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CARDIAC ARRYTHMIASIS AND ITS DETAILED PATHOPHYSIOLOGY - A REVIEW

Dr. Fazlu Rahaman[1], Dr Pilli Yesupadam[2], Mrs.Aqsa Farheen[3]

ABSTRACT: Amid the previous couple of years, the improvement of powerful, experimental innovations for treatment of cardiovascular arrhythmias has surpassed the pace at which itemized information of the basic science has aggregated. Cardiac arrhythmia is a group of conditions in which the heartbeat is irregular, either too fast, or too slow and is characterized by abnormal electrical conduction in the heart resulting in ineffective pumping heart rate and rhythm that is not physiologically justified. Cardiac arrhythmias are commonly classified as tachycardia. (supraventricular or ventricular) or bradycardias. The specialist management of arrhythmias has changed significantly over the past decade. Abnormal impulse conduction results in reentrant excitation. Usually a combination of slowed conduction and unidirectional conduction block provides the conditions necessary for re-entry to occur. This article outlines current management strategies for atrial flutter and atrial fibrillation, with particular emphasis on curative strategies with catheter ablation and the recent data on rhythm compared with rate control strategies. The biology of arrhythmia is largely quantifiable, which allows for systematic analysis that could transform treatment strategies that are often still empirical into management based on molecular evidence.

Keywords: Cardiac arrhythmia, Ventricular arrhythmia, Ventricular myocardium and morbidity

INTRODUCTION:

While ischemic and hemorrhagic strokes can cause cardiovascular problems, there have been studies looking at abnormal ECGs and arrhythmias after a severe stroke. The irregularity of drive initiation, motivation conduction, or both may be caused by arrhythmias. At this time, it is believed that a number of devices may induce strange drive initiation or conduction. The discovery of its complex nature has likely set us on the correct path to understand the factors that really cause certain clinical arrhythmias.

Many different types of particle channels work together to interrupt the heart's activity potential. The sarcolemma of cardiomyocytes contains protein structures called cardiovascular particle directs. These structures guide a specific and rapid flow of particles via a focused pore by means of highly controlled opening and closing, also known as gating. A well-organized constriction 1 is ensured by the spatial heterogeneity of particle channel articulation, which is based on the varied activity potential architecture of the various sections of the heart.

professor[1],Associate professor[2], Assistant professor[3]

Department of Pharmacy Practice,

Global College of Pharmacy, Hyderabad. Chilkur (V), Moinabad (M), Telangana- 501504.

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1. Cardiac Arrhythmias: Normally, while an adult is at rest, impulses between 60 and 100 beats per minute (bpm) originate in the sinoatrial (SA) node, which initiates the heart's rhythm. As you sleep, your heart rate might drop to 30–50 beats per minute, and you could have short pauses (sinoatrial block), rhythms at the junctions of your heart's chambers, and even first- and second-degree atrioventricular nodal blocks. recurring often enough (especially in competitive athletes) to be classified as typical variations 2.

In arrhythmias, there are two main types.

When the heart rate falls below 60 beats per minute, it is known as bradycardia.

When the heart rate exceeds 100 beats per minute, it is known as tachycardia.

The conventional approach for identifying cardiac arrhythmias is electrocardiography, however there are several physical symptoms that might aid in the diagnosis.

