INTRODUCTION

Supraventricular tachycardia (SVT) includes a variety of reentrant fast heart rhythms originating in any part of the heart's conduction system above the ventricles. The major types of SVT include atrioventricular reentrant tachycardia, atrioventricular reciprocating tachycardia (antidromic and orthodromic), focal atrial tachycardia, atrial flutter (typical and atypical) and atrial fibrillation [1].

AV nodal reenterant tachycardia (AVNRT) is the most common type of reentrant supraventricular tachycardia (SVT) as it represents up to 60% of patients presented with paroxysmal supraventricular tachycardia (PSVT). In AV nodal reentry, the reentrant circuit is usually said to be enclosed within the AV node. In patients with AV nodal reentry, the AV node is functionally divided into two longitudinal pathways (Fast and slow pathways) which is called dual AV nodal pathways and these pathways formed together the reentrant circuit [2].

AV Reentrant tachycardia (AVRT) is the second most common type of PSVT representing about 30% of the cases. In AVRT, an accessory pathway (AP) of atrioventricular (AV) conduction may develop circus movement tachycardia. Orthodromic AVRT is the most common form. It occurs as a result of antegrade conduction through the normal AV conduction system and retrograde

conduction to the atria via the AP. Less commonly, conduction occurs in the opposite direction resulting in antidromic AVRT [3].

Distinction between atrioventricular node re-entrant tachycardia (AVNRT) and orthodromic AVRT using concealed (AP) is sometimes difficult using location of retrograde P waves on ECG [4].

As the diagnosis of concealed atrioventricular accessory pathway frequently requires some electrophysiological work up, since the electrographic signs of preexitation during sinus rhythum are lacking and the ability of standard surface ECG to recognize its presence during tachycardia is limited[5].

Recently, several phenomena have been described (usually as indices) that can aid in distinguishing AVNRT from orthodromic form of AVRT [6].

There is a recent study that has been performed using Tachycardia entrainment from the right ventricular (RV) apex and again from its base using pacing at 10-40-ms faster than the tachycardia cycle length then stimulus-atrial (SA) interval was measured from stimulus to the earliest atrial electrogram and Ventricle to atrium (VA) interval was measured from the ventricular electrogram at RV catheter in both sites; RV apex and RV base; to the earliest

atrial electrogram during tachycardia. Finally The SA-VA interval was calculated from both sites and the difference between them was calculated. The [SA-VA] apex–[SA-VA] base was -9.4 ± 6.6 in AVNRT and 10 ± 11.3 in AVRT and the difference was negative for all AVNRT cases and positive for all septal accessory pathways (APs)[7].

Another way for differentiation between both tachycardias in electrophysiology lab is to measure stimulo-atrial time during RV apical pacing and during RV basal pacing where this interval should be shorter at basal site than apical sites in case of presence of septal AP[8].

Another way for differentiation is by measuring the PPI (Post-pacing index technique) which is the time required for the last stimulated orthodromic wavefront to reach the excitable gap of a circuit, travel around the circuit, and return to the pacing site. If the pacing site is in the circuit, then the PPI= tachycardia CL (TCL). The farther a pacing site is from a circuit, the greater the PPI-TCL difference will be. Because the RV apex is close to orthodromic AVRT circuits involving right sided or septal APs, yet relatively far from AVNRT circuits, the PPI-TCL difference after entrainment from the RVA can be used to distinguish AVNRT from AVRT (particularly when atrial activation is concentric). A PPI-TCL difference > 115 ms is

consistent with AVNRT, while a PPI-TCL difference < 115 ms is consistent with AVRT[9].

Another technique for diagnosing the participation of retrograde concealed bypass tract (CBT) is the ability of a ventricular premature beat (VPB) to depolarize the atrium with the same atrial activation sequence as SVT when the His bundle is refractory (Atrial pre-excitation) [10].

The response most commonly seen and easiest to demonstrate is atrial capture by VPBs when the His-Purkinje system is known to be refractory, that is, when antegrade His bundle depolarization is already manifest. As long as the antegrade His bundle deflection is not influenced by ventricular stimulation, excitation of the atria by a VPB must be over a bypass tract. It is possible to deliver the stimulus 35 to 55 msec before the inscription of the His deflection because retrograde conduction from ventricle-to-His invariably exceeds the H-V interval. Preexcitation of the atrium when the His bundle is refractory may not always be possible by RV stimulation if the bypass tract is left sided [10].

