Role of Atrial Electrophysiology and Autonomic Nervous System in Patients With Supraventricular Tachycardia and Paroxysmal Atrial Fibrillation

YI-JEN CHEN, MD,* SHIH-ANN CHEN, MD, CHING-TAI TAI, MD, ZU-CHIN WEN, MD, AN-NING FENG, MD, YU-AN DING, MD, MAU-SONG CHANG, MD

Taipei, Taiwan

**Objectives.** The purposes of this study were to evaluate the atrial electrophysiology and autonomic nervous system in patients who had paroxysmal supraventricular tachycardia (PSVT) associated with paroxysmal atrial fibrillation (PAF).

**Background.** PAF frequently appeared in patients with PSVT. However, the critical determinants for the occurrence of PAF were not clear.

**Methods.** This study population consisted of 50 patients who had PSVT with (n = 23) and without (n = 27) PAF. Atrial pressure, atrial size, atrial effective refractory periods (AERPs), and AERP dispersion were evaluated during baseline and PSVT, respectively. Twenty-four hour heart rate variability and baroreflex sensitivity (BRS) were also examined.

**Results.** There was greater baseline AERP dispersion in patients with PAF than in those without PAF. The atrial pressure, atrial size, AERPs in the right posterolateral atrium and distal coronary sinus, and AERP dispersion were increased during PSVT as compared with those during baseline. Patients with PAF had greater AERP dispersion than those without PAF during PSVT. The differences of atrial size, right posterolateral AERP, and AERP dispersion between baseline and PSVT were greater in patients with PAF than in those without PAF. BRS, but not heart rate variability, was higher in patients with PAF than in those without PAF. Univariate analysis showed that higher BRS (>4.5 ms/mm Hg, p = 0.0002, odds ratio = 16.1), AERP dispersion during PSVT (>40 ms, p = 0.0008, odds ratio = 9.7), and increase of right atrial area during PSVT (>2 cm², p = 0.016, odds ratio = 10.7) were significantly correlated with the occurrence of PAF in patients with PSVT.

**Conclusions.** Disturbed atrial electrophysiology and higher vagal reflex could play important roles in the genesis of PAF in patients with PSVT.

(J Am Coll Cardiol 1998;32:732–8)

©1998 by the American College of Cardiology

---

Paroxysmal atrial fibrillation (PAF) frequently occurred in patients with paroxysmal supraventricular tachycardia (PSVT) (1–4). However, the mechanisms responsible for the occurrence of PAF were not fully understood. Although previous studies have suggested that atrioventricular (AV) accessory pathway or slow AV node pathway itself may play an important role in the genesis of PAF (5–11), disturbed atrial electrophysiology during PSVT may be the other mechanism of initiation of PAF (1,5,6,12). Klein et al. (13) have shown increased atrial refractoriness during PSVT and considered mechanoelectrical feedback a cause of PAF during PSVT. Satoh et al. (14) demonstrated that increasing atrial dispersion from atrial stretch in canine heart could induce atrial fibrillation (AF). In humen, increase of atrial size correlates well with an increased vulnerability to AF (15). Thus, possible alteration of atrial size and atrial pressure during PSVT may change the atrial electrophysiologic properties and result in the occurrence of PAF. Another possible mechanism is that autonomic nervous system may be related to the genesis of PAF during PSVT. Although Coumel et al. (16) have described different types of PAF resulting from increased vagal tone or increased adrenergic tone, there is no report about the role of autonomic nervous system in patients who have PSVT associated with PAF.

Therefore, the purposes of this study were 1) to evaluate the baseline atrial pressure, atrial size and atrial electrophysiologic characteristics and to evaluate these changes during PSVT in patients with and without PAF; 2) to investigate the role of the autonomic nervous system in the occurrence of PAF in these patients; 3) to study the critical determinant of the occurrence of PAF in the patients with PSVT.

