronary disease because of the influence of atrial repolarisation in these leads. Greater ST segment depression involving multiple leads usually predicts extensive myocardial ischemia.

Approximately 8% of individuals develop ST segment depression during recovery rather than during exercise. Patient with ST-depression restricted to recovery phase have highest specificity, positive predictive value, and negative predictive value. However, accuracy was highest in-patient group with both recovery phase and exercise ST-segment depression.

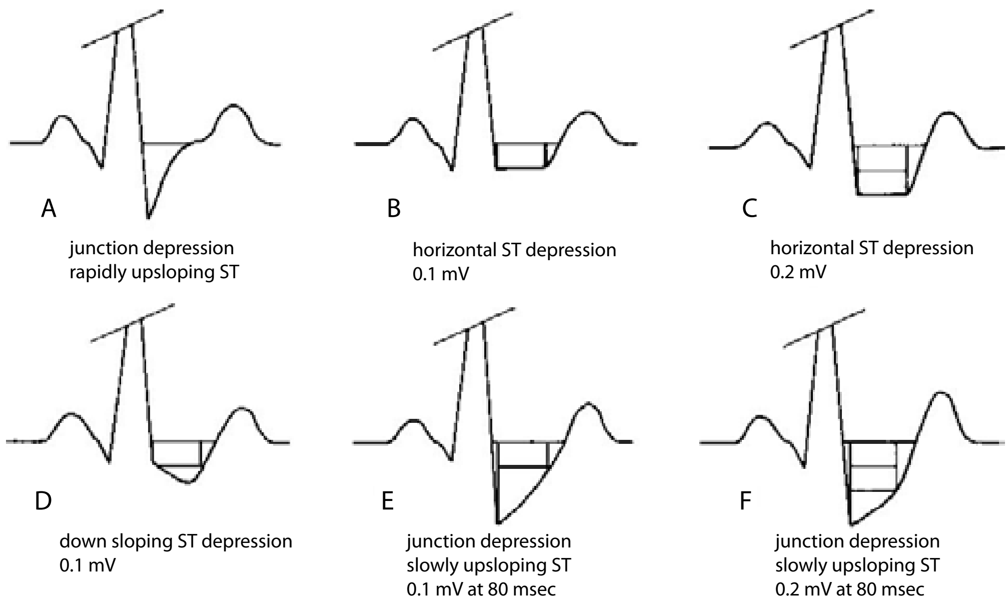
- UPSLOPING ST DEPRESSION: Depression of the J point is normally seen with increasing heart rates during exercise and is related to atrial repolarization rather than ischemia. In this circumstance, the ST segment rapidly (within 80 milliseconds) returns to baseline. However, ST segment depression that is slowly upsloping (0.5 to 1.0 Mv/sec) may be considered abnormal, especially if it occurs in the early stages of exercise. Upsloping ST depression have a very low positive predictive value for diagnosis of obstructive coronary artery disease.

- RESTING ST DEPRESSION: In presence of baseline ST segment depression due to LBBB, LVH or digoxin therapy, ST segment changes may not be quite reliable. However, exacerbation of even minor degrees of ST depression will have prognostic value.

- ST ELEVATION: It was considered positive is there is 1mm or greater or 0.1mV of ST segment elevation above the PQ point at 60milliseconds after the J point in at least three consecutive beats. ST elevation signifies either a significant proximal artery stenosis or severe coronary vasospasm.

  When Exercise-induced ST segment elevation is seen in leads with a pre-existing Q wave it would suggest either LV aneurysm or regional wall motion abnormality rather than inducible ischemia. However, if ST segment elevation is associated with reciprocal depression in the non-infarcted area they may be indicative of transmural ischemia of a particular vascular territory.

- ST SEGMENT ELEVATION IN LEAD aVR: ST elevation of 1mm or greater may be a predictor of left



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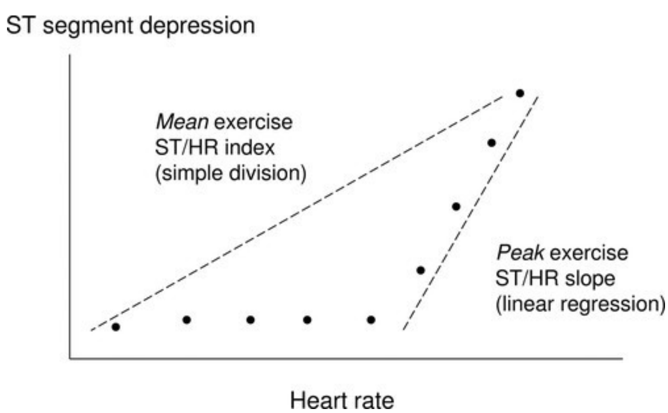

Figure 1: Schematic definition of the ST/HR slope and the ST/HR index. Because the rate of change of ST depression in relation to change in heart rate during ischemia is greatest at the end of exercise, the ST/HR index systematically underestimates the ST/HR slope. (ST depression is plotted as positive on the y axis.[3]

main disease, or proximal LAD disease or multivessel CAD. It is a sensitive, moderately specific parameter with High negative predictive value for inducible ischemia.

- ST SEGMENT ADJUSTMENTS: Its an alternative method to analyse ST Segment depression. ST depression during exercise-induced ischemia is based on the presence of coronary obstruction or increased demand of myocardial oxygen demand. As ST segment depression does change throughout the stages of exercise, it may reflect more than just coronary obstruction. Because changes in heart rate are related to changes in myocardial oxygen demand, in the presence of limited coronary blood flow there should be a progressive relationship between the degree of ST depression and increasing heart rate. Adjustment of ST depression for changes in myocardial oxygen demand related to heart rate is therefore physiologically logical.

  Heart rate adjustments can be done using two methods. The complex method, ST/HR slope, is automated, easily available on the testing machines. It plots ST depression as a function of HR at numerous points during exercise and generated the terminal ST/HR slope of each lead. A value of 2.4µV/beat/min is considered abnormal. The other simple method is ST/HR index. It is calculated by dividing the maximum ST segment in microvolts by the difference in resting peak heart rate. A value of 1.6µV/Beat/min is considered abnormal.

QRS Duration

Conduction velocity increases with sympathetic stimulation, whereas ischemia tends to decrease conduction velocity by slowing the rapid upstroke (phase 0) of the ventricular action potential. It has been stated that differences in QRS duration from rest to exercise serve as a marker of ischemia.

Normally there is a shortening of QRS duration. However, Exercise induced LBBB can occur in <0.5%. When LBBB occurs with higher Heart rates of >125bpm, it is not of much significance. Whereas if LBBB is noted at lower heart rates, the incidence of underlying CAD is higher. In contrast, exercise induced RBBB is not a reliable parameter to assess ischemia.

ORS Amplitude: Normal finding is an increase in R-wave amplitude during submaximal exercise, with a decrease at maximum exercise.

T WAVE ABNORMALITIES: Transient conversion of a negative T wave at rest to Positive T wave in exercise is called pseudo normalisation. It's a Nonspecific finding in patients without prior MI. It does not have a diagnostic or prognostic value.

Exercise Induced Arrhthymia:

- ECTOPY: Suppression of resting ectopy during exercise is a normal finding and does not predict possibility of underlying CAD. Whereas, ventricular ectopic beats Can occur in up to 20% of patients undergoing exercise ECG. Ventricular ectopy occurring during exercise or recovery predicts overall increased mortality. Atrial ectopy is also quite frequent during exercise however does not predict adverse outcome
- NON-SUSTAINED OR SUSTAINED VT: Exercise induced VT may be monomorphic, polymorphic or bidirectional. It is reported in patients with underlying structural heart disease or presence of ischemia. However, occurrence of non-sustained VT during exercise ECG is not of much significance.
- SUPRAVENTRICULAR TACHYCARDIA: exercise induced SVT does not predict ischemia. However, could be a marker of later occurrence of atrial fibrillation.

Exercise ECG Artifacts:

Electrocardiographic artifacts are defined as electrocardiographic alterations, and are not related to cardiac electrical activity. The two main artifacts are Motion artifacts and the presence of other electrical devices in the room which may cause electrical interference. Consequence to these artefacts, interpretation of exercise ECG may be difficult.

Differentiation of ventricular tachycardia artifacts[4]

Some ECGs may resemble specific arrhythmias, such as VT and atrial flutter or atrial fibrillation. It is important to make a correct diagnosis, otherwise patient will end up undergoing unnecessary investigations and medications. The characteristics that help to differentiate VT from an artifact include:

- Absence of hemodynamic impairment during the event;
- Presence of normal beats in any lead;
- An unstable baseline in the ECG before or after the event or both.
- Association with body movement.

Some signs of pseudo-VT are:

- *Sinus sign*: one of the frontal leads (I, II, and III) may present normal sinus rhythm with P, QRS, and T waves.
- *Spike sign*: presence of small regular or irregular peaks between wide QRS complexes.
- *Notch sign*: overlapping notches in the artifact, appearing to be a wide QRS complex, coinciding with the duration of the cycle in which the sinus rhythm was recorded.

Diagnostic Value of Exercise Ecg:

The diagnostic accuracy of exercise ECG is lower in women compared to men. The sensitivity and specificity among women being 61% and 70%, respectively; compared to 68% and 77%, respectively, in men.[5]

Summary

Exercise electrocardiography is underutilized as the initial test modality in patients with interpretable ECG who are able to exercise. Data derived from exercise ECG can yield substantial data for risk stratification. In addition to exercise-induced ischemic ST-segment depression, such markers as ST-segment elevation in lead aVR, abnormal heart rate recovery post-exercise, failure to achieve target heart rate, and poor exercise capacity improve risk stratification of Exercise ECG. For example, patients achieving ≥10 metabolic equivalents on Exercise ECG have less possibility of CAD and an excellent prognosis. Its also vital to select patients appropriately and interpret the test in a Holistic manner.

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ECG in Cardiac Emergencies

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Introduction

The 12 lead Electrocardiogram is a noninvasive diagnostic tool to gather important information regarding not merely the cardiac status but also that of the systemic events affecting the cardiac system. Widely used in the hospital settings, the EKG is an important preliminary investigative tool in the emergency room (ER). The role of the EKG in the initial evaluation of patients presenting with Chest Pain, Dyspnoea, Syncope, Blunt trauma, Hypotension, Syncope, Cardiac arrest is profound. It helps the physician to focus his attention and narrow down the differential diagnosis to situations that warrant immediate attention and treatment. In addition, it also aids the physician in monitoring the treatment response in ACS patients, diagnosing and monitoring specific arrhythmias, electrolyte abnormalities and drug intoxications.

The ECG truly shines in the fast-paced environment of the emergency department (ED). Here, it plays a starring role in a variety of diagnostic dramas, helping doctors decipher the mysteries of: Chest pain, Shortness of breath, Dizziness, fainting, and even confusion that can sometimes be traced back to the heart's electrical rhythm. The ECG can help connect the dots in Poisoning and overdoses, Trauma, Shock and of course the varying causes of Chest Pain.

In a study[1] that looked at the impact of ECG in the ER the following data was observed: Physicians found that a primary diagnosis could be established through ECG in 10.9% of the total. Out of 95 ECGs identified as normal (31.3%), (2.3%) facilitated a rule-out diagnosis. Among the total of 209 ECGs deemed abnormal (68.7%), 72 (23.6%) were considered to have "diagnostic significance." Thus of the total ECGs 23.6% were deemed as Normal, 31.3% as Abnormal but not diagnostic, while 45% were regarded as Abnormal and Diagnostic.

ECG is requested to unravel a clinical syndrome like Chest Pain or Dyspnoea or Syncope. Based on the clinical scenario, the differential diagnosis warrants a pattern of ECG changes that will assist in focusing the problem. The subsequent sections will examine the individual emergency situations rather than the clinical scenario, and the role of the ECG in unravelling it.

Aortic Dissection

Aortic Dissection is a life threatening cardiac emergency that presents with features of Chest pain accompanied by with any or all of these features: Hypertension, Hypotension, Cardiac Tamponade, Shock. It is critical to diagnose the condition urgently as mortality increases with time. Clinical features to suggest Dissection includes the characteristic a. Pain that is Precordial radiating posteriorly down to abdomen, the character being tearing or ripping. b. Hypertension with asymmetric pulse c. Aortic regurgitation d. Pericardial effusion, Tamponade.

ECG features are related to Ischemia arising from the dissected flap extending to the coronaries often involving the coronary ostia with resultant ischemia. The features of Aortic Regurgitation and Pericardial effusion can also be contributary. The following ECG features are noted (1) Ischemic ST depression global or related to Inferior or Anterior territory 20-50%. (2) ST segment elevation 10%; an independent predictor of mortality and is related to coronary occlusion. ST elevation aVR can be associated with diffuse ST depression or ST elevation in Anterior leads. In a multivariate analysis aVR ST elevation was the strongest predictor of in hospital mortality. (3) The other ECG changes include LVH, LAE, LBBB, PR prolongation and Diffuse T inversions.

The suspicion of Aortic Dissection based on the character of Pain, Assymetric Pulses, Aortic regurgitation, should prompt the cardiologist to perform echo to rule out dissection and if necessary CT, to confirm the same.

Acute Pulmonary Embolism

Pulmonary Embolism relates to the sudden obstruction of the Pulmonary Vascular tree by a thrombus majority of the time and rarely by tumour or fat emboli. The hemodynamic consequences relate to the obstruction and the sudden rise in Pulmonary artery Pressure followed by rise in RV systolic pressure and consequent RV strain. This can result in RV decompensation and RV failure, Hypotension and even Asystole. Patient presents with Acute onset of Breathlessness, Chest discomfort, hypotension and even collapse and cardiac asystole if the embolism is massive. Clinical and Echo findings are suggestive of Pulmonary Hypertension, RV Dysfunction, Tachypnea and Cyanosis. CT Pulmonary angio findings are confirmative.

ECG changes often reflect the findings of Acute RV strain consequent to the acute rise in Pulmonary vascular resistance. The changes are (1) RA enlargement (2) Right Axis deviation 3. RVH 4. RV strain 5. RV Ischemia. Prominent tall P in Inf leads reflect RAE. (3) Rt axis, RBBB and RVH : R/S ratio in V1V2 >1. 4. ST depression or elevation in VI V2 suggestive of RV Free wall ischemia. 5. The Classic Pul embolism pattern is S1Q3T3: S waves in Lead I : Rt axis; Q3T3: RV strain. 6. Sinus tachycardia, Atrial Fibrillation and terminal Bradycardia.

Both S in I and RBBB can be attributed to delayed RV Depolarisation. ST elevation or Depression in V1V2 can lead to a mistaken diagnosis of ACS, often associated with mild elevation of Troponin. D Dimer, is significantly increased in PE.

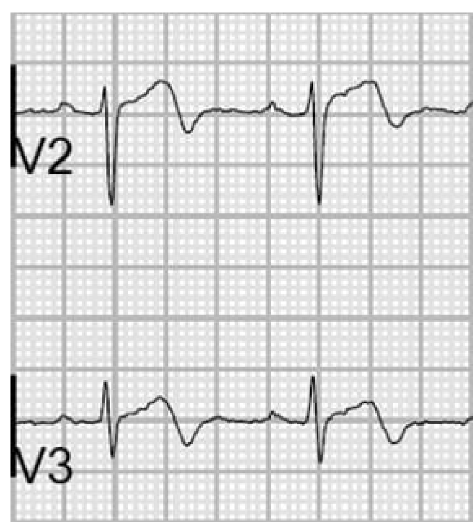
ECG in ACS

Chest pain is one of the most common reasons for emergency department (ED) visits. It is one of the most challenging conditions to evaluate, and often leads to ED overcrowding, inefficient use of resources, and delays in diagnosis. Chest Pain can result from a variety of causes that includes: (1) Cardiac etiology related: Acute Coronary Syndrome, Aortic Dissection, Myopericarditis, Pulmonary embolism. (2) Musculoskeletal causes: Costo-chondritis (3) Pulmonic causes: Pneumothorax, Pleuritis (4) Gastrointestinal causes: Oesophagitis, Gastritis, Cholecystitis

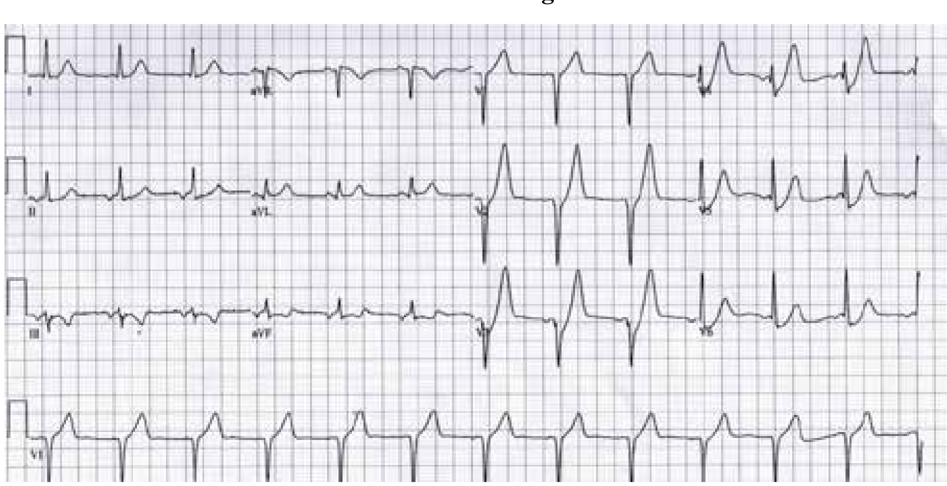
The ECG is an important tool in the assessment and management of patients with ACS and therefore should be performed and interpreted within 10 minutes of arrival to the ED. Symptoms described as a pressure, tightness, squeezing, heaviness, or burning should be considered consistent with ACS. Pain in locations away from the chest can occur and include the shoulder, arm, neck, back, upper abdomen, or jaw. Other associated symptoms include shortness of breath, nausea, vomiting, diaphoresis, fatigue, and mental status changes. The HEART[2] score is a useful approach to include in the ER that will ensure inclusion of all ACS features. THE HEART score takes into account: (1) History (2) ECG (3) Age (4) Risk Score (5) Troponin each of which is scored from 0-2, the lowest score being 0 and the highest score being 10. Low risk score of 3 or less has a MACE rate 1.7% for discharge from ED; Moderate Risk score 4-6 has a Mace rate 12-17% requiring observation and further testing and a High risk score 7-10 has a Mace rate of 50-60% requiring urgent intervention. The ECG features in the score includes Score 0=Normal ECG, Score 1: Abnormal ECG with repolarisation abnormalities without ST depression; Score2: Abnormal ECG with ST depression or elevation.

The EKG changes seen in ACS include: Normal EKG in 1-6% accounting for 2% of missed AMI. The subsequent evolution of STEMI includes: 1. Prolongation of QT interval 2. Hyperacute T waves 3. ST segment and J point elevation 4. Increase in R wave amplitude 5. Q wave formation. Hyperacute T waves: are broad based and symmetrical with increased amplitude in precordial leads. They can also be seen in Hyperkalemia, Early Repolarisation, LVH and Myopericarditis. ST segment elevation of 1mm should be in at least two contiguous leads, 1.5 mm in women and 2.5mm in men age <40 yrs. ST segment depression is due to subendocardial ischemia associated with T inversions. It can also be a feature of reciprocal ST depression. Loss of Precordial T wave balance is also suggestive of ischemia. Normally T wave is inverted in V1 and upright in V6. If this pattern is reversed , it suggests ischemia. Certain specific diagnostic ECG constellations include Wellens Sign and De Winters sign. Wellens syndrome is a pattern of inverted or Biphasic T in V2 to V3 that is suggestive of a Proximal LAD lesion. De winters sign consists of the following pattern: Pronounced upsloping ST depression V2-V4, Tall symmetrical upright T waves and ST elevation aVR.

Wellens sign



De winters sign



Artificial Intelligence (AI) based ACS detection

Diagnosis of ACS in the Emergency is always a challenge. Current recommendations rely on the ST segment amplitude and T wave morphology for ACS diagnosis: with a sensitivity of only 40% for a 12 lead ECG. Computational algorithms however can identify hundreds of features from a 12 lead ECG. Machine learning models based on these can achieve nearly 50% increased sensitivity to pick up ACS. These include Nondipolar electrical dispersion, VAT, QRS and T angles and principal component analysis ratio of ECG wave forms. It has the potential to improve ACS diagnosis in the ER and reduce the dwell time of patients. The software can be easily integrated into ECG platforms for easy interpretation.

Myocarditis Pericarditis, Pericardial Effusion and Tamponade

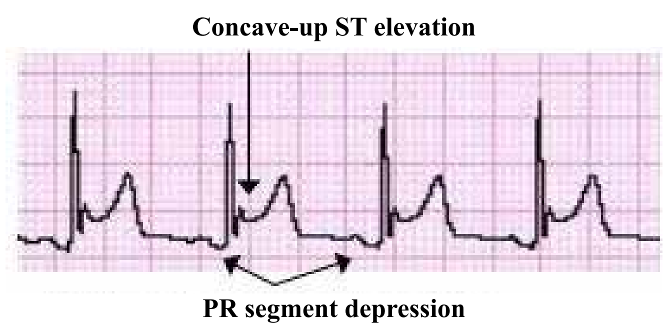
Acute Myocarditis presents to the ER with features of LV Dysfunction and Cardiac Failure with Dyspnea; Acute Pericarditis on the other hand presents to the ER with Chest Pain or with features of Pericardial Effusion or Tamponade. The Chest Pain is precordial and partly positional. The pericardial sac is a protective double-layered membrane, and when fluid accumulates excessively and rapidly in this space, it can compress the heart, leading to a decrease in preload and cardiac output which can further lead to shock and even cardiac arrest. With Pericardial Effusion and Tamponade, patients present to the ER with Dyspnea, Restlessness, Hypotension and even shock. Physical examination may reveal Triphasic Pericardial rub or Beck's triad, which consists of hypotension, distended neck veins, and muffled heart sounds accompanied with Pulsus paradoxus.

