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Cardiac Arrhythmias, Types, Complication and Treatment

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Abstract: **A cardiac arrhythmia simply defined is a variation from the normal heart rate and/or rhythm that is not physiologically justified. Arrhythmias are normally divided into 2 categories: ventricular and supraventricular. The present paper intended to fogous' on discussing the cardiac arrhythmias from different aspects, most important the treatment approaches of most common types of this devastating condition which is, supraventricular tachycardia (SVT). We conducted a computerized Literature search involving human subjects, published in English until December 2016, and indexed in MEDLINE (through PubMed), EMBASE, and the Cochrane Library. We reviewed articles related to supraventricular tachycardia (SVT) previously published by the ACC, AHA, and Heart Rhythm Society (HRS). References selected and published in each identified study was manually searched for more relevant articles. Understanding of the mechanism of each SVT is essential in figuring out management at the bedside and in the electrophysiology laboratory. Acknowledgment, identification, and differentiation of the various SVTs are of excellent significance in formulating an effective treatment method. Developments over the past 4 years have enabled the precise diagnosis of SVTs. Today, In the acute setting, both vagal maneuvers and pharmacologic therapy can be effective in arrhythmia termination most SVTs effective and safe.**

Keywords: **Supraventricular Tachycardia (SVT), Heart Rhythm Society (HRS).**

1. INTRODUCTION

A cardiac arrhythmia simply defined is a variation from the normal heart rate and/or rhythm that is not physiologically justified. Arrhythmias are normally divided into 2 categories: ventricular and supraventricular. Ventricular arrhythmias happen in the lower chambers of the heart, called the ventricles. Supraventricular arrhythmias take place in the location above the ventricles, normally in the upper chambers of the heart, called the atria [(1)]. The irregular beats can either be too slow (bradycardia) or too quick (tachycardia). The four primary types of arrhythmia are premature (extra) beats, supraventricular arrhythmias, ventricular arrhythmias, and bradyarrhythmias [(1).] Clinical evaluation can help, whether an ECG is offered or not. The key thing to bear in mind is that there are couple of parts in the electrical "wiring" of the heart (**Figure 1**) [(2)] and the pulse can only be quick or slow, routine or irregular. If an ECG is offered, Broad (> 0.12 seconds) is more likely to be ventricular tachycardia (VT) and will typically be more concerning than narrow, which recommends a high depolarization site and is likely to be supraventricular tachycardia (SVT) [(1,2)]. Supraventricular tachycardia (SVT) is a basic term describing a group of arrhythmias whose mechanism is or includes above the atrioventricular node. SVT is tachycardia having an electropathologic substrate occurring above the bundle of His and triggering heart rates exceeding 100 beats per minute [(3)]. Orejarena et al [(3)] released an epidemiologic study of SVT in the general population. Based on 1990 census information, the incidence of SVT is estimated to be 36/100,000 person-years and the prevalence is 2.29/ 1000 individuals [(3)].

Accelerated rhythms can be frightening to the patient if recurrent or relentless, and can cause significant morbidity. This short article concentrates on the most typical types of paroxysmal SVT: atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), and atrial tachycardia (AT). Atrial fibrillation and flutter are categorized as types of SVT, they will not be gone over in this short article and are evaluated elsewhere [(4,5)]. (**Table 1**) lists the two common types of SVT and typical mechanisms and their incidence in general population [(6,7)]. (**Figure 1**) [(2)] illustrates AVNRT, AVRT, AT, and normal sinus rhythm.

Table1: Common Types of Supraventricular Tachycardia and Usual Characteristics [4,5]

TYPE	*EPIDEMIOLOGY*	*MECHANISM*
AVNRT	Most common SVT (approximately 50 to60%) [4] Occurs more often in younger women	Reentry caused by nodal pathways or tracts (two types): atypical (fast/slow) represents 10% and typical (slow/fast) represents 90% of all AVNRT
AVRT	Second most common SVT (approximately 30%) Orthodromic most common type (81 to 87%)Occurs more often in younger women and childrenMay be comorbid with Wolff-Parkinson-White syndrome [4,5]	Reentry caused by accessory pathways (two types): orthodromic (antegrade conduction through atrioventricular node) and antidromic (retrograde conduction through atrioventricular node)

Objective:

The present paper intended to fogous' on discussing the cardiac arrhythmias from different aspects, most important the treatment approaches of most common types of this devastating condition which is, supraventricular tachycardia (SVT).

