Effect of Atrioventricular Interval During Pacing or Reciprocating Tachycardia on Atrial Size, Pressure, and Refractory Period

Contraction-Excitation Feedback in Human Atrium

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To determine whether a contraction-excitation feedback mechanism exists in human atrium, we investigated the effects of varying the atrioventricular (AV) interval from 0 to 360 msec during AV pacing at a cycle length of 400 msec on atrial pressure, size, and refractoriness in 10 patients (group 1, without supraventricular tachycardia). The same parameters were determined in another 10 patients (group 2, with different spontaneous AV relations) during AV reciprocating tachycardia or AV nodal reciprocating tachycardia and during high right atrial (RA) pacing at the tachycardia cycle length. In group 1 patients, peak and mean RA pressure, RA effective refractory period (RA-ERP), and left atrial (LA) size increased to minimal values at an AV interval of 120 msec and remained low as the AV interval was increased and approached 400 msec. The increase in each of the variables from its lowest to greatest value was as follows: Mean systemic blood pressure, 20.9±3.1 mm Hg; LA size, 0.55±0.05 cm; RA peak pressure, 10.4±1.8 mm Hg; RA mean pressure, 3.5±0.6 mm Hg; and RA-ERP, 22.5±3.0 msec, p<0.001 for each. The weighted mean correlation coefficient with RA-ERP was significant for RA peak pressure and LA size (p<0.001 for each). These same relations were investigated in five patients with the Wolff-Parkinson-White syndrome and AV reciprocating tachycardia and five patients with AV nodal reciprocating tachycardia (group 2). In the patients with Wolff-Parkinson-White syndrome, the increase in each variable measured during tachycardia versus that measured during RA pacing (at the tachycardia rate) was as follows: LA size, 0.45±0.28 cm (p<0.05); RA peak pressure, 4.5±2.0 mm Hg (p<0.01); RA mean pressure, 1.7±1.2 mm Hg (p<0.05); and RA-ERP, 30.0±23.2 msec (p<0.05). The mean blood pressure did not change. In the patients with AV nodal reciprocating tachycardia, similar increases occurred during tachycardia (as compared with atrial pacing at the same rate) as follows: LA size, 0.59±0.20 cm (p=0.03); RA peak pressure, 8.0±5.2 mm Hg (p=0.03); RA mean pressure, 3.6±1.1 mm Hg (p=0.03); and RA-ERP, 57±56 msec (p=0.03). Again, the mean blood pressure did not change. We conclude that 1) a very short or very long AV interval causes an increase in RA pressure, LA size, and RA refractoriness, and 2) during reciprocating tachycardia, LA size, RA pressure, and RA-ERP are all greater than during atrial pacing at the same cycle length, probably due to the AV relations characteristic of these arrhythmias. Thus, a contraction-excitation feedback mechanism exists in the human atrium and might be partly responsible for some arrhythmias in man. (Circulation 1990;82:60–68)

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Contraction-excitation feedback has been previously demonstrated in canine atrium but not human atrium, and its role in human arrhythmias has never been fully investigated. For instance, many patients with supraventricular tachycardia but otherwise structurally normal hearts can develop atrial fibrillation. It has been suggested that a contraction-excitation feedback mechanism might be responsible for this and a variety of other arrhythmias.12

Profound hemodynamic alterations might accompany the development of atrioventricular (AV) recip-
rocati ng tachycardia (AVRT) or AV nodal reciprocating tachycardia (AVNRT) in susceptible patients. In addition to the well-known autonomic changes, an increase in atrial pressure and the development of atrial stretch could result from the rapid heart rate and altered AV relations. Contraction-excitation feedback might occur if an increase in atrial pressure and wall stretch altered atrial properties of conduction and refractoriness that, particularly if not uniform, might be conducive to the development of atrial tachyarrhythmias.

