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Typical AVNRT—An Update on Mechanisms and Therapy

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Abstract. **Typical atrioventricular reentrant tachycardia (AVNRT) is the most common paroxysmal supraventricular tachycardia among adults, and accounts for considerable morbidity. The concept of dual pathway physiology remains useful, although this physiology likely results from the functional properties of anisotropic tissue within the triangle of Koch, rather than anatomically distinct tracts of conduction. Also, there remains debate regarding whether the critical reentrant circuit path requires participation of the atrium. In our opinion, current evidence favors functional anisotropic reentry limited to the subatrial tissues as the arrhythmia mechanism. Reasons for this are reviewed. Fortunately, typical AVNRT is readily amenable to definitive therapy by catheter-based radiofrequency energy delivery at the so-called slow pathway region located at the posterior Triangle of Koch. Anterior or left-sided approaches are very rarely indicated. Results from multiple series have shown this strategy to be both safe and effective, therefore ablation therapy should now be considered as the definitive therapy of choice for the majority of patients.**

Key Words. **atrioventricular node, atrioventricular nodal reentry tachycardia, dual atrioventricular nodal physiology, radiofrequency catheter ablation**

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common paroxysmal supraventricular tachycardia, diagnosed in over 50% of adult cases [1,2]. Women are affected twice as frequently as men [3–9], and both symptoms and inducibility can follow a menstrual pattern in some women [10]. Most patients present between late adolescence and age 40, although the arrhythmia can occur at any age [1,3–9], including, more rarely, in children [11–13]. Catheter-based radiofrequency ablation is now well established as the definitive treatment of choice for most symptomatic patients, with excellent success rates (>90–97% cure or significant palliation) and a low incidence of serious complications [7,8]. In the common or "typical" form of AVNRT, which accounts for $\sim$ 90% of cases, the antegrade limb of the reentrant circuit usually conducts slowly whereas the retrograde limb is fast. However, controversy remains as to whether the "upper common pathway" resides completely within the subatrial nodal structures (which include transitional cells within the triangle of Koch (TOK)), with the muscular atrium a passive bystander [14], or whether the adjacent or overlying atrial tissue is an obligate part of the reentrant circuit [3]. Furthermore, although AVNRT is classically depicted as following an anatomically-defined circuit along slow (SP) and fast pathways (FP), there is mounting evidence that the circuit is functionally-based, rather than anatomic. Discontinuous, non-uniform anisotropy within the TOK is a substrate that can support functional, anisotropic reentry which can exhibit dual pathway physiological characteristics. This article reviews recent clinical and experimental findings which have added to our understanding of AVNRT, and discusses implications and techniques for contemporary catheter-based therapy.

Electrophysiology

The circuit in AVNRT is traditionally depicted as being anatomically constrained to a longitudinally dissociated slow pathway (SP) and fast pathway (FP) conducting around a central obstacle, with proximal and distal connections referred to as the upper and lower final common pathways. Dual pathways, anatomic or functional are consistent with several observations. Delivery of increasingly premature atrial complexes results in a discontinuous "jump" (>50 ms over a 10 ms increase in prematurity) in the A-V conduction curve and sudden prolongation of the PR interval, consistent with block in a refractory FP, and conduction over a SP with a shorter refractory period. If sufficient time elapses for the FP to recover retrograde excitability, then the SP impulse may conduct retrogradely over the FP, and produce an atrial echo beat if conducted to the atria. Reentry back into a recovered SP initiates AVNRT. Rarely, a single atrial complex results in two ventricular responses, or a single ventricular complex yields a pair atrial responses, so-called antegrade and retrograde "double-fire" phenomena [9]. Finally, it is possible to preempt atrial echoes and entrain AVNRT by ventricular pacing during SP conduction at critical cycle lengths. Together, these findings confirm the validity of the dual pathway physiology concept in AVNRT.