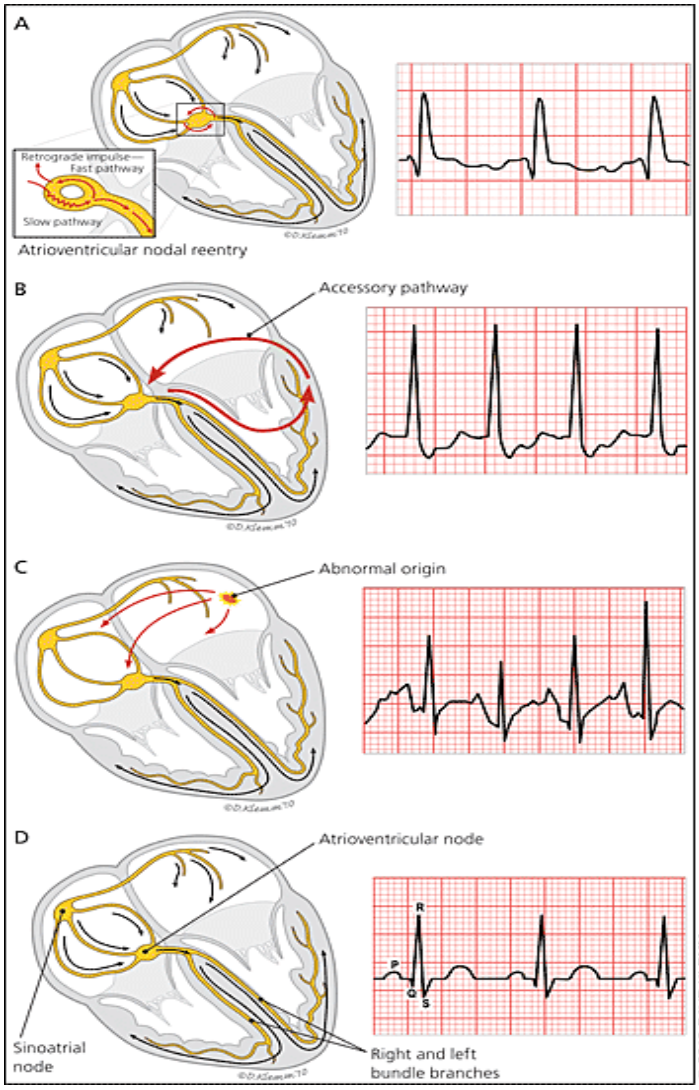

Figure1: *(A)* In typical atrioventricular nodal reentrant tachycardia, the retrograde P wave may not be seen or may be visible early after the QRS complex. *(B)* In atrioventricular reciprocating tachycardia, there is typically a short RP interval *(C)* Atrial tachycardia typically produces variable RP and PR intervals. In atrial tachycardia, the morphology and axis of the P wave are influenced by atrial site of origin and tachycardia mechanism. Short- and long-term therapies are discussed in the text. *(D)* Normal sinus rhythm.

2. METHODOLOGY

We conducted a computerized Literature search involving human subjects, published in English until December 2016, and indexed in MEDLINE (through PubMed), EMBASE, the Cochrane Library. We reviewed articles related to supraventricular tachycardia (SVT) previously published by the ACC, AHA, and Heart Rhythm Society (HRS). References selected and published in each identified study was manually searched for more relevant articles.

3. RESULTS

Overview:

Recent years have actually witnessed essential advances in everybody understanding of the electrophysiologic mechanisms underlying the development of a range of heart arrhythmias. The systems responsible for heart arrhythmias are usually divided into 2 significant classifications: (A) enhanced or abnormal impulse formation (ie, focal activity) and (B) conduction disruptions (ie, reentry) (**Figure 2**) [(3)].