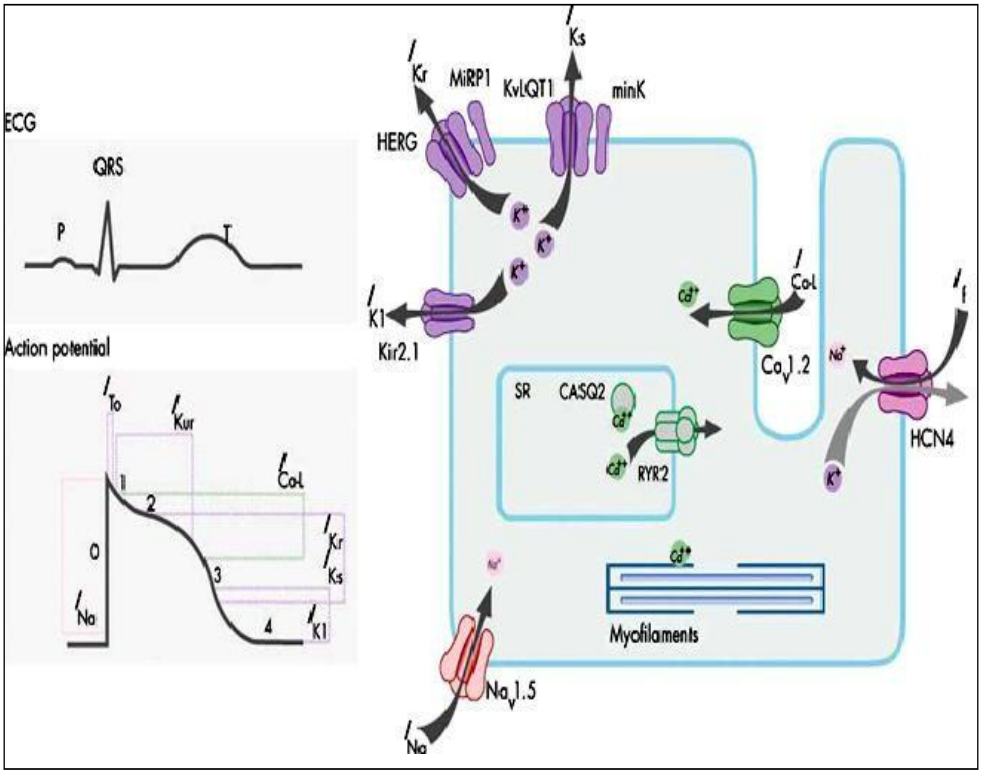

FIG. 1: A) SCHEMATIC REPRESENTATION OF A CARDIOMYOCYTE DISPLAYING (ONLY) THOSE PROTEINS INVOLVED IN THE PATHOGENESIS OF INHERENT ARRHYTHMIA SYNDROMES. B) IN PANEL A, THE ACTION POTENTIALS ARE AN ALIGNED WITH ITS APPROXIMATE TIME OF ACTION DURING THE ECG. IN A PANEL B, ANKYRIN-B, ADAPTER PROTEIN INVOLVED IN THE LONG QR SYNDROME TYPE 4, IS NOT DEPICTED

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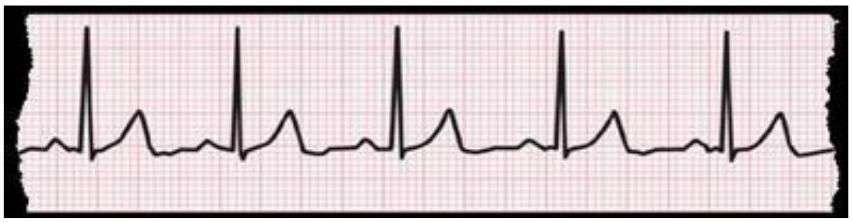

FIG. 2: ECG STRIP SHOWING A NORMAL HEARTBEAT

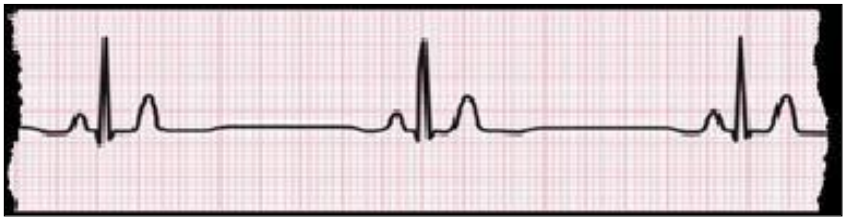

FIG. 3: ECG STRIP SHOWING BRADYCARDIA

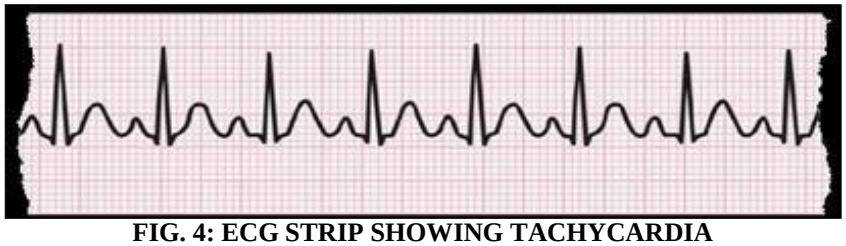

FIG. 4: ECG STRIP SHOWING TACHYCARDIA

Description of an Arrhythmia: Arrhythmias maybe described from their following characteristics:

1. Rate (*e.g.* tachycardia or bradycardia)

a. Tachycardia is defined as three or more consecutive impulses from the same pacemaker at a rate exceeding 100 bpm in adults (*i.e.* > 8 years of age).

b. Bradycardia is defined as three or more consecutive impulses from the same pacemaker at a rate less than 60 bpm.

2. Rhythm (*e.g.* regular or irregular)
3. Origin of impulse (*i.e.* supraventricular,ventricular, or artificial pacemaker)
4. Impulse conduction (*i.e.* atrioventricular,ventriculo-atrial or block)
5. Ventricular rate [3].