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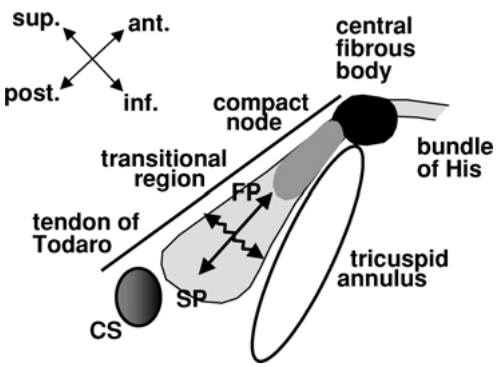

Fig. 1. *Schematic of the triangle of Koch and anisotropic conduction. Koch's triangle contains the region bounded by the tendon of Todaro, the tricuspid annulus, and the coronary sinus (CS) ostium. Transitional fibres are oriented with the their long axis parallel to annulus within the triangle. Impulses travelling along the longitudinal axis of the transitional fibers conduct rapidly (straight line), whereas impulses perpendicular to the fibers conduct slowly (zig-zag line). FP and SP denote the sites typically referred to as fast and slow pathway regions. Details in text.*

It should be stressed, however, that in clinical usage, the terms slow and fast pathway refer to neither areas with different conduction velocities nor to specific electrical pathways in the sense of accessory pathways (bypass tracts). Rather, slow and fast pathways have come to refer to specific *anatomic sites* where the earliest retrograde AV nodal activations are recorded within the Triangle of Koch (TOK) during atypical ("fast orthograde/slow antegrade") or typical ("slow orthograde/fast antegrade") AVNRT, respectively. The TOK consists of the region bounded by the tricuspid annulus inferiorly, the tendon of Todaro superiorly, the os of the coronary sinus posteriorly (base of the triangle) and the compact AV node and central fibrous body anteriorly, at the apex (Figure 1). Because the earliest site of retrograde atrial activation shifts from the apex of the TOK in typical AVNRT to the coronary sinus os during the atypical tachycardia, separate posterior/inferior SP and anterior/superior FP inputs to the AV node have been postulated [5]. Furthermore, low frequency, multiphasic potentials have been recorded in the posterior TOK by a His catheter and thought to represent specific SP potentials [3,4]. Radiofrequency ablation of sites with such potentials, located several millimeters away from the compact node resulted in elimination of AVNRT without PR interval prolongation or AV block. Though initially interpreted as supporting the presence of discrete pathways, these findings are not unique to that mechanism, and also consistent with functional reentry as explained below.

Not all patients with AVNRT exhibit clear-cut dual pathways, however, some have smooth, continuous AV conduction curves while in others, multiple discontinuities are found. Though the latter could occur from multiple pathways, functionally-based pathways that do not depend on anatomical tracts best explain this observed variability.

Histologically, the AV node is a complex and variable structure which exhibits variable posterior atrial extensions engaged by intercalating transitional tissue [15,16]. While it is tempting to equate the anatomical nodal extensions with physiologic dual pathways, this relationship has not been conclusively demonstrated [17]. Sodium channels and gap junctions are concentrated along the periphery, but are scarce central midnodal region [18]. Since transitional fibers separated by connective tissue travel parallel to the tricuspid valve from the inferoposterior base of the TOK near the CS towards the compact node, dual pathway physiology could also result from discontinuous anisotropic conduction along (FP) and across (SP) these fibers (Figure 1). Spach and associates [19] showed experimentally that non-uniform anisotropy exists in the TOK, and that dual pathway responses may be explained on this basis. Others have also characterized the TOK as anisotropic, but found the directional differences in conduction time insufficient to account for the full discontinuity in A-H intervals [20] .

The anisotropic model helps explain several important observations. Zig-zag propagation transverse to fiber orientation could give rise to low-amplitude, fractionated or multiphasic electrograms in the TOK attributed to conduction in a slow pathway [3,4], as it does in other models [21–23]. The majority of AVNRT patients have multiple, heterogeneous sites of early atrial activation during the arrhythmia rather than highly focal breakthrough sites, also consistent with such a mechanism [24] (Figure 2). With distinct anatomical tracts, junctional tachycardia arising from the compact node following SP ablation [25] would show the identical patterns of atrial activation (both qualitatively and quantitatively) as during AVNRT, yet this is often not the case [26].

If anisotropy and cell-coupling are relevant to the genesis of AVNRT, then its epidemiology and electrophysiologic characteristics should change with age, as fatty infiltration and microfibrosis progresses. Morphometric studies by Waki et al. [16] demonstrated increased fibrofatty infiltration, widening of the transitional cell zone along with relative elongation of the compact node and right-sided extension with advancing age between infancy and 20 years. Among children, AVNRT accounts for $<5\%$ of SVTs among infants and becomes increasingly common with age [11]. AVNRT cycle lengths appear to increase with age [12,27] and one study, this was attributable to prolonged retrograde HA conduction time at the anterior TOK [27] .