AIM OF THE WORK

To compare the" (SA-VA interval) after resetting of the tachycardia" to"(RV pacing from apex and base)" in differentiating AVNRT from orthodroimc AVRT using concealed APs in the electrophysiology lab as regard their specificity and sensitivity. **These techniques are:**

1- Entrainment of the tachycardia from RV apex and from RV base with 10-40 ms faster than TCL and measuring SA, VA interval and SA-VA interval and calculating the difference between them in RV apex and base.

2- Delivering single or double ventricular extra stimuli ,measuring the SA interval after resetting of the tachycardia, measuring the VA during the tachycardia, and their subtraction, if the difference is >110, it's AVNRT, and if <110, it's AVRT.

ATRIOVENTRICULAR NODAL RE-ENTRANT TACHYCARDIA (AVNRT)

The AV-Node:

Anatomy: The first descriptions of the anatomy of the nodes and atrioventricular conduction system appeared nearly 100 years ago[11]. The AV-node is a subendocardial right atrial structure that measures approximately 6 x 4 x 1.5 mm. The AV-node is located within the triangle of koch (which is bordered by the tendon of Todaro anterosuperiorly, septal tricuspid annulus inferiorly and anterior margin of coronary sinus ostium postero-superiorly) and penetrates the right fibrous trigone (central fibrous body) [12].

Histology: *The AVN may be divided into three layers:*

1- ***Superficial or subendocardial:*** The atrial approaches from various directions merge gradually with the superficial part of the AV node. These fibers are loosely arranged with smaller node like cells oriented along the atrial cells, some intermingling with the atrial cells, fat, elastic tissue, collagen, and nerve fibers. A distinct increase in fat occurs with normal aging of the heart.

2- ***Intermediate or midzone:*** AV nodal cells are more or less compact; however, the orientation and arrangement of the cells vary considerably with fewer nerve cells.

3- ***Deep or innermost layer:*** AV nodal cells are tightly arranged and may be considered compact. At the light microscopic level, the nodal fibers vary from the periphery toward the central fibrous body. At the electron microscopic level, the innermost layer shows fewer myofibrils and mitochondria, which are randomly arranged. The cytoplasmic reticulum is poorly developed with no transverse system. The gap junctions are scarce, but the desmosomes are frequent[13].

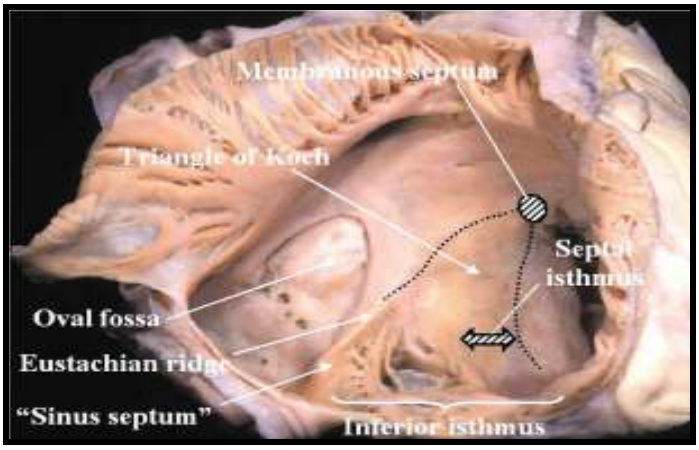

Figure (1): The heart has been positioned in appropriate orientation, and the right atrium opened through a window in the appendage. The septal surface of the atrium is located posteriorly, and the apex of the triangle of Koch (dotted lines) points upwards. The important atrial landmarks are shown[12].

Based on the morphology of the action potentials recorded by microelectrodes, the following three distinct regions of the AV node have been identified: 1) the atrionodal (AN), 2) true nodal or compact nodal (N), and 3) nodo-His (NH). N-response was characterized by a relatively slow rate

of rise during upstroke and small amplitude of the action potential. AN and NH action potentials showed an intermediate morphology between the N- region and the atrial muscle (AN) and the N-region and His bundle (NH), respectively[14].