Unusual automaticity consists of both decreased automaticity, which causes bradycardia, and increased automaticity, which causes tachycardia. Arrhythmias caused by unusual automaticity can result from diverse systems (**Figure 2**) [(3)]. Modifications in sinus rate can be accompanied by shifts of the origin of the dominant pacemaker within the sinus node or to subsidiary pacemaker sites elsewhere in the atria. Impulse conduction from the SA mode can be impaired or obstructed as a result of disease or increased vagal activity causing advancement of bradycardia. AV junctional rhythms occur when AV junctional pacemakers situated either in the AV node or in the His bundle speed up to exceed the rate of SA node, or when the SA nodal activation rate was too sluggish to reduce the AV junctional pacemaker [(8)].

Due to the fact that of genetic anomalies that result in abnormalities of either membrane clock or Ca clock systems of automaticity, Bradycardia can happen in structurally typical hearts. One example is the anomaly of hyperpolarization-activated nucleotide-gated channel (HCN4), Mutations of the HCN4 may trigger familial bradycardia as well [(8,9)].

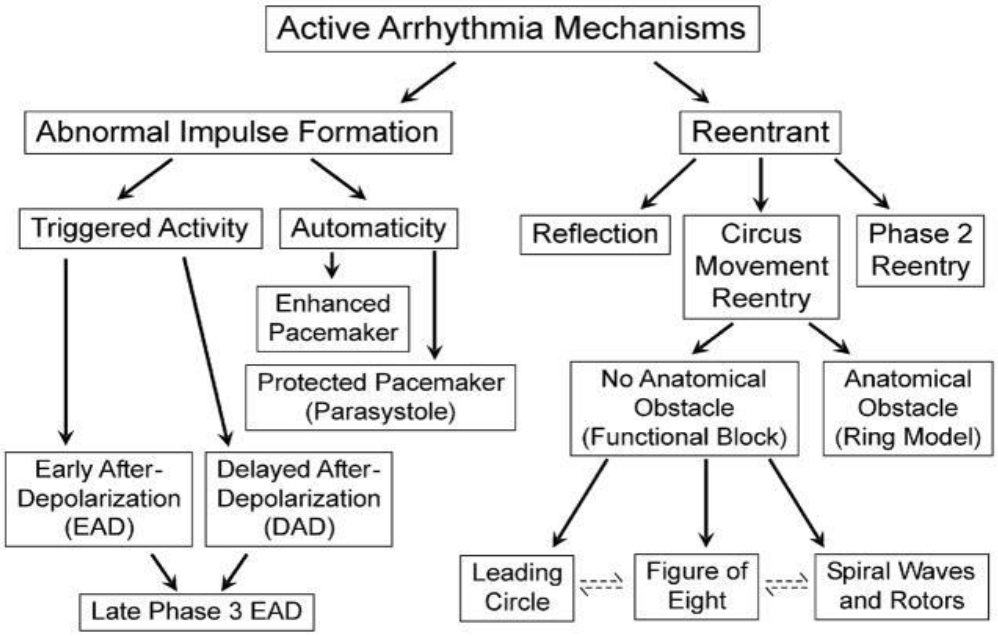

Figure 2: Classification of active cardiac arrhythmias. [(3)]

Mechanisms of supraventricular tachycardia (SVT):

SVT is caused by 1 of 3 mechanisms as discussed above: reentry, increased automaticity, or triggered activity [(10,11,12)].

Reentry includes repetitive impulse propagation around 2 limbs or paths, separated by a zone of non-excitability [(10,11,12,13)]. For reentry to happen, the 2 limbs should have differing electrophysiologic properties (conduction speed and refractory duration). Atrial or ventricular early beats or runs of tachycardia (automated or set off in origin) lead to a unidirectional block in one limb with circular repeated reentry in the opposite instructions (**Figure 1 & 2**). Reentry is the system of all cases of AVNRT and AVRT and of some cases of atrial tachycardia [(10,11,12,13)].