ECG in Acute Pericarditis consist of (1) PR segment depression maximal in II, aVF, V4-V6. (2) Diffuse ST segment elevation that is concave upwardly, not confined to coronary artery distribution, ST/T ratio >0.25 in V6 and ST depression in aVR. (3) T inversion . The ST elevation is related to the Pericardial inflammation that extends to the Epicardium. Spodik[3] described four stages starting with ST elevation and T wave concordance with ending with T inversion without Q waves. Electrical alternans is a characteristic finding in cardiac tamponade. It refers to alternating amplitude of the QRS complexes due to swinging of the heart within the pericardial fluid. The Triad of Electrical Alternans, Low QRS voltage and PR segment depression is a classic combination but with low sensitivity 17% and high specificity 90%.With associated Myocarditis there can be sinus Tachycardia, AV Blocks, Bundle branch blocks, Prolonged QT, Arrythmias (SVT /AF/VT) and ST changes elevation or depression. Bedside Echo is diagnostic in these cases

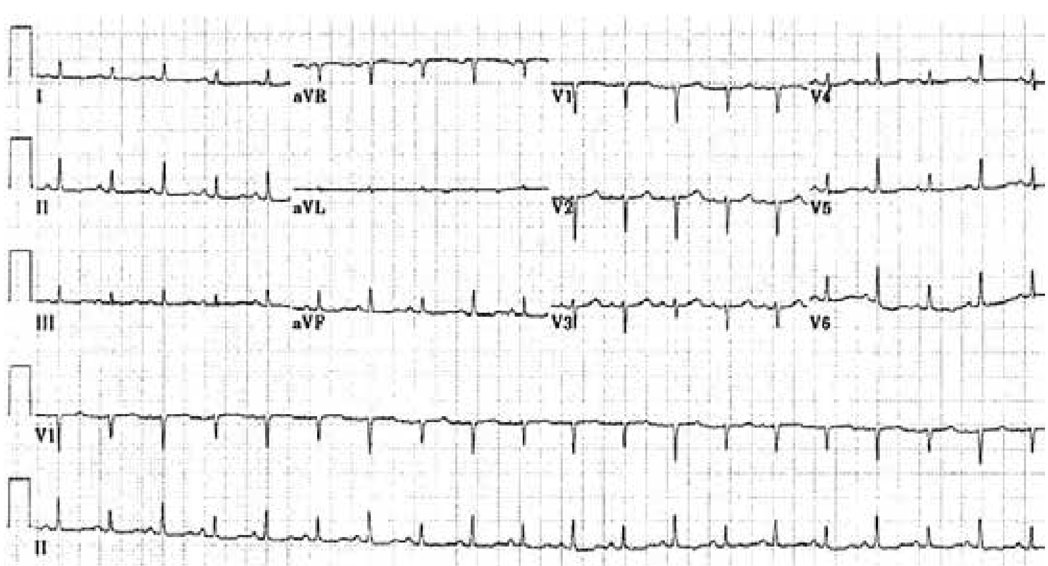
Syncope

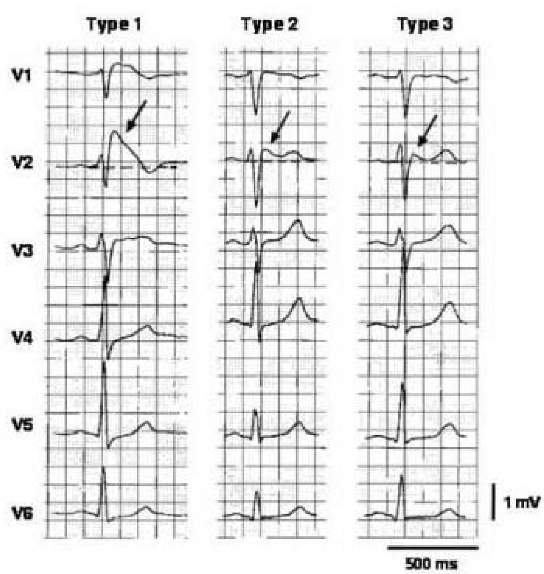
Patients are often rushed to the ER following an episode of Syncope or Resuscitated Cardiac Arrest. The earliest response is to note the Blood pressure, Saturation and the Rhythm as the immediate requirement is to stabilise the respiration, blood pressure and rhythm to sustain life. An immediate ECG can reveal any abnormal rhythm that may have resulted in a Tachy or Brady Arrhythmia leading to the cardiac event. Rather than dwelling on the clinical syndrome that has resulted in the event, we will discuss the individual disease and the ECG manifestations.

Sudden Cardiac Death can occur in the background of LV dysfunction related to Ischemic Heart Disease, and



(Picture taken from ECG Library.Courtesy-Internet)





(Courtesy:Medscape)

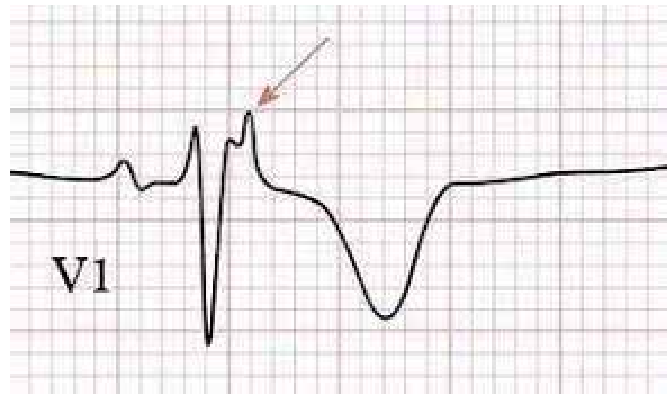
a variety of inherited conditions that includes Brugada syndrome, Hypertrophic Cardiomyopathy, Arrhythmogenic Cardiomyopathy, Long QT syndrome.

Brugada Syndrome is a genetic channelopathy related to autosomal dominant mutation of the cardiac sodium channel gene SCN5A. The ECG features are 1. RBBB with ST elevation in V1-V2 with downsloping morphology and inverted T waves. There is often additional PR prolongation. Type 1 has ST segment elevation convex upward and T inversion V1-V3. Type2 has a concave upward ST elevation that does not reach the baseline ; Type3: Concave ST elevation with ST reaching the baseline. S wave >80ms in V1, ST elevation >80ms from the J point are indicative of high risk for ventricular arrhythmias.

Hypertrophic Cardiomyopathy is an autosomal dominant mutation that has variable hypertrophy patterns with variable prognosis. ECG features include High amplitude QRS suggestive of LVH, Dominant R waves V1V2, abnormal Q waves, Deep T inversions and non specific IV conduction delay.

Arrhythmogenic Cardiomyopathy is an autosomal dominant genetic cardiomyopathy with features of RV Dysplasia and Ventricular Arrhythmias. EKG features include 1. Repolarisation abnormalities: Inverted T V1-V3, Inverted T V1-V4 with RBBB. 2. Depolarisation abnormalities: Epsilon wave V1V2. 3. Arrhythmias: Nonsustained VT: LBBB LAD. 4. QRS dur. V1 > V6.

Long QT Syndrome: Long QT syndrome associated with Syncope, Cardiac Arrest is characterised by (1) Prolonged QT interval and Bradycardia related Torsade de pointes Ventricular Tachycardia.. Drug and Electrolyte related (hypokalemia and hypomagnesemia) Torsade de pointes is often a forme fruste variant or related to a less Dominant gene



that manifests during medication or electrolyte deficiency. Corrected QTc longer than 440ms is prolonged; but the risk is highest when more than 500ms. Lead II is the best single lead for measuring QT interval. (2) T wave abnormalities: T wave alternans, Notched T wave . T morphology correlates with the Type of LQT: a. LQT1: Broad T wave. IKs channel and KCNQ1 gene. B. LQT2 Split or low amplitude late T wave, IKr channel and HERG gene. c. LQT3: Late onset Biphasic or peaked T with bradycardia, INa channel and SCN5A gene, bradycardia with Long short sequences (3) Clinical history of SCD in the family and Congenital deafness.

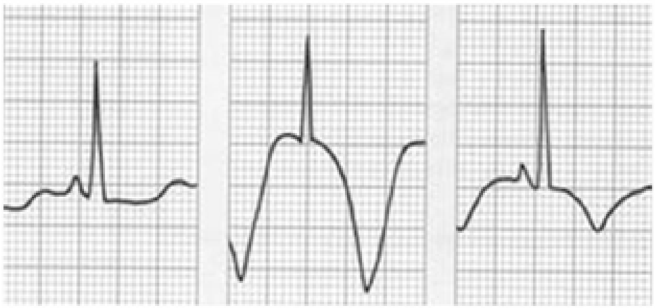
Abnormal ECG Finding	CM (%)	ARVC (%)	DCM (%)
T-wave inversions	54	15-85	15-45
Pathologic Q waves	42	-	10-25
ST-segment depression	46	-	-
Conduction delay	18	29-64	-
LBBB (Left bundle branch block)	2	-	9-20
QTc prolongation	13	-	-
Left axis deviation	15	-	15-25
Prolonged S-wave upstroke	-	24-95	-
Epsilon waves	-	9-33	-
Goldberger's triad	-	-	90*
Goldberger's triad	(1) SV1 or SV2 + RV5 or RV6 > 3.5 mV, (2) Total QRS amplitude in limb leads <0.8 mV, (3) R/S ratio <1 in V4		

ECG in Other Medical Emergencies

CVA

The relationship between acute strokes, particularly subarachnoid hemorrhage, and electrocardiographic (ECG) abnormalities is complex and often presents a diagnostic challenge. The sustained sympathetic stimulation can be triggered by various factors associated with stroke, including increased intracranial pressure, hypothalamic stimulation,

ECG Changes in stroke



Picture courtesey: www.medscape.com

From Burch GE, Meyers R. Abildskov JA: A new electrocardiographic pattern observed in cerebrovascular accidents. Circulation 9:720, 1954)

cardiac nerve stimulation, or activity in an arrhythmogenic center in the insular cortex.

In SAH, the incidence of ECG abnormalities ranges from 49% to 100%, including changes in ST segment (15% to 51% of patients), T-waves (12% to 92%), prominent U-waves (4% to 47%), QT prolongation (11% to 66%), pathological Q waves, and sinus dysrhythmias. Abnormal T-wave was the most abnormal ECG findings in patients with ischemic stroke. The characteristic ECG changes seen with raised intracranial tension are widespread giant T wave inversions ("cerebral T waves"), QT prolongation and bradycardia (The Cushing Reflex which indicates imminent brain herniation).

Dyselectrolytemia

a. Hypokalemia - Hypokalaemia is defined as a serum potassium level of < 3.5 mmol/L. ECG changes generally do not manifest until there is a moderate degree of hypokalaemia (2.5-2.9 mmol/L). The earliest ECG manifestation of hypokalaemia is a decrease in T wave amplitude. Other ECG changes seen are PR interval prolongation, widespread ST depression or T wave inversion / flattening, prominent U waves and prolonged QT interval. Hypokalemia is mostly seen associated with hypomagnesemia.

b. Hyperkalemia - Hyperkalaemia is defined as a serum potassium level of > 5.2 mmol/L. ECG changes generally do not manifest until there is a moderate degree of hyperkalaemia (≥ 6.0 mmol/L). The earliest manifestation of hyperkalaemia is an increase in T wave amplitude. Other changes seen are PR interval prolongation, bradyarrhythmias, conduction blocks and QRS widening with bizarre morphology. With worsening hyperkalemia, the QRS further widens causing the appearance of sine wave (pre terminal rhythm) and dangerous VF can also occur.

c. Hypomagnesemia - Normal serum magnesium levels are generally considered to be 0.8 – 1.0 mmol/L. Hypomagnesaemia, defined as a level < 0.8 mmol/L, is associated with QT interval prolongation and an increased risk of ventricular arrhythmias. Other changes seen are PR interval prolongation and predisposition to ventricular arrhythmias.

d. Hypercalcemia – 90% of all causes of hyercalcemia are caused by primary hyperparathyroidism or malignancies. Some of the common ECG changes seen are shortening of the QT interval, prolongation of the QRS duration and ST – T changes and even malignant ventricular arrhythmias.

e. Hypocalcemia – Renal failure, poor gastrointestinal absorption, sepsis, acute pancreatitis etc are some of the common causes of hypocalcemia. Some of the common ECG changes are prolonged QT interval, AV blocks and sinus bradycardia.

ECG in Medical Emergencies related to Febrile Illness

It is not unusual to have patients report to ER with a febrile illness and have cardiac symptoms in the form of Chest Pain, Dyspnea , Hypotension. The following common episodes are discussed

Sepsis: ECG changes can occur in septic patients due to reduced excitability of myocardium. Rich et al. demonstrated significant reduction in amplitude of QRS during sepsis which normalised after recovery.28 Some accompanying ECG changes observed in the cases of sepsis include prolonged QTc interval, bundle branch blocks, Osborn waves, nonspecific ST changes, and very rarely ST-segment elevations.29 Rare cases of Enteric fever associated sepsis, encephalopathy and reversible first degree AV block with right bundle branch block not attributable to myocarditis have been reported.[4]

COVID-19 and ECG: The JAMA in 2020 reported that arrhythmia (16.7%) and acute cardiac injury (7.2%) are the most prevalent cardiac complications in COVID patients.[5] 31 The ECG in COVID infection may mimic ventricular strain patterns, especially when associated with elevated Troponins. A variety of arrhythmias may be manifest on the ECG tracing, with Atrial fibrillation the most common. COVID myocarditis may manifest as abnormalities in the T wave and ST segment, or, sinus tachycardia and conduction defects (such as right- and left-bundle branch blocks and atrioventricular blocks) may also indicate myocarditis. The ACC has cautioned of AMI going undiagnosed on the background of COVID, especially with the non-specific elevation of biomarkers, and reminded Cardiologists that pathologic Q waves (as with prior scar tissue or myocardial infarction) and RSR' patterns might predict cardiac arrest in COVID patients. Workers from Tamil Nadu, in south India studied 315 COVID patients, and found sinus tachycardia (23.8%), sinus bradycardia (12.7%), atrial arrhythmia (3.5%), ischemic changes (32.4%), QT segment changes (16.2%), with QTc prolongation, ST-T changes and sinus tachycardia being most strongly associated with COVID related mortality. In a publication from Washington, the authors have identified QRS and QTc intervals are early markers for COVID-19 disease progression and mortality.34

Dengue

Dengue often presents to the ER with features of Haemorrhagic shock and Hypovolemia. The following are noted: Tachycardia: Increased heart rate is a common finding in Dengue fever, reflecting the body's response to the infection. Bradycardia: In some cases, particularly during the critical phase of Dengue fever, bradycardia (abnormally slow heart rate) may be observed. Arrhythmias: Dengue infection can lead to various arrhythmias, including atrial fibrillation, atrial flutter, and other supraventricular and ventricular arrhythmias. ST-segment Changes: ECG may show ST-segment changes, such as ST-segment depression or elevation, which can be indicative of myocardial involvement. QT Prolongation: Dengue fever may cause prolongation of the QT interval on the ECG, which is associated with an increased risk of arrhythmias. Atrioventricular (AV) Conduction Abnormalities: Some patients may experience disturbances in AV conduction, leading to conditions like heart block.

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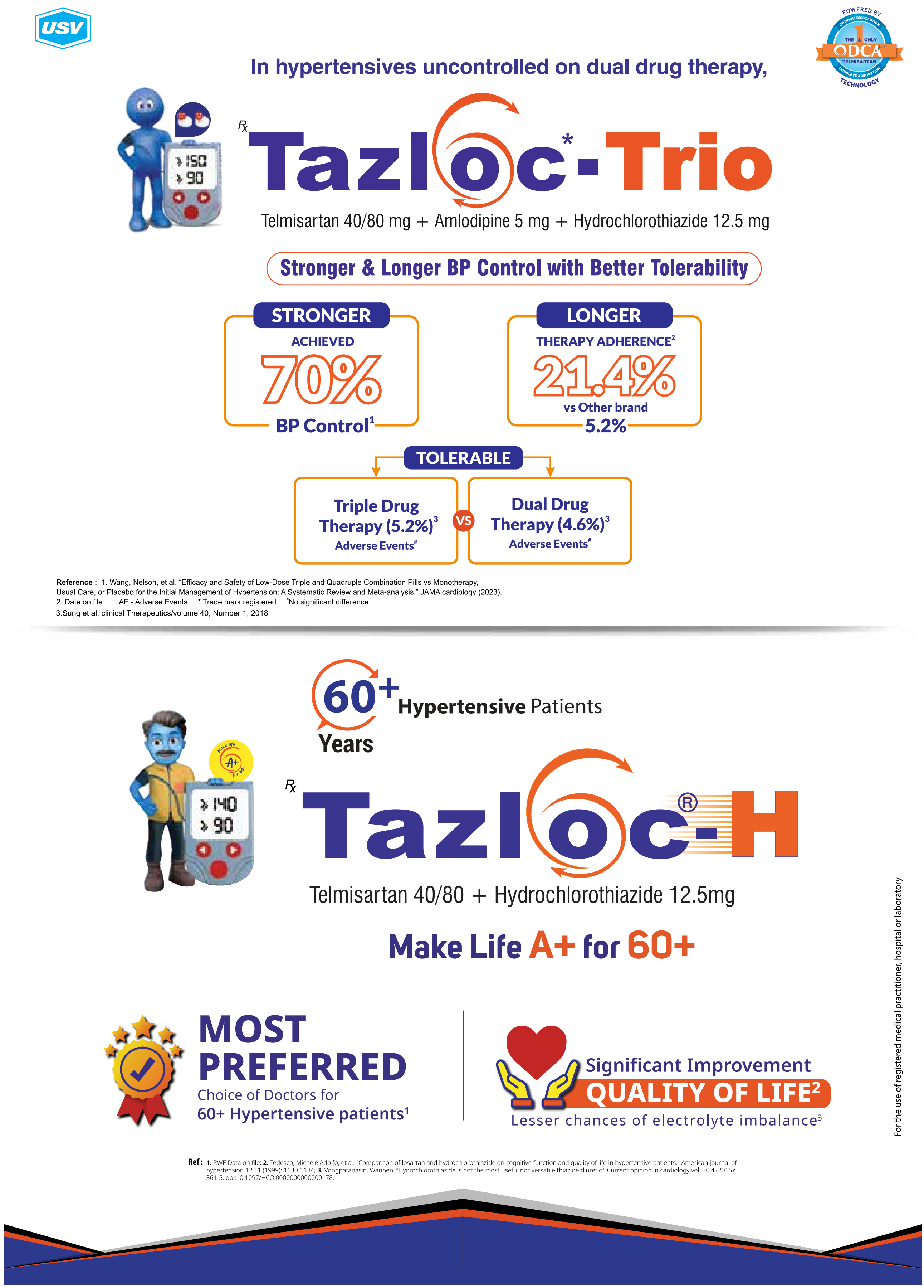
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3.Sung et al, clinical Therapeutics/volume 40, Number 1, 2018
60+ Years Hypertensive Patients
140
90
Make life A+ for 60+
Rx Tazloc®-H
Telmisartan 40/80 + Hydrochlorothiazide 12.5mg
Make Life A+ for 60+
MOST PREFERRED
Choice of Doctors for
60+ Hypertensive patients[1]
Significant Improvement
QUALITY OF LIFE[2]
Lesser chances of electrolyte imbalance[3]
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*ARB + BB **BB *Telmisartan + Metoprolol: ARB+B **Metoprolol: BB
In hypertensive patients with CAD,
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In AMI* patients undergoing PCI**1
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POST PCI** METOPROLOL2
Reference :
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*AMI: Acute Myocardial Infarction # Telmisartan + Metoprolol: ARB + BB **PCI: Percutaneous coronary intervention

All About Premature Ventricular Contractions: A Short Review

Sheikh Mohamad Tahir, Daljeet Kaur Saggu
MD, DM, AIG Hospital, Hyderabad, India

Abstract

Sixty-one-year gentleman a case of coronary artery disease (CAD) with old anterior wall myocardial infarction, severe left ventricular (LV) dysfunction admitted with refractory heart failure (HF). He was started on dobutamine and a diuretic infusion. His baseline investigations including renal parameters and electrolytes were essentially normal. There was no evidence of any underlying uncorrected ischemia. He was implanted with cardiac resynchronization pacemaker with defibrillator (CRT-D) a year back and was on guideline directed medical therapy (GDMT) for HF till hospitalization. His ECG (figure1a, b) showed frequent premature ventricular contractions (PVCs) which were hindering his biventricular pacing (BiVP). These PVCs were refractory to multiple antiarrhythmic drugs (AADs). Apart from this, we could not find any other obvious reversible cause of HF for him.

How to approach this case?

Introduction

Premature ventricular complexes are common and can occur in a wide variety of clinical scenarios and in a diverse population. They can present in patients both with and without preexisting cardiac disease. Whenever possible, investigating for the underlying reversible cause of PVCs is of outmost clinical significance.