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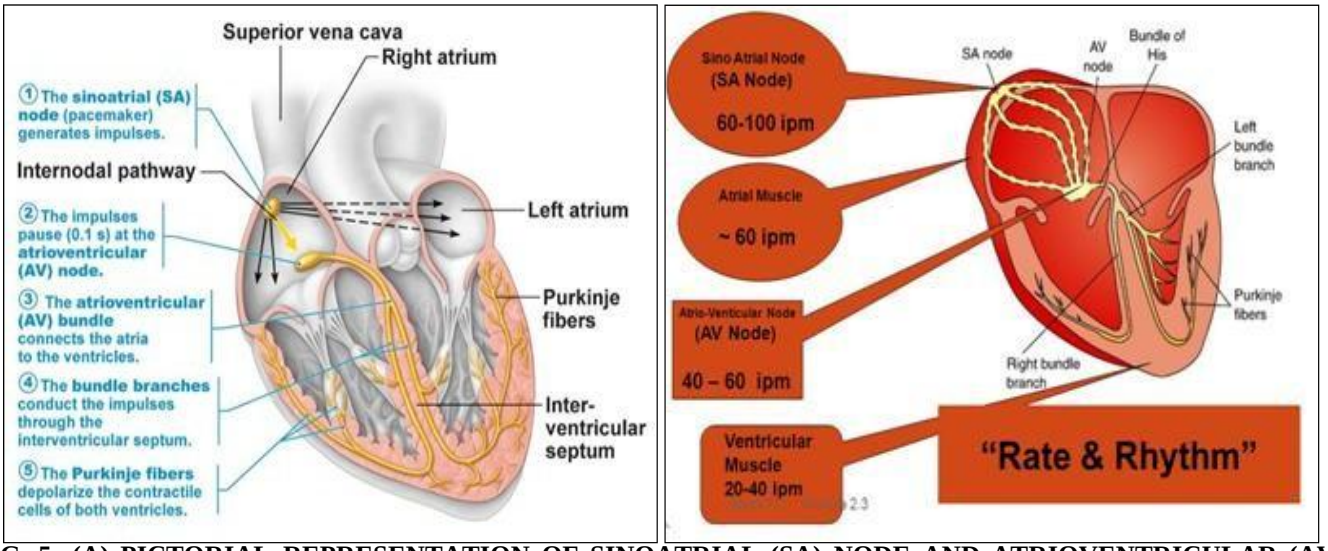

FIG. 5: (A) PICTORIAL REPRESENTATION OF SINOATRIAL (SA) NODE AND ATRIOVENTRICULAR (AV) NODE (B) INTRINSIC RATES (SA) NODE AND (AV) NODE

Descriptions of Premature Ventricular Contractions: Premature ventricular contractions (PVCs), sometimes called ventricular extra beats (VEBs). Premature ventricular beats occurring after every normal beat are termed "ventricular bigeminy". PVCs that occur at intervals of 2 normal beats to 1 PVC are termed "PVCs in trigeminy". Three premature ventricular grouped together is termed a "run of PVCs" in general, runslasting longer than three beats are referred to as

- ✓ Ventricular tachycardia
- ✓ Accelerated idioventricular rhythm
- ✓ Monomorphic ventricular tachycardia
- ✓ Polymorphic ventricular tachycardia
- ✓ Ventricular fibrillation [4].

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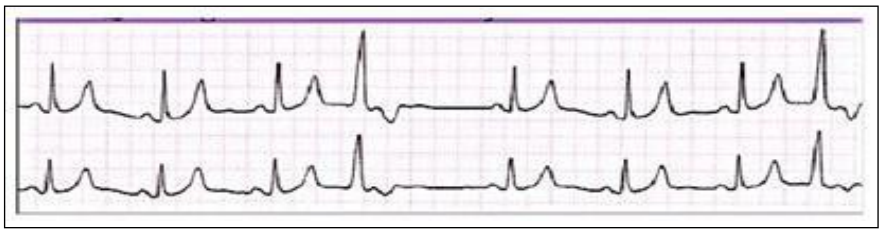
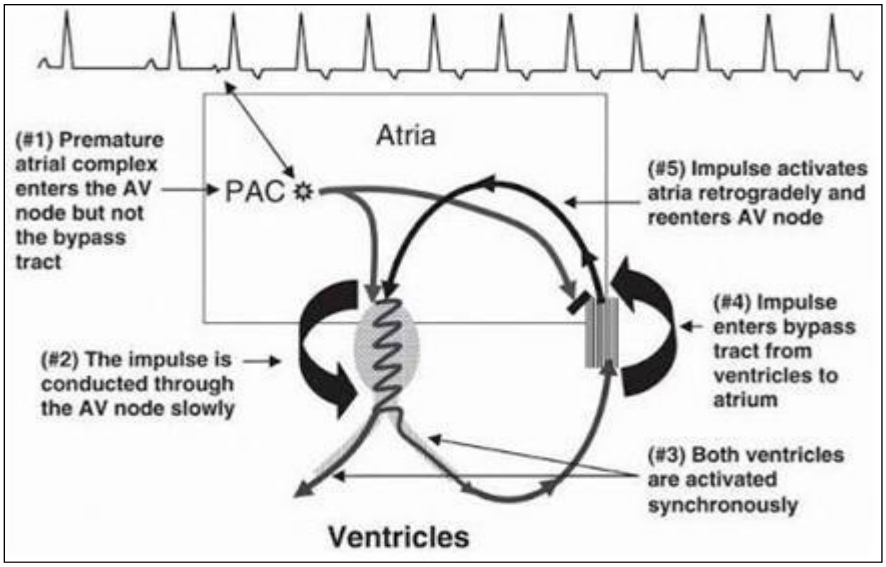