The term automaticity refers to the phenomenon of spontaneous tachycardia arising from a group, or focus, of cells and can lead to ventricular and atrial arrhythmias. These foci of myocardial tissue show pathologic modifications in resting membrane capacity and can develop the capacity to depolarize quickly and become the dominant rhythm. Automaticity can happen in the setting of electrolyte abnormalities, changes in free tone, and other regional factors, much of which are not understood. Automatic foci are the mechanisms of more than 70% of cases of focal atrial tachycardia [(11)]. Automatic arrhythmias and arrhythmias caused by triggered activity matter due to the fact that in addition to being the systems of sustained arrhythmias, brief bursts of these arrhythmias are sets off for reentrant SVTs [(11,12)]. Arrhythmias due to triggered activity are triggered by extra depolarizations instantly following cellular repolarization (called delayed afterdepolarizations) that may lead to extra-systoles and sustained tachyarrhythmias. Activated activity can happen in action to enhancement of intracellular calcium (eg, with heart glycosides) and is the mechanism of approximately 30% of focal atrial tachycardia. Triggered activity is likewise implicated in the initiation of atrial fibrillation [(11,12)].

A. Atrioventricular Nodal Reentrant Tachycardia (AVNRT):

AVNRT is the commonest form of routine supraventricular tachycardia in human beings, and provides as routine narrow complex tachycardia on the ECG. AVNRT can be common (also described as the common form) or atypical (the uncommon type) depending on the area of the atrial deflection between successive QRS complexes. This is believed to reflect direction of reentrant excitation with the AV nodal circuit [(14)]. Normal AVNRT or the "slow-fast" type is the commonest (> 80%) type of AVNRT. The earliest site of retrograde atrial activation in this type is seen in the region of the fast path near the pinnacle of the triangle of Koch. It is generally initiated by an atrial premature depolarization which, by virtue of prematurity, discovers the fast pathway refractory and conducts to the ventricles by means of the slow path (**Figure 3**) [(15)], middle panel); the surface area ECG records a prolonged PR interval. If adequate time has actually lapsed to allow healing of the fast path, the impulse can perform quickly up the fast path, leading to a common AV nodal "echo" beat; the surface area ECG records a short RP period. AVNRT is initiated if this pattern perpetuates. Typical AVNRT can be initiated by numerous or single atrial premature complexes [(15)]. An estimated 32%-40% of the typical population have dual atrioventricular nodal physiology, although just a minority of these develop AVNRT [(16)]. The quick path inputs into the peak of the triangle of Koch near the compact atrioventricular node. The sluggish pathway inputs near the os of the coronary sinus at the base of the triangle. In the common kind (slow-fast AVNRT), conduction occurs antegrade through the sluggish pathway and retrograde via the fast path. Irregular types of AVNRT such as fast-slow and slow-slow are likewise encountered and are among the causes of long RP tachycardia [(17,18)].

