Enhanced Atrioventricular Conduction in Patients Without Preexcitation Syndrome: Relation to Heart Rate in Paroxysmal Reciprocating Tachycardia

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SUMMARY We studied the electrophysiologic characteristics of atrioventricular (AV) nodal conduction in patients with reciprocating tachycardia (RT) without ventricular preexcitation, and the relation of these characteristics to RT cycle length (CL). Thirty-five symptomatic patients who had a normal PR interval (0.13–0.20 second) during sinus rhythm underwent detailed intracardiac electrophysiologic study during which ventricular preexcitation was excluded, and the RT mechanism was determined. RT was due to reentry using an accessory AV pathway capable of conduction only in the retrograde direction (concealed AP) in 13 patients (37%) and to reentry within the AV node in 22 (63%). Dynamic properties of AV conduction (assessed by degree of AH prolongation during progressive increase in atrial pacing rate) were normally distributed ($p < 0.005$); 12 patients (34%) fulfilled the criteria for enhanced AV conduction (EAVC).

The patients with EAVC had a shorter RT CL than did patients without EAVC (294 ± 43.3 msec vs 360 ± 68.1 msec, $p < 0.01$). However, CL differences were primarily due to the influence of EAVC in the subgroup of patients with RT using a concealed AP (EAVC CL, 274 ± 35.1 msec; without EAVC, 326 ± 15.8 msec, $p < 0.005$). The RT CL in patients with reentry within the AV node was not measurably influenced by concomitant EAVC (EAVC, 314 ± 43.8 msec; without EAVC, 374 ± 76.8 msec) (NS).

This study suggests that despite the presence of a normal PR interval during sinus rhythm, dynamic AV conduction responses can vary widely in patients with RT. In patients with RT using a concealed AP, but not in those with reentry within the AV node, coexisting diminished physiologic AV conduction slowing may be associated with more rapid tachycardia rates.

THE VENTRICULAR RATE is a principal factor in the hemodynamic effects and severity of symptoms in patients with paroxysmal supraventricular tachyarrhythmias. Improved understanding of the determinants of heart rate during supraventricular tachycardias may lead to more effective application of antiarrhythmic therapy.

The relation between the electrophysiologic properties of atrioventricular (AV) conduction and the tachycardia rate in patients with certain supraventricular tachyarrhythmias is controversial. Gallagher et al., studying patients with Wolff-Parkinson-White (WPW) syndrome, and Benditt et al., studying patients with Lown-Ganong-Levine (LGL) syndrome, reported that tachycardia rates were faster in patients who had diminished conduction delay within the AV node, which was termed enhanced AV nodal conduction (EAVC). Bauerfeind et al., evaluating patients with reciprocating tachycardia (RT) due to reentry within the AV node, could not substantiate a relation between electrocardiographic and electrophysiologic descriptors of AV nodal function and the associated tachycardia rate.

To further evaluate these apparently divergent views, we examined the ventricular response during supraventricular tachyarrhythmias in patients without ventricular preexcitation.

Methods

Patient Selection

The patients were selected according to the following criteria: (1) history of documented symptomatic paroxysmal regular narrow QRS tachycardia; (2) normal PR interval (0.13–0.20 second) documented by
12-lead ECG; (3) electrophysiologic documentation of reciprocating supraventricular tachycardia due to reentry within the AV node or to reentry using a concealed accessory AV pathway; 4-11 (4) absence of ventricular preexcitation (i.e., accessory AV, nodoventricular, or fasciculoventricular pathways with antegrade conduction properties) documented by detailed electrophysiologic study and by absence of a delta wave on the 12-lead ECG; (5) withdrawal of all cardioactive medications for at least three drug half-lives before intracardiac electrophysiologic study.

Thirty-five patients, 24 women and 11 men, mean age 40 ± 15.2 years, were included in this study. Five patients had known underlying organic heart disease (three coronary artery disease, one cardiomyopathy and one cardiomyopathy with mitral valve prolapse). Apart from palpitation, these five patients were asymptomatic at time of study and were not receiving cardioactive glycosides. Of the remaining 30 patients, one had mitral valve prolapse but was otherwise normal. All patients gave informed consent for these studies.