Heart Rate	Rhythm	P wave	PR interval (sec.)	QRS (sec.)
Var.	Irregular	No P waves associated with premature beat	NA	Wide > .12

Side Effects Associated with Arrhythmias:

- Chest pain
- Fainting
- Swelling of the feet or legs
- Shortness of breath
- Abnormally fast heartbeat
- Abnormally slow heartbeat
- Dizziness or light-headedness
- Cough



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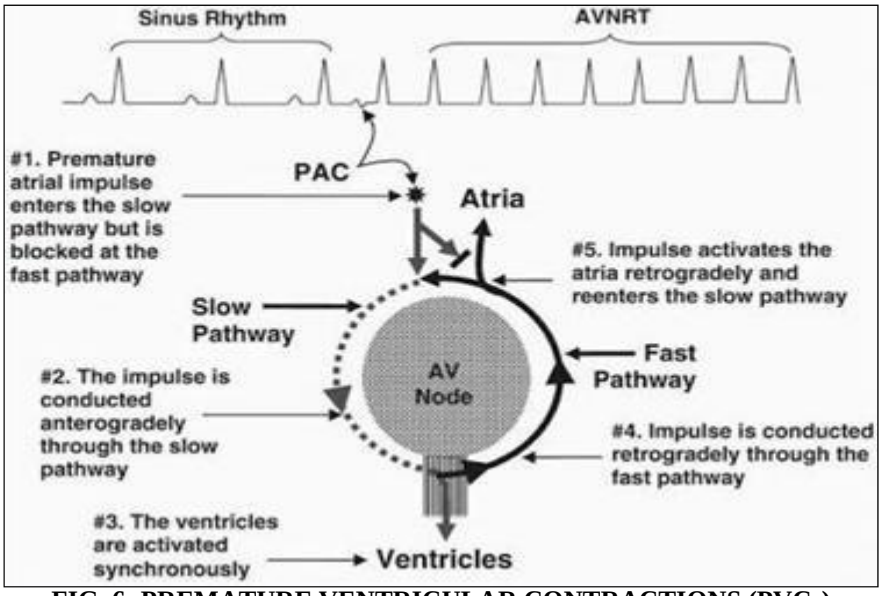

FIG. 6: PREMATURE VENTRICULAR CONTRACTIONS (PVCs)

Signs and symptoms: Some arrhythmias have no signs or symptoms, but when present the most common signs or symptoms a person will experience are:

- Palpitations (a feeling that your heart hasskipped a beat or is beating too hard)
- A slow heartbeat
- An irregular heartbeat

TABLE 1: SIGNS AND SYMPTOMS

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- Heart failure or cardiomyopathy, which weakens the heart and changes the way electrical signals move around the heart
- Heart tissue that is too thick or stiff or thathasn't formed normally
- Leaking or narrowed heart valves, which make the heart work too hard and can, lead to heart failure [5].

Symptoms	Risk Factors
Fatigue	Cardiomyopathy
Dizziness	Hyperthyroidism and hypothyroidism
Light headedness	CAD
Fainting [syncope] or near – fainting Spells	Smoking or alcohol consumption or caffeine drug abuse
Rapid heartbeat or pounding	Sleep apnea
Shortness of breath	Genetics
Chest pain	Stress
In extreme cases, sudden and cardiac arrest	Diabetes

Patho physiological of Arrhythmias: Heart arrhythmias caused by anomalous driving forcesstart irregular motivation conduction or the two components. Strange drive start incorporates improved ordinary automaticity, irregular automaticity and activated action coming about because of after depolarization. Though, the unusual driving forces are directed incorporates conduction piece and reentry. Albeit every one of these components is causes arrhythmias, it's unrealistic to demonstrate which instrument is in charge of an arrhythmia. Be that as it may, the conceivable system of assurance of numerousclinical arrhythmias in view of their qualities and conduct and to list rhythms most steady with known electrophysiological components [6 - 8].