Prevalence

Many of the studies that describe the prevalence of PVCs, arise from the databases of ECGs or Holter monitor studies among those seeking clinical care, where the prevalence is expected to be elevated. It is not surprising that the prevalence of PVCs increases as the duration of ECG monitoring increases. In the ARIC study, a 2-minute ECG detected PVCs in 5.5% of the population.[1] In the Framingham Heart Study, over one hour of monitoring evidence of PVCs was observed in approximately 12% of individuals without CAD.[2]

Predictors

An analysis using the ARIC study cohort demonstrated that older age, male sex, taller height, black race, a history of hypertension, lower left ventricular ejection fraction (LVEF), evidence of any cardiac disease, a higher heart rate, and hypomagnesemia were each associated with the presence of at least 1 PVC during a 2-minute ECG recording, after multivariable adjustment.[3,4,5] Potentially modifiable risk factors observed across various cohorts and methodological approaches include higher blood pressure, less physical activity, and smoking.[5]

Mechanism

The actual mechanism of PVCs in many cases is not known, and more than 1 process may ultimately be responsible. There are 3 basic mechanisms that might be operative including triggered activity, automaticity, and reentry. Triggered activity is generally attributed to after depolarizations initiated by increases in intracellular calcium.[6] Early afterdepolarizations (EADs), occurring in the plateau phase of the action potential, classically arise in the setting of prolonged repolarization, resulting in the PVCs that may initiate torsades de pointes (TdP) in those with congenital or acquired forms of the long-QT syndrome (LQTS) (Figure 2a, b).

Delayed afterdepolarizations (DADs) occur at repolarized membrane potentials and are classic manifestations of digitalis toxicity[7] or catecholaminergic polymorphic ventricular tachycardia (CPVT) (figure 2c).[8] Caffeine is also known to result in DADs through the release of calcium from the sarcoplasmic reticulum.[9] In contrast with a triggered mechanism, PVCs arising from automaticity may exhibit parasystole. Here, the PVCs should march through at the same cycle length, independent of the underlying rhythm. It is important to note that the absence of PVC may occur intermittently because of ventricular refractoriness related to the separate underlying rhythm, and therefore multiples of the parasystolic PVC interval should be considered before excluding automaticity.[10-12] The clear and consistent predilection for PVCs to arise from certain anatomic regions, such as the outflow tract, has led to a speculation that the origins of the ectopic focus may relate to shared embryological development with cell or tissue types physiologically destined for automaticity, such as specialized conduction tissue.[13]

Reentry requires 2 distinct electric pathways with different conduction properties and refractoriness which can cause transient or permanent unidirectional block in one limb. In general, an area of scar, or a series of electrically connected cardiomyocytes meandering through an area of fibrosis, may provide a pathway that conducts much more slowly than surrounding healthier tissue during a sinus beat, such that the resultant exiting depolarizing wave front would meet

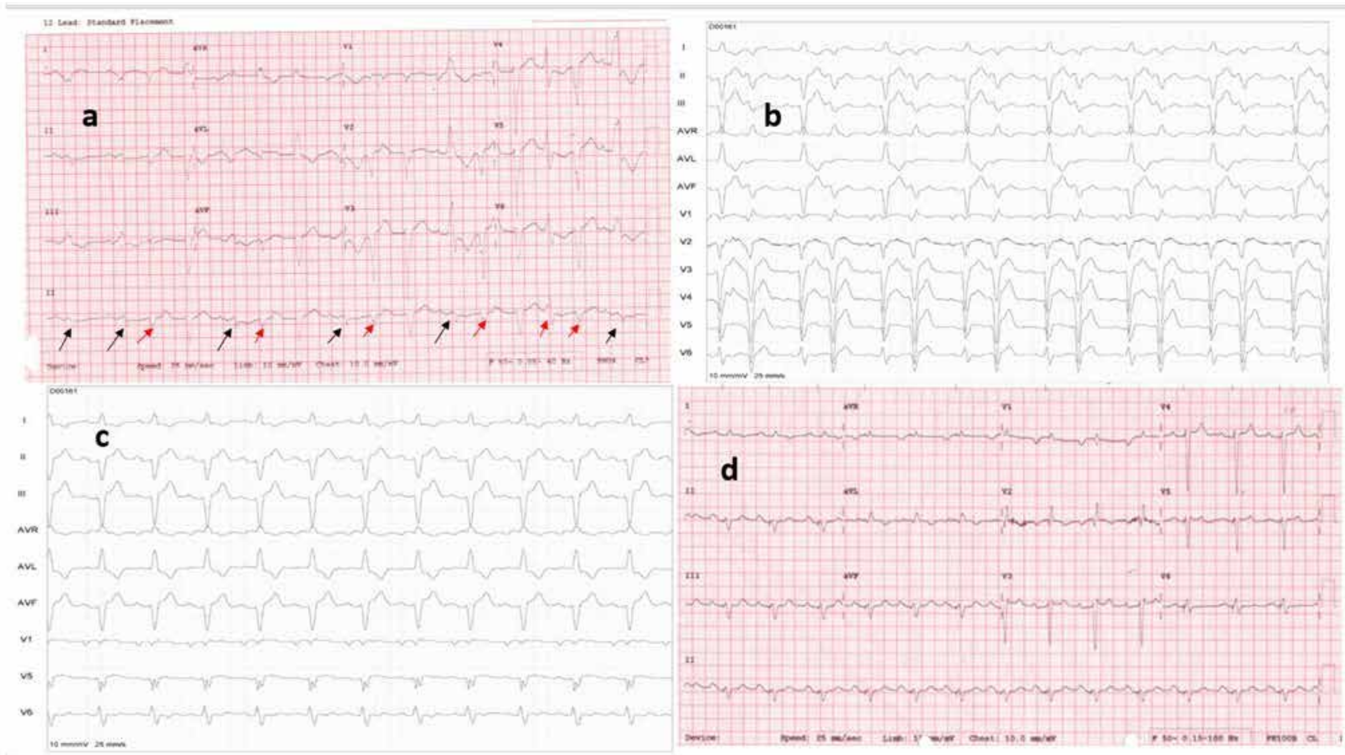

Figure 1: 12 lead surface electrocardiograms (ECGs) of our patient who presented with refractory heart failure (HF). a: Baseline 12 lead ECG when patient presented with HF. Red arrows showing frequent premature ventricular contractions (PVCs), black arrows showing biventricular paced (BIVP) beats and remaining beats are intrinsic beats conducting with long PR interval. b: 12 lead ECG with BIVP switched off, showing frequent PVCs in the form of ventricular bigeminy. c: 12 lead ECG after catheter ablation for PVCs showing clean baseline ECG with underlying left bundle branch block. d: 12 lead ECG of same patient after catheter ablation for PVC with BIVP switched on, showing narrow QRS suggestive of optimal BIVP. See text for details.

myocardium that was no longer refractory, producing a PVC.[14] Although re-entry is usually considered most pertinent to sustained arrhythmias, it can play a role in single PVCs.

Evaluation

Patients with PVCs typically come to clinical attention either because of symptoms or an incidental finding during clinical examination. PVCs can present with highly varied clinical symptoms ranging from a feeling of skipped beats, palpitations, chest discomfort, fatigue, tiredness to shortness of breath especially if frequent PVCs are contributing to arrhythmia induced cardiomyopathy (AICMP). Patients can present with giddiness, blackouts, syncope, and sudden cardiac arrest (SCA) if PVCs are inducing sustained ventricular arrhythmias (VAs) (figure 2d).[15-18] Although there are no established guidelines to identify patients with PVC at the highest risk of PVC triggered ventricular fibrillation (VF), the clinical pearl is to consider PVCs as potentially playing a causal role when VF is observed. A family history is important to elucidate any possible inherited disorders that may be associated with PVCs and a risk for SCA. As an initial screen, it is often useful to recommend asking about any first-degree family members who either died suddenly or at an early age. A positive family history in a patient with PVCs should heighten suspicion for inherited cardiomyopathies or channelopathies. In the absence of symptoms, PVCs may manifest as an irregular pulse or as an incidental finding on ECG. Both patients and healthcare providers may be alarmed by apparent bradycardia ascertained by a palpable pulse alone, occurring because of intermittent, poorly perfused PVCs.

Initial evaluation of PVC is best performed by a baseline 12 lead ECG. When a PVC is observed, it is useful to run a 12-lead rhythm strip over 30 to 50 seconds. On this long rhythm strip a clinician can evaluate frequency, morphology (single versus multiple, outflow versus non outflow morphology PVC), short or long coupled, fixed or variable coupling interval of PVCs. Attention to the remainder of the ECG may also reveal clues to the underlying substrate: careful measurement of the QT interval is mandatory (figure 2a), precordial T-wave inversion beyond lead V2 or right ventricular conduction delay may be indicative of arrhythmogenic right ventricular dysplasia (ARVD), pathological Q waves can reflect discrete areas of scar, an early precordial transition accompanied by a prominent S wave in V6 may signal a basolateral scar observed in nonischemic cardiomyopathy, and conduction disease may be a manifestation of cardiac sarcoidosis (CS) or myocardial tuberculosis (MTB).[19-21] PVCs arising from common sites, such as the right ventricular outflow tract (RVOT), may sometimes trigger VF in patients with underlying Brugada syndrome and TdP in patients with LQTS.[22,23] Recent

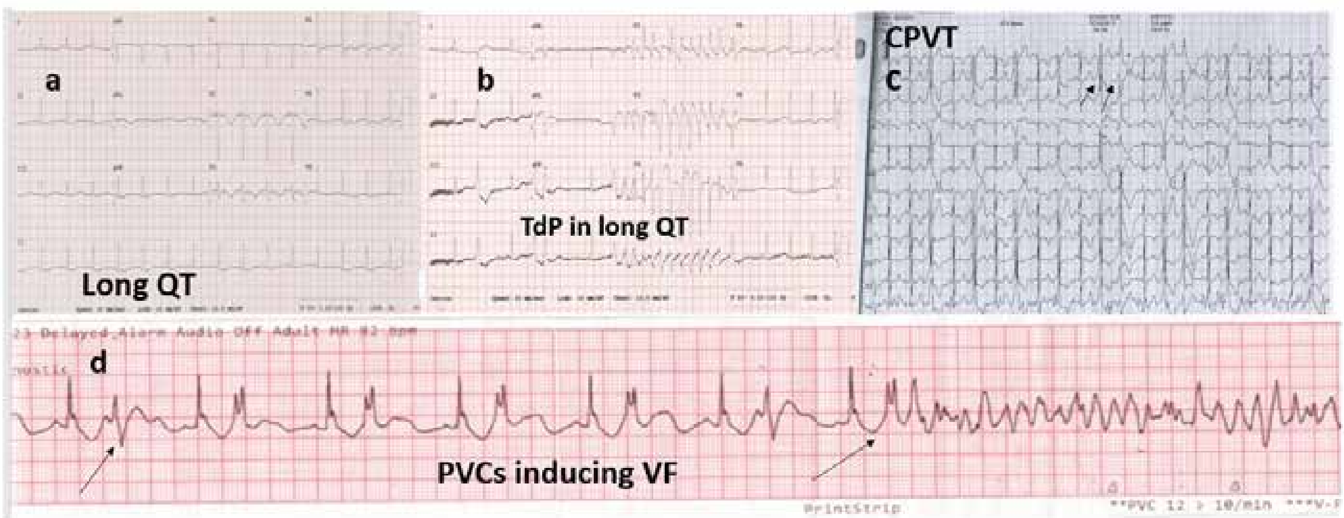

Figure 2: a: Baseline 12 lead electrocardiogram (ECG) of a patient with long QT syndrome. **b:** 12 lead ECG of same patient as in 'a' showing prolong QT and Torsades de Pointes(TdP) induced by premature ventricular contractions (PVCs). **c:** 12 lead ECG during stage 3 exercise of a case of catecholaminergic polymorphic ventricular tachycardia (CPVT) showing bidirectional (black arrows) PVCs. d: Single lead ECG of a diagnosed case of Brugada syndrome showing ventricular bigeminy (of more than one morphology) inducing ventricular fibrillation(VF) because of R on T phenomenon.

evidence has demonstrated that substantial daily variation may occur and a week of extended ECG monitoring may be required before the maximum daily PVC frequency is observed.[24] Longer duration of monitoring will also be helpful to determine symptom correlation, different morphologies of PVCs, fixed or changing coupling interval PVCs, nocturnal variation, relation with exertion and thus help to risk stratify them. PVCs from the outflow tract appear to be the most common[25] followed by fascicular, papillary muscle and mitral annular PVCs.[26-32] Other common sites of PVC origin are in proximity to venous structures, such as the great cardiac vein and anterior intraventricular vein and from the crux of the heart, where all 4 cardiac chambers meet.[33-36]

Imaging

Following clinical evaluation and ECG, echocardiography (echo) is the next most important imaging modality to evaluate for any obvious structural heart disease(SHD). A cardiac magnetic resonance imaging with late gadolinium enhancement (CMRI-LGE) should be considered when the PVC is not arising from a common location (such as RVOT) or when sustained VA or reduced ventricular function is present. Of note, CMRI images may be difficult to interpret in the context of frequent PVCs because of gating difficulties. The CMRI can be helpful in making a diagnosis of ARVD or when there is a suspicion for CS/MTB.[38-39] Even in the absence of a clear underlying diagnosis, LGE observed on CMRI can help visualize cardiac scar, potentially helping to plan for and guide catheter ablation (CA) procedures.[40,41] Among those with evidence of LGE, a positron emission tomography (PET) scan may be particularly useful in assessing infiltrative and inflammatory processes.[39]

Prognosis

Data from a community-based study, Cardiovascular Health Study, and armed with baseline echo data with a median follow-up of nearly 14 years, it is demonstrated that a greater frequency of PVCs is associated with a 5-year reduction in LVEF, increased risk of HF and an increased risk for death.[42] Among patients presenting for clinical care for their HF and PVCs, a higher burden of PVCs has consistently been shown to be an important risk factor.[43] Although there is no clear single threshold cutoff, studies have suggested that optimal test characteristics for a PVC related AICMP occur at a PVC burdens of >10%.[44-48] Additional factors reported to be associated with AICMP include male sex, asymptomatic PVCs, longer duration of palpitations, interpolated PVCs, variable PVC coupling interval, longer PVC QRS duration **and** PVCs arising from epicardium.[49-55]

Management

For the clinical management of patients presenting with PVCs, 3 key pieces of information are needed: (1) information regarding symptoms, (2) the burden and morphology of PVCs, and (3) the presence or absence of SHD. If a patient is otherwise healthy without any systemic disease, is physically active without limitation, does not have a history of syncope or symptoms compatible with VA, does not have a family history suggestive of SCA, and neither a resting ECG (with a long rhythm strip as described earlier) nor physical examination reveal any PVCs (in support of a low PVC burden) or other ECG abnormalities (discussed earlier), it is reasonable to stop there without ordering further testing. Patients can always be encouraged to return if, after reassurance, they develop worsening symptoms. Sometimes, PVCs are incidental finding in an otherwise sick patient e.g. in ongoing ischemia, multiorgan dysfunction, sepsis, acute kidney injury, electrolyte imbalance, autoimmune disorders etc. In such instances it is important to treat underlying condition first and then reassess the frequency and the effect on hemodynamic of PVCs and decide accordingly. The goal ultimately is to determine the genetic and environmental factors responsible for problematic PVCs, so that underlying cause can be treated precisely. Expert guidelines commonly call for avoidance of caffeine.[56)] If a patient has bothersome symptoms despite reassurance or a reduced LVEF, either medical treatment or CA are reasonable first options. In general, CA exhibits superior effectiveness.[46,57,58]

Medical therapy

Adenosine, via action on the adenosine A1 receptor, which itself inhibits production of adenylyl cyclase (and hence cAMP), can terminate triggered PVCs.[59] Because catecholamines also activate cAMP, β-blockers (BB) may reduce PVCs arising from triggered activity**.** Last, nondihydropyridine calcium channel blockers (CCBs: diltiazem and verapamil) may prevent triggered PVCs by reducing cytosolic calcium accumulation through blockage of L-type calcium channels.[60] Thus, either BB or nondihydropyridine CCBs are considered first-line medicines for PVCs. Both have a long track record of safety in structurally normal hearts, and BB may have additional benefits in the setting of CAD or reduced LVEF. β-Blockers are particularly effective for sympathetically mediated, triggered PVCs, with data demonstrating effectiveness specifically in outflow tract PVCs.[56,61] The nondihydropyridine CCBs have similarly demonstrated effectiveness in outflow tract PVCs and are considered particularly useful for fascicular VA.[56,62,63] Second line drugs if BB or CCBs fails, are flecainide, propafenone, sotalol, and amiodarone. If AADs fail, CA should be considered next. Mexiletine may be used rarely, but its effectiveness is inferior to either other AADs or CA.[46, 64] Flecainide and propafenone are well-tolerated, in general, and are often efficacious.[46, 58, 64] Sotalol can suppress PVCs in most patients and is a particularly reasonable choice in the presence of CAD but, careful monitoring of the QT interval is critical.[65-67] Although amiodarone is efficacious and is one of the few AADs that can be safely administered in the setting of severe LV dysfunction the associated side-effect profile, in particular, with long-term use, makes it substantially less preferable in younger patients. In general, amiodarone is reserved for older patients and for those with no other options.[68] Cather ablation is more efficacious than medicines to treat PVCs particularly for a monomorphic target.[46, 57,58] Success of PVC ablation procedures ranges from approximately 80% to 95%.[25] CA is listed as a class I indication to treat PVCs if medicines are not tolerated, not effective, or preferred by the patient.

Going back to our clinical case who had persistent PVCs refractory to multiple AADs and was in refractory HF: CA successfully eliminated these PVCs (figure 1c, d) and patient achieved >99% BiVP. There was immediate improvement in clinical symptoms and over more than 4years of follow-up there is not even a single HF related hospitalization and patient is in New York heart failure (NYHA) class II on GDMT.

Conclusions

PVCs are a commonly observed phenomenon that are best evaluated with a thorough history and physical examination and a 12-lead ECG, usually supplemented with ambulatory ECG monitoring and an echo. A CMRI-LGE is usually reserved for cases with suspected underlying SHD. Although many patients with PVCs may require only reassurance, treatment is generally reserved for underlying SHD, channelopathies, symptomatic PVCs or concern for AICMP. PVC burden remains the most reliable marker for PVC related AICMP. Observational data suggest that tobacco smoke and a sedentary lifestyle may promote more PVCs. β-Blockers, CCBs, or CA are each reasonable first-line strategies to treat PVCs. The mechanisms explaining the presence or absence of symptoms, and the optimal risk stratification for AICMP need to be further understood which will help in the development of targeted prevention strategies and therapeutics.

Conflicts of interest

None to declare.

Acknowledgement

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ECG in Sinus Node Dysfunction

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Abstract

Sinus Node Dysfunction previously known as Sick Sinus Syndrome, caused by the inability of sinus node to perform pacemaker function and impulse transmission leading to a variety of abnormal rhythms. Most of the time, it remains asymptomatic and needs no therapy. However when symptomatic, proper medications and need for pacemaker is to be considered.

Sinus Node Dysfunction (SND) is the inability of the heart's natural pacemaker (Sinus node) to create a heart rate that's appropriate for the body's needs. It causes irregular heart rhythms. Sick Sinus Syndrome is synonymous with sinus node dysfunction and can present with a constellation of abnormal rhythms :

- Sinus bradycardia
- SA arrest
- SA exit block
- Chronotropic incompetence
- Combination of SA/AV nodal conduction
- Paroxysmal Atrial tachyarrhythmias
- Tachy-Brady Syndrome
  - The rhythm disorder switches between very fast and very slow rates in an irregular way.
  - Appears as tachycardia and bradycardia episodes on an ECG.

Etiology

Sinus node dysfunction can occur because of :

a. Intrinsic factor – fibrosis of sinus nodal tissue itself because of
   - Age-related Degeneration[1]
   - Congenital Disorders
   - Infiltrative Disorders like sarcoidosis, amyloidosis etc.
   - Infections – Bacterial Endocarditis, Chagas Disease
   - Cardiac Surgery e.g. Valve replacement surgery, correction of congenital heart disease or heart transplant
   - Familial – Gene Mutations

b. External factors –
   - Increased Vagal Tone
   - Metabolic – Hypothyroidism, Hyperkalemia, Hypokalemia, Hypocalcaemia, Hypoxia, Hypothermia
   - Obstructive Sleep Apnea
   - Increased Intracranial Pressure
   - Drugs and Toxins like antiarrhythmic drugs, digoxin, lithium and sympatholytic medications

Incidence

Sick Sinus Syndrome is commonly seen in elderly persons, however it can occur at any age. The mean age of presentation is 68 years. SND is observed in one of every 600 cardiac patients of ≥ 65 years. Both genders are equally affected. More than 50% pacemakers were implanted in US in 1990 for SND.[2]

In India, approximately 1.1 Million people in our total population suffers from Sinus Node Dysfunction. As per data from various economic studies, approximately 15% of our population will be able to get a pacemaker implanted as a therapy for this indication. However, as per the rough estimate, annually only about 2500 pacemakers must be getting implanted on account of SND in India and the balance 140K patients are left untreated even though they have the means to afford pacemaker therapy.

Pathophysiology

Sinoatrial (SA) node have 2 specialized functional cells, a collection of atrial myocytes –

- "P cells" - the pacemaker cells for the intrinsic pacemaker function
- "T cells" – the cells responsible for propagating the impulse

Dysfunction of these cells results in several arrhythmias.