Functional properties of the AV-node:

The AV-node produces a wide variety of conduction delays as well as beat-to-beat changes in delay. This rate induced variations in nodal conduction time are governed by three intrinsic nodal properties called recovery, facilitation and fatigue which were first described by Leuis and Master in 1925. The recovery property is related to the slow rate of recovery of excitability in the node which can be assessed by periodic premature stimulation at a slow basic rate[15]. However, the nodal conduction time decreases with the shortening of the last cycle before the premature cycle. This is called the "facilitatory" effect[16]. A rapid stimulation also initiates an "exhaustion" of the node, called "fatigue" which is associated with a progressive prolongation in the conduction time. The induction of fatigue can be elicited during stimulation for 5 minutes at a fast rate. Facilitation and fatigue usually occur together during stimulation at a fast rate[17].

Reentry:

Definition: Reentry is the reactivation of fibers for a second time or repeatedly by the same wave front[18].

Prerequisites for reentry:

There are three prerequisites for the development of reentry:

1. An available circuit with unidirectional block.
2. A difference in the refractory periods of the two pathways (limbs) in the circuit.
3. Conduction that is sufficiently slow somewhere in the circuit to allow the remainder of the circuit to recover its responsiveness by the time the impulse returns[18].

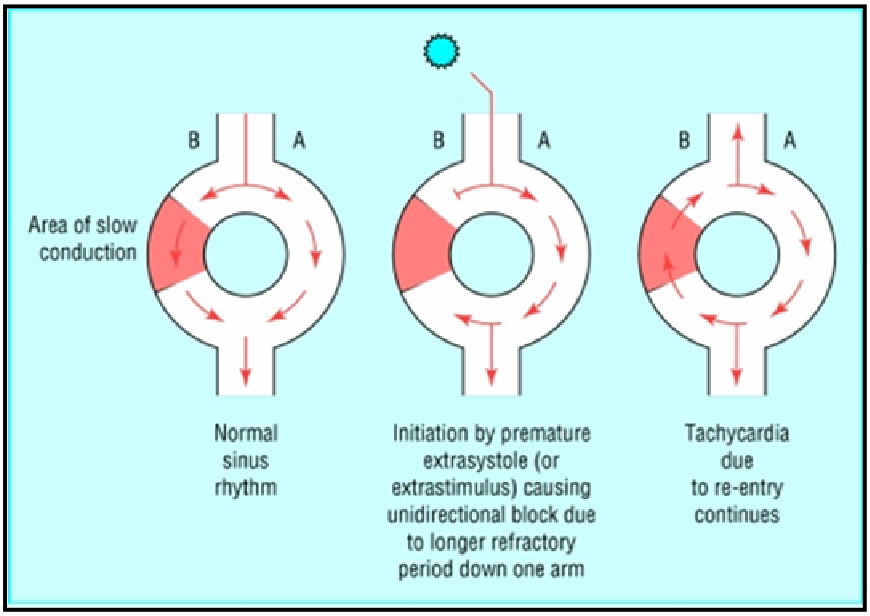

Figure (2): Mechanism of a re-entry circuit. An excitation wave is propagated at a normal rate down path A, but slowly down path B. An excitation wave from an extrasystole now encounters the slow pathway (B), which is still refractory, creating unidirectional block. There is now retrograde conduction from path A, which coincides with the end of the refractory period in path B. This gives rise to a persistent circus movement[19].

Atrioventricular nodal reentrant tachycardia (AVNRT):

Definition:

Atrioventricular nodal reentrant tachycardia (AVNRT) is a common form of paroxysmal supraventricular tachycardia, based on a substrate characterized by two functionally and anatomically distinct atrioventricular (AV) nodal pathways. The exact boundaries of the reentrant circuit in AVNRT have been subject of debate for many years, while some investigators supported the idea that the circuit was confined within the AV node, others suggested that atrial tissue and transitional cells formed the upper part of the reentrant circuit [20].

Epidemiology:

AVNRT is prevalent in 60% of patients presenting with paroxysmal SVT in the USA with a female predominance. Internationally, Frequency is similar to that in the United States[21].