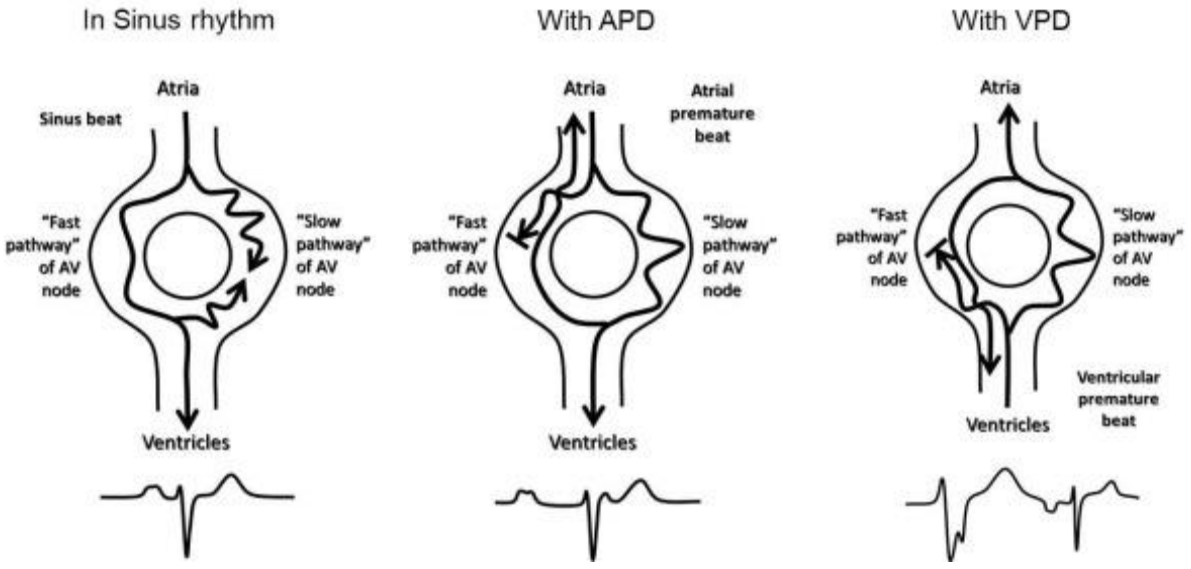

Figure3: Model of dual AV nodal pathways physiology in sinus rhythm, with an atrial premature beat (APD) which initiates typical "slow-fast" AVNRT, and with a ventricular premature beat (VPD) which initiates atypical "fast-slow" AVNRT. [(15)]

B. Atrioventricular Reentrant Tachycardia (AVRT):

Accessory paths are anomalous, extranodal conduction pathways between atrium and ventricle. The accessory path and the atrioventricular node can function as the 2 limbs of the AVRT reentrant circuit. Device pathways are classified based upon their place along the tricuspid or mitral annuli, conduction characteristics, and directionality of conduction

(antegrade, retrograde, or both). Accessory paths capable of antegrade conduction will show a delta wave called the Wolff-Parkinson-White (WPW) pattern on the surface electrocardiogram (ECG) (**Figure 4**) [19]. Device paths capable only of retrograde conduction do not exhibit a delta wave on the surface ECG (hidden pathways) but can still form a reentrant loop with the atrioventricular node and lead to AVRT [19,20]. AVRT is classified as orthodromic if antegrade conduction occurs through the atrioventricular node and antidromic if antegrade conduction takes place via the accessory pathway.