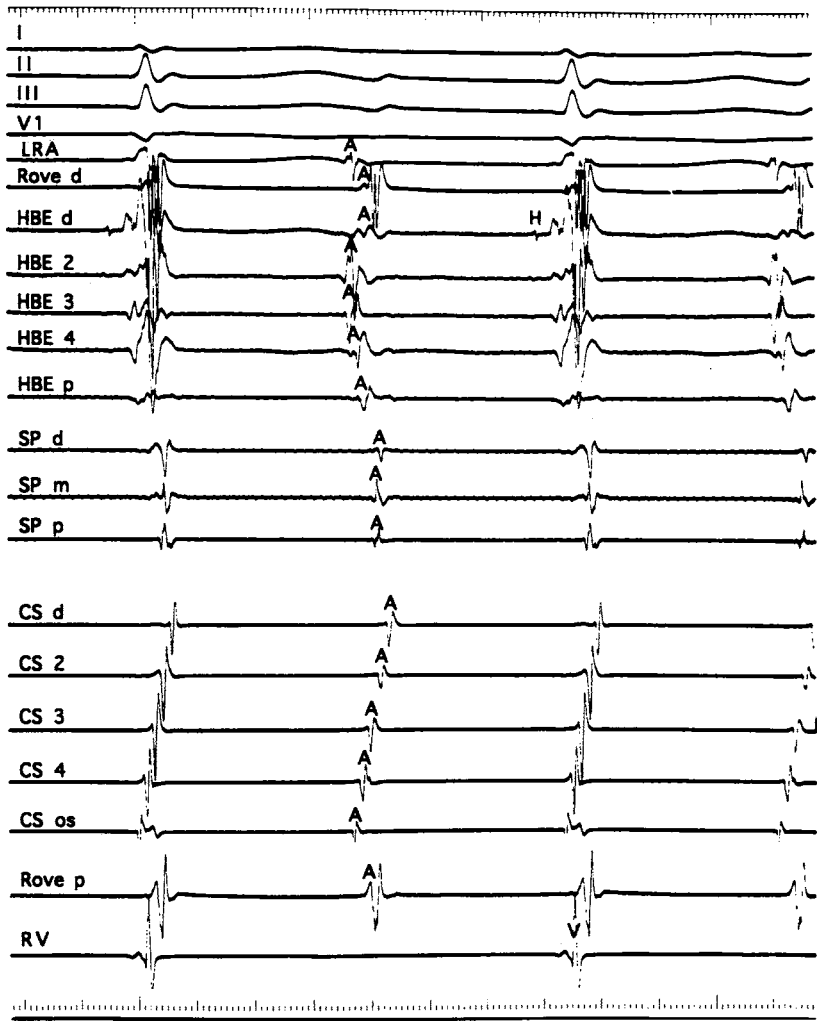

Fig. 2. *Retrograde atrial activation pattern during AVNRT. From top to bottom: leads I,II,III and V1 of surface electrocardiogram; low right atrium (LRA) recording, distal roving catheter (Rove d) recording, 5 His bundle electrograms (HBE) from distal (TOK apex) to proximal (TOK base), slow pathway (SP) region recordings (distal, mid, and proximal), 5 coronary sinus (CS) recording from distal (d) to proximal (os), proximal recording from the roving catheter (Rove p) and a right ventricular (RV) apex recording. The earliest sites of activation are both broad, recorded near-simultaneously at more than two adjacent electrode (e.g. HBE 2-4) and multiple, with early breakthrough sites recorded at more than one separate location (e.g., LRA, HBE, CS os).*

Finally, several observations are difficult to reconcile with a significant part of the AVNRT circuit being supranodal. Persistent AVNRT in the absence of 1:1 atrial activation strongly argues that atrial participation is unnecessary, or if so, then only such a small part that would be both electrocardiographically silent and demonstrate block to the remainder of the atrium. AVNRT persisting in the face of atrial fibrillation [28] is stronger evidence still, as one would have need to invoke a region of atrium somehow protected from the bombardment of high frequency fibrillation wavefronts to support atrial participation. Figure 3 shows intracardiac recordings from two patients with AVNRT persisting despite lack of one-to-one nodo-atrial conduction.