AVNRT may occur in persons of any age. Although it is common in young adults, some patients do not present until their seventh or eighth decade or older[21]. The incidence of dual AV nodal pathways decreases with aging, suggesting that the AV node undergoes age-related electrophysiological changes. This finding could explain the uncommon onset of AV nodal re-entrant tachycardia in the elderly [22]. Over 50%

of the patients become symptomatic before the age of 30 years, the onset of this arrhythmia is uncommon in the elderly even though the greater incidence of ectopic beats with aging plays a facilitating role in giving rise to the tachycardia [23].

Although uncommon, atrioventricular nodal reentry tachycardia (AVNRT) may occur in infants. In pediatric patients, AVNRT typically presents beyond 5 years of age with a gradual increase in frequency with advancing age. Episodes presenting in infancy tend to have a lower recurrence risk compared with SVT presenting in childhood[24].

AVNRT is usually well tolerated; it often occurs in patients with no structural heart disease. In patients with coronary artery disease, AVNRT may cause angina or myocardial infarction. Prognosis for patients without heart disease is usually good [21].

Pathophysiology:

The substrate for AVNRT is the presence of dual AV nodal pathways and it may be functional rather than anatomical. The AV node is functionally divided into 2 longitudinal pathways that form the reentrant circuit. In the majority of patients, during AVNRT, antegrade conduction occurs to the ventricle over the slow (alpha) pathway and retrograde conduction occurs over the fast (beta) pathway. In approximately two thirds of patients, the tachycardia is

initiated when an appropriately timed atrial premature complex is blocked in the fast pathway (longer refractory period) and conducts in the slow pathway (shorter refractory period). While the impulse conducts to the ventricle in the slow pathway (antegrade conduction), the fast pathway recovers so that the impulse can conduct retrograde up the fast pathway to the atrium and the atrial end of the slow pathway (retrograde conduction) and in approximately one third of patients, AVNRT is induced by premature ventricular stimulation. This sets up the reentrant circuit and is called slow/fast AV nodal reentrant tachycardia.

In addition to the typical mechanism of AV nodal reentry described above, atypical AV nodal reentry can occur in the opposite direction, with antegrade conduction in the fast pathway and retrograde conduction in the slow pathway, the so-called fast /slow AV nodal reentry.

Less commonly, the reentrant circuit can be over 2 slow pathways, the so-called slow-slow AV node reentry[21].

Initiation of AVNRT:

For AVNRT initiation, there must be a critical delay in AV nodal conduction time[30].

During electrophysiologic study, patients with AVNRT were found to have distinct pattern of AV nodal conduction. This pattern can be demonstrated by atrial extra stimulation, when the atrial extrastimulus coupling interval (A1-A2) is shortened by 10 msec, the AV nodal conduction time is

prolonged (A2-H2) gradually till a certain decrement is reached, the A2H2 interval is increased markedly (50 msec). This is the so called AH jump, from the electrophysiological point of view. This AH jump shows that the conduction is no longer across the fast pathway and is now across the slow pathway[30].

AV nodal reentrant tachycardia is usually initated by atrial premature beat that is early enough so that it encounters the fast pathway during its refractoriness, thus the impulse is conducted to the ventricles in the slow pathway which is vulnerable and excitable. After passing through the slow pathway the impulse encounter the fast pathway from its ventricular end after it regains excitability and so the impulse pass retrogradely through the fast pathway reactivating both atria forming the atrial echo beat, if these sequences are repeated AVNRT becomes sustained**.** Autonomic nervous system has a great role for initiation and maintenance of tachycardia [30]**.**

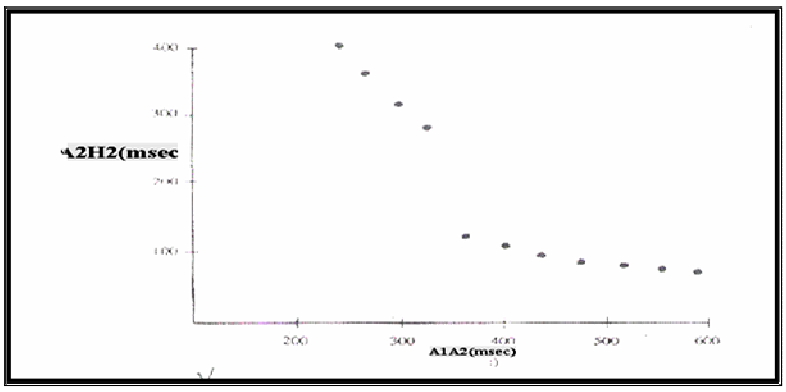

Figure (3): A typical AV conduction curve from a patient with AV nodal reentrant tachycardia. On the AV conduction curve, this jump from faster to slower conduction is manifested by sudden discontinuity in the curve [2].