Normal Automaticity: Normal automaticity involves the slow progressive depolarization of the membrane potential (spontaneous diastolic depolarization or phase four depolarization) until a threshold potential is reached, at which point an action potential is initiated. Although automaticity is an intrinsic property of all myocardial cells, the occurrence of spontaneous activity is prevented by the natural hierarchy of pacemaker function. The spontaneous discharge rate of the sinoatrial (SA) nodal complex exceeds that of all other subsidiary or latent pacemakers. As a result, the impulse initiated by the SA node depresses the activity of subsidiary pacemaker sites, before they can spontaneously depolarize to threshold. However, slowly depolarizing and previously suppressed pacemakers in the atrium, AV node, or ventricle can become active and assume pacemaker control of the cardiac rhythm if the SA node pacemaker becomes slow or unable to generate an impulse or if impulses generated by the SA node are unable to activate the surrounding atrial myocardium. The emergence of subsidiary or latent pacemakers under such circumstances is an appropriate fail-safe mechanism which assures that ventricular activation is maintained [9 - 12].

Role of Latent Pacemaker Cells in the Generation of Escape Beats: Application of increasing intensity of vagal stimulation (to elicit acetylcholine release and promote bradycardia) results in a shift of the origin of the heart beat from its normal site in the SA node to other sites in the atria or in the AV node. Further increases in stimulation intensity lead to very prolonged cardiac arrest and initiation of escape beats from the AV junction or the ventricles. Note: see the lack of p- wave in ECGs recorded during intense vagal stimulation (delay time above dotted line).

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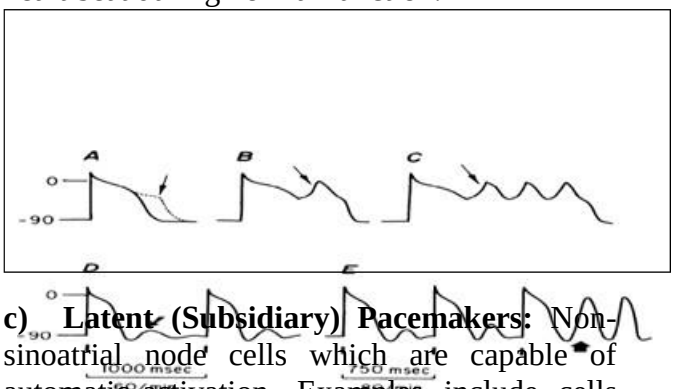
Abnormal Impulse Initiation:

a) Altered Normal Automaticity: Abnormal automaticity includes both reduced automaticity, which causes bradycardia, and increased automaticity, which causes tachycardia. Arrhythmias caused by abnormal automaticity can result from diverse mechanisms.

b) Enhanced Automaticity:

Primary Pacemakers: Cells from the sinoatrial node exhibiting normal automaticity. These cells are responsible for initiating the heart beat during normal function.



c) Latent (Subsidiary) Pacemakers: Non-sinoatrial node cells which are capable of automatic activation. Examples include cells from the atrioventricular (AV) junction, some fibers/cells at the pulmonary veins, and cells from the His- Purkinje system amongst others. Enhanced pacemaker can occur via three mechanisms: A negative shift in the threshold potential (TP), a positive shift in the maximum diastolic potential (MDP), and an increased rate of phase 4 depolarization. Atrial and ventricular myocardial cells do not display spontaneous diastolic depolarization or automaticity under normal conditions, but can develop these characteristics when depolarized, resulting in the development of repetitive impulse initiation, a phenomenon termed depolarization-induced automaticity.

- ✓ An increase in extracellular potassium, which reduces the reversal potential for I_{K1}, the outward current that largely determines the resting membrane or maximum diastolicpotential;
- ✓ A reduced number of I_{K1}channels;
- ✓ A reduced ability of the I_{K1} channel to conduct potassium ions; or
- ✓ Electro tonic influence of neighboring cells in the depolarized zone [13 - 15].

Triggered Rhythms: Triggered activity results from the premature activation of cardiac tissues by after depolarization's, which are depolarizations triggered by one or more preceding action potentials. One occurs early during the repolarization of the membrane, an early after depolarization, and the other occurs after the repolarization is complete or nearly completed, a delayed after depolarization [16]. When either type is large enough to reach threshold, the resulting action potential is called a triggered action potential. Triggered activity will cause arrhythmias when impulse initiation shifts from the sinus node to the triggered focus. For this, the rate of triggered impulses must be faster than the rate of the sinus node, an event that may be brought about when the sinus node has been slowed or inhibited, when it has been blocked, or when the triggered focus is intrinsically faster [17 - 19].