Recently, elegant studies applying optical mapping techniques to the rabbit AV node showed AV conduction to be clearly three-dimensional, not planar or bi-cable like [29–31]. One study confirmed apparent retrograde atrial breakthroughs for FP conduction anteriorly near the apex and SP conduction posteriorly at the base of the TOK. Three-dimensional reconstruction analysis of surface fluorescent signals recorded during apparent AV nodal reentry was consistent with the participation of a thin superficial layer of atrial or rapidly conducing transitional cells in the circuit within the TOK [32]. Of note, sustained AVNRT is difficult to induce and is rarely sustained in this rabbit model [29,32], perhaps because of such rapid conduction in the superficial limb of the circuit.

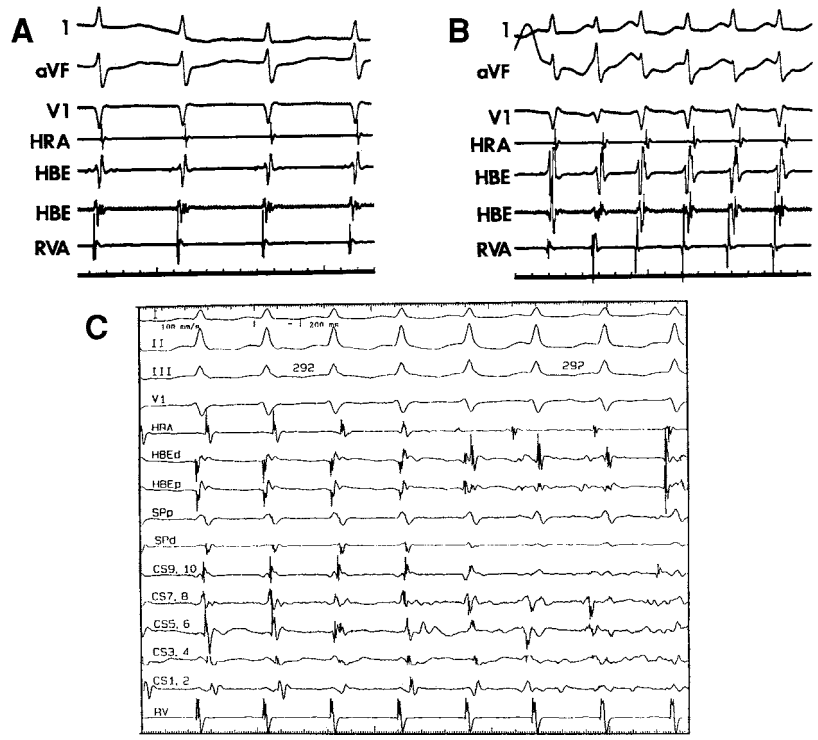

Fig. 3. *Persistence of AVNRT despite loss of 1 nodoatrial coupling. Panels A and B show 2 sets of recordings from the same patient with AVNRT. Tracings shown from top to bottom are leads I, aVF and V1 from the surface electrocardiogram, distal and proximal His bundle elctrograms (HBE), and right ventricular apex (RVA) recording. (A) Atrial, His and ventricular electrograms are near synchronous, supporting a junctional source with cycle length 510 ms. (B) A slight perturbation of the His bundle catheter moments later reveals results in a similar tachycardia but with a cycle length slightly greater than half the first (285 ms). Interpretation was AVNRT with 2:1 nodo-atrial and 2:1 nodo-ventricular block in (A), with slowing caused by the catheter movement allowing 1:1 antegrade and retrograde conduction in (B). (C) Co-existence of AVNRT during atrial fibrillation in a different patient. Shown from top to bottom are recordings from surface electrocardiogram leads I, II, III and V1, high right atrium (HRA), distal and proximal HBE and slow pathway (SP) regions, 5 coronary sinus bipoles and the right ventricle (RV). Electrograms of the first 2 QRS complexes show typical AVNRT with cycle length 292 ms. Subsequent HBE and CS recordings then changed to atrial fibrillation. However, both the ventricular and SP cycle length remained 292 ms and regular, suggesting that ventricular activation remained driven by continuing AVNRT. (Reproduced with permission [28].)*

Earlier studies in the rabbit AV node, mapped the site of alternation in nodal conduction with premature stimuli to the node proper, not to its atrionodal inputs [33]. A high-density conventional electrode study in the canine heart found that reentrant ventricular echo beats did not require perinodal or endocardial tissues [34].