Clinical Electrophysiologic Procedure

The electrophysiologic techniques used in this laboratory have been described in detail. 7, 12 Two patients with coronary artery disease did not undergo pacing at the minimal cycle length with 1:1 AV conduction, and their data are not included in figures 1 and 2. In four additional patients, the AH interval could not be clearly measured at three or more paced cycle lengths. Data from these four patients are not included in figure 2.

In 18 patients, ventricular responses during atrial fibrillation were recorded and both the shortest RR interval and average RR interval were measured by examining rhythm strips (paper speed 50 mm/sec) lasting 1-3 minutes. 6, 7

Statistical Analysis

Data are presented as mean ± SD. The equality of mean values of refractory periods, cycle lengths and RR intervals was determined by t test. The t statistic and associated degrees of freedom were calculated using a method for comparing independent groups of unequal variances and sample sizes. 14 The slope of pacing frequency–AH interval curves was determined by computer-based linear regression analysis using 95% confidence limits, and normality was assessed by the chi-square goodness of fit test. 14

Definitions

Enhanced AV conduction (EAVC). 6, 7 was diagnosed if the following criteria were met: AH interval in sinus rhythm ≤ 60 msec; 1:1 AV conduction during right atrial pacing at cycle lengths ≤ 300 msec (200 beats/min); between sinus cycle length and a cycle length of 300 msec, AH prolongation ≤ 100 msec.

RT due to reentry using a concealed accessory AV pathway was diagnosed if ventricular preexcitation was excluded and at least three of the following criteria were satisfied: (1) Induction of premature ventricular depolarizations at progressively shorter coupling intervals during tachycardia resulted in preexcitation of the atria with an activation sequence identical to that of the tachycardia when the His bundle was refractory. 10 (2) Ventriculoatrial atrial electrogram interval > 61 msec during narrow QRS RT. 16 (3) Increased ventriculoatrial interval after onset of functional bundle branch block during RT. 17 (4) Eccentric atrial activation sequence during tachycardia. 17, 18 These criteria do not absolutely exclude the remote possibility that reentry within the AV node might occur in a patient with a concomitant non-participating concealed septal accessory AV pathway.

RT due to reentry within the AV node was diagnosed if the retrograde atrial activation sequence was initiated earliest in the low septal right atrium, and was indistinguishable from the normal retrograde conduction sequence (thereby excluding reentry within the region of the sinus node), and if participation of an accessory AV pathway in the tachycardia (by criteria noted above) was excluded or if the ventriculoatrial interval was ≤ 61 msec. 16

RT due to reentry solely within the base of the atria cannot be distinguished by these criteria from tachycardia due to reentry within the AV node. We did not attempt to exclude an atrial contribution to a reentry circuit by routine use of the atrial extrastimulus technique.

Results

Patient Subgroups

Thirty-five patients with paroxysmal RT were included in this study.* Each patient was assigned to one of four subgroups based on the mechanism of RT, and electrophysiologic characteristics of AV nodal conduction. Reentry within the AV node was diagnosed in 22 patients (63%); 16 of these patients (nine women and seven men, mean age 42 ± 16.1 years) had normal AV conduction and six (five women and one man, mean age 49 ± 7.1 years) had EAVC. In 13 patients (37%), RT was due to reentry using a concealed accessory AV pathway; seven of these patients (five women and two men, mean age 38 ± 11.3 years) had normal AV nodal conduction and six (five women and one man) had EAVC.

There was no significant age difference between patients with (39 ± 16.8 years) and without (41 ± 15.2 years) EAVC. The five patients with concomitant cardiac disease and the patient with mitral valve prolapse did not have EAVC. The mean PR interval in patients with EAVC (0.14 ± 0.01 second) was shorter than in patients without EAVC (0.16 ± 0.02 second, p < 0.025).

AV Nodal Conduction Properties

Figure 1 illustrates the effect of progressive reduction of atrial pacing cycle length on AH interval dura-

*Detailed clinical electrophysiologic findings in each patient are available upon request.
tion in 33 of 35 patients. In all but four of 33 patients, the AH interval was measurable to the shortest atrial pacing cycle length with 1:1 AV conduction. Although the AH interval response to incremental atrial pacing encompassed a wide range (fig. 1), the distribution of AV nodal conduction properties appeared to be unimodal. To further assess the nature of this distribution, AH intervals at each paced rate were plotted against the pacing frequency for each patient, and the slope of the relation was evaluated by computer-based linear regression analysis. A minimum of three AH interval–pacing frequency data points was required for this analysis, thereby excluding six of 35 patients. The resulting distribution of slopes (msec/Hz) of each AH interval–pacing frequency curve (fig. 2) was analyzed by chi-square goodness-of-fit test and was consistent with a normal distribution of AV conduction characteristics in this patient population.