FIG. 7: A, B, C SHOWS THE EARLY AFTER DEPOLARIZATION AND D AND E SHOWS DELAYED AFTER DEPOLARIZATION

After - depolarization Phenomena

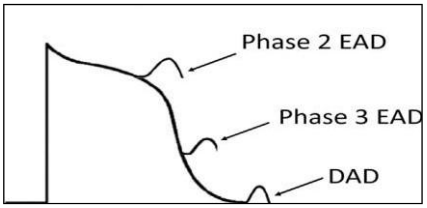


FIG. 8: AFTER DEPOLARIZATION PHENOMENA: EARLY AFTER DEPOLARIZATION (EAD) OCCURS EARLY (PHASE 2) OR LATE (PHASE 3), AND DELAYED AFTER DEPOLARIZATION (DAD) OCCURS DURING PHASE 4 OF THE ACTION POTENTIAL

EADs is typically observed in cardiac tissues exposed to injury, altered electrolytes, hypoxia, acidosis, catecholamines, and pharmacologic agents, including anti-arrhythmic drugs. Ventricular hypertrophy and heart failure also predispose to the development of EADs. EAD characteristics vary as a function of animal species, tissue or cell type, and the method by which the EAD is elicited. Early after depolarizations (EADs) can develop before full repolarization, corresponding to phase 2 or phase 3 of the cardiac action potential in humans [20]. They are usually but not exclusively associated

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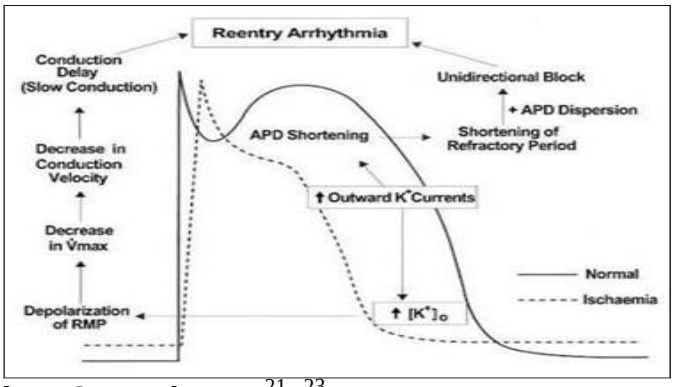
with prolonged action potential durations (APDs), which occur when the inward current is greater in amplitude than the outward current. Whatever be the underlying mechanism, if the change in membrane potential brought about by the EAD is sufficiently large, it will activate I_{Na}, resulting in triggered activity. EADs and their resulting triggered activity is thought underlie the arrhythmogenesis observed in heart failure and



long QT syndromes [21 - 23].

Delayed After Depolarizations: DADs and DAD-induced triggered activity are observed under conditions that augment intracellular calcium, $[Ca^{2+}]$, such as after exposure to toxic levels of cardiac glycosides (digitalis) or catecholamine. This activity is also manifest in hypertrophied and failing hearts as well as in Purkinje fibers surviving myocardial infarction. In contrast to EADs, DADs are always induced at relatively rapid rates. Delayed after depolarizations (DADs) were first described as oscillatory after potentials. They can develop after full repolarization, corresponding to phase 4 of the cardiac action potential in humans. It is worth noting that DADs and late EADs are somewhat similar. Both occur under conditions of intracellular calcium over load and involve spontaneous release of calcium from the sarcoplasmic reticulum. The difference appears to be the timing of this release, which occurs during their polarizing phase of the action potential in the case of late EADs, and at the resting membrane potential for DADs. Indeed, for atrial fibrillation, both EADs and DADs have been implicated as the mechanisms of arrhythmogenesis [24 - 25].

Re-entry: Re-entry occurs when an action potential fails to extinguish itself and reactivates a region that has recovered from refractoriness. It can be divided into two types:

- Re-entry that occurs in the presence of an obstacle, around which an action potential can travel (circus-type)

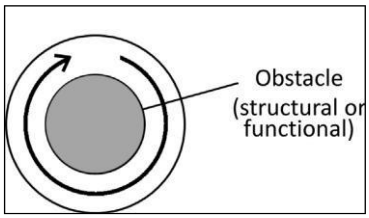


FIG. 9: CIRCUS - TYPE RE-ENTRY

- Re-entry that occurs without an obstacle(reflection or phase 2).