The purpose of this study was twofold as follows: First, to test the hypothesis that varying AV intervals at rapid pacing rates (by modeling the values found during AVRT and AVNRT) increases atrial pressure and size, which then alters atrial refractoriness, and second, to test whether similar responses occurred during AVRT or AVNRT by investigating the effect of AVRT or AVNRT (versus atrial pacing at the same rate) on atrial size, pressure, and refractory period in patients with the Wolff-Parkinson-White syndrome (WPW) and patients with sustained AVNRT.

Methods

Patient Population

Ten patients without spontaneous tachycardia (six men and four women) with a mean age of 50 years (range, 28–62 years) underwent electrophysiological study in the postabsorptive nonsedated state after oral and written informed consent. Patients with WPW were excluded from this portion of the investigation so that many AV intervals could be investigated without limitation by rapid AV or ventriculo-atrial conduction over an accessory pathway. Seven patients had no structural heart disease, two patients had mild mitral valve prolapse by echocardiography, and one patient had a previous myocardial infarction. No patient was receiving cardioactive medications except one who was receiving metoprolol for angina. All patients were being studied because of syncope (nine patients) or presyncope (one patient).

Five of 13 consecutive patients with WPW and AVRT (spontaneous and electrically induced) were also studied. There were three males and two females with a mean age of 24 years (range, 15–37 years). These patients were receiving no cardioactive medications. None had identifiable structural heart disease, and only one patient had a known history of atrial fibrillation. Eight of the 13 patients with WPW and manifest preexcitation had to be excluded from the study because sustained atrial fibrillation was repeatedly induced either during diagnostic testing or during the study protocol.

Five of seven consecutive patients with AVNRT (spontaneous and electrically induced) completed the study. There were two men and three women with a mean age of 57 years (range, 40–68 years). These patients were also receiving no cardioactive medications, and none had identifiable structural heart disease. None of the seven patients had a history of atrial fibrillation. As with the WPW patients, two of the seven patients with AVNRT had to be excluded from the study because sustained atrial fibrillation was repeatedly induced during diagnostic testing or during the study protocol.

Pacing Protocol

For all patients, a 6F multipolar electrode catheter (USCI Corp., Billerica, Massachusetts) was positioned in the high right atrium and in the right ventricular apex. One Goodale-Lubin 7F luminal catheter (USCI) was positioned in the mid-right atrium to obtain atrial pressure recordings. Pacing was performed with a custom-built stimulator (Medical Engineering, Krannert Institute, Indianapolis, Ind.) using 2-msec rectangular pulses at twice late diastolic threshold. Surface leads I, II, III, and V1 were displayed on a multichannel oscilloscope (VR-16, Electronics for Medicine PPG Biomedical Systems, Hershey, Pennsylvania) simultaneously with high right atrial and ventricular electrograms and right atrial pressure. All parameters were recorded at a paper speed of 50 or 75 mm/sec.

In the 10 patients without WPW, AV pacing was performed at a drive cycle length of 400 msec with the AV interval varying in 40-msec steps from 0 to 360 msec. The high right atrial effective refractory period (RA-ERP) was determined at each AV interval by introducing progressively more premature atrial extrastimuli (5-msec decrements) after every eighth paced beat until the extrastimulus failed to capture the atrium twice in succession. Pacing was performed at each AV interval for 2 minutes at which time values stabilized and systemic blood pressure, atrial pressure, size, and refractory period were determined.

In the five patients with WPW, AVRT was initiated and observed for 2 minutes. Atrial pressure and size, and systemic blood pressure were measured after which atrial extrastimuli (2-msec pulse duration, twice diastolic threshold) were introduced, starting in early diastole (no atrial capture) and scanning diastole in 5-msec increments until atrial capture occurred twice in succession. These same measurements were obtained during right atrial pacing (at the AVRT rate) in these patients. If tachycardia was initiated (testing during atrial pacing) or terminated (testing during tachycardia) before determination of the refractory period, repeat testing was performed after a 2-minute equilibrium period.

The five patients with AVNRT were studied in a manner identical to the five patients with WPW and AVRT.