**AV Nodal Refractory Periods**

Refractory period data were obtained during extrastimulus testing at a fixed atrial pacing cycle length (500 msec, 120 beats/min), permitting comparison of findings in different patients. AV effective and functional refractory periods (AVERP and AVFRP) in patients with EAVC (244 ± 23.4 msec and 324 ± 41.6 msec, respectively) were shorter (p < 0.05) than in patients without EAVC (293 ± 43.1 msec and 396 ± 65.4 msec, respectively). Although there were insufficient data for statistical analysis, the relationship between EAVC and shorter refractory periods appeared to be independent of the type of RT. In the EAVC groups, the mean AVERP was 255 ± 14.7 msec in patients with RT due to reentry within AV node and 233 ± 27.5 msec in those with RT due to a concealed accessory AV pathway. In patients without EAVC, these values were 293 ± 45.9 msec and 290 ± 35.5 msec, respectively. AVFRP behaved in a similar fashion, but was less clearcut (EAVC: AV nodal reentry 293 ± 50.0 msec, accessory pathway reentry 348 ± 10.4 msec; without EAVC: AV nodal reentry 404 ± 68.1 msec, accessory pathway reentry 352 ± 21 msec). Atrial effective and functional refractory periods did not differ in the various patient subgroups.

**Cycle Length During RT**

In figure 3, RT cycle lengths are compared in patients with and without EAVC for all patients, for patients with RT due to reentry using a concealed accessory AV pathway and for patients with RT due to reentry within the AV node. Patients with EAVC had shorter cycle lengths during RT (i.e., faster heart rates) than did patients without EAVC (294 ± 43.3 msec vs 360 ± 68.1 msec, p < 0.01). However, this finding was largely due to differences in cycle length in patients with RT using a concealed accessory AV pathway (EAVC, 274 ± 35.1 msec; without EAVC, 326 ± 15.7 msec, p < 0.005).

In contrast to findings in patients with concealed accessory AV pathways, there was no statistically significant difference in tachycardia rates in patients with or without EAVC when the RT mechanism was due to reentry within the AV node (fig. 3).

**Mean and Shortest RR Interval in Atrial Fibrillation**

The ventricular response during pacing-induced atrial fibrillation was assessed in 18 of 35 patients (51%) — five with and 13 without EAVC (fig. 4). The mean RR interval (363 ± 32.5 msec vs 457 ± 52.1 msec, p < 0.005) and the shortest RR interval (252 ± 35.6 msec vs 374 ± 65.4 msec, p < 0.001) were shorter in patients with EAVC than in patients without
EAVC. These findings are consistent with the differences in AVERP and AVFRP and tend to support the concept that diminished capacity to induce physiologic conduction delay within the AV junction may be associated with more rapid heart rates during supraventricular tachyarrhythmias.

**Discussion**

In the present study, we examined the relation between AV nodal conduction properties in the antegrade (atrioventricular) direction and cycle length during RT in patients without ventricular preexcitation. Two principal observations were made. First, dynamic AV nodal conduction properties, as assessed by the degree of AH interval prolongation during incremental atrial pacing, encompassed a broad (fig. 1) but normally distributed (fig. 2) spectrum of responses. Consequently, patients exhibiting AV conduction properties characterized by the term EAVC appeared to constitute an arbitrarily defined subset, not a distinct electrophysiologic entity. Second, when RT cycle length was examined in the context of AV nodal conduction properties, AV nodal conduction characteristics correlated with tachycardia rate in patients with reentry using concealed accessory AV pathways, but not in patients with RT due to reentry within the AV node. This observation appears to reconcile apparently divergent findings in patients with paroxysmal RT.