FIG. 10: RE - ENTRY OCCURS WITH AN OBSTACLE

Recently, a different type of reflection, called the expansion- type, has been demonstrated. Ante grade propagation of an impulse occurs from an arrow isthmus region to an expanded distal region. The activation wave front has an outward curvature (convex) and stimulates a higher number of cells in the expanded region, where the source-sink mismatch is greatest. This causes the direction of electro tonic currents to be reversed, intern prolonging the action potential. This in turn initiates retro grade propagation long the same path [26].

3. Cardiac Ablation: Cardiac ablation is a strategy that can rectify arrhythmias. This is a method to treat atrial fibrillation (AF), a kind of sporadic pulse. It can help keep your pulse in an ordinary mood. Removal, for the most part, utilizes long, adaptable tubes (catheters) embedded through a vein in your crotch and strung to your heart to adjust auxiliary issues in your heart that reason an arrhythmia. It works by decimating tissue in the heart that triggers an unusual heartbeat. Now and again, removal keeps irregular electrical signs from going through the

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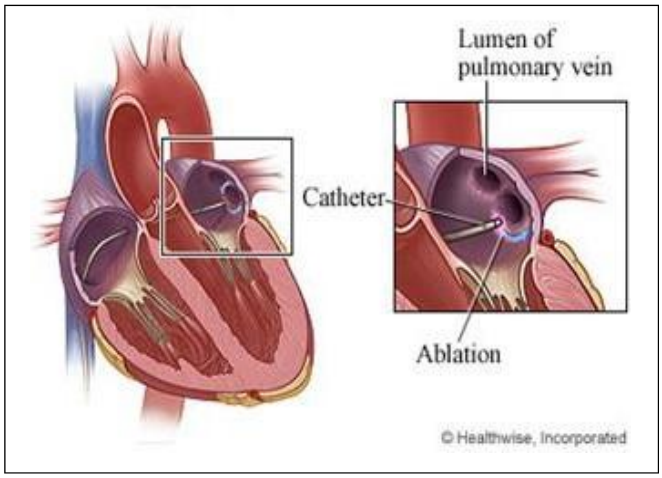
heart and, along these lines, stopsthe arrhythmia. Cardiovascular removal is in some cases done through open-heart surgery, however, it's frequently done utilizing catheters, making the technique less obtrusive and shortening recuperation times [27 - 29].

About Catheter Ablation: Catheter removal is likewise used to help control other heartbeat issues,

for example, atrial shudder and atrial fibrillation. Catheter removal devastates the anomalous tissue without harming whatever is left of the heart. Extraordinary cells in the heart make electrical signs that go along pathways to the assemblies of your heart. These signs make the hearts upper and lower chambers beat in the correct arrangement. Anomalous cells might be made by sorted out electrical signs that reason sporadic or fast heartbeats called arrhythmias. At the point when this happens,



the heart may not pump blood successfully and you may feel blackout, shy of breath and frail. Prescriptions to treat fast and sporadic heartbeats work exceptionally well for the vast majority. Be that as it may, they don't work for everybody, and they may cause symptoms in a few people. In these cases, specialists may recommend catheter removal. The system is utilized regularly to treat a condition called supraventricular tachycardia, which happens due to irregular conduction strands in the heart [28].

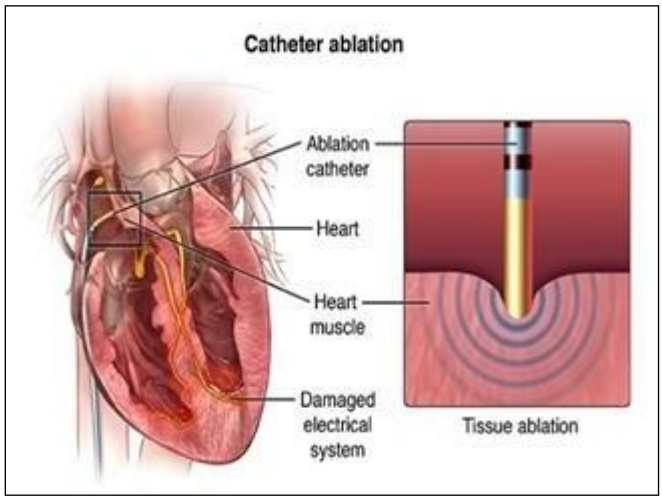


FIG. 11: REPRESENTATION OF CATHETER ABLATION IN LUMEN OF PULMONARY VEIN

Before AF Ablation:

- **An EKG (Electrocardiogram):** This simple, painless test records the heart's electrical activity. The test shows how fast the heart is beating and its rhythm. An EKG also records the strength and timing of electrical signals as they pass through your heart.
- **Echocardiography:** This is a painless test that uses sound waves to create of the heart & also

  show how well the heart's chambers and valves are working. Stress testing - Some heart problems are easier to diagnose when the heart is working hard and beating fast [29].