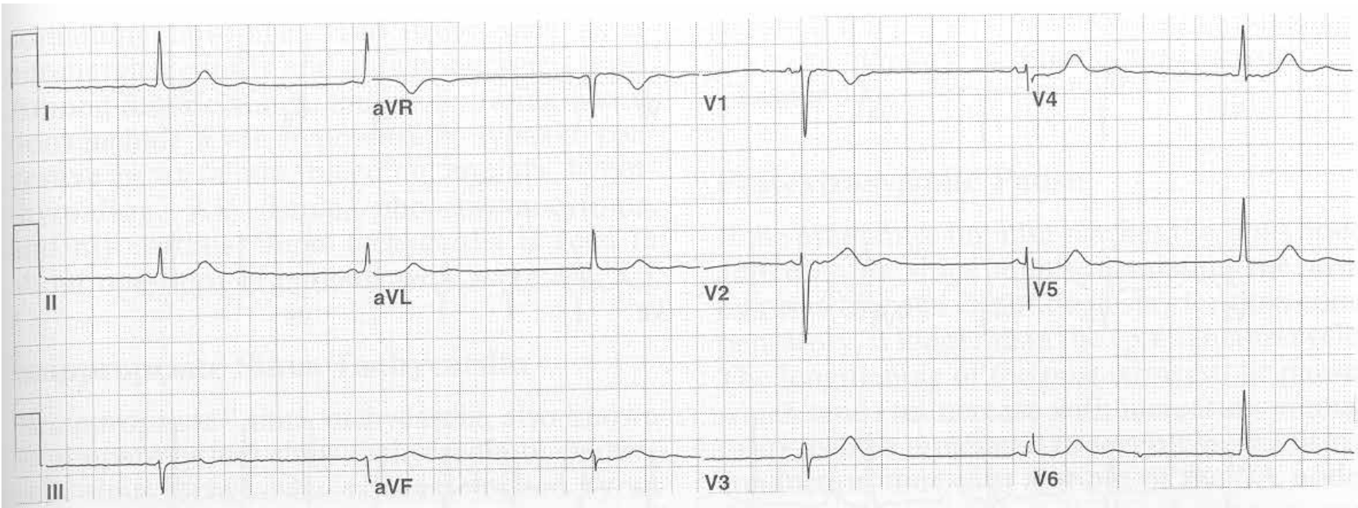

Figure 1: ECG showing severe sinus bradycardia

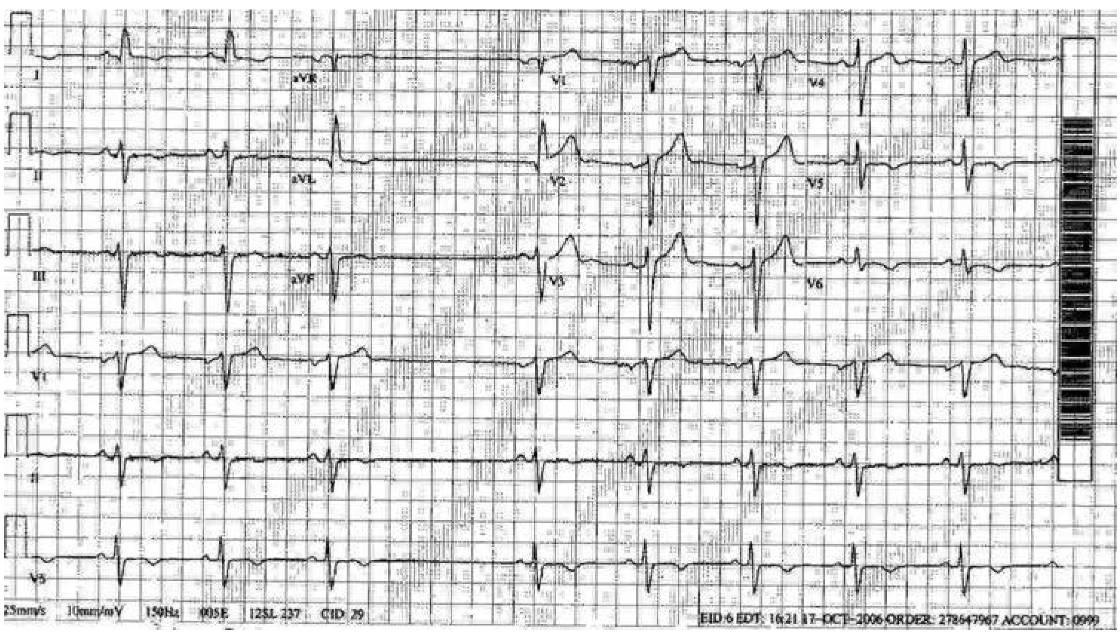

Figure 2: ECG showing sinus pause

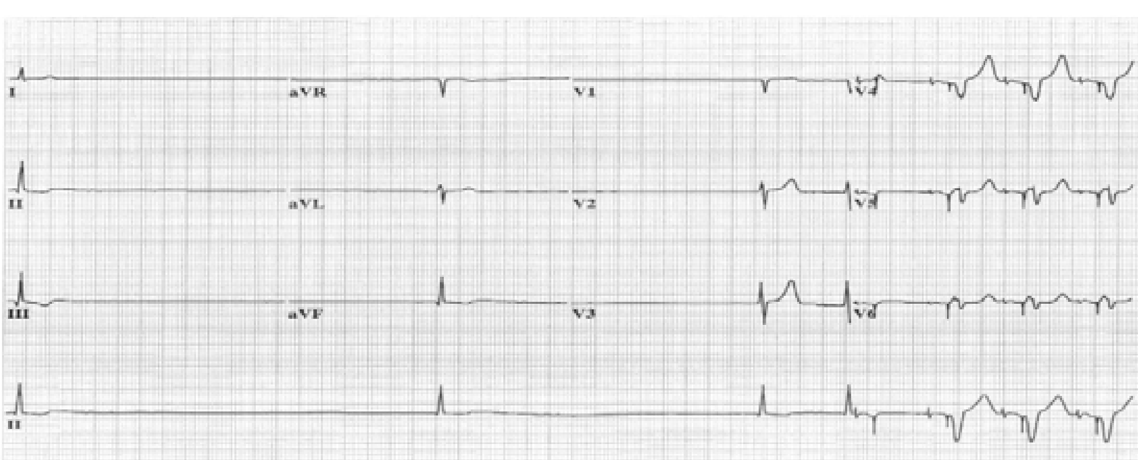

Figure 3: ECG showing sinus arrest needing pacemaker support

- Severe sinus bradycardia, which may be abrupt and inappropriate
- Sinus pause or sinus arrest, usually rescued by escape rhythm, atrial, junctional or ventricular
- SA Exit Blocks, first-, second- or third-degree, because of failure of "T" cells to transmit the impulse from SA node to right atrium
- Tachy-brady syndrome – Alternating bradycardia with supraventricular arrhythmias, most frequently atrial fibrillation. At least 50% of the patients of SND present with tachy-brady syndrome.
- Atrial fibrillation with slow ventricular response, not on AV node blocking drugs, likely because of the simultaneous affection of AV node. Annually, up to 4.5% with median of 0.6% of SND patients will develop AV blocks.[3]
- Chronotropic incompetence is seen in 20 to 60% of the patients[3].

Clinical

SND usually progresses slowly over decades and in early stages, patients of Sick Sinus Syndrome are usually asymptomatic. But depending on the heart rate, symptoms of Bradycardias and Tachycardias can occur. However, symptoms include :

- Syncope
- Pre-syncope
- Lethargy
- Fatigue,
- Exercise intolerance
- Unexplained falls
- Memory loss
- Palpitations

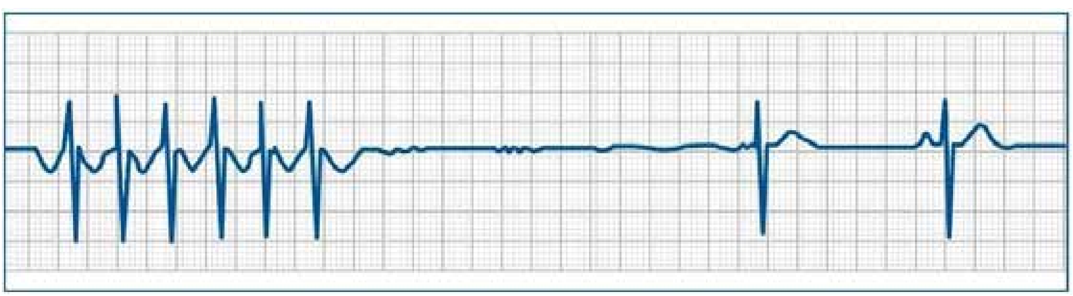

Figure 4: ECG showing Tachy-Brady Syndrome

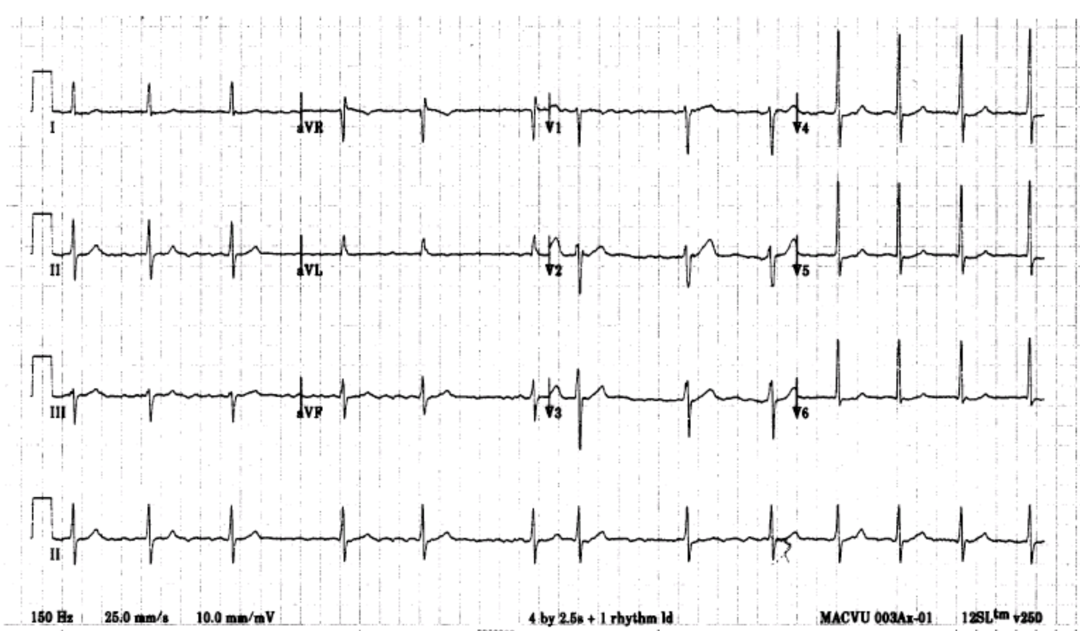

Figure 5: ECG showing atrial fibrillation with slow ventricular response

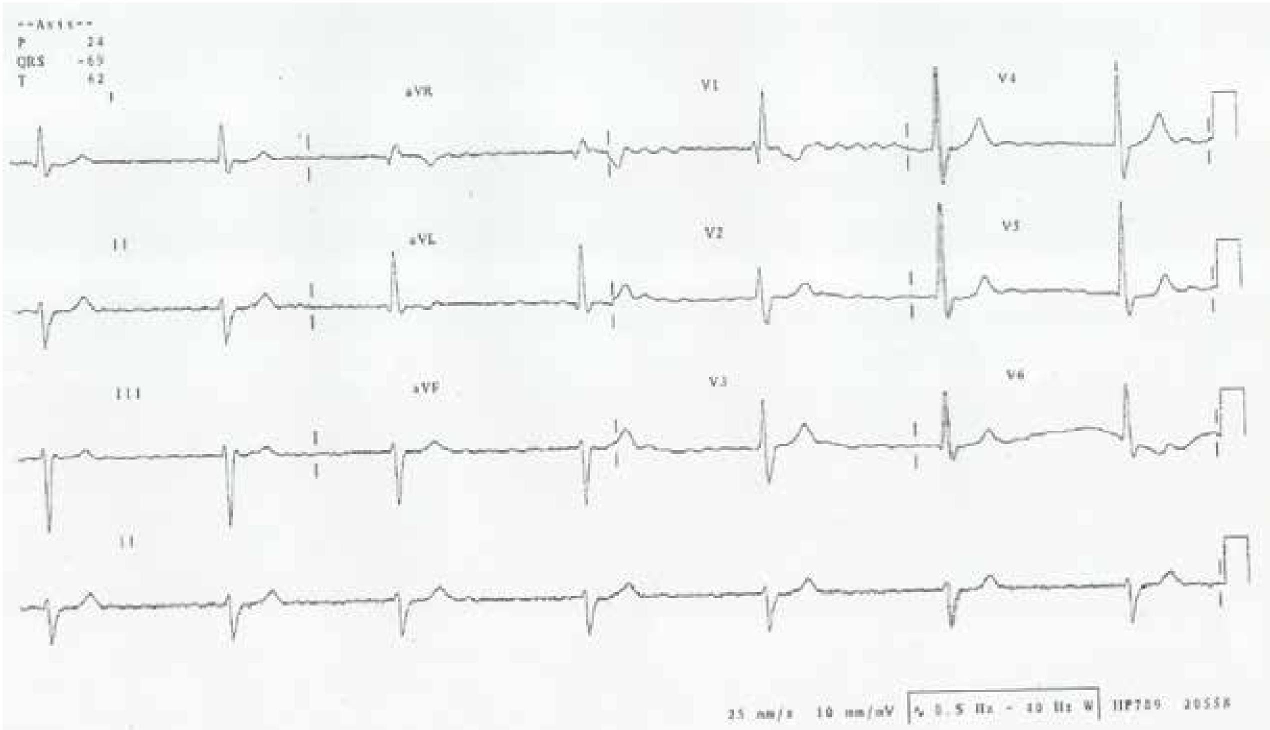

Figure 6: ECG showing atrial fibrillation with complete AV block

- SOB
    - Angina, Heart Failure

Central nervous system underperfusion because of severe sinus bradycardia or sinus pauses, manifests as presyncope or syncope. Prolonged sinus pause and failure to return to sinus rhythm after sudden termination of atrial fibrillation in tachy-brady syndrome may result in syncope. There is increased risk of thromboembolism due to paroxysmal atrial fibrillation or atrial flutter which may lead to transient ischemic attacks or cardioembolic stroke.

Diagnostic Work-up

Primary aim to evaluate the patients of SND :

1. To exclude the reversible causes
    a. Electrolyte disturbances
    b. Metabolic abnormalities
    c. Hypothyroidism
    d. Raised intracranial pressure
    e. Jaundice
    f. Obstructive sleep apnea
    g. Offending drugs like β-blockers, digoxin, calcium channel blockers, certain anti-arrhythmic drugs etc.
2. To correlate the patient's symptoms with the rhythm disturbances

As the nature of the disease is episodic, diagnosing sick sinus syndrome on 12-lead ECG is unusual and patient needs prolonged ECG monitoring to document the episodes and correlate the symptoms. Holter monitoring (24-72 hours), event recorders or external loop recorders (7-30 days monitoring) will help in achieving the diagnosis. Rarely, one may have to resort to further long ECG monitoring with internal loop recorders (1-3 years).

Whenever, there is strong suspicion of sick sinus syndrome and the investigations do not reveal any arrhythmias, then it is advised to go for electrophysiogical (EP) study. Treadmill exercise test can be done to look for chronological incompetence. When the patient achieves less than 80% of maximal predicted heart rate, chronological incompetence is diagnosed.

Treatment

Management of sinus node dysfunction is 3-pronged approach:

1. To identify and treat the reversible causes
2. To treat symptomatic tachycardia especially paroxysmal atrial fibrillation
3. To treat symptomatic bradycardia

After managing the reversible factors, the most important consideration is placement of permanent pacemaker in symptomatic patients of sinus node dysfunction, who have documented bradycardia responsible for the symptoms and in patients with proven chronic incompetence. Even patients

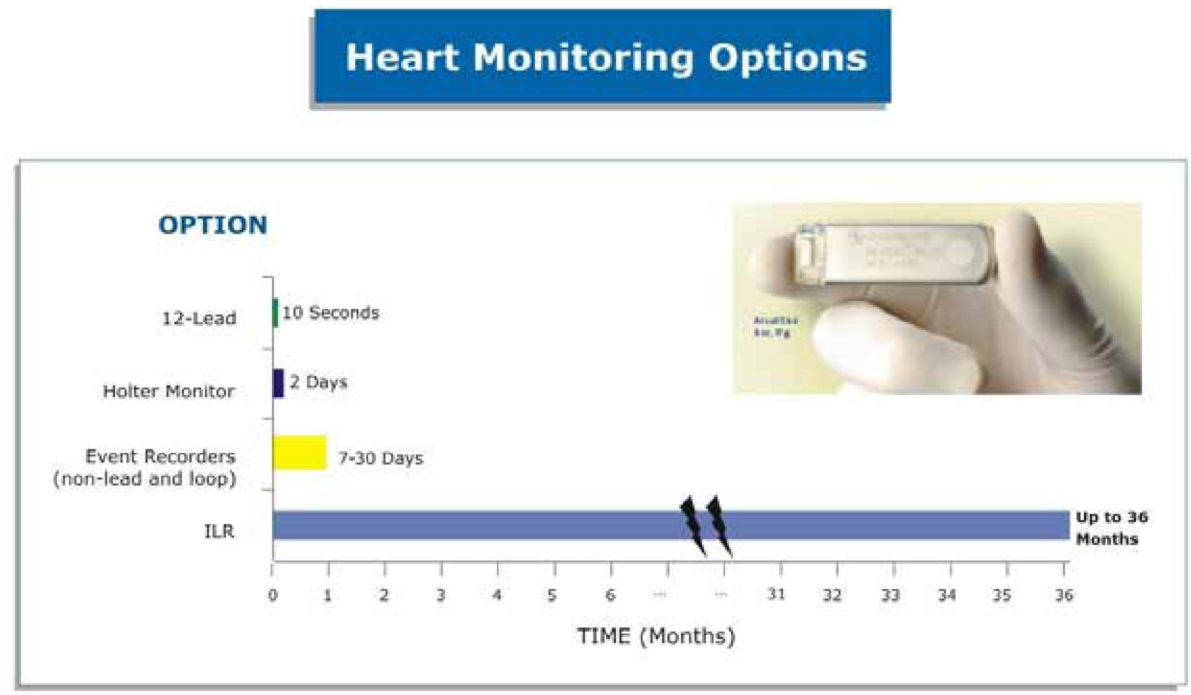

Figure 7: Showing ECG monitoring options

necessitating treatment for their tachycardia symptoms, will need permanent pacemaker implantation because the therapy will induce bradycardia, making patients symptomatic. Other medical conditions like thyrotoxicosis, migraine etc. requiring β-blocker therapy in patients with sinus node dysfunction will also need permanent pacemaker implantation. Even, permanent pacemaker implantation is indicated in symptomatic patients who have bradycardia with heart rate less than 40 beats per minute. Further, it is reasonable to consider permanent pacemaker implantation who have diagnostic EP study.

ACC/AHA Guidelines (2002) for permanent pacemaker implantation in patients with sinus node dysfunction[4]

Recommendation for Pacing in Sinus Node Dysfunction

Class I *- Evidence and/or agreement that permanent pacing is useful and effective.*

- Sinus node dysfunction with documented symptomatic bradycardia, including frequent sinus pauses that produce symptoms. In some patients, bradycardia is iatrogenic and will occur as a consequence of essential long-term drug therapy of a type and dose for which there are no acceptable alternatives.
- Symptomatic chronotropic incompetence.

Class IIa *- Conflicting evidence/ divergence of opinion but weight of evidence / opinion in favour*

- Sinus node dysfunction occurring spontaneously or as a result of necessary drug therapy, with heart rate less than 40 bpm when a clear association between significant symptoms consistent with bradycardia and the actual presence of bradycardia has not been documented.
- Syncope of unexplained origin when major abnormalities of sinus node function are discovered or provoked in electrophysiological studies.

Class IIb *- Conflicting evidence/divergence of opinion where usefulness / efficacy is less well established*

- In minimally symptomatic patients, chronic heart rate less than 40 bpm while awake.

Class III *- Permanent pacing is not useful/effective and in some cases may be harmful.*

- Sinus node dysfunction in asymptomatic patients, including those in whom substantial sinus bradycardia (heart rate less than 40 bpm) is a consequence of long-term drug treatment.
- Sinus node dysfunction in patients with symptoms suggestive of bradycardia that are clearly documented as not associated with a slow heart rate.
- Sinus node dysfunction with symptomatic bradycardia due to nonessential drug therapy.