Hemodynamic Measurements

Peak and mean right atrial pressures were measured by using the fluid-filled luminal catheter (Goodale-Lubin) in the mid-right atrium. Systemic systolic and diastolic blood pressure (brachial cuff pressure) were measured at each AV pacing interval after a 2-minute equilibration period. Mean arterial
FIGURE 1. Photomicrographs and analog data from one patient without supraventricular tachycardia showing changes in right atrial effective refractory period (RA-ERP), left atrial size, and right atrial pressure at atroventricular (AV) intervals of 0 (lower panel) and 120 msec (upper panel), respectively. The format for each panel is identical, showing (from top to bottom) surface electrocardiographic lead I, and high right atrial (HRA) and right ventricular (RV) electrogram recordings. In the lower right portion of the figures, electrograms are reproduced and superimposed on a pressure recording from the right atrium (RA) to illustrate the timing of the cardiac cycle. A two-dimensional echocardiogram is shown for each AV pacing interval with the left atrium shown by the arrow. The measurement of left atrial size is depicted to the right of the picture. Upper panel: During pacing at an AV interval of 120 msec, the RA-ERP is 245 msec, the peak atrial pressure is 9 mm Hg, and the left atrial size is 3.9 cm. Lower panel: At an AV interval of 0 msec (simultaneous AV pacing), the RA-ERP is 255 msec, the peak atrial pressure is 23 mm Hg, and the left atrial size is 4.4 cm. S_s, drive train pacing; S_a, atrial extrastimulus.
blood pressure was calculated as diastolic pressure plus one third the pulse pressure. Systemic blood pressure in the patients with AVRT and AVNRT was measured with intra-arterial catheters.

**Atrial Size**

Left atrial size was determined in all patients at end systole by two-dimensional echocardiography (Advanced Technology Laboratories, Bothell, Washington, or Hewlett-Packard Corporation, Palo Alto, California) by using the parasternal long-axis view (digitized still frame, electronic calipers, Microsonics Corporation, Mahwah, New Jersey). Echocardiographic pictures were obtained after a 1-minute equilibration period at each AV pacing interval before refractory period determinations, or after 2 minutes of AVRT, AVNRT, or atrial pacing in the patients with AVRT or AVNRT.

**Definitions**

In the 10 patients without WPW, the RA-ERP was defined as the longest extrastimulus interval (S1-S2) during which S2 failed to produce a propagated atrial response. In the 10 patients with AVRT or AVNRT, however, to compare the effective refractory period determination during atrial pacing and AVRT or AVNRT, the RA-ERP was defined as the longest atrial electrogram to extrastimulus interval (A1-S2) during which S2 failed to capture the atrium, both during AVRT or AVNRT and atrial pacing.

**Statistical Analysis**

The significance for individual variables was determined by a single variable t test (mean value for each variable versus zero). The change in the measured parameters during AVNRT were analyzed using the Wilcoxon signed rank test. Correlations were determined by comparing the weighted mean correlation coefficients of RA-ERP versus that of each of the other variables. Statistical significance was defined as p values of less than 0.05.

**Results**

**Effects of Variations of the Atrioventricular Interval**

Analog data from one patient without supraventricular tachycardia are illustrated in Figure 1, showing RA-ERP, right atrial pressure, and left atrial size at AV intervals of 0 and 120 msec. This patient was typical for the group as a whole in that maximal and minimal values for these measured parameters were found at 0 and 120 msec, respectively, that is, there was a larger left atrium, a higher right atrial pressure, and a longer RA-ERP at an AV interval of 0 msec (simultaneous AV pacing) than at an AV interval of 120 msec.

Data from the 10 patients without supraventricular tachycardia are shown in Figure 2. As the AV interval increased from 0 to 400 msec (abscissa), right atrial peak and mean pressures initially declined but subsequently increased as the AV interval was prolonged more. Left atrial size and RA-ERP followed a similar although less consistent pattern. Mean systemic blood pressure, however, initially increased before gradually decreasing at longer AV pacing intervals.