Expect After AF Ablation:

- Chest pain is common
- Arrhythmia
- Resting heart rate changes
- Digestive problems

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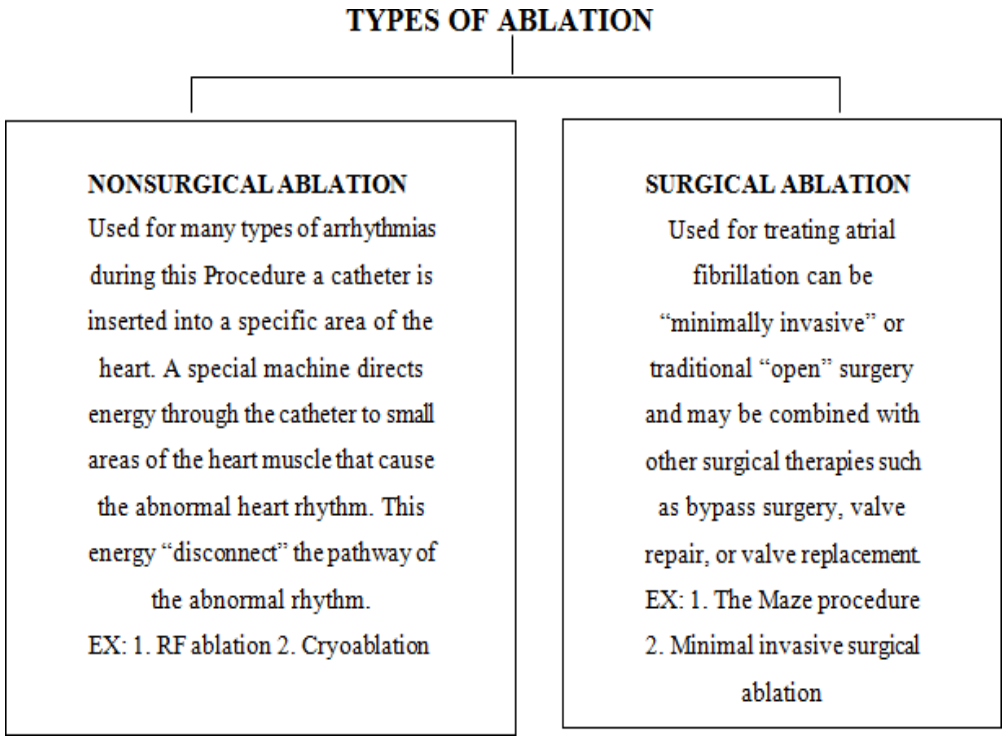

4. Ablation for Supraventricular Arrhythmias: Although an established therapy for many years, improvements in electrophysiological techniques have led to catheter ablation becoming first linetherapy for most patients with AV nodal re-entrant tachycardia (AVNRT) or WPW and for many patients with atrial flutter.

Atria Ventricular Nodal Re-entrant Tachycardia (AVNRT): AVNRT is caused by re- entry within the AV node, using ‘‘fast’’ and ‘‘slow’’ pathways. Ablation of the slow pathway to cure AVNRT has become a comparatively straightforward and routine procedure. The most significant complication associated with slow pathway ablation is damage to the fast pathway and hence AV blocks requiring pacing. In one largeseries of more than 8000 patients, long term cure was achieved in 99% of patients with high grade AV block necessitating pacing occurring in only 0.4%. 1. Catheter ablation has become the treatment of choice in patients with symptomatic AVNRT. Indeed, many patients and their physicians choose ablation asfirst line therapy to avoid the need for anti-arrhythmic drug therapy [30 -34].

CONCLUSION: Cardiac arrhythmias and ECG abnormalities occur frequently in patients with acute stroke, either with or without coexisting cardiac diseases. The incidence and prevalence of arrhythmias and significantly abnormal ECGs depend on the types and on set of strokes. Theduration and equipment used for cardiac monitoring and criteria used for determination and classification of arrhythmias various steps involved in pathophysiological of arrhythmias to cause the AF and finally sudden death. The master administration of arrhythmias has changed altogether finished the previous decade. This is halfway a result of a noteworthy increment in the confirmation base, mainly in the rate contrasted and mood wrangle for the administration of atrial fibrillation. It is our goal and hope as a next step to promote catheter ablation as a first-line therapy in more patients with AF based on definitive evidence that the procedure reduces mortality and morbidity. In addition, as technology has advanced ablation has found a significant role in the management of all arrhythmias.

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