**Methods**

**Patients characteristics.** This study included 50 patients who had PSVT and were divided into two groups. Group I...
blood pressure was measured by using intraarterial catheters by using the fluid-filled luminal catheter. The patient’s mean atrial pressure and pulmonary wedge pressure were measured after discontinuation of antiarrhythmic drugs. Informed consent for the study and ablation was obtained from each patient. Four multipolar, closely spaced electrode catheters (inter electrode space = 2 mm, Boston Scientific, MA) were introduced from the right and left femoral veins and placed in the high right atrium, low right posterolateral atrium, His bundle area and right ventricle for programmed electrical stimulation and recording. One sterecable decapolar catheter (2-5-2-5-2-5-2 configuration, Daig) was introduced from the right internal jugular vein and placed in the coronary sinus. Intracardiac electrograms were displayed simultaneously with electrocardiographic leads I, II and VI on a multichannel oscilloscopic recorder (MIDAS system series 2000) and were recorded on paper at a speed of 100 to 150 mm/s. A programmed digital stimulator (DTU-210 or 215, Bloom Associates Ltd., Reading) was used for atrial and ventricular incremental pacing and extrastimulation to induce tachycardia. The patients who needed isoproterenol or atropine to induce tachycardia were not included in this study.

**Study protocol.** After PSVT was initiated and sustained for 5 min, we measured hemodynamic parameters (mean blood pressure, right atrial pressure, and pulmonary wedge pressure), bilateral atrial size, AERPs, and stimulation thresholds. The data of baseline study were obtained again from the same parameters measured during sinus rhythm after PSVT terminated more than 10 min.

1) **Measurements of hemodynamic parameters.** The right atrial pressure and pulmonary wedge pressure were measured by using the fluid-filled luminal catheter. The patient’s mean blood pressure was measured by using intraarterial catheters from femoral artery.

2) **Measurement of AERP.** The AERP and stimulation threshold in high right lateral atrium, low right posterolateral atrium, and distal coronary sinus were measured during PSVT and during baseline study, respectively. Measurement of the AERPs was made from an atrial extrastimulus (2 ms pulse duration, twice threshold) introduced from early diastole after every eighth atrial beat (during PSVT or during atrial pacing with a rate similar to that of PSVT) until atrial capture occurred twice in succession (in 2 ms increments). The measured AERPs were acceptable for analysis only if these data were identical during two repeated measurements. AERP dispersion was defined as the maximal difference of AERPs at the three stimulation sites. Atrial vulnerability was defined as the inducibility of PAF sustained more than 30 s from single atrial extrastimulus during PSVT or during baseline study (a basic atrial pacing cycle length similar to that during PSVT). The patients with PAF sustained more than 10 min would receive electrical cardioversion to restore to sinus rhythm. If PAF was induced during the study protocol, repeated testing was performed 15 min after PAF was spontaneously or electrically converted to sinus rhythm.

3) **Measurement of atrial size.** Left atrial size and right atrial size were measured at end systole by two-dimensional echocardiography (Hewlett-Packard Sonus 1000 ultrasound system) in the apical four chamber view (19).

4) **Baroreflex sensitivity study.** Baroreflex sensitivity study (BRS) was determined only when the patients had stable heart rate and blood pressure by using intravenous phenylephrine after the baseline electrophysiologic study. Patients were placed in supine position and were instructed to breathe regularly. Continuous arterial pressure and surface electrocardiogram were recorded simultaneously at a paper speed of 25 mm/s. The blood pressure was increased by 15 to 30 mm Hg by intravenous injection of phenylephrine (2–4 µg/kg body weight). The dose found by this procedure was repeated at least twice. Beat-by-beat changes in baseline systolic blood pressure (in mm Hg) and in the lengths of the RR intervals (in milliseconds) was calculated from records. Each RR interval was plotted against the preceding arterial pressure pulse, and linear regression analysis was performed. Only regression lines exhibiting a significant correlation coefficient (p < 0.05) were accepted for analysis. The final slope represents the mean value of three or more successive determinations.