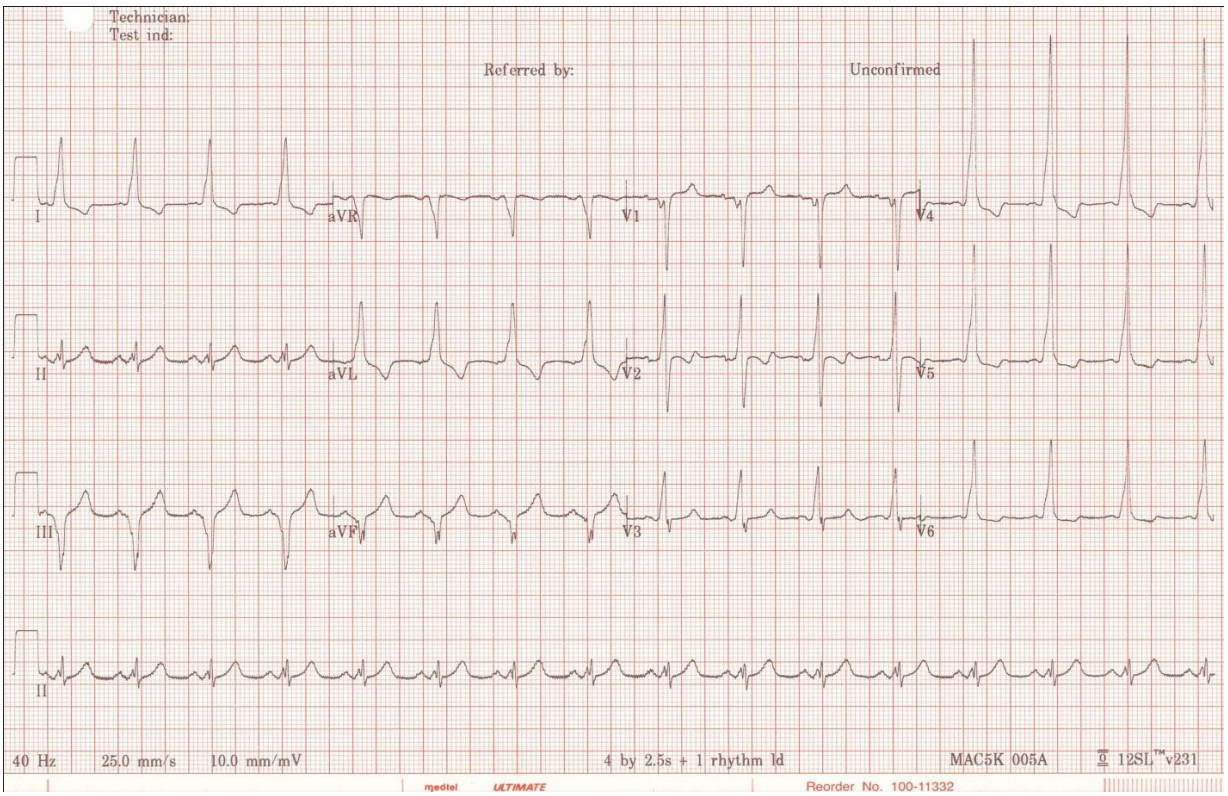

Figure 4: Electrocardiogram (ECG) showing a short PR interval and slurred R wave upstroke (delta wave) characteristic of the Wolff-Parkinson-White pattern. [19]

C. Atrial Tachycardia (AT):

The term atrial tachycardia explains a group of atrial arrhythmias whose systems might be macroreentrant or focal. Focal atrial tachycardia leads to a centrifugal wavefront spread throughout the atria from a focus of (A) improved automaticity, (B) triggered activity, or (C) a small area of reentry (microreentry). Macroreentrant atrial tachycardia arises from a large intraatrial reentrant loop > 2 cm in length. Note that the term macroreentrant atrial tachycardia is associated with the term atrial flutter. Both focal and macroreentrant atrial tachycardia may result from previous structural heart problem or surgical treatment for congenital heart disease, may follow surgical or catheter ablation of atrial fibrillation, or may be due to idiopathic phenomena. Atrial tachycardia often emerges from the crista terminalis however can likewise occur from the tricuspid annulus, compact atrioventricular node, and coronary sinus ostium [21,22]. Left atrial tachycardias typically arise from the pulmonary veins, as well as the mitral annulus and left atrial appendage [22].

Treatment approaches of SVT:

Vagal Maneuvers approach for treatment of SVT.

Vagal maneuvers are an appropriate first treatment option in patients with hemodynamically steady SVT. Research studies report an approximately 25% success rate, although reported rates differ widely in the literature (6%-54%) [23,24,25]. The most commonly performed maneuvers are the Valsalva maneuver and carotid sinus massage. The boost in intrathoracic pressure resulting from the Valsalva maneuver promotes aortic and carotid baroreceptors, causing a boost in vagal input into the atrioventricular node. Latest research studies advocate placing the patient in a supine position and attempting the maneuver for 15-20 seconds [23,24,25]. The Valsalva maneuver has actually typically been revealed to be most reliable in adults, having an exceptional effect on SVT termination compared to carotid sinus massage. Care is encouraged when thinking about whether to try carotid sinus massage in older patients, as there is a risk of carotid

atheroembolism and stroke even in the absence of an audible bruit. No standards currently exist regarding the appropriate variety of attempts prior to initiating other therapies, although a lot of companies will attempt an optimum of 2 efforts. A number of research studies have actually suggested that vagal maneuvers are more reliable in the termination of AVRT compared with AVNRT [23,24,25,26,27].