These and other lines of clinical evidence for and against anatomical versus functional dual AV nodal reentry are summarized in Table 1. Note that each observation predicted by (or consistent with) anatomically separate atrionodal pathways can also be explained by discontinuous anisotropic conduction within the node, although the reverse is not true. Table 2 lists clinical observations that support the notion that the atria are not a necessary part of the reentrant circuit in AVNRT. It should be said that wholly intranodal anatomic pathways, and functional anisotropic pathways involving the atria are also theoretical possibilities. Figure 4 depicts four potential reentrant schemes predicted by combining anatomic or functional reentry with or without an atrial component to the circuit path. While the issue remains unresolved, we believe that the weight of current evidence favors a functional, anisotropic basis for reentry contained wholly within the subatrial tissues (e.g., Figure 4(B)).

Catheter Ablation Therapy

Catheter ablation therapy should be offered to all patients with symptomatic AVNRT barring the standard contraindications. Fortunately, given the usual manifestation of the arrhythmia in early to middle adulthood, most patients have little in the way of comorbidities, including structural heart disease. Ablative therapy is by far more successful in controlling symptoms and improving quality of life than current

Table 1. *Evidence supporting functional versus anatomic dual pathways*

Observation	Functional dual pathways	Anatomic dual pathways
Post-SP ablation accelerated junctional rhythms show different atrial retrograde activation patterns from typical AVNRT	Consistent	Inconsistent
Broad and or multiple retrograde atrial activations	Consistent	Inconsistent
AVNRT incidence and characteristics change with advancing age (i.e., microfibrosis)	Consistent	Less consistent
Posterior TOK ablation can eliminate "FP" conduction in some patients and produce heart block	Consistent	Consistent only if atypical FP or compact node location
FP ablation can cure AVNRT without causing PR interval prolongation or heart block	Consistent	Consistent only if FP ERP > SP ERP
Low frequency, double or fractionated "SP" potentials	Consistent	Consistent
Singly discontinuous AV conduction curve	Consistent	Consistent
Continuous or multiply discontinuous AV conduction curve	Consistent	Only if multiple (>2) distinct pathways present

SP = slow pathway, AVNRT = atrioventricular nodal reentrant tachycardia, TOK = triangle of Koch, FP = fast pathway, AV = atrioventricular.

medical therapy. Moreover, it is cost-effective in patients with frequent episodes refractory to drug therapy [35,36]. One study concluded that even employed as initial therapy, ablation was cost-effective in the long run (i.e.,within ~10 years) compared to medical therapy given the cumulative drug costs saved [37].

Fluoroscopically-guided radiofrequency catheters remain the work-horse technology for AV node modification. Nonfluorscopic electroanatomic mapping [38] and newer energy sources such as lasers [39] and cryothermy [40] have been used with success in smalls numbers of AVNRT patients, however, any clear advantages to these techniques for this particular arrhythmia have yet to be demonstrated.

Theoretically, AVNRT can be cured by ablation of any requisite portion of the reentrant circuit. Since "modification" the potential circuit often suffices to render the arrhythmia non-inducible, it is possible that ablation energy can adequately uncouple key superficial transitional cells from the compact node, although this remains conjecture. We and others typically target the SP region for ablation/modification by positioning the catheter at the posterior and middle thirds of the TOK, anterior to the coronary sinus os just superior to the tricuspid annulus insertion into the inferior interatrial septum. Some operators are guided by the presence of "SP potentials" which

Table 2. *Evidence that atrial participation is unnecessary in AVNRT*

Observation
Initiation of AVNRT by an APD or atrial pacing without an atrial echo beat
AVNRT persistence despite lack of 1:1 VA or nodo-atrial relationship (i.e, during 2:1 or variable VA block, VA dissociation, atrial flutter, atrial fibrillation, adenosine)
Atrial pacing caputure without affecting AVNRT
Resetting of tachycardia by ventricular stimulation without atrial activation
Heterogenous retrograde earliest atrial activation pattern during tachycardia incompatible with atrial participation
Atrial pacing at AVNRT cycle length yields longer A-H interval than during AVNRT proper
Spontaneoue changes in HA or VA intervals without AVNRT cycle length changes
Atrial pacing produces AV nodal Wenchebach phenomenon at a cycle length greater than the AVNRT cycle lenget

AVNRT = atrioventricular nodal reentrant tachycardia, APD = atrial premature depolarization, VA = ventriculoatrial, HA = His-atrial.