5) **Heart rate variability study.** The 24-h electrocardiographic recordings were performed on the day before electrophysiologic study. Tape-recorded electrocardiograms for each subject were digitally processed and annotated by manual editing with a Holter Analysis System (Oxford Medilog Excel, Oxford Med. Instruments). After QRS configuration classification, the longest and the shortest RR intervals on the RR interval histogram, and the largest and the smallest RR ratios on the RR ratio histogram were manually confirmed until no QRS complex was mislabeled as either an artifact or an ectopic beat. In this laboratory, time domain variables considered in this study were mean RR interval (the mean of all coupling intervals between normal sinus beats expressed in milliseconds), SDNN (the standard deviation of the mean RR interval expressed in milliseconds), rMSSD (the root mean square of differences of successive RR intervals), and PNN50 (the percentage of adjacent RR intervals that differed by more than 50 ms). In addition, three frequency-domain measurements,
namely, low-frequency component (0.04 to 0.15 Hz), high frequency component (0.15 to 0.40 Hz), and low/high frequency ratio were computed.

**Statistical analysis.** All data are expressed as mean ± SEM. Comparisons of the data measured during PSVT and baseline study were analyzed by paired t test for numerical data. The differences of the measured numerical data between different groups were analyzed by unpaired t test. Chi-square test with Yates’ correction or Fisher exact test were used for the categorical data. Univariate and multivariate analysis was used to analyze the variables (tachycardia cycle length, hemodynamics, atrial size, AERPs, ERP dispersion, BRS, and heart rate variability) which could predict the occurrence of PAF in patients with PSVT. A p value less than 0.05 was considered to be statistically significant.

**Results**

**Baseline study.** In Group I (with PAF), there were 10 patients who had AV nodal reentrant tachycardia and 13 patients who had concealed accessory pathways associated with AV reentrant tachycardia. In Group II (without PAF), there were 15 patients who had AV nodal reentrant tachycardia and 12 patients who had concealed accessory pathways associated with AV reentrant tachycardia. The age (45 ± 4, versus 43 ± 3 years, p > 0.05) and sex ratio (M/F, 12/11 versus 14/13, p > 0.05) were similar between Group I and Group II. The mean blood pressure, mean right atrial pressure, pulmonary capillary wedge pressure, and bilateral atrial size were also similar between Group I and Group II (Table 1). Moreover, AERPs and stimulation thresholds in the high right atrium, right posterolateral atrium, and distal coronary sinus were also similar between the two groups. However, AERP dispersion was higher in Group I than in Group II (31 ± 3 versus 22 ± 2 ms, p < 0.05). Furthermore, atrial vulnerability was higher in Group I (6 of 23, 26%) than in Group II (1 of 27, 4%, p < 0.05).

**During SVT.** The tachycardia cycle lengths were similar between Group I and Group II (329 ± 8 versus 336 ± 7 ms, p > 0.05). The mean blood pressure, mean right atrial pressure, pulmonary capillary wedge pressure and bilateral atrial size were similar between Group I and Group II (Table 2). The AERPs and stimulation thresholds in high right atrium, right posterolateral atrium and distal coronary sinus were similar between the two groups. However, AERP dispersion was greater in Group I than in Group II (47 ± 3 versus 31 ± 2 ms, p < 0.0001). Furthermore, atrial vulnerability was significantly higher in Group I (19 of 23, 83%) than in Group II (3 of 27, 11%, p < 0.0001).

**Baseline study versus PSVT.** As compared with those during baseline study, Group I and Group II patients had lower mean systolic blood pressure (Group I, 101 ± 3 vs 95 ± 3 mm Hg, p < 0.05, Group II, 103 ± 3 vs 98 ± 3 mm Hg, p < 0.05), higher mean right atrial pressure (Group I, 3.2 ± 0.6 vs 8.2 ± 0.7 mm Hg, p < 0.01, Group II, 3.4 ± 0.5 vs 7.5 ± 0.8 mm Hg, p < 0.01), higher pulmonary capillary wedge pressure (Group I, 9.9 ± 0.9 vs 21.6 ± 1.2 mm Hg, p < 0.01, Group II, 9.4 ± 0.8 vs 18.5 ± 1.0 mm Hg, p < 0.01), and greater atrial size (left or right) during PSVT (Fig. 1). The high right AERP during baseline study was similar to that during PSVT; however, the AERPs in the right posterolateral atrium and distal coronary sinus were longer during PSVT (Fig. 2). The stimulation thresholds in the high right atrium (Group I, 0.78 ± 0.05 vs 0.66 ± 0.05 mV, p < 0.05, Group II, 0.72 ± 0.04 vs 0.64 ± 0.04 mV, p < 0.05), right posterolateral atrium (Group I, 0.72 ± 0.05 vs 0.63 ± 0.03 mV, p < 0.05, Group II, 0.70 ± 0.04 vs 0.61 ± 0.03 mV, p < 0.05), and distal coronary sinus (Group I, 0.90 ± 0.07 vs 0.75 ± 0.05 mV, p < 0.05, Group II, 0.88 ± 0.05