Choice of pacemakers lies between atrial based pacing and dual-chamber pacing. Both have advantages and disadvantages. The basic idea is:

- Provide Atrial Pacing
- Minimize Ventricular Pacing

Table 1: ACC/AHA Guidelines 2018 regarding permanent pacing for SND patients[5]

Recommendations for Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to SND Referenced studies that support recommendations are summarized Online Data Supplements 24 and 25		
COR	**LOE**	**Recommendations**
I	**C-LD**	1. In patients with symptoms that are directly attributable to SND, permanent pacing is indicated to increase heart rate and improve symptoms.[S5.4.4-1,S5.4.4-2]
I	**C-EO**	2. In patients who develop symptomatic sinus bradycardia as a consequence of guideline- directed management and therapy for which there is no alternative treatment and continued treatment is clinically necessary, permanent pacing is recommended to increase heart rate and improve symptoms.
IIa	**C-EO**	3. For patients with tachy-brady syndrome and symptoms attributable to bradycardia, permanent pacing is reasonable to increase heart rate and reduce symptoms attributable to hypoperfusion.
IIa	**C-EO**	4. In patients with symptomatic chronotropic incompetence, permanent pacing with rate- responsive programming is reasonable to increase exertional heart rates and improve symptoms.
IIb	**C-LD**	5. In patients with symptoms that are likely attributable to SND, a trial of oral theophylline may be considered to increase heart rate, improve symptoms, and help determine the potential effects of permanent pacing.[S5.4.4-3,S5.4.4-4]

Table 2: ACC/AHA Guidelines 2018 regarding choice of pacemaker for SND patients[5]

Recommendations for Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to SND Referenced studies that support recommendations are summarized in Online Data Suppliment 25.		
COR	**LOE**	**Recommendations**
I	**B-R**	1. In symptomatic patients with SND, atrial- based pacing is recommended over single chamber ventricular pacing.[S5.4.4.1-1–S5.4.4.1-4]
I	**B-R**	2. In symptomatic patients with SND and intact atrioventricular conduction without evidence of conduction abnormalities, dual chamber or single chamber atrial pacing is recommended.[S5.4.4.1-5]
IIa	**B-R**	3. In symptomatic patients with SND who have dual chamber pacemakers and intact atrioventricular conduction, it is reasonable to program the dual chamber pacemaker to minimize ventricular pacing.[S5.4.4.1-6]
IIa	**C-EO**	4. In symptomatic patients with SND in which frequent ventricular pacing is not expected or the patient has significant comorbidities that are otherwise likely to determine the survival and clinical outcomes, single chamber ventricular pacing is reasonable.

- Provide Dual Chamber Pacing if AV Block or Bradycardia due to AF develops

In the Danish study[6], single center study, in 225 patients of sick sinus syndrome followed up to 5.5 years, concluded that over long term follow-up, AAI pacing is better than VVI pacing in SSS patients :

- Reduced overall mortality
- Reduced cardiovascular mortality
- Reduced heart failure
- Reduced atrial fibrillation
- Reduced thromboembolism

Potential problems of AAI pacing was :

- Development of AV Block requiring ventricular pacing
- Development of atrial fibrillation with bradycardia requiring ventricular pacing
- Atrial lead dislodgement

Conventional dual chamber pacemakers

- are designed to pace the ventricle
- may cause harmful effects of ventricular dyssynchrony by unnecessarily pacing the ventricles in SND patients when they have intact AV conduction

Therefore, conventional dual chamber pacemakers are not optimal for the treatment of SND. Now, we have advanced dual chamber pacemakers with MVP (Managed Ventricular Pacing) mode. These pacemakers provide:

- Optimal AAI/R pacing whenever AV conduction is intact
- Dual chamber ventricular support during transient or persistent loss of conduction
- Minimizes unnecessary ventricular pacing & its long term harmful effects of increased AF and HF

- MVP provides functional AAI/R pacing with the safety of dual chamber ventricular support in the presence of transient or persistent loss of conduction
- DDD/DDDR with MVP :
  - Minimize ventricular pacing
  - provide full atrial support
  - provide full safety of DDD/DDR

Patients of sick sinus syndrome with paroxysmal atrial arrhythmias will need anticoagulants for prevention of stroke.

Conclusion

Primarily, the elderly population faces the brunt of sinus node dysfunction. SA node degeneration is the essential factor and may be because of intrinsic or extrinsic causes. There may be occasional affection of the AV node in the course of the disease. Tachy-brady syndrome seems to be the synonym for sick sinus syndrome. Patients of SND may be asymptomatic or may have symptoms because of organ hypo-perfusion. Permanent pacemaker implantation is the main treatment, once the reversible causes are ruled out. In SND, risk of sudden cardiac death is low.

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QT Prolongation

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Introduction

One of the most challenging aspects of ECG reading is measurement and interpretation of the QT interval. It signifies the time taken for the ventricles to complete repolarization after activation. Abnormal prolongation of the QT interval can lead to torsades de pointes (TDP), a form of potentially life threatening polymorphic ventricular tachycardia (VT). Automated ECG printouts cannot be relied upon to diagnose QT interval prolongation; thus, the onus is on the clinician to identify it and prevent catastrophic consequences.

During tachycardia, the QT interval decreases, and in bradycardia, it lengthens. Thus, there is a requirement to correct it where the resting heart rates of patients may not be normal. The corrected QT interval is known as QTc, and it's prolongation, rather than the QT, that determines the risk of TDP.

Measurement of the QT interval

It should be measured from the start of the QRS complex to the end of the T wave and averaged over 3 to 5 beats in a single lead. Prominent U waves should be included in the measurement if they merge into the T wave. The determination of the end of the T wave is difficult at times as it often flattens towards its end. Hence several investigators have proposed the use of a "tangent technique" (see Fig. 1), where the steepest part of the T wave curve intersects the baseline.[1]

Manual Measurement of QTc

The three steps are required to correctly measure the QTc interval[2]

1. Select the appropriate ECG lead (usually II or V5)
2. Manually measure the QT interval (the duration from the start of the QRS complex to the end of the T wave)
3. Apply a correctional formula or use the QT nomogram to adjust for the current heart rate.

Since it is influenced by heart rate, RR interval preceding the QT interval should be measured for rate correction.

The most commonly used formulae are Fridericia's cube root formula (QTc = QT/RR1/3) and Bazett's square root formula (QTc = QT/RR1/2). There is no consensus on best method, Bazett's formula is considered the gold standard, even though it may overestimate QT prolongation. As a rule, QT prolongation is considered when the QTc interval is greater than 450 ms in males and >460 ms in females , but arrhythmias

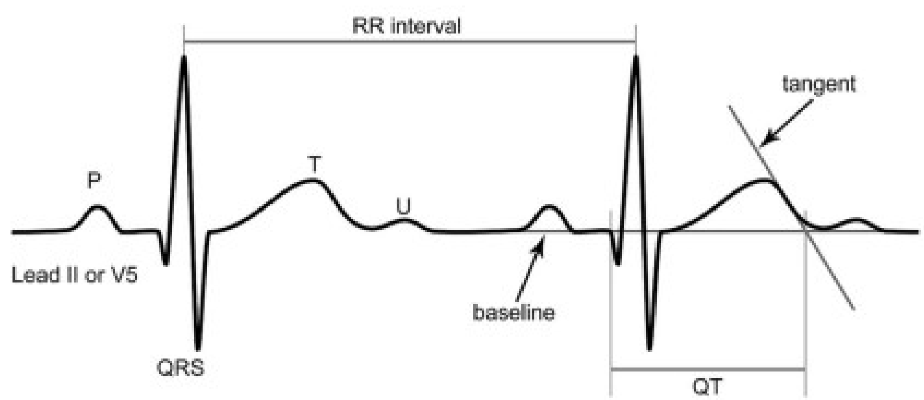

Figure 1

Formula	Equation	Strengths	Limitations	When to use
Bazett	$QT_c = \frac{QT}{\sqrt{RR}}$	• Simplest formula • Widely accepted and used	• Tendency to over-diagnose long QT as it overcorrects at high heart rates and undercorrects at low heart rates	• Best used when HR is between 50 and 70 bpm
Fridericia	$QT_c = \frac{QT}{\sqrt{RR}}$	• More accurate than Bazett formula at abnormal heart rates	• Tendency to overcorrect at high heart rates (i.e. over-diagnosis of long QT)	• Useful in bradycardic patients (HR < 50 bpm)

are most often associated with values of 500 ms or more.[3]

The "Half RR" method If QT is less than half the RR interval, it is normal. No calculation required If the QT is greater than half the RR interval, it may still be normal. Cannot be used at HR < 60 bpm. Useful as a "screening" test, especially in AF but not as a true diagnostic test.

Measurement of QT interval is particularly challenging with atrial fibrillation because it varies from beat to beat depending upon interval in between R waves. It is thus desirable to measure QT interval which follows the shortest and longest R-R interval and divide each by square root of R-R interval preceding it (Figure2).The average is then used to calculate the interval.

Clinical Scenarios

Congenital long QT syndrome (CLQTS)- includes sixteen identified genetic abnormalities that give rise to abnormal myocardial repolarisation. Management includes avoidance of classic arrhythmia triggers, particularly swimming or water sports. Beta blocker therapy is the mainstay of treatment, and the use of implantable cardioverter/defibrillators is necessitated in selected patients assessed to be at high risk of TDP.

Several forms of CLQTS are recognized and LQTS1, LQTS2, LQTS3 are now well understood. They seem to have different clinical manifestation and outcomes.

LQTS1 is triggered by physical activity, LQTS2 by auditory stimuli and LQTS3 occurs at rest or during sleep.[4]

The T wave is of long duration in LQTS1, small and notched in LQTS2 and has a long onset in LQTS3 (Figure 3).

Each genotype has a different clinical outcome as observed in international registry. Although the risk of cardiac events is observed to be higher in LQTS 1 &2, lethal arrhythmias are significantly higher in LQTS3 group.[5]

Long QT syndrome may also be acquired in the setting of cardiomyopathy, myocardial ischaemia, severe intracranial injury, electrolyte abnormalities and several medications including, various antimicrobials, antipsychotics, antidepressants, antiarrhythmics and antiemetics.

Risk factors for drug-induced torsade de pointes:[6]

Female gender, hypokalemia, bradycardia, Congestive heart failure, Left ventricular hypertrophy, Base-line QT prolongation, Subclinical long QT syndrome, Ion-channel polymorphisms Severe hypomagnesemia.

Drugs implicated in torsades de pointes:

1. Antiarrhythmic medications Class IA (Quinidine, Procainamide, and Disopyramide) Class III (Dofetilide, Ibutilide, Sotalol, and Amiodarone) Class IV (Verapamil)
2. Promotility medications Cisapride
3. Antimicrobial medications Macrolides Erythromycin Clarithromycin Fluoroquinolones Antiprotozoals Pentamidine Antimalarials Chloroquine
4. Antipsychotic medications Phenothiazine neuroleptics Thioridazine Chlorpromazine Butyrophenone neuroleptics Haloperidol
5. Miscellaneous medications Arsenic trioxide Methadone

More than 100 FDA-approved drugs marketed in the United States, affecting all disciplines of medicine, have either QTc-prolonging or torsadogenic potential.

Mechanism of drug induced QT prolongation

QT interval is the summation of action potentials in myocardial cells. It is primarily due to action potential prolongation, that results from an increase in inward current (e.g., through sodium or calcium channels) or a decrease in outward current (e.g., through potassium channels). Myocardial repolarization is primarily mediated by efflux of potassium ions. Two subtypes of the delayed rectifier potassium current, IKr (rapid) and IKs (slow), are responsible for it.

The hallmark mechanism of acquired LQTS and TdP is the blockade of IKr by specific drugs. This causes a delay in phase 3 rapid repolarization of the action potential, which is reflected by QT prolongation (Figure 4). Prolonged repolarization can cause early afterdepolarizations (EADs) due to activation of inward depolarizing currents (L-type calcium channels or sodium calcium exchange current).[7]

Repetitive extrasystoles, unidirectional block and zones of slow conduction can lead to reentry and Torsades de pointes.

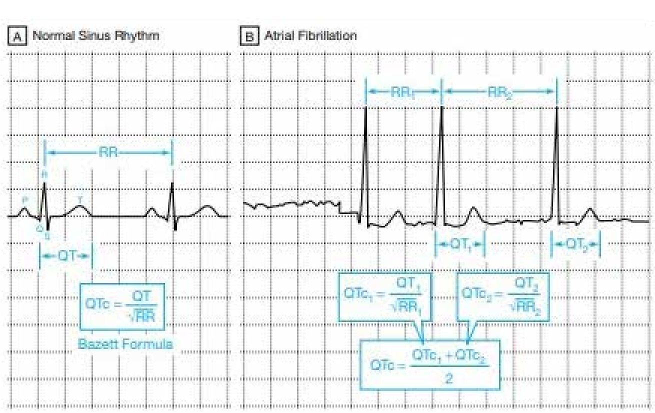

Figure 2

Figure 3

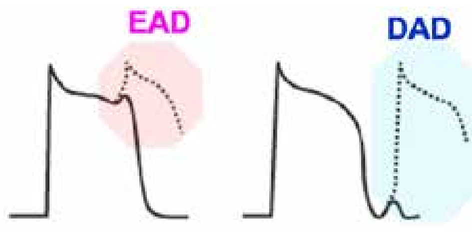

Figure 4: EAD-early after depolarization, DAD-delayed after depolarization

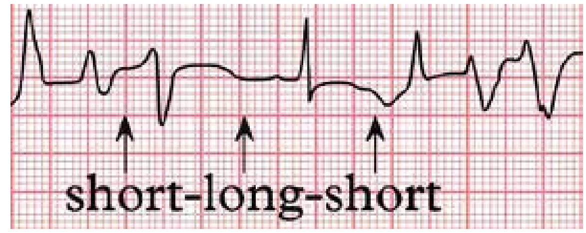

Figure 5

It is usually preceded by a short-long-short ECG sequence (Figure 5).[8]

Management of ventricular arrhythmias with acquired LQTS:

Most of the episodes of TDP are short-lived and terminate spontaneously.If prolonged can cause haemodynamic compromise and will require prompt cardioversion.

By far the most important measure is withdrawal of offending drugs.

Identification and vigorous correction of electrolyte abnormalities like hypokalemia and hypomagnesemia is life saving. Intravenous magnesium is the agent of choice for immediate treatment of TDP irrespective of the serum magnesium level. 2 g bolus of magnesium sulfate is followed by intravenous infusion at a rate of 2–4 mg per minute . The mechanism by which it prevents the recurrences is unclear. Its action is probably mediated through blockage of sodium or calcium currents. The only side effect is flushing during the infusion.

Serum potassium should be maintained in the high normal range.

Overdrive transvenous pacing shortens QTc and is highly effective in preventing recurrences of TDP, especially when they are precipitated by a pause or bradycardia. Short-term pacing rates of 90 to 110 beats/min are recommended.

Intravenous isoproterenol is sometimes needed. It is useful if temporary pacing is unavailable or while preparing for transvenous catheter insertion . It is contraindicated in patients with congenital LQTS or ischemic heart disease. Side effects include palpitations and flushes.

Conclusion

Recognition of QT prolongation by clinicians as a cause for clinical deterioration in hospital settings particularly due to drugs or electrolyte abnormalities is crucial.If the measurement is made with reasonable accuracy it can help to estimate the risk of torsades de pointes. Knowledge of drugs that causes QT prolongation and safer alternatives is required to manage this challenging clinical situation.

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Normal Rhythm - But Why Has The QRS Widened?

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Introduction

The QRS complex represents the electrical forces generated by ventricular depolarization. The duration of the QRS complex is measured in the lead with the widest complex and should not exceed two and a half small squares (0.10 s). Delays in ventricular depolarization gives rise to abnormally wide QRS complexes (>0.1- 0.12 s).

Some conditions may lead to wide QRS in normal sinus rhythm. Starting with one case which is discussed below.

Case scenario

The patient is a 76-year-old male that was admitted to emergency department (ED) with palpitation, disorientation and vomiting. He had a history of diabetes, hypertension and CKD. Patient was hypotensive, heart rate 145 bpm, SO2 92% and GCS 10/15. In ED it was diagnosed as a case of wide complex tachycardia according to ECG (Fig.1) and 150 mg of amiodarone IV was given. The patient was shifted to CCU. On closure look, presence of hyperacute T, prolong PR which was merged with T, broadened QRS and sinus tachycardia. Immediately ABG was done which revealed a potassium level of 8.5mmol/L. Treatment of the patient's hyperkalemia with insulin, beta-agonists, and calcium was initiated. After initial management patients ECG became normal (Fig. 2).

Discussion

Above mention case, Hyperkalemia is a potentially life-threatening condition that can be difficult to diagnose due to the paucity of specific signs and symptoms. So clinical suspicion of hyperkalemia requires an immediate 12-lead ECG manifestations of electrolyte imbalance. Hyperkalemia shortens Action potential duration (APD) and alters ERP thus slowing of conduction velocity. ECG changes, including peaked T wave (K+ 5.5 – 6.5 mmol/L), flattened P wave with prolonged PR interval & ST depression (6.5 – 7 mmol/L), wide QRS duration (7 – 9 mmol/L) and sine wave, asystole or VF (> 9 mmol/L).[1]

Evaluation of wide complex QRS, there should be both an immediate recognition as well as a more step by step deliberate assessment. Wide complex may immediately sort it into the most common categories: LBBB morphology, RBBB morphology (+/- LAFB or LPFB), ventricular paced rhythm (based on pacer spikes and appropriately wide QRS immediately following), or something that doesn't fit into any of those categories.

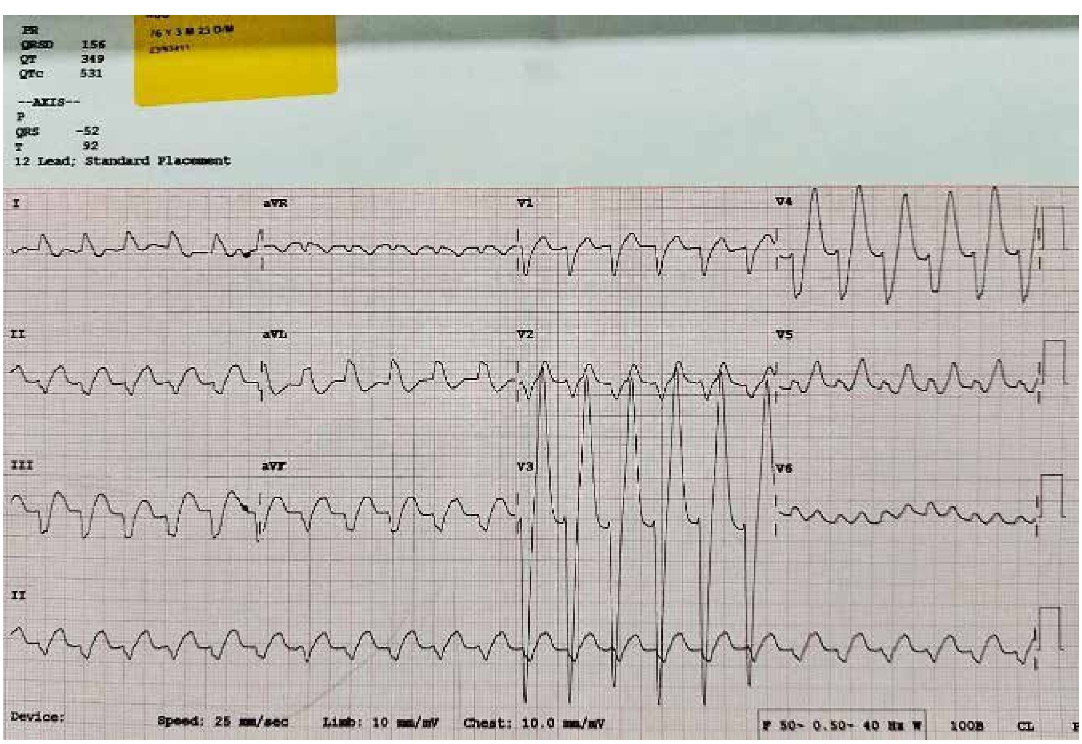

Figure 1: ECG showing hyperacute T, prolong PR, Wide QRS and sinus tachycardia.

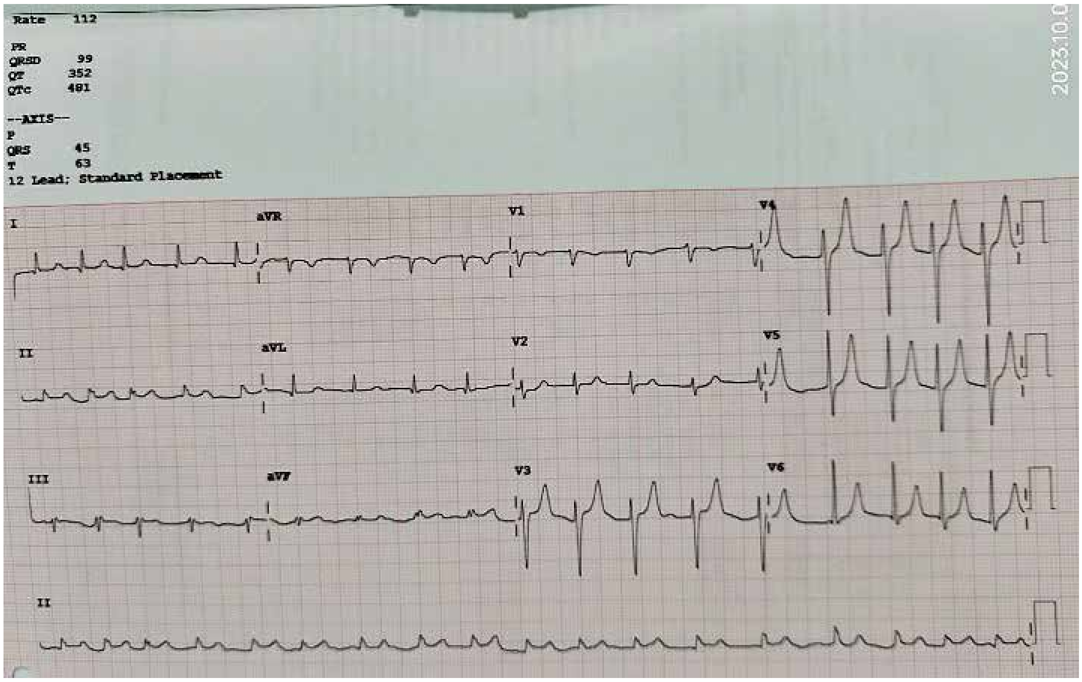

Figure 2: Normalization of ECG after treatment.