Pharmacologic Treatment option for SVT:

Pharmacologic treatment for severe termination of SVT is appropriate in patients when vagal maneuvers stop working. The preferred preliminary agents are intravenous (IV) adenosine or a nondihydropyridine calcium channel blocker. Adenosine's impacts are moderated by membrane hyperpolarization that generally happens within 15-30 seconds after administration. Adenosine has a powerful result on the atrioventricular node and is highly effective in causing short-lived, total atrioventricular nodal block. Short-term sinus bradycardia or sinus arrests often happen but are short lived [28,29]. The ECG needs to be constantly taped during adenosine administration to document the impact of the drug on SVT and to keep an eye on for the uncommon incident of proarrhythmia. Patients must be cautioned that they might experience temporary sensations of claustrophobia, chest, and dyspnea discomfort. The initial dose of adenosine is 6 mg IV bolus (flushed), utilizing a big vein. Subsequent dosages of 12 mg or even 18 mg boluses may be needed. SVT resolution following adenosine administration establishes AVNRT or AVRT as the most likely SVT mechanism, although occasionally some focal atrial tachycardias will terminate with adenosine. In macroreentrant and most focal atrial tachycardias, adenosine administration leads to transient slowing down of the ventricular rate, revealing the underlying atrial activity. Adenosine is contraindicated in patients with WPW and atrial fibrillation due to the fact that it shortens the refractory duration of the path that can result in more quick conduction of atrial fibrillation, increasing the opportunities of hazardous ventricular arrhythmias [28,29]. Because propagation of the action potential through the atrioventricular node is calcium-channel reliant, the nondihydropyridine calcium channel blockers verapamil and diltiazem are extremely efficient in the termination of AVNRT and AVRT [30]. Verapamil is infused at a dose of 5 mg during 2 minutes, followed by another 7.5 mg after 5-10 minutes if needed. Diltiazem is given at a dose of 20 mg IV, with another 25-35 mg provided if SVT persists. As with adenosine, short-term arrhythmias might be seen, although hypotension happens more commonly in patients receiving calcium channel blockers, particularly if SVT continues after administration. Previous studies have actually demonstrated comparable effectiveness between adenosine and calcium channel blockers, [28] although adenosine is normally preferred provided its lower incidence of hypotension and its exceptionally short half-life. Beta blockers such as IV metoprolol or esmolol infusion are frequently used in severe SVT, however information regarding this practice are restricted. Because of the marked supremacy of diltiazem [31], a little randomized study comparing IV diltiazem to esmolol for severe SVT was terminated early.

Other drugs which are; long-acting calcium channel blockers and beta blockers enhance symptoms in 60%-80% of patients with SVT [28,32]. Flecainide and propafenone are class Ic antiarrhythmic drugs that slow conduction and reduce automaticity and can thus lead to marked reduction in frequency and period of SVT episodes in some patients. These agents are contraindicated in patients with recognized structural cardiovascular disease because of an increased risk of ventricular arrhythmias [32,33]. Flecainide may lead to headaches, visual disturbances, moderate stomach pain, and nausea. These negative effects may require its discontinuation. Propafenone may be somewhat more widely tolerated however might be connected with a metal taste. These antiarrhythmic drugs need to be initiated and subsequented by an electrophysiologist experienced in their usage. For example, both drugs can result in assistance of 1:1 conduction of atrial flutter and for that reason need to be coprescribed with an atrioventricular nodal blocker in the setting of certain atrial arrhythmias, including atrial fibrillation.

Alternative antiarrhythmic drugs consist of sotalol or dofetilide, both class III agents. Like the class Ic agents, these drugs have actually been used successfully in patients with AVRT, AVNRT, and atrial tachycardia. Class III antiarrhythmic drugs exert their impact in this setting through prolongation of refractory period, prevention of reentry propagation, and suppression of automaticity [34].

4. CONCLUSION

Understanding of the mechanism of each SVT is essential in figuring out management at the bedside and in the electrophysiology laboratory. Acknowledgment, identification, and differentiation of the various SVTs are of excellent significance in formulating an effective treatment method. Developments over the past 4 years have enabled the precise diagnosis of SVTs. Today, In the severe setting, both vagal maneuvers and pharmacologic therapy can be effective in arrhythmia termination most SVTs effective and safe. And newer techniques have considerably enhanced ablation effectiveness and reduced periprocedural problems and treatment times.

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