---

**Table 1. Hemodynamics, Atrial Size, Electrophysiologic Characteristics During Baseline Study**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group I</th>
<th>Group II</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>45 ± 3</td>
<td>43 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>12/11</td>
<td>14/13</td>
<td>NS</td>
</tr>
<tr>
<td>MBP (mm Hg)</td>
<td>101 ± 3</td>
<td>103 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>MRAP (mm Hg)</td>
<td>3.2 ± 0.6</td>
<td>3.4 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>9.9 ± 0.9</td>
<td>9.4 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>RAA (cm²)</td>
<td>11.0 ± 0.5</td>
<td>11.3 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td>LAA (cm²)</td>
<td>10.6 ± 0.5</td>
<td>10.9 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>ERP (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRA</td>
<td>187 ± 3</td>
<td>190 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>RPL</td>
<td>197 ± 4</td>
<td>200 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>DCS</td>
<td>203 ± 5</td>
<td>196 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>Dispersion</td>
<td>31 ± 3</td>
<td>22 ± 2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Threshold (mV)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRA</td>
<td>0.78 ± 0.05</td>
<td>0.72 ± 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>RPL</td>
<td>0.72 ± 0.05</td>
<td>0.70 ± 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>DCS</td>
<td>0.90 ± 0.07</td>
<td>0.88 ± 0.05</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Table 2. Hemodynamics, Atrial Size, Electrophysiologic Characteristics During PSVT**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group I</th>
<th>Group II</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MBP (mm Hg)</td>
<td>95 ± 3</td>
<td>98 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>MRAP (mm Hg)</td>
<td>8.2 ± 0.7</td>
<td>7.5 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>21.6 ± 1.2</td>
<td>18.5 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>RAA (cm²)</td>
<td>13.6 ± 0.5</td>
<td>13.1 ± 0.5</td>
<td>NS</td>
</tr>
<tr>
<td>LAA (cm²)</td>
<td>13.8 ± 0.6</td>
<td>13.2 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>ERP (ms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRA</td>
<td>189 ± 4</td>
<td>194 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>RPL</td>
<td>222 ± 5</td>
<td>213 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>DCS</td>
<td>217 ± 4</td>
<td>209 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>Dispersion</td>
<td>47 ± 3</td>
<td>31 ± 2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Threshold (mV)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HRA</td>
<td>0.66 ± 0.05</td>
<td>0.64 ± 0.04</td>
<td>NS</td>
</tr>
<tr>
<td>RPL</td>
<td>0.63 ± 0.03</td>
<td>0.61 ± 0.03</td>
<td>NS</td>
</tr>
<tr>
<td>DCS</td>
<td>0.75 ± 0.05</td>
<td>0.77 ± 0.04</td>
<td>NS</td>
</tr>
</tbody>
</table>

DCS = distal coronary sinus, ERP = effective refractory period, HRA = high right atrium, LAA/RAA = left/right atrial area, MRAP = mean right atrial pressure, MBP = mean blood pressure, PCWP = pulmonary capillary wedge pressure, PSVT = paroxysmal supraventricular tachycardia.
vs 0.77 ± 0.04 mV, p < 0.05) were lower during PSVT whereas AERP dispersion was greater during PSVT (Fig. 3). Furthermore, atrial vulnerability was higher during PSVT (22 of 50, 44%) than during baseline study (7 of 50, 12%, p < 0.005).