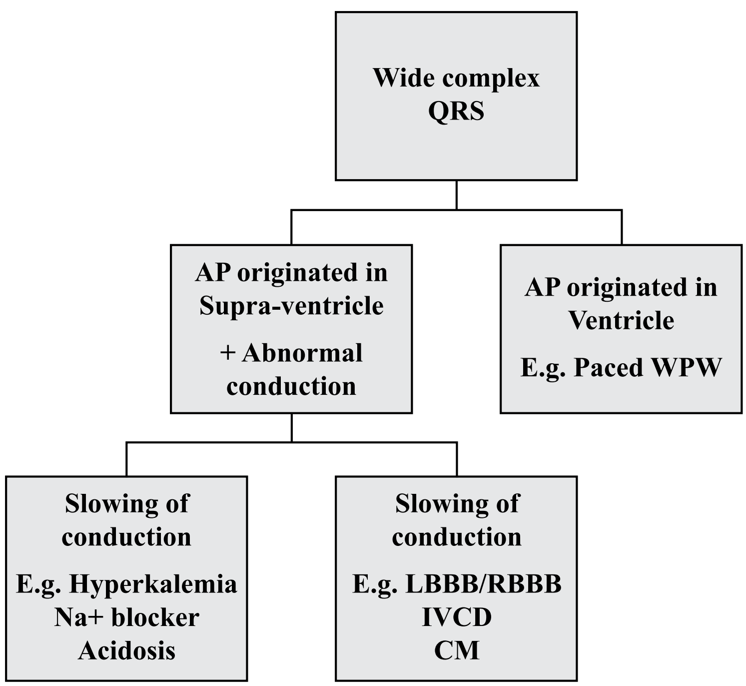

Figure 3: Step by step evaluation of wide complex QRS.

The action potential (AP) originated from the supra-ventricles, entered the conduction system correctly, then either of two things can happened. (1) normal speed of conduction, but structural blockade somewhere within the conduction system such as: LBBB (Fig.4), RBBB, intraventricular conduction delay or cardiomyopathy. (2) Normal conduction system, but slowed speed of conduction; examples: Hyperkalemia (Fig 1), Na channel blockade (medication, toxin) and severe acidosis.

The AP originated in the ventricles, outside of conduction system, or in case of WPW reached the ventricles without using the conduction system; will cause widen QRS complex. When an accessory pathway is present, the sinus node action potential can pass through the bypass tract before the AV node, resulting in the ventricles becoming rapidly depolarized. This is termed "pre-excitation" and results in a shortened PR interval on the ECG (Fig 5).

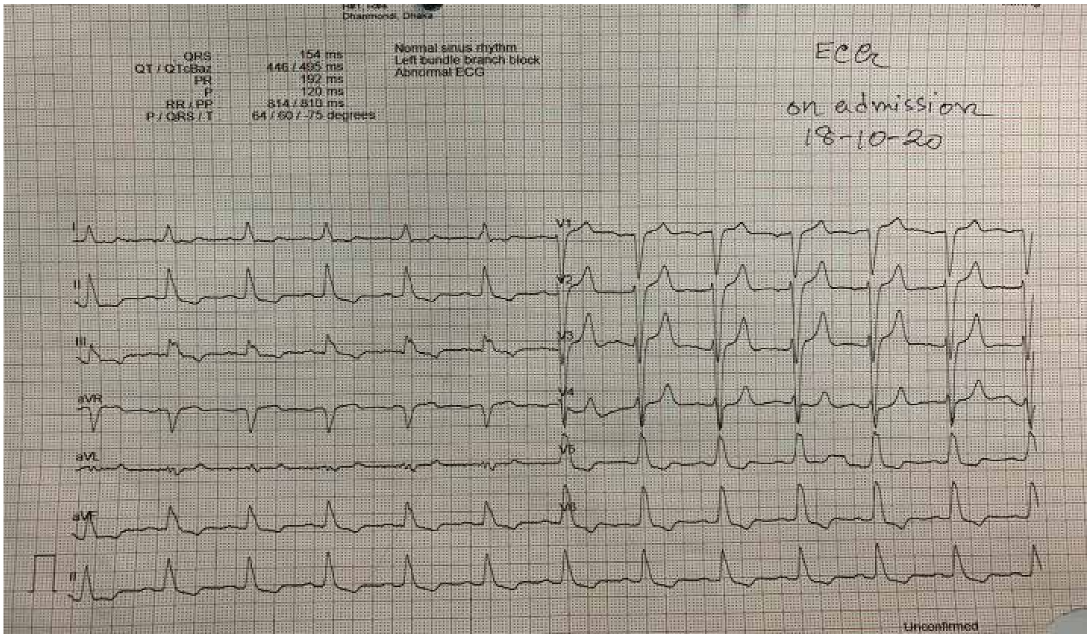

Figure 4: ECG showing LBBB evidence by QRS ≥ 120ms, prominent S in V1, broad R wave in I, aVL & V5-6 and . absence of Q waves in lateral leads.

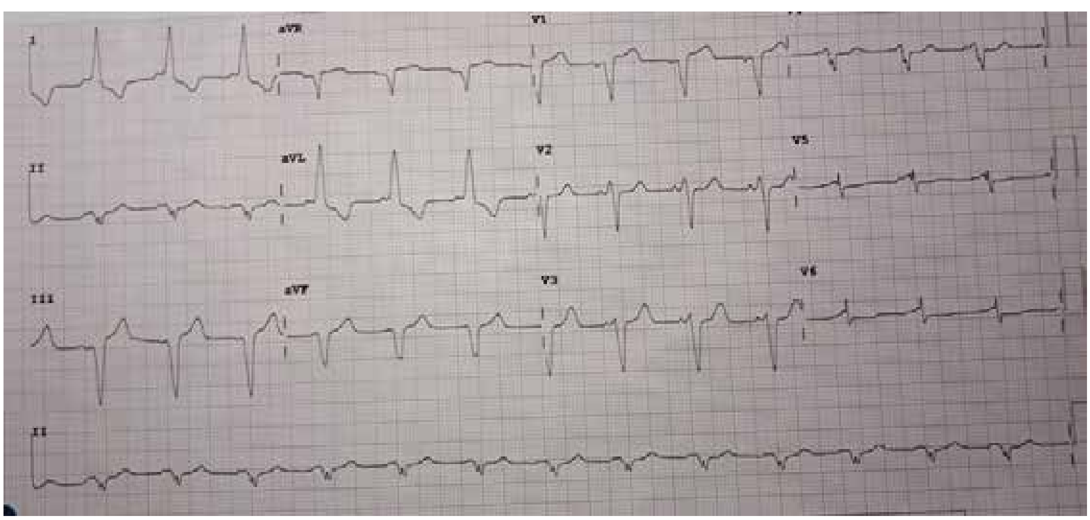

Figure 5: ECG showing WPW evidence by: (1) Short PR interval (2) delta wave, (3) wide QRS (4) secondary repolarization changes -ST segment.

Wide QRS ECG can give additional information on bundle branch block (BBB) and etiology behind it. Some of the causes for BBB include heart attack, LVH, DCM, lung disease, myocarditis or valvular/congenital heart diseases. Sometime aberrant conduction may occur during fast tachycardias or very slow bradycardia (phase 4 block).

Conclusion

One must become oriented with recognizing RBBB, LBBB, and paced rhythms in order to correctly diagnose wide QRS complexes. The differential of widening of the QRS includes aberrant conduction, hyperkalemia, accessory pathway conduction and antiarrhythmic use, but also ischemia. Some important causes of a wide QRS complex are also life threatening, immediately recognizable on ECG and immediately treatable. So, wide QRS has a great importance and we have to have a better grasp of its morphology & etiology behind it.

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27%
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References: 1. Hypertension 38.4 (2001): 852-857.
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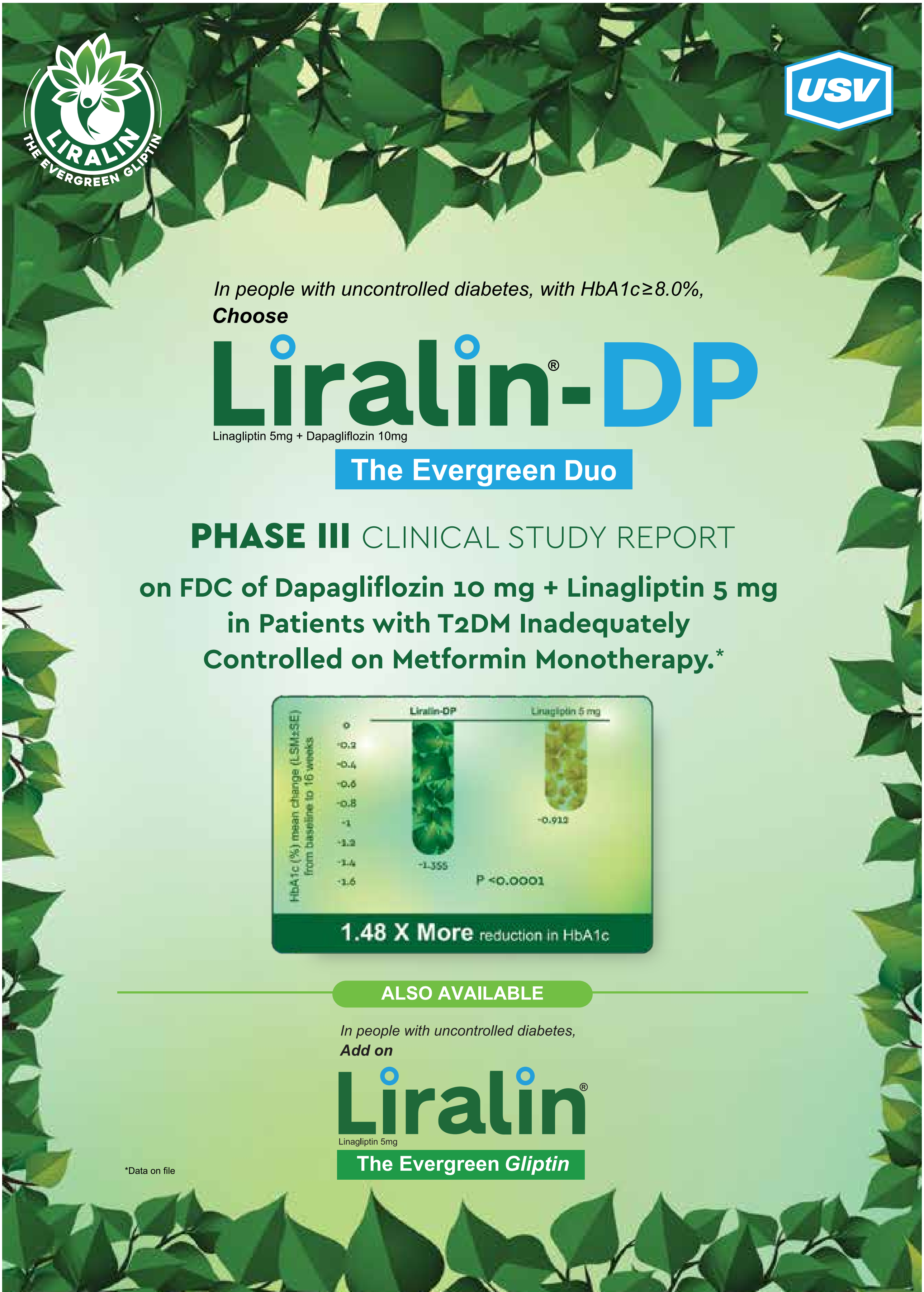

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Linagliptin 5 mg
0
-0.2
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-0.6
-0.8
-1
-1.2
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-1.6
-1.355
-0.912
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Management of Recurrences of Atrial Fibrillation

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Introduction

Atrial fibrillation is one of the most common heart rhythm disorders that increases in prevalence with increasing age, body mass index, and poor physical activity. Increase in survival in patients with structural heart disease and in patients with cardiac surgery predisposes to atrial fibrillation.[15] Approximately 1% of all the patients globally are less than 60 years old. However there is an increase to 12% and 35% in patients with the age group of 75-84 years and ≥80 years respectively.[16] By 2030, over 12 million people are projected to have atrial fibrillation globally.

AF is associated with 1.5 - 2-fold higher risk of mortality (Figure1). There is also a higher risk for stroke in patients where the burden of atrial fibrillation is high and this is independent of the use of anticoagulants. There is therefore the need to not only consider CHADS2 as the risk for stroke but also add the burden of AF burden to it, now called as CHA2DS2VASc AF burden score.[1,5,7]

The pathophysiology of atrial fibrillation involves complex interactions between genetic, structural, and functional factors (Figure 2). Modern contributors to it include physical inactivity, sleep disorders as well as a role for visceral fat mediated inflammatory cytokines.[3,15]

Strategies for AF Management

The cornerstones of AF management are prevention of stroke, rate control for management of the ventricular response to atrial fibrillation, rhythm control for maintenance of sinus rhythm and lifestyle interventions for the prevention of recurrences after the use of medications and/or ablation for the maintenance of sinus rhythm.

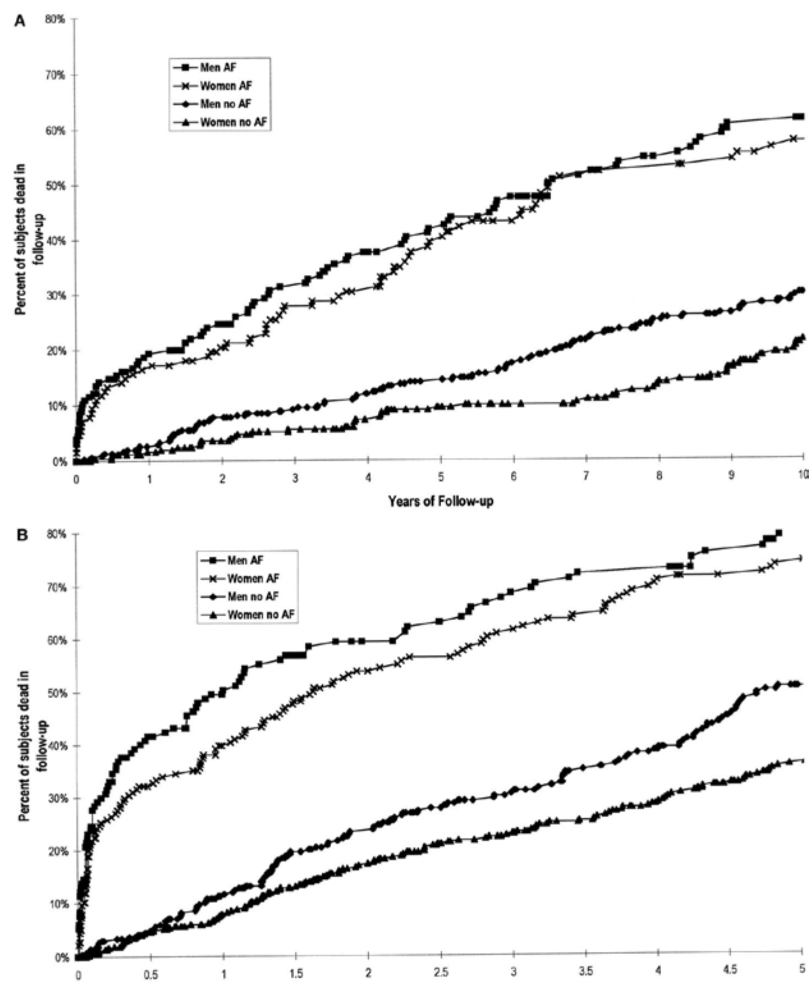

Figure 1: Kaplan-Meier mortality curves for subjects 55 to 74 years of age. Vertical axis shows percent of subjects dead at follow-up (0% to 80%); horizontal axis, up to 10 years of follow-up.[7]

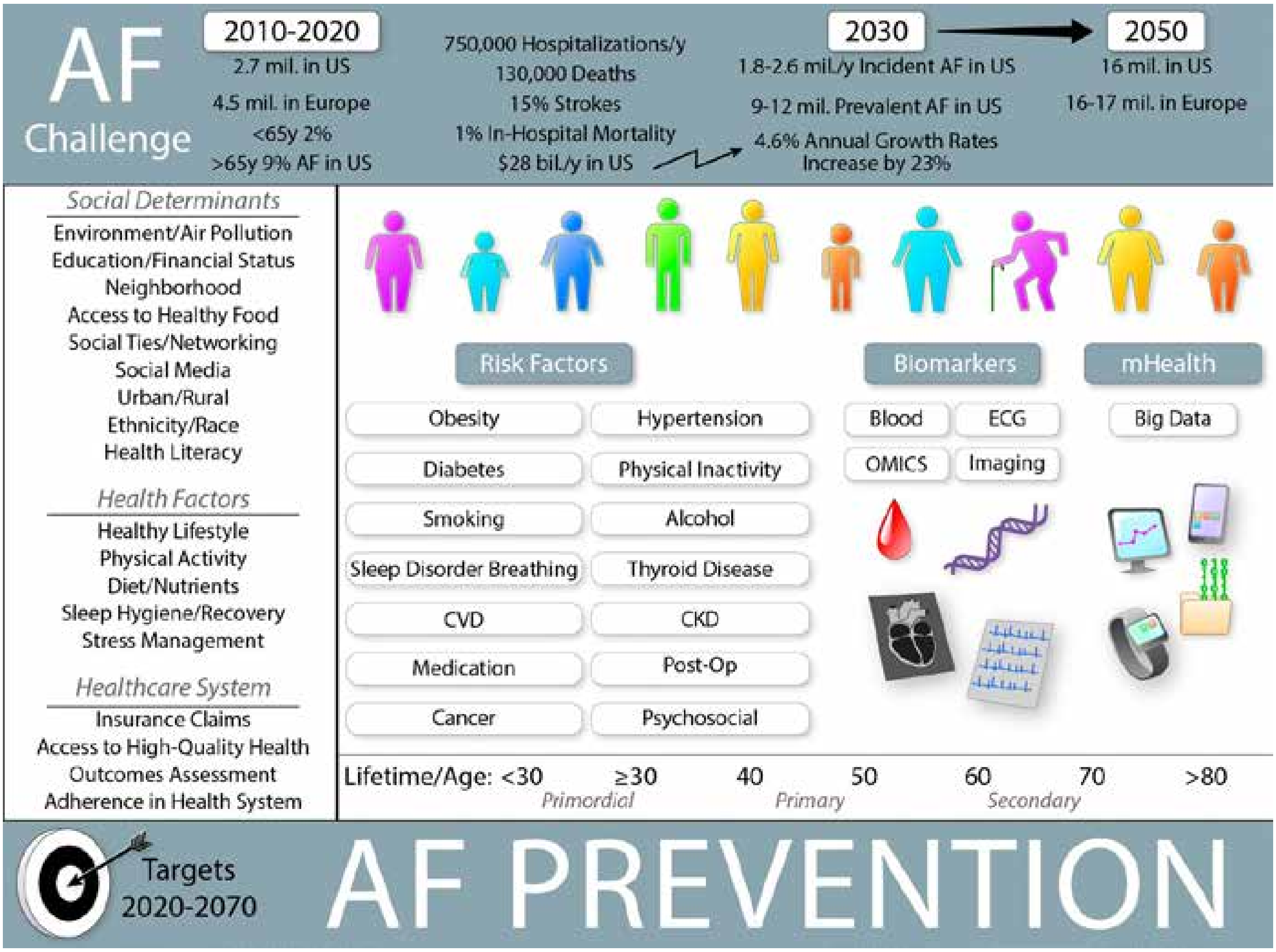

Figure 2: An overview of challenges in AF epidemiology[3]

Risk of stroke: *CHA_2DS_2-VASc score* should be considered to assess stroke risk. Warfarin, Dabigatran, Rivaroxaban or Apixaban should be considered in patients with prior stroke, transient ischemic attack or *CHA_2DS_2-VASc* ≥2.[15] In patients at risk for stroke and intolerant to medications or in whom there is a relative/absolute contraindication to oral anticoagulants or if there is history/risk of a life-threatening bleed when on anticoagulation, a left atrial appendage occluder may be considered.