**AV Conduction Characteristics and Tachycardia Rate**

AV conduction characteristics were examined primarily in terms of the effect of progressive increases of atrial pacing rate on the duration of the AH interval. Clearly, a wide spectrum of AV node conduction responsiveness was present (fig. 1). Despite the presence of a normal PR interval during sinus rhythm, many patients had minimal AH interval prolongation until relatively rapid atrial pacing rates (pacing cycle lengths < 350 msec, heart rates > 170 beats/min) were achieved. In fact, comparison of the AH interval—pacing cycle length relationships in these patients with previously published curves in patients with short PR intervals alone49 or with LGL syndrome1 reveals considerable overlap. Consequently, electrophysiologic characteristics of AV conduction in man might constitute a spectrum encompassing findings in patients with LGL syndrome, patients with EAVC, and patients with apparently normal capacity for initiating physiologic AV conduction delay. The relative importance of neural, anatomic, and developmental factors21–24 in effecting this wide range of AV junctional conduction properties is still unknown.

**AV Conduction Characteristics and Ventricular Response During Supraventricular Tachyarrhythmias**

Although AV conduction properties in patients in this study appeared to be normally distributed with no discrete subgroups, it was useful for analytical purposes to subdivide the patient population by the presence or absence of EAVC. Patients who had EAVC had more rapid heart rates during RT (fig. 3). This finding was due primarily to the subgroup of patients with RT due to reentry using a concealed accessory AV pathway.

The apparent relation between dynamic properties of AV nodal conduction as reflected by the presence or absence of EAVC, and RT cycle length in patients...
with accessory AV pathways is consistent with our understanding of orthodromic RT mechanisms in patients with either concealed accessory AV pathways or with WPW syndrome. In both conditions, the reentry pathway usually uses the AV node in the antegrade (AV) direction and the accessory AV pathway in the ventriculoatrial direction. Consequently, during RT, conduction in the antegrade direction traverses the entire AV node from atria to ventricles. Therefore, electrophysiologic properties of AV conduction that can be assessed by atrial pacing techniques would be expected to influence RT cycle length directly. However, there was no apparent correlation between the presence or absence of EAVC and tachycardia cycle length in patients with RT due to reentry within the AV node. This observation parallels the report by Bauernfeind et al., in which linear regression analysis showed no significant relation between static electrophysiologic measurements during sinus rhythm (i.e., PR and AH interval) and RT cycle lengths in patients with presumed reentry within the AV node.

The absence of a close correlation between AV nodal conduction properties and tachycardia cycle length in patients with RT due to reentry within the AV node is not unexpected. Given the complex histology of AV junctional tissues and the multiplicity of cellular electrophysiologic interactions that can result, many potential reentry pathways may exist.

The possibility that EAVC in patients with RT is due to AV conduction over the so-called fast AV nodal pathway merits consideration. Although too few of our patients exhibited discontinuous AV conduction curves to permit direct evaluation of a relationship between EAVC and fast-pathway conduction characteristics, Bauernfeind et al. found no statistically significant relation between fast pathway conduction and RT cycle length in patients with RT due to reentry within the AV node.

**Ventricular Response During Atrial Fibrillation**

Evaluation of the ventricular response during atrial fibrillation revealed that both the mean RR intervals and shortest RR intervals were shorter in patients with EAVC than in those without EAVC. Although the number of patients was too small for statistical analysis, the relation between EAVC and ventricular response in atrial fibrillation appeared to hold in patients who had exhibited RT due to reentry within the AV node and in those who had RT using an accessory AV pathway and paralleled findings in AV refractory period studies. Consequently, fundamental differences in AV nodal characteristics of conduction and refractoriness do not seem to explain the discrepancy in the relation between the presence of EAVC and tachycardia rate in these two forms of RT.

In conclusion, interaction of developmental, anatomic, electrophysiologic, and neural influences results in a spectrum of physiologic conduction delay responses within AV junctional tissue in man. An abnormality in one or more of these factors may contribute to diminished AV conduction delay capability and permit rapid tachycardia rates in certain patients. Because therapy of supraventricular tachycardias relies on drugs that either alter excitable membrane properties directly (e.g., quinidine, procainamide and verapamil) or modulate neural influences within the AV node (e.g., digoxin and propranolol), a more complete understanding of the basis for diminished physiologic conduction delay within the AV junction may have important therapeutic implications.

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