Analysis of all the measured parameters showed that Group I patients had greater difference of atrial size (left and right), AERP in the right posterolateral atrium, and AERP dispersion between baseline study and PSVT than Group II patients (Figs. 1–3).

Parameters of autonomic nervous system. As shown in Figure 4, the BRS was significantly higher in Group I than in Group II (6.3 ± 0.3 versus 3.3 ± 0.2, p < 0.000001); furthermore, the incidence of PAF was significantly higher in patients with a BRS more than 4.5 ms/mm Hg than those with a BRS less than 4.5 ms/mm Hg (78% versus 19%, p < 0.05). Analysis of the 24-h heart rate variability showed that all the parameters were similar between Group I and Group II (Table 3).

Determinants of PAF. Univariate logistic regression analysis showed that higher BRS (>4.5 ms/mm Hg, p = 0.0002, odds ratio = 16.1), AERP dispersion during PSVT (>40 ms, p = 0.0008, odds ratio = 9.7), and increase of right atrial area during PSVT (>2 cm², p = 0.016, odds ratio = 10.7) were significantly correlated with the occurrence of PAF, whereas
higher BRS was associated with the highest odds ratio. The other factors including age, tachycardia cycle length, heart rate variability and other hemodynamic parameters did not correlate with the occurrence of PAF. Furthermore, multivariate analysis could not find any independent factor correlated with the occurrence of PAF.

**Discussion**

**Main findings.** This study demonstrated that AERP dispersion was increased as the atrial pressure was elevated during PSVT and the patients who were associated with PAF had greater AERP dispersion and greater increase of atrial size during PSVT. Moreover, the patients who were associated with PAF had a higher BRS than those without PAF. Univariate analysis showed that BRS was the most powerful determinant of the occurrence of PAF.

**Role of atrial electrophysiologic characteristics.** Atrial refractoriness and dispersion. Electrophysiologic characteristics of atrium have been considered to play an important role in the genesis of AF in patients with PSVT (1,5,6,12). In this study, we evaluated AERPs in three atrial sites, and found no significant difference between patients with and without PAF during baseline study and PSVT. Della Bella et al. (6) also showed that atrial refractoriness was not different between patients with and without a history of PAF. However, Fujimura et al. (5) demonstrated a shorter atrial functional refractory period in patients who had Wolff-Parkinson-White syndrome with PAF.

It has been demonstrated that an increase of atrial pressure would result in a significant prolongation of monophasic action potential duration at 90% repolarization and produce atrial arrhythmias (20). Klein et al. (13) have indicated that high right AERP was increased during PSVT (both in AV reentrant tachycardia and AV nodal reentrant tachycardia) and during pacing at certain AV interval. In this study, an increase of atrial pressure during PSVT did increase AERPs in the right posterolateral atrium and distal coronary sinus; however, high right lateral AERP was not significantly changed during PSVT. Calkins et al. (21) also reported a similar result that high right lateral atrial AERP did not change during different atrial pressure caused by alterations in the AV interval. The electrophysiologic basis for the different changes of effective refractory periods among different atrial sites was not clear. Satoh et al. (14) have demonstrated that atrium was stretched unequally during increasing atrial pressure, which would result in an unequal prolongation of refractoriness between crista terminals and right atrial free wall in canine hearts. In this study, the effects of increasing atrial pressure on atrial refractoriness were different among different atrial sites, which may arise from the effect of heterogeneous atrial tissue characteristics, or an unequal distribution of autonomic innervation in human atrium (22,23). In addition, this study showed that patients with PAF had a greater increase of right posterolateral AERP during PSVT than those without PAF. It is possible that the greater increase of right posterolateral AERP results in greater AERP dispersion in patients with PAF.