Classification and types of AVNRT:

AVNRT classification depends on the direction of the depolarizing wave in the different components of the reentrant circuit, thus two forms are known of AVNRT.

(A) Typical form of AVNRT (Slow/fast AVNRT):

It is the commonest type of AVNRT, accounting for 90% of the cases. In this form, the fast pathway serves as the retrograde limb of the circuit, whereas the slow pathway is the anterograde limb (i.e. slow-fast AV-node re-entry)[26].

Electrocardiographic criteria:

The 12-lead ECG shows a narrow complex regular tachycardia and the P-wave is usually obscured as the atria are activated by the retrograde fast pathway simultaneously with the ventricles, however, sometimes, evidence of retrograde P-waves can be detected on the surface ECG as pseudo-S waves on inferior leads or pseudo-R' in V1[26].

The onset is abrupt with an atrial premature complex, which conducts with a prolonged PR interval, abrupt termination occurs with a retrograde P wave, sometimes followed by a brief period of asystole or bradycardia before the sinus node recovers from its tachycardia-induced suppression.

The cycle length may vary, especially at the beginning and the end of the tachycardia. This variation reflects the variable antegrade AV nodal conduction time. Vagal maneuvers may slow or terminate the tachycardia[29].

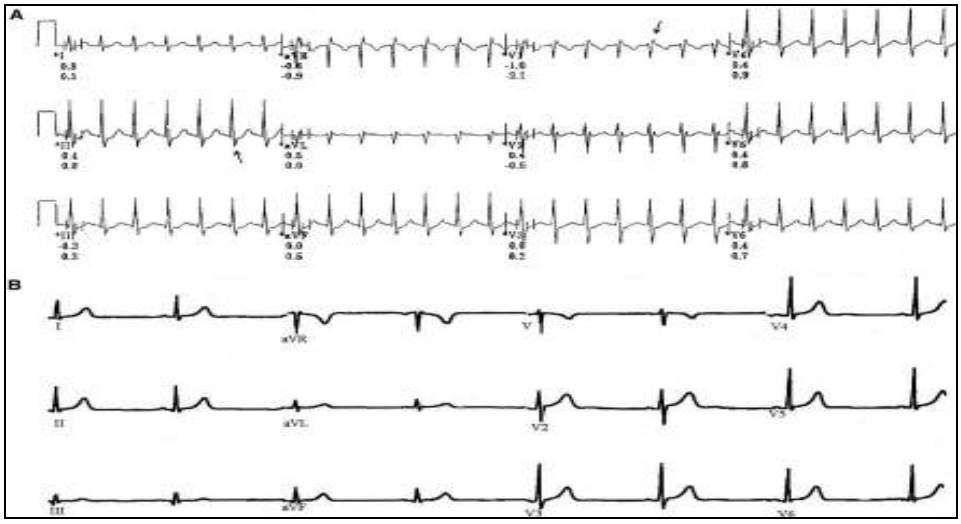

Figure (4): ECG pattern of typical AVNRT. Panel A: 12-Lead ECG shows a regular SVT recorded at an ECG paper speed of 25 mm/sec. Panel B: After conversion to sinus rhythm, the 12-lead ECG shows sinus rhythm with narrow QRS complexes. In comparison with Panel A: Note the pseudo r' in V1 (arrow) and accentuated S waves in II, III, aVF (arrow). These findings are pathognomonic for AVNRT. AVNRT indicates atrioventricular nodal reciprocating tachycardia; mm/sec, millimeters per second; QRS, ventricular activation on ECG; SVT, supraventricular tachycardia; VF, ventricular fibrillation [26].

Electrophysiological criteria:

- Initiation of tachycardia by AV-delay: the initiation of slow/fast AVNRT is dependent on a critical delay in AV-nodal conduction time with sudden increment in AH interval (AH-jump) suggesting antegrade conduction over the slow pathway [26].