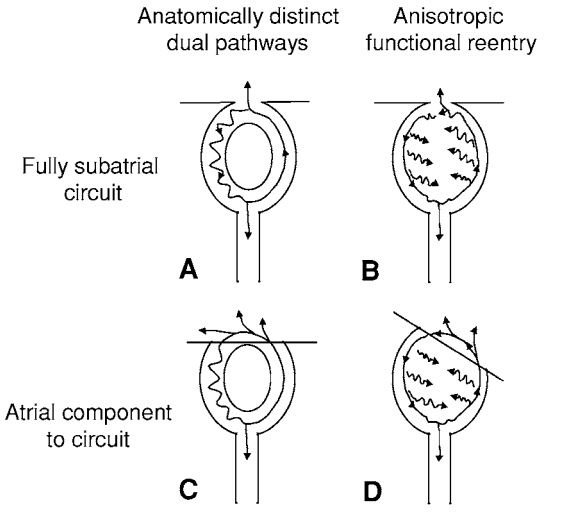

Fig. 4. *Schematic representation of AVNRT occurring via anatomic or functional dual pathways including or excluding the atrium within the circuit. (A) Reentry within anatomically defined pathways around a central obstacle, contained within the nodal tissues. (B) Anisotropic functionally-based reentry within the nodal tissues. (C) Anatomically based reentry with the atrium a necessary part of the circuit. (D) Functional longitudinal block within the node with the atrium required to complete the circuit.*

can be recorded there, though their elimination has not been proven necessary to cure the patient. Similar potentials can be recorded in all patients, whether or not they have supraventricular arrhythmias; experimental data suggest they likely represent a composite signal from the various superficial and deeper tissues in that area [41]. Since the incidence of heart block increases the further anterior the ablation within the TOK, we systematically begin posteriorly, near the coronary sinus os, and proceed anteriorly until either the arrhythmia becomes non-inducible by programmed stimulation with or without beta-adrenergic agonists, or heart block threatens, often heralded by a transient junctional tachycardia [42]. The SP is said to be ablated if dual pathway physiology is entirely abolished and modified if the SP conduction time is lengthened or echo beats are still inducible, but without sustained arrhythmia. With this posterior approach or slow-pathway ablation or modification, cure rates from several different clinical series have been in excess of 90%, and as high as 97% [3–5,7,8,43,44]. The risk of permanent heart block requiring permanent pacemaker placement has been less than 1% [7,8,44]. The risk of this complication appears greater in patients who are older [45], have a prolonged PR interval at baseline [46], show near-simultaneous activation of the CS os, SP and apex of TOK [40] or receive complete SP ablation rather than modification [46].

Anterior approach or FP ablation is reserved for highly symptomatic patients in whom SP ablation is unsuccessful, or who possess a prolonged P-R interval "consistent with" dependence on the SP for antegrade AV conduction at baseline. Even then, SP ablation can still be curative, further supporting the functional nature of AV nodal propagation.

Although early atrial activation may be recorded in the coronary sinus in nearly 50% of patients, this "breakthrough" appears to be independent from His bundle activation, tachycardia cycle length, or other activations within the TOK (which are themselves correlated), suggesting that while left posterior atrionodal connections exist, they are not part of the reentrant circuit per se, but rather are activated as a bystander [47]. We have not required left-sided ablations, though case reports exist of AVNRT not responsive to right-side therapy being successfully ablated from the left [48,49]. It is likely that this represents participation of left sided extensions of the node. Because of the attendant risks of bleeding and stroke, left sided ablation should be reserved for only the most recalcitrant cases in highly symptomatic individuals, in whom ablation inside the os of the coronary sinus has also failed. Care must be taken to prove that a slowly conducting left posteroseptal bypass tract is not responsible for the arrhythmia.

Conclusion

AVNRT is a common arrhythmia responsible for considerable morbidity. Although its underlying mechanism remains incompletely understood, substantial evidence favors functional, anisotropic reentry occurring within the subatrial nodal structures. A critical part of the functional circuit seems to involve or be influenced by the posterior to midportion of the TOK, since radiofrequency energy delivered there is highly successful in treating the arrhythmias, even when dual pathway physiology is not completely abolished. Catheter-based ablation therapy is both safe and effective and should be considered the definitive therapy of choice for most symptomatic patients.

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