**Correlation of Atrial Pressure and Size With Refractory Period**

The maximal change in each of the parameters for the 10 patients without supraventricular tachycardia, and the correlations with RA-ERP, are shown in Table 1. As the AV interval varied, there was a significant change in mean blood pressure, left atrial size, peak and mean right atrial pressure, and RA-ERP. An attempt was made to correlate the effect of AV interval variation on each of the parameters with the change in the RA-ERP. As Table 1 shows, RA-ERP correlated significantly with left atrial size and right atrial peak pressure, although the correlation was not strong for each parameter. There was no
correlation between RA-ERP and either right atrial mean pressure or mean systemic blood pressure.

**Effect of Reciprocating Tachycardia**

During AVRT, left atrial size increased, right atrial peak and mean pressure increased, and RA-ERP increased (Table 2). There was no change in the mean systemic blood pressure. Analog data from a patient (patient 5, Table 2) whose measured changes were similar to the mean change typical for the group as a whole are shown in Figure 3. Although there was a great deal of variability between patients, no quantitative relation existed between the degree of change in atrial refractory period and either right atrial pressure or left atrial size, nor was there any relation between the AVRT rate, accessory pathway location or history of atrial fibrillation, and the measured changes (Table 2).

During AVNRT (as during AVRT), left atrial size increased, right atrial peak and mean pressures increased, and RA-ERP increased (Table 3). There was no change in the mean systemic blood pressure. Again, because of the great variability between patients, no identifiable quantitative relation existed between the degree of change in atrial refractory period and either right atrial pressure or left atrial size, nor did the measured changes in any of the patients correlate with the rate of tachycardia or atrial pacing. Analog data from one patient (patient 5, Table 3) whose measured changes were also similar to the mean change typical for the group as a whole are shown in Figure 4.

**Discussion**

**New Observations**

This study demonstrates in humans that the RA-ERP increases as does the right atrial pressure and left atrial size during pacing at certain AV intervals, and during AVRT and AVNRT. In our model, as the AV interval during AV pacing prolonged from 0 to 120 msec, minimal values for right atrial pressure and RA-ERP were obtained. These values increased as the AV interval prolonged more. Consistent with these data, right atrial pressure, left atrial size, and RA-ERP all increased in patients during AVRT or during AVNRT compared with values obtained during atrial pacing at the tachycardia cycle lengths.

**Contraction-Excitation Feedback in Atrial and Ventricular Myocardium**

We have previously investigated the immediate effects of an increase in atrial pressure on atrial refractoriness by determining the relation between the atrial pressure and effective refractory period in dogs. During AV pacing at a cycle length of 300

### Table 1. Maximal Change in the Parameters for the Ten Patients Without Tachycardia and Correlation With Right Atrial Effective Refractory Period

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Max Δ (±SEM)</th>
<th>p</th>
<th>r-RA-ERP</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP mean (mm Hg)</td>
<td>20.9±3.1</td>
<td>0.001</td>
<td>0.04</td>
<td>NS</td>
</tr>
<tr>
<td>LA size (cm)</td>
<td>0.55±0.05</td>
<td>0.001</td>
<td>0.33</td>
<td>0.001</td>
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<tr>
<td>RA peak (mm Hg)</td>
<td>10.4±1.8</td>
<td>0.001</td>
<td>0.34</td>
<td>0.001</td>
</tr>
<tr>
<td>RA mean (mm Hg)</td>
<td>3.5±0.6</td>
<td>0.001</td>
<td>0.11</td>
<td>NS</td>
</tr>
<tr>
<td>RA-ERP (msec)</td>
<td>22.5±3.0</td>
<td>0.001</td>
<td>. . .</td>
<td>. . .</td>
</tr>
</tbody>
</table>

Max Δ, Maximal change in each parameter for all 10 patients; p, statistical significance of maximal change in each parameter for all patients; r-RA-ERP, correlation of changes in each variable with the RA-ERP as atrioventricular interval changed; RA-ERP, effective refractory period of the right atrium; BP, blood pressure; LA, left atrium; RA, right atrium.