Rate control: This strategy focuses on controlling the heart rate to a target range using multiple agents like beta blockers, calcium channel blockers, digoxin, in isolation or in combination. The ventricular rate should be monitored at rest and during activity (six-minute walk) and kept to a lenient target of 110 beats per minute when in atrial fibrillation. An extreme form of rate control is ablation of the AV node and implantation of a pacemaker. This is the 'pace and ablate' approach. This remains the intervention of choice in patients who are frail, have complex surgically repaired cardiac anatomy, post recurrent ablations for maintenance of sinus rhythm at risk for stiff left atrial syndrome or in those who develop heart failure with repeated episodes of fast atrial fibrillation. A randomized control trial demonstrated mortality benefit in patients with AF using this strategy in the latter group as compared to medical therapy alone.6,15,16,17,20

Lifestyle and risk factor management (LRFM): This set of interventions should be considered to reduce the risk of AF onset particularly in the population at increased risk of AF. LRFM includes maintenance of ideal weight, and weight loss if overweight or obese; pursue a physically active lifestyle; receive smoking/alcohol/drugs cessation counseling and/or medication, control diabetes and BP in accordance with guideline directed medical therapy (GDMT). Sleep study should be considered in patients with sleep disordered breathing. These form an important set of interventions for the management of AF and are aimed at maintenance of sinus rhythm.[15]

Rhythm Control: This aims to restore and maintain normal sinus rhythm using a combination of approaches, including cardioversion, antiarrhythmic drugs, and radiofrequency catheter ablation in the setting of appropriate anticoagulation and rate control. Catheter Ablation is often considered in cases of symptomatic AF refractory to medications.[6,15,16,17]

Covariate	P	HR	HR: 99% Confidence Limits	
			Lower	Upper
Age at enrollment*	<0.0001	1.06	1.05	1.08
Coronary artery disease	<0.0001	1.56	1.20	2.04
Congestive heart failure	<0.0001	1.57	1.18	2.09
Diabetes	<0.0001	1.56	1.17	2.07
Stroke or transient ischemic attack	<0.0001	1.70	1.24	2.33
Smoking	<0.0001	1.78	1.25	2.53
Left ventricular dysfunction	0.0065	1.36	1.02	1.81
Mitral regurgitation	0.0043	1.36	1.03	1.80
Sinus rhythm	<0.0001	0.53	0.39	0.72
Warfarin use	<0.0001	0.50	0.37	0.69
Digoxin use	0.0007	1.42	1.09	1.86
Rhythm-control drug use	0.0005	1.49	1.11	2.01
*Per year of age.				

Figure 3: Probability values, HRs, and their 99% confidence limits for these 12 covariates are shown. Importance of sinus rhythm is highlighted.[14,18]

Rate Vs Rhythm Control

The Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study (14) compared the effectiveness two distinct management strategies for atrial fibrillation (AF): rhythm control and rate control. The primary objective of the AFFIRM trial was the assessment of whether one approach was superior in reducing mortality and morbidity in patients diagnosed with AF. This study included patients who are 65 years or younger and have at least one of the risk factors for stroke or death. Sinus rhythm was maintained using antiarrhythmic drugs in the rhythm control cohort of patients. Cardioversion or catheter ablation was considered whenever required to maintain the sinus rhythm. Beta blockers and calcium channel blockers were used to control the rate in the rate control cohort of patients. The primary goal of this cohort was to keep the heart rate controlled and decrease symptoms. in the patients who were drug refractory, AV nodal ablation was considered followed by pacemaker implantation. At a five year follow up of rate control cohort, 34.6 percent patients were in sinus rhythm and over 80% of those in AF had controlled heart rate. Whereas in the rhythm control cohort at one, three and five years' follow up, prevalence of sinus rhythm was 82.4%, 73.3 percent and 62.6 percent respectively.

The primary outcome measure of the AFFIRM study was all cause mortality. The key findings revealed that overall mortality was not statistically different between the two strategies. However, the rate control strategy demonstrated advantages such as fewer hospitalizations and a lower risk of adverse drug effects. In contrast, the rhythm control strategy was associated with a higher rate of antiarrhythmic drug use, procedural interventions, and potential adverse effects arising from these interventions.

Limitations of AFFIRM study

The primary outcome was not statistically different in both the patient cohorts. Prior to randomization, 17% of patients had failed the antiarrhythmic drug therapy. At 5 years follow up, sinus rhythm in rate control and rhythm control patient cohorts was 34.6% and 62% respectively. The crossover rate was 14.9% vs 37.5% in rate control patient cohort and rhythm control cohort respectively, mainly because of intolerance to drug and failure of antiarrhythmic therapy. Anticoagulation protocol was followed in 85% and 70% in rate control patient cohorts and rhythm control patient cohorts respectively. 70% patients had dilated LA with LA size greater than 5 cm being one of the inclusion criteria. Also, it's worth noting that survival curves in this study appeared to be diverging, not converging.

In the primary results of the AFFIRM Study, treatment strategy (rate-control versus rhythm-control) was not related to death, but during the analysis it was observed that, the presence of sinus rhythm[14,18] was associated with a lower risk of death, and the use of AADs was associated with increased mortality. (Figure 3)

AF Induced Cardiomyopathy

AF induced cardiomyopathy (AF-CM) is defined as LV systolic dysfunction in patients with paroxysmal or persistent atrial fibrillation despite appropriate rate control. Despite AF being the most prevalent arrhythmia, the prevalence and factors that predispose or prevent AF-CM are unknown. (Figure 4) A predominant clinical challenge is to understand whether HF and cardiomyopathy is caused due to AF or vice versa. Myocardial contractility can be impaired because of persistent tachycardia, either directly or through alterations in cellular

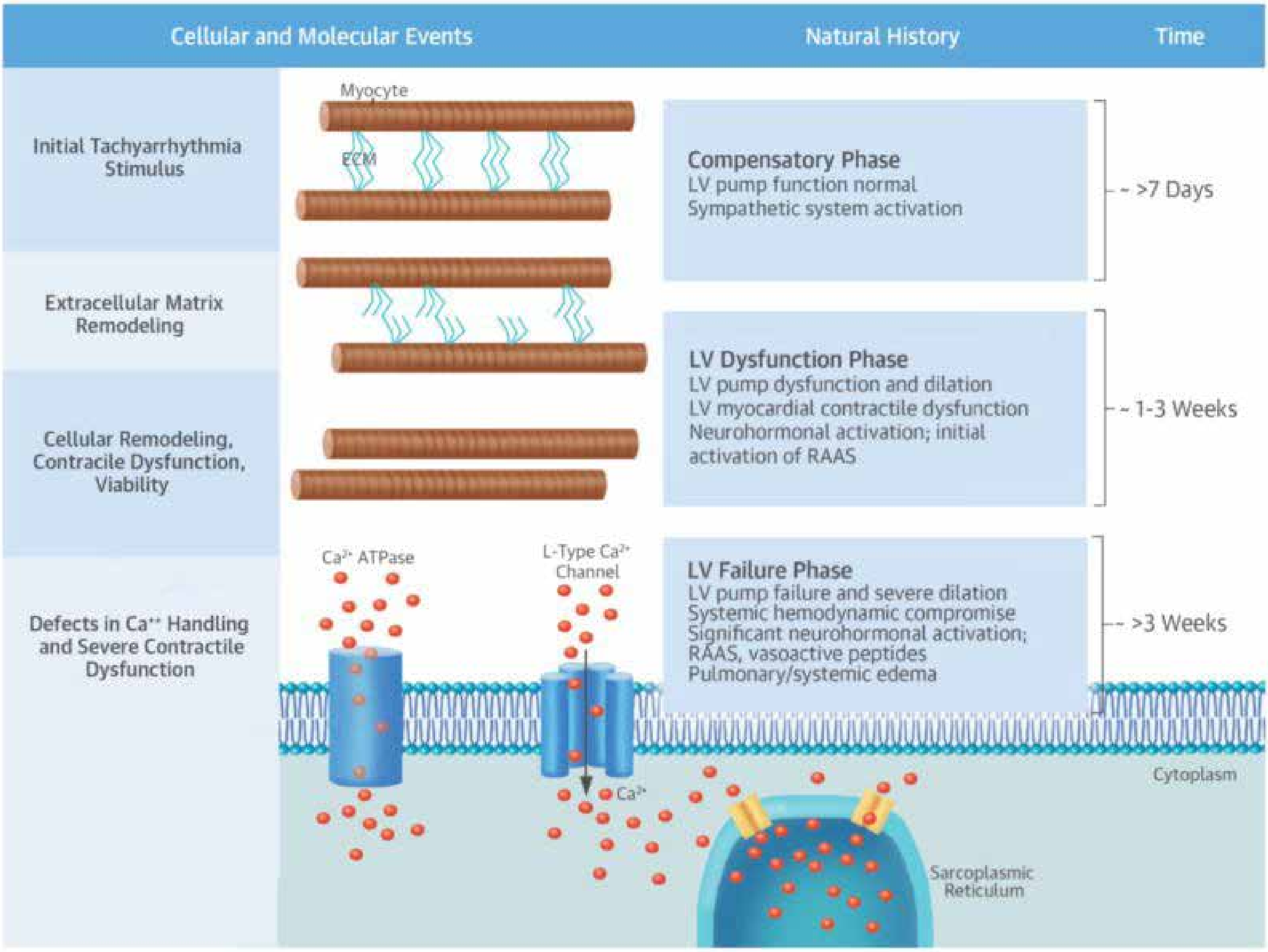

Figure 4: Mechanism of AF induced cardiomyopathy and LV dysfunction.[2]

and neurohormonal mechanisms with calcium mishandling. Diastolic filling can be impaired because of resting tachycardia and rapid increase in heart rate with exercise. Lack of an atrial kick can further worsen diastolic function. HF and increased left sided filling pressures along with functional mitral regurgitation can result in mechanoelectrical changes in the left atrium, perpetuating AF, and cardiomyopathy.[2,4] There is also evidence to suggest that the variability in ventricular rates (delta RR interval) as seen in AF leads to worsening of the cardiomyopathy.[21]

Higher the AF burden , higher the chances of hospitalization in patients with heart failure (EF<35%). This finding remained independent of whether the patients were ablated.[22]

Early Rhythm Control Therapy

A randomized trial comparing patients with recurrent AF in the early stages of disease(<1y) treated with either rhythm control with medications/ablation or rate control therapy with medications was stopped early for efficacy in the rhythm control arm. There were lesser deaths from cardiovascular causes, strokes and hospitalizations' for heart failure or acute coronary syndrome in patients randomized to the rhythm control arm as compared to the rate control strategy (Figure 5). Although events of syncope, acute hospitalization for AF were higher in the rhythm control arm, incidence of stroke was independently reduced in the arm undergoing rhythm control.[13]

Comparison of strategies to maintain Sinus Rhythm

The CABANA trial and the CASTLE HF trials showed benefit in the use of the ablation strategy in the maintenance of sinus rhythm over the use of medications (Figure 6a and b). This benefit was primarily driven by the benefits in the patients with heart failure, a finding that was reinforced in the recent CASTLE HTx study conducted in patients with symptomatic AF and HFrEF suitable for cardiac transplants [composite of all cause death, worsening HF needing transplant or LVAD at 1 year follow-up lower in the rhythm control via ablation group {8.2% vs 29.9% HR 0.24 ; 95% CI 0.11-0.52}] The trial was stopped early by the data safety monitoring board in view of the efficacy of the ablative therapy for rhythm control.[11,19,21]

In patients with paroxysmal AF, Carlos et al[10] demonstrated

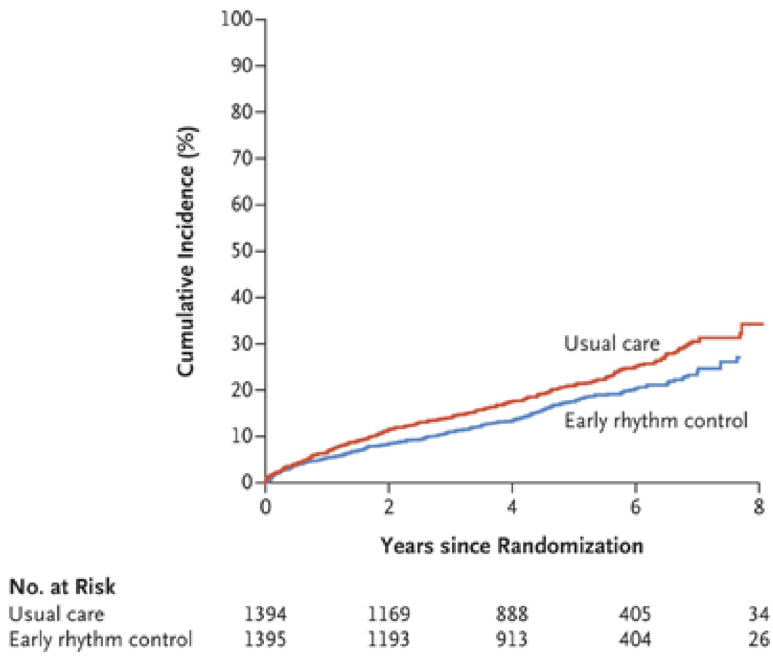

Figure 5: Aalen–Johansen Cumulative-Incidence Curves for the First Primary Outcome. The first primary outcome was a composite of death from CVD, stroke, or hospitalization with worsening of heart failure or acute coronary syndrome.[13]

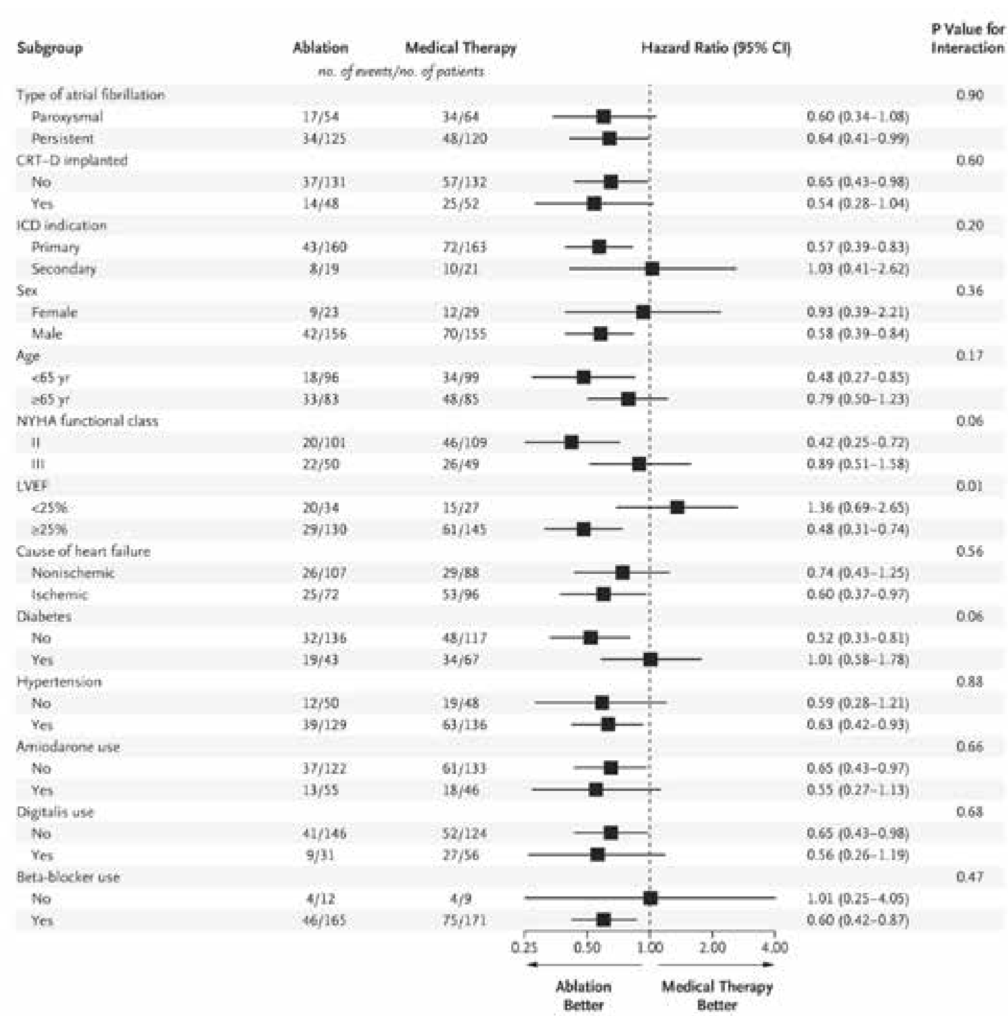

Figure 6a: Subgroup Analyses of the Primary End Point.[19]

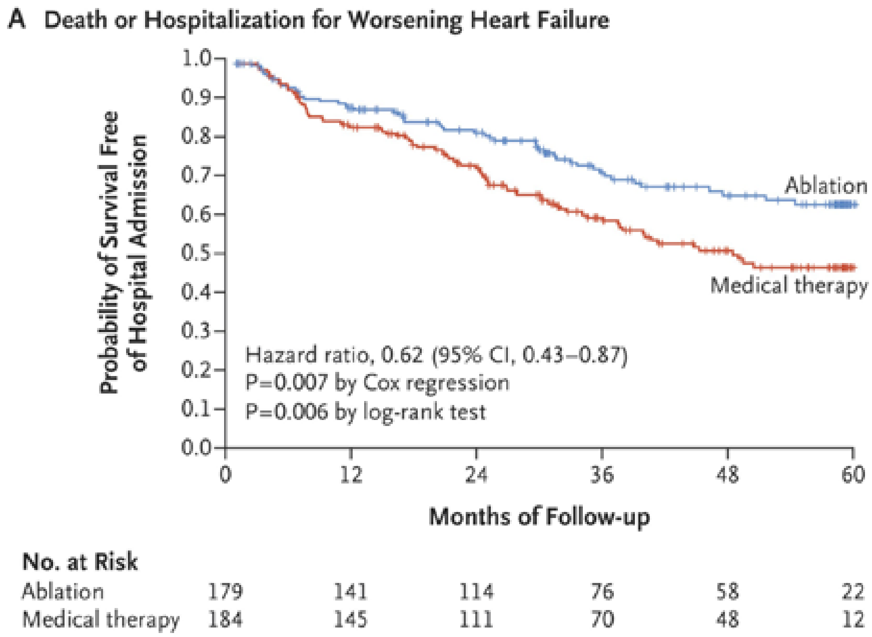

Figure 6b: Kaplan–Meier Curves Comparing Survival Free of the Primary End Point which shows the probability of freedom from death from any cause or admission for worsening heart failure.[19]

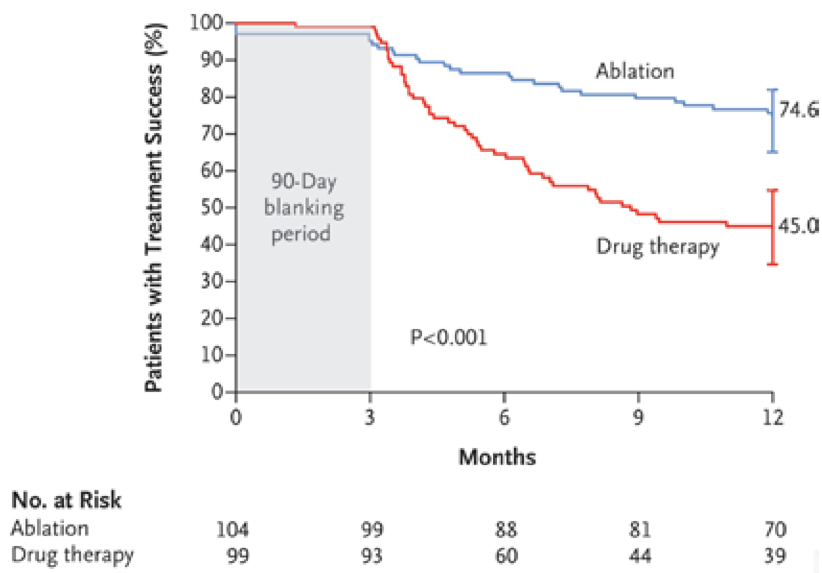

Figure 7: Oussama M.W. et.al. Cryoballoon ablation as initial therapy for AF ablation. NEJM 2021

that the patients with paroxysmal AF without previous antiarrhythmic drug treatment, radiofrequency ablation compared with antiarrhythmic drugs resulted in a lower rate of recurrent atrial tachyarrhythmias at 2 years.