Moe et al. (24,25) have demonstrated that dispersion of atrial refractory period was a key factor for initiation of atrial arrhythmia. Misier et al. (26) found that patients with idiopathic AF had more atrial dispersion of refractoriness than patients without AF. Moreover, experimental observations also suggested that AERP dispersion was important in determining the ability to sustain AF, which was significantly affected by vagal activity (27,28). This study showed that AERP dispersion during baseline study was greater in patients with PAF than in those without PAF. Moreover, the greatest AERP dispersion was found during PSVT in patients with PAF. These findings suggested that the significant increase of AERP dispersion may create more heterogeneous conduction, which would favor the induction and maintenance of PAF. Thus, the increase of AERP dispersion during PSVT in this study might suggest that PAF was more likely to originate from degeneration of PSVT rather than de novo as another primary arrhythmia (2).

**Atrial vulnerability.** Several investigators have demonstrated the role of atrial vulnerability in the occurrence of PAF (1,6,12). Della Bella et al. (6) have demonstrated that a single atrial extrastimulus could induce PAF more easily in patients with a history of PAF than in those without a history of PAF. However, these studies did not evaluate the difference of atrial vulnerability between baseline and PSVT. In this study, during baseline study and PSVT, AF was induced more easily in patients with a history of PAF than those without a history of PAF. Furthermore, our results showed that AF was induced more frequently during PSVT than during baseline study. These findings suggested that increased atrial vulnerability during PSVT could be a trigger factor of AF.

**Role of tachycardia cycle length.** Cycle length of AV reentrant tachycardia has been considered to be shorter in patients with a history of PAF than in those without a history of PAF (7). On the contrary, both Della Bella et al. (6) and Fujimura et al. (5) showed that the occurrence of PAF in AV reentrant tachycardia was not related to the cycle length of tachycardia. Hamer et al. (4) also showed that the occurrence of PAF was not related to the rate of PSVT. In this study, the tachycardia cycle length was similar between patients with and without PAF.
Role of atrial size and atrial pressure. Previous study has shown that a critical mass of atrial tissue was necessary to produce AF (17,29). Although this study showed that the atrial size was similar between patients with and without PAF during baseline study and PSVT, the patients with PAF had greater difference of atrial size between baseline study and PSVT than those without PAF. Thus, an acute increase of atrial size in these patients may provide more wavelets for the sustenance of AF. Moreover, PAF could arise from enhancement of vagal activity caused by acute increase of atrial size (30,31).

Role of autonomic nervous system. Autonomic nervous system was proposed to be involved in the genesis of AF. Coumel et al. (16) have demonstrated that some cases of lone AF were associated with periods of enhanced vagal tone. However, the role of autonomic nervous system in the occurrence of PAF in patients with PSVT has not been evaluated.

It has been demonstrated that increase of atrial pressure during tachycardia can activate cardiac mechanoreceptors and lead to an enhancement of vagal activity (30,31). In addition, it is known that tachycardia generally causes a fall of blood pressure at their onset and progressive elevation of blood pressure is followed with increase of vagal activity (32). Furthermore, previous studies have shown that administration of acetylcholine or methylcholine to the atrium could induce AF, and vagal stimulation could induce prolonged episodes of AF (33,34). In this study, patients who had PSVT associated with PAF had a better BRS than patients without PAF. Besides, univariate regression showed that BRS was the most correlated with the occurrence of PAF in patients with PSVT. Although we could not evaluate dynamic vagal activity directly using nerve recording during PSVT, it is possible that patients with a better BRS have higher vagal activity during the tachycardia.

Although this study showed that a BRS test (measures of autonomic reflex modulation) was correlated well with the occurrence of PAF, the study found no difference in the measurement of heart rate variability, including time domain measures and power spectral measures between patients with and without PAF. Analysis of heart rate variability has been used as a noninvasive index of parasympathetic tone and sympathetic activity during the tachycardia. Moreover, univariate regression showed that BRS was the most correlated with the occurrence of PAF in patients with PSVT.

Conclusions. This study demonstrated the importance of abnormal atrial electrophysiology in the genesis of PAF in patients with PSVT; furthermore, a higher BRS was closely related to the occurrence of PAF in PSVT.

References


28. Liu L, Nattel S. Differing sympathetic and vagal effects on atrial fibrillation...