### Table 2. Changes in Left Atrial Size, Right Atrial Pressure, and Right Atrial Effective Refractory Period From Atrial Pacing at the Tachycardia Rate to Atrioventricular Reciprocating Tachycardia in the Wolff-Parkinson-White Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>ΔLAD (cm)</th>
<th>ΔRAM (mm Hg)</th>
<th>ΔRAP (mm Hg)</th>
<th>ΔMBP (mm Hg)</th>
<th>ΔRA-ERP (msec)</th>
<th>RT_{CL} (msec)</th>
<th>RT AV_{min} (msec)</th>
<th>Atrial pace AV (msec)</th>
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<tr>
<td>1</td>
<td>0.39</td>
<td>0</td>
<td>4</td>
<td>−11</td>
<td>30</td>
<td>350</td>
<td>220</td>
<td>75</td>
</tr>
<tr>
<td>2</td>
<td>0.55</td>
<td>1.5</td>
<td>4</td>
<td>2</td>
<td>65</td>
<td>330</td>
<td>240</td>
<td>80</td>
</tr>
<tr>
<td>3</td>
<td>0.07</td>
<td>3</td>
<td>8</td>
<td>−1</td>
<td>25</td>
<td>440</td>
<td>280</td>
<td>95</td>
</tr>
<tr>
<td>4</td>
<td>0.85</td>
<td>2.5</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>300</td>
<td>235</td>
<td>110</td>
</tr>
<tr>
<td>5</td>
<td>0.37</td>
<td>1.5</td>
<td>3.5</td>
<td>8</td>
<td>30±23.2</td>
<td>354</td>
<td>250</td>
<td>84</td>
</tr>
</tbody>
</table>

Mean±SEM 0.45±0.2 1.7±1.2 4.5±0.3 −0.2±6.9 30±23.2 354 250 84

The accessory pathway location was left free wall in all patients except patient 3, whose accessory pathway was located in the right free wall. Note the increases in left atrial dimension, right atrial mean and peak pressure, and right atrial effective refractory period were all significant. There was no significant change in mean systemic blood pressure. ΔLAD, Change from atrial pacing (at the tachycardia rate) to atrioventricular reciprocating tachycardia (AVRT) in the left atrial dimension; ARAM, right atrial mean pressure; ΔRAP, right atrial peak pressure; ΔMBP, mean systemic blood pressure; ΔRA-ERP, right atrial effective refractory period for each of the five patients with Wolff-Parkinson-White syndrome and sustained AVRT. RT_{CL}, AVRT cycle length; RT AV_{min}, minimal measured atrioventricular interval during AVRT; atrial pace AV, AV interval during atrial pacing.
FIGURE 3. Photomicrographs and analog data from one patient with Wolff-Parkinson-White syndrome (patient 5, Table 2) showing changes in right atrial effective refractory period (RA-ERP), left atrial size, and right atrial pressure during atioventricular reciprocating tachycardia (AVRT) (lower panel) when compared with atrial pacing at the same rate (upper panel). The format and abbreviations are the same as in Figure 1; however, the RA-ERP has been redefined (see “Methods”). Upper panel: The atrium is paced at a cycle length of 350 msec. Peak atrial pressure is 13 mm Hg, the RA-ERP is 120 msec, and the left atrial size is 2.54 cm. Lower panel: AVRT is present at a cycle length of 350 msec. Peak right atrial pressure has increased to 16 mm Hg, RA-ERP has increased to 150 msec, and left atrial size has increased to 2.91 cm. I, Surface electrocardiographic lead I; HRA, high right atrial; RV, right ventricular; RA, right atrium.
TABLE 3. Changes in Left Atrial Size, Right Atrial Pressure, and Right Atrial Effective Refractory Period From Atrial Pacing at the Tachycardia Rate to Atrioventricular Nodal Reciprocating Tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>ΔLAD (cm)</th>
<th>ΔRAM (mm Hg)</th>
<th>ΔRAP (mm Hg)</th>
<th>ΔMBP (mm Hg)</th>
<th>ΔRA-ERP (msec)</th>
<th>RTCL (msec)</th>
<th>RT AV (VA)₉₅min (msec)</th>
<th>Atrial pace AV (msec)</th>
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<td>1</td>
<td>0.65</td>
<td>2</td>
<td>12</td>
<td>4</td>
<td>60</td>
<td>370</td>
<td>330 (40)</td>
<td>290</td>
</tr>
<tr>
<td>2</td>
<td>0.30</td>
<td>4</td>
<td>12</td>
<td>2</td>
<td>15</td>
<td>280</td>
<td>270 (10)</td>
<td>170</td>
</tr>
<tr>
<td>3</td>
<td>0.71</td>
<td>3</td>
<td>4</td>
<td>8</td>
<td>10</td>
<td>280</td>
<td>240 (40)</td>
<td>165</td>
</tr>
<tr>
<td>4</td>
<td>0.49</td>
<td>2</td>
<td>10</td>
<td>150</td>
<td>300</td>
<td>270 (30)</td>
<td></td>
<td>210</td>
</tr>
<tr>
<td>5</td>
<td>0.82</td>
<td>5</td>
<td>5</td>
<td>50</td>
<td>390</td>
<td>350 (40)</td>
<td></td>
<td>300</td>
</tr>
<tr>
<td>Mean±SEM</td>
<td>0.59±0.20</td>
<td>3.6±1.1</td>
<td>8.6±5.2</td>
<td>2.6±6.6</td>
<td>57±56</td>
<td>324</td>
<td>292</td>
<td>227</td>
</tr>
</tbody>
</table>