On the similar lines, Oussama MW et al[8] observed that cryoballoon ablation as initial therapy was superior to drug therapy for the prevention of atrial arrhythmia recurrence at 12 m follow up in patients with paroxysmal atrial fibrillation (Figure 7)

A NNT of 5 patients with ablation instead of antiarrhythmic drugs is present to prevent one recurrence of paroxysmal atrial fibrillation[23]

In patients with HFpEF, ablation for persistent AF improved invasive exercise hemodynamic parameters, exercise capacity and quality of life in patients randomised to rhythm control versus medical management. 50% patients in the ablation arm did not meet the exercise right catheterisation criteria for HFpEF compared to 7% in the medical management arm.[26]

Prevention of disease progression

Ablation therapy has been demonstrated to decrease the progression of paroxysmal AF to persistent AF over 3 years of follow-up (HR 0.25; 95% CI 0.09-0.7). This effect was more pronounced with the use of radiofrequency energy as compared to cryoenergy.[24] Patients with drug refractory

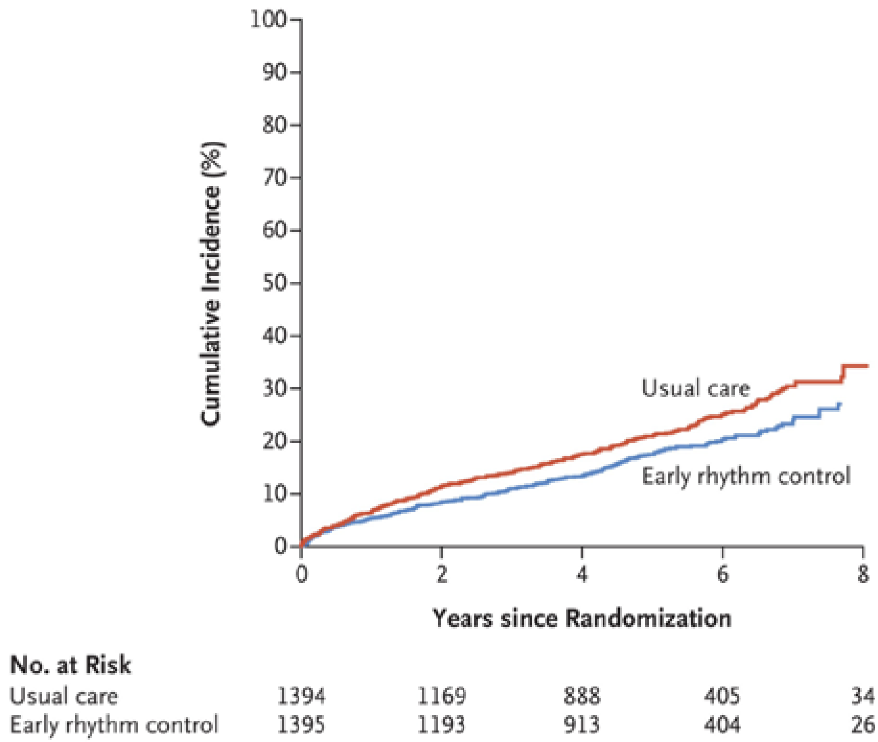

Figure 8: Winkle et al; Very long term outcomes of AF ablation HRJ 2023; [PAF 33.6%; persistent AF[psAF] 56.4%;long standing AF [lsAF] 9.9% distribution of population]

paroxysmal AF >60 years undergoing ablation were 10 times less likely to develop persistent AF/AT at 3 years follow up. (HR=0.1; CI= 0.024-0.47) when compared to treatment with antiarrhythmic drugs.[28]

Role of substrate in the management of recurrent atrial fibrillation

AF is frequently associated with LA myopathy . Data regarding the choice of therapy for the management of the disease needs evaluation of the substrate also . Bifid P waves, interatrial blocks, atrial dilation, reduced atrial strain or atrial MR as measured by echo, atrial fibrosis estimated by MRI and elevation in LA/PCWP v wave relative to LV EDP indicate the possible presence of LA myopathy. This has predictive value for future AF risk, risk of stroke and systemic embolism independent of AF burden and remains an important variable to consider when ablation strategies are used which by creating more scar in LA may paradoxically worsen hemodynamics.[25]

Very Long term outcomes of AF ablation

Recurrences post AF ablation determine the acceptability and the health care utility outcomes for the intervention. When followed up over 15 years across various technological changes, The recurrence rates are highest in the first 2 years after which the incidence of the same is 2%/y. This is driven by use of contact force sensing technology. Across eras, outcomes improved for paroxysmal and persistent AF but long standing AF outcomes remained similar indicating the need for better strategies for management of same (Figure 8)[27]

Conclusion

Atrial fibrillation is not a benign arrhythmia. Increasing burden increases the risk of mortality and stroke independent of the use of anticoagulation. Early judicious use of rhythm control shows mortality benefits especially in patients with heart failure and it also prevents progression of disease. Ablation is safer than medications in the rhythm control strategy. Success of ablation is driven by use of contact force technology with 2%/year recurrence in arrhythmia after 2 years post ablation. Underlying atrial cardiomyopathy should be assessed actively and should factor in the decision making for management strategy for AF. Rate control strategy has a small but important niche in the management of patients with the 'pace and ablate' strategy showing mortality benefits over medical therapy in patients with heart failure attributable to atrial fibrillation. Lifestyle measures targeting pre-specified goals performed under the aegis of a supervised program shows independent contribution towards the maintenance of sinus rhythm

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Sinus or Atrial Tachycardia

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Sinus tachycardia (ST) is characterized by a sinus P wave at a rate of more than 100 per minute, usually followed by a QRS complex that is usually narrow (but may be wide in the presence of an underlying BBB). Sinus Tachycardia is common and ubiquitous. It is present during exercise but can occur at rest when associated with any number of conditions like fever, Sepsis, dehydration, anxiety, drugs, hyperdynamic states like anaemia, hyperthyroidism, heart failure and other conditions of high sympathetic tone.

A certain condition called Inappropriate sinus Tachycardia (IST) is seen often in the clinical practice. The prevalence estimated to be around 1%. The prognosis of IST is benign but the symptoms could be debilitating. It is a poorly characterised condition that may be due to multiple mechanisms. Salient features include persistently elevated resting Heart rate (>90bpm), associated with an exaggerated response to minimal exertion or postural change (>25bpm). It is a diagnosis of exclusion. The postulated mechanisms are: Imbalance in the sympatho-vagal system of the SA node, hyper response to adrenergic influences, channelopathy involving the Potassium channel. Electrocardiographic features of inappropriate sinus tachycardia are similar to sinus tachycardia. However inappropriate sinus tachycardia can rarely have a sudden in onset and offset. Another tachycardia which involves the sinus node is sinus node re-entrant tachycardia (SNRT).

Sinus node re-entry tachycardia (SNRT) is an uncommon form of paroxysmal supraventricular tachycardia, can mimic sinus tachycardia in P-wave morphology. A frequent premature atrial complex at the initiation or termination of the tachycardia. The usual rate of SNRT is between 110 and 150bpm. The exact mechanism of SNRT remains controversial. The rentrant circuit involves SA node and the perinodal tissue. It mimics atrial tachycardia in many aspects.

Atrial tachycardia is defined as regular atrial activation from atrial areas with centrifugal spread. Atrial tachycardia can be a result of one or a combination of the mechanisms leading to arrhythmia: automatic, triggered activity, or re-entry. In some cases, the mechanism remains undetermined[1]. Electrophysiologic features may overlap if there is a small reentrant circuit, as in micro-reentry. It is useful to categorize atrial tachycardia in two broad groups: macro re-entrant or focal. Focal AT can present at any age and can occur in both (idiopathic) and diseased hearts (structural heart disease). It represents 3-17% of supraventricular tachycardia cases referred for ablation.[2] Macroreenterent AT are frequently associated with regions of atrial scar and are often noted in the context of structural heart disease, prior cardiac surgery and increasingly after prior catheter ablation of Atrial fibrillation (AF). Focal AT usually has benign prognosis. However, 8-10% of patients with focal AT may develop a tachycardia induced cardiomyopathy[3].

The ECG features of atrial tachycardia are:

- Atrial rate- Can occur between 100 to 250 BPM
- Ventricular conduction can be variable
  - Regular if 1 to 1, 2 to 1, or 4 to 1 AV block
  - Irregular or irregularly irregular in the setting of variable AV block
- P wave morphology
  - Unifocal, but similar in morphology to each other
  - Differs from normal sinus P wave
- May exhibit either long RP or short RP intervals

It is not often difficult to differentiate atrial tachycardia from Sinus tachycardia from the 12 lead electrocardiogram (ECG). But sometimes, it can become a difficult task to decide the plan of management. This situation is commonly seen in intensive care units where the component of haemodynamic compromise is added. The biggest confusion arises when the P wave morphology is similar to sinus tachycardia or if the P waves are not clearly seen. A careful analysis of the 12 lead ECG and telemetry recordings is critical in these circumstances.

The following can help in differentiation of Atrial tachycardia from sinus tachycardia from the ECG.

Ventricular rate

The usual ventricular rate of atrial tachycardia is 150-200bpm. Whereas the rates of sinus tachycardia is between 100-150bpm. Ventricular rates in sinus tachycardia doesn't usually exceed 160bpm, but it can go higher even up to 180bpm in subjects with high sympathetic tone. At the same time, the ventricular rate of AT can be lower than usual either due to drugs or could be due to 2:1 conduction. The below (Figure 1) 12 lead electrogram of a 18 year old girl shows narrow complex long RP tachycardia with a ventricular rate of 130bpm who was on anti arrhythmic drugs. Even though the rate is less than 150bpm, it is atrial tachycardia as the P wave morphology suggests it to be atrial tachycardia. She underwent successful radiofrequency ablation of atrial tachycardia subsequently. Hence ventricular rate should not only be considered for the differentiation of AT from ST.

P wave Morphology

P waves in sinus tachycardia are upright in inferolateral leads

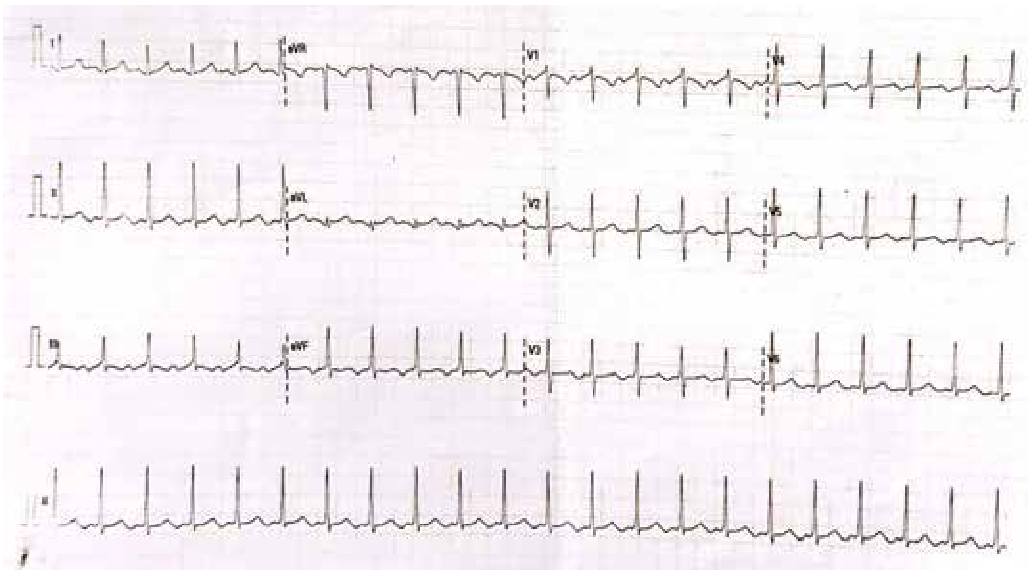

Figure 1: 12 lead electrogram of a 18 year old girl with history of palpitations showing Long RP Tachycardia with P wave morphology suggestive of atrial tachycardia.

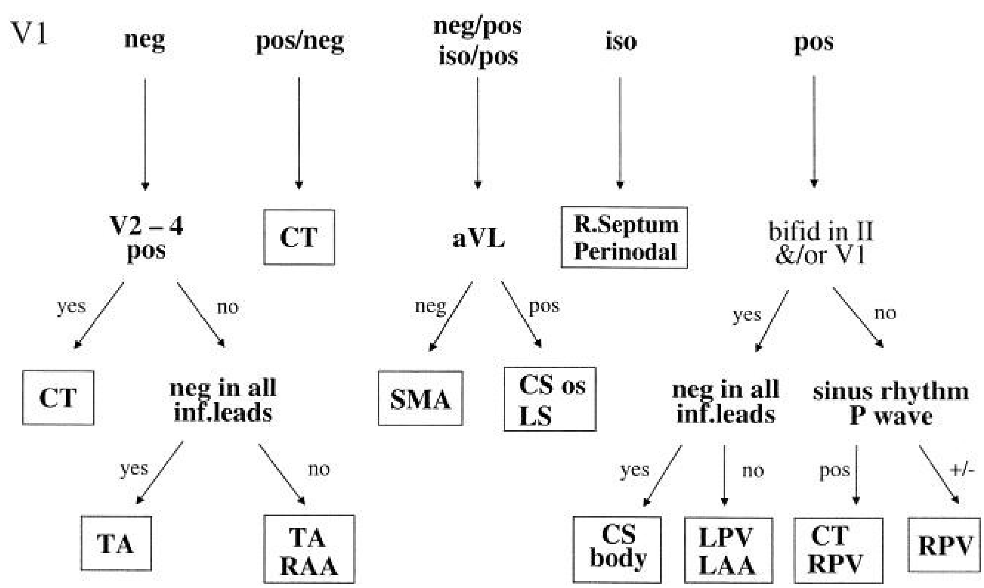

Figure 2: Algorithm to detect the anatomic site of origin of focal atrial tachycardia (taken from Kristen etal.[4]

and V2-V6, Inverted in aVR, Biphasic (Positive/negative). But in atrial tachycardia, the P wave morphology varies from it. The P wave morphology depends on the origin of the tachycardia. In focal atrial tachycardia, the P wave morphology differs from the sinus tachycardia. The commonly used algorithm to identify the origin of atrial tachycardia in focal AT is given by Kristen etal[4] (Figure 2)

In atrial tachycardia, there will be an isoelectric line between the P waves. Saw tooth appearance is typical of atrial flutter where no isoelectric line is seen between the flutter waves. Figure 1 shows a narrow complex long RP tachycardia with a ventricular rate of 130bpm. The differential diagnosis could be Atrial tachycardia or sinus tachycardia. P wave is inverted in inferior leads, positive in I, aVL and biphasic in V1, V2. This P wave morphology is different compared to sinus tachycardia which suggests the tachycardia to be atrial tachycardia.

Perinodal Atrial tachycardia appears identical to sinus tachycardia on surface ECG with an exception of taller P waves in Lead II and deeper negative P wave in V1. This could be difficult to appreciate on a routine 12 lead ECG.

P waves may not be clear on 12 lead ECG in some conditions which makes the diferentiation difficult. Figure 3 shows a 12 lead elctrocardiogram taken from a 65year old male admitted to Medical intensive care unit with Pneumonia. ECG shows narrow complex tachycardia with long RP interval. P wave morphology is not clear to diagnose it. In that scenario, the onset and offset of the tachycardia and response to Adensoine/ carotid sinus massage helps to differentiate.

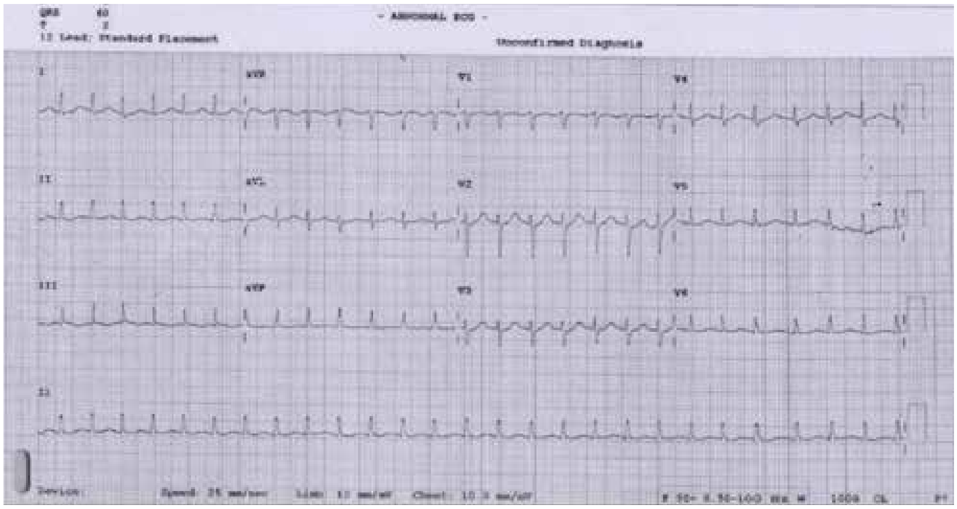

Figure 3: 12 lead electrocardiogram of a 65 year old male admitted to medical intensive care unit with pneumonia.

Figure 3: Holter recording of 18 year old girl shows abrupt onset and offset of the tachycardia.

Onset and Offset

Differentiation of atrial tachycardia from sinus tachycardia may not be clearly possible in conditions like perinodal atrial tachycardia (The P wave morphology is similar to sinus tachycardia) or Sinus node reenterant Tachycardia (The P wave morphology is similar to sinus tachycardia) or when the P waves are not clearly seen on surface ECG. In these situations, the onset of the tachycardia which is recorded in the Holter recording or central telemonitor or CIEDs is helpful in differentiating in ST from AT.

Sinus tachycardia has gradual onset and offset where as AT is sudden in onset and offset. Figure 3 is a holter recording of 18 year old girl with documented narrow complex tachycardia. The tachycardia is abrupt in onset and offset.

AT may demonstrate warm up phenomenon at onset and Cool down at termination which can cause change in the cycle length at the onset and offset of tachycardia which is a characteristic feature of focal atrial tachycardia.

Response to Adenosine and Carotid sinus massage (CSM)

Sinus node re-entry tachycardia usually terminates with CSM. Similarly focal atrial tachycardia may get terminated with CSM. Sinus tachycardia will slow down with CSM. Similarly, adenosine can terminate the focal atrial tachycardia (characterises the focal mechanism of the atrial tachycardia) or SNRT. Adenosine in atrial flutter or atrial tachycardia with re-entry as the mechanism causes atrioventricular block with prominent flutter waves or P waves respectively. Where as sinus tachycardia will be terminated followed by a gradual recurrence.

Below ECG recording (Figure 4) shows the response of the tachycardia with carotid sinus massage (CSM). Tachycardia was terminated with carotid sinus massage and resumed spontaneously which suggests focal mechanism of the tachycardia.

Conclusion

Often physicians and cardiologists are called to differentiate sinus tachycardia from atrial tachycardia. Sometimes it would be difficult to differentiate these two tachycardias accurately. P wave morphology on 12 lead ECG helps in most of the cases to distinguish these two. But response to adenosine, carotid sinus massage (CSM) also helps, as focal atrial tachycardia terminates but not sinus tachycardia. Whenever available, onset and offset of the tachycardia recorded in the holter/ central telemonitor/ CIED also helps in differentiation.

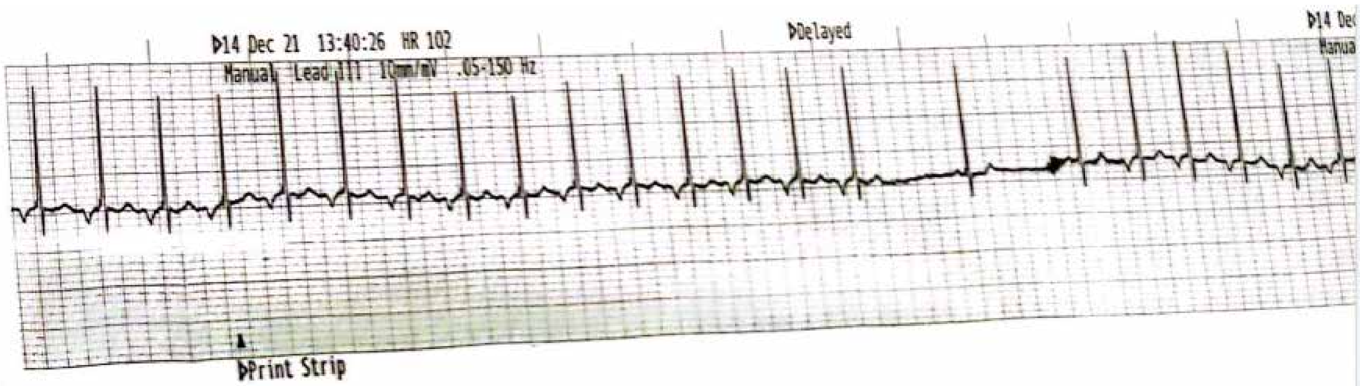

Figure 4: ECG recording during carotid sinus massage shows tachycardia termination followed by recurrence of the tachycardia.

Sinus node re-entry tachycardia and inappropriate sinus tachycardia are the two types of tachycardias involving the sinus node with P wave morphology similar to sinus tachycardia. SNRT terminates with CSM where as inappropriate sinus tachycardia doesn't. Perinodal atrial tachycardia mimics sinus tachycardia but behaves as focal atrial tachycardia. Its always intelligent to give adenosine or to do CSM whenever in doubt.

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Name in full (Surname first) ______________________________

Qualifications ______________________________

University (Post-Graduate obtained) ______________________________

Year of obtaining first Post-Graduation ______________________________

Mailing Address ______________________________

____

Tel. No. Hospital ______________ Clinic ______________ Residence ______________

Fax ______________ E-Mail ______________________________

Enclose cheque/draft for Life Membership: Rs. 4720/- or Fellowship: Rs. 7080/- towards Membership of the

Society ______________________________ Dated ______________

of ______________________________

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

Thanking you,

Yours sincerely

Signature of the Applicant

Proposed by (the Member of the Society)

Name ______________________________

Address ______________________________

Signature ______________________________

FOR OFFICE USE ONLY

Recommendations of the Executive Body / Credential Committee

Accepted / Not Accepted

Life Membership No. or Fellowship No.

____

Hon. Secretary, ISE

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

***Life Members :**

1. Person should be a Post-Graduate in Medicine/ Pediatrics/Anaesthesia/ Physiology or other allied subjects from an University recognised by Medical Council of India, with interest in Cardiology / Electrocardiology.
2. Candidates are requested to submit self-attested **Xerox** copies of the PG Certificate and Medical Council of India Registration Certificate alongwith Application Form.
3. Life Membership Fees: Rs.4,000/- + 18% GST. Total Rs. 4720/- only. In case, Life Membership is not approved by the Credential Committee, the cheque / draft will be returned.

****Fellowship:**

1. Person should be a Member of the Society.
2. He/She should be of atleast 7 years of standing after Post-Graduation.
3. He/She should have minimum 3 publications In Cardiology In Indexed Journals (Not Abstracts)
4. List of Publications to be submitted for the Fellowship.
5. Fellowship Fees: Rs.6,000/- + 18% GST. Total Rs. 7080/- only. In case, fellowship is not approved by the Credential Committee, the cheque / draft will be returned.

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


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SECRETARIAT

Ketan K. Mehta

HON. GENERAL-SECRETARY

Indian Society of Electrocardiology

Health Harmony, 2-Dattani Chambers, S V Road, Malad (W), Mumbai 400064

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