Changes from atrial pacing (at the tachycardia rate) to atrioventricular (AV) nodal reciprocating tachycardia (AVNRT) in the left atrial dimension (ΔLAD), right atrial mean (ΔRAM) and peak (ΔRAP) pressures, mean systemic blood pressure (ΔMBP), and right atrial effective refractory period (ΔRA-ERP) for each of the five patients with AVNRT. The minimal ventriculoatrial (VA) interval in all five patients was short (<60 msec). Note the increases in left atrial dimension, right atrial mean and peak pressures, and right atrial effective refractory period were all significant. As expected, the AV interval was longer (i.e., VA interval was shorter) during tachycardia than during atrial pacing at the same rate. There was no significant change in mean systemic blood pressure. RTCL, AVNRT cycle length; RT AV (VA)₉₅min, minimal measured atroventricular interval during AVNRT [(VA), ventriculoatrial interval]; Atrial pace AV, AV interval during atrial pacing.

msec, mean left atrial pressure and refractory period were found to be lowest at a short AV interval (47 msec) and highest at a longer AV interval (147 msec). Left atrial diameter increased by more than 4 mm. These changes were not affected by surgical decentralization of effenter vagal and sympathetic innervation to eliminate baroreceptor reflex influences on refractoriness. Refractory period increases were noted in several atrial locations (right and left atrial free wall and interatrial septum). Thus, an increase in atrial pressure lengthened the refractory period of both atria and the interatrial septum, suggesting a contraction-excitation feedback mechanism dependent on atrial stretch and secondarily on the AV relation.1

In the present human study, we also found changes in atrial pressure, size, and refractory period with variation of the AV interval. Not only were these changes not uniform from patient to patient, however, the AV interval at which these changes were maximal was not in midcycle but rather during simultaneous AV pacing. Possible explanations for the difference between the previous canine and present human results include a different pacing rate (300 msec in the canine study versus 400 msec in this human study), species differences, and the fact that the dogs were anesthetized. Nevertheless, both the canine and present human study showed an increase in atrial size and pressure was associated with an increase in atrial refractoriness.

Role of Contraction-Excitation Feedback

We found that the RA-ERP increased during AVRT more than during atrial pacing at the same rate. Similar increases occurred in patients with AVNRT, and therefore, these results were most likely due to the differences in the AV interval of tachycardia versus atrial pacing. The increase in RA-ERP as right atrial pressure and left atrial size increased during AVRT and AVNRT is consistent with the data obtained during AV pacing in patients without tachycardia (Figure 2). Obstruction to atrial emptying due to closed AV valves at the AV intervals found during reciprocating tachycardia and replicated during AV sequential pacing must stretch the atria. We hypothesize that atrial stretch occurs unequally, perhaps more in the free wall than in the atrial septum or in thick muscle bundles, possibly involving the accessory pathway directly or its insertion site or sites. During a reciprocating tachycardia, unequal changes in the atrial refractory period might result in regional alterations or dispersion of refractoriness that predispose to the development of atrial fibrillation.13 Whether patients with reciprocating tachycardia who develop atrial fibrillation have a greater increase in atrial size and refractory period or greater dispersion of refractoriness during spontaneous tachycardia than do those patients who do not develop atrial fibrillation remains to be tested. It would be possible, during surgery, to test the effects of atrial stretch at multiple atrial sites for interruption of an accessory pathway or during surgery for AV nodal reentry. Anisotropic conduction14 might similarly be affected heterogeneously by stretch, leading to areas with conduction delay or block that could predispose to the development of atrial fibrillation.

Limitations of the Study

Pressure and refractory period changes were measured from the left atrium, whereas atrial size was measured from the left atrium with the assumption that an overall change in one atrium reflects similar direction although perhaps not quantitative changes in the other. Because we were not able to measure refractory period changes reliably in discrete locations, that is, atrial septum versus free wall or thick muscle bundles (e.g., crista terminalis) versus thin atrial walls, we could not search for quantitative differences between atrial sites. In fact, not knowing the type of atrial architecture at the electrode site might explain the RA-ERP changes that seemed inconsistent quantitatively with the amount of pres-
sure or size change in patients 3 and 4 (Table 2) with AVRT or patient 3 with AVNRT (Table 3). It might also explain why the increase in left atrial dimension was greater in patients during AV pacing that modeled reciprocating tachycardia than during AVRT or AVNRT, whereas the increase in RA-ERP was less (Tables 1–3).

The drive cycle length of 400 msec used to investigate AV relations in this study exceeds the typical AVRT cycle length. Shorter cycle lengths might have resulted in more profound changes in atrial pressure, size, and refractory period, possibly peaking at a different part of the cardiac cycle. Because most6,8,9 but not all15 studies of patients with WPW (for example) and spontaneous atrial fibrillation, however, have not found the cycle length of AVRT to be a risk factor for the development of atrial fibrillation, and because we found the same directional changes in patients with reciprocating tachycardia as we did during variations of the AV interval, it is not likely that more rapid pacing would have resulted in qualitatively different findings.
We cannot be certain that our observed changes in refractory period during tachycardia were not due to directional changes in atrial activation (i.e., retrograde atrial activation during tachycardia). Our data in normal hearts (Figure 2), however, showed similar refractory period increases during AV pacing at short AV intervals (when there was no change in atrial activation). Further, these changes correlated with changes in atrial pressure and size, supporting our hypothesis.

Finally, patients who had easily inducible (and often spontaneous) atrial fibrillation had to be excluded from the study because of repetitive induction of atrial fibrillation that precluded continuation of the study protocol. This occurred frequently in patients with WPW as well as in patients with AVNRT. Induced atrial fibrillation in these patients severely limited our ability to determine whether patients with spontaneous atrial fibrillation were more likely to have greater changes in the measured parameters than patients without a history of spontaneous atrial fibrillation. Thus, the data were skewed in favor of the patients who could complete the study protocol, that is, patients who were not prone to atrial fibrillation.

Clinical Relevance

Data from our animal study1 and the present clinical study demonstrate that the change in atrial size, pressure, and refractory period in patients during tachycardia depends on the AV interval, indicating that a contraction-excitation feedback mechanism exists in atrial muscle and might influence atrial electrophysiology during AVRT or AVNRT.

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References


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