Clinical and Electrophysiologic Characteristics Before and After Radiofrequency Ablation of Sustained Slow Atrioventricular Nodal Pathway Conduction

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ABSTRACT

OBJECTIVES This study examined the clinical and electrocardiographic characteristics and electrophysiologic determinants of sustained slow pathway conduction (SSPC) during sinus rhythm.

BACKGROUND SSPC during sinus rhythm in presence of dual atrioventricular (AV) nodal pathways has not been thoroughly studied.

METHODS We studied 30 consecutive patients (19 men), whose median age was 31 years (interquartile range: 22 to 48 years); their electrocardiograms revealed 2 different PR intervals during nearly identical sinus rates. We measured the short and long PR intervals and their differences and examined the electrophysiologic determinants of SSPC during slow pathway (SP) ablation in 12 patients.

RESULTS Among the 30 patients, 21 (70%) complained of major symptoms. The short and long PR intervals measured 202±42 ms and 472±110 ms, respectively, and their mean difference measured 270±101 ms. During electrophysiologic studies, dual AV nodal and SSPC were observed in all patients. A markedly prolonged refractory period (593±116 ms) and retrograde conduction block over the fast pathway (FP) were observed over a range of sinus cycle lengths (CLs). Ablation of the SP in 11 patients promoted FP conduction and shortened its effective refractory period from 593±116 ms to 288±90 ms. Over a median follow-up of 2 years (interquartile range: 1 to 3 years), all patients remained asymptomatic and without recurrences of SSPC or AV block.

CONCLUSIONS Two distinct PR intervals during sinus rhythm indicated the presence of dual AV nodal pathways. SSPC was promoted by a markedly impaired bidirectional conduction over the FP at critical sinus CL. SP ablation safely and effectively eliminated SSPC in symptomatic patients. (J Am Coll Cardiol EP 2016;2:367–74) © 2016 by the American College of Cardiology Foundation.
Between 1998 and 2014, we studied 30 consecutive patients (19 men) whose median age was 31 years (interquartile range: 22 to 48 years) and who presented with short and long PR intervals during nearly identical sinus rates, in the absence of structural heart disease and cardioactive medication that influenced AV nodal conduction. All patients underwent detailed cardiac investigations, including history, physical examination, transthoracic echocardiography, 24-h cardiac investigations, including history, physical examination, transthoracic echocardiography, 24-h ambulatory electrocardiogram, exercise treadmill examination, transthoracic echocardiography, 24-h ambulatory electrocardiogram, exercise treadmill testing, and, if needed, isoproterenol infusion test.

Our institutional review board reviewed and approved the protocol of this study, which was conducted in accordance with institutional policies, national legal requirements, and the revised Declaration of Helsinki.

**ELECTROCARDIOGRAPHIC OBSERVATIONS AND MEASUREMENTS.** Stable, short and long PR intervals during sinus rhythm were initially documented by 12-lead electrocardiograms in 13 patients and by 24-h ambulatory electrocardiograms in 17 patients. The measurements included the short and long PR intervals, their difference (Δ), and their corresponding sinus rates after the long PR interval had remained stable for ≥30 s. The appearance and disappearance of the long PR interval on the 24-h ambulatory electrocardiogram were analyzed in 28 patients.

**ELECTROPHYSIOLOGIC STUDIES AND CATHETER ABLATION.** Electrophysiologic studies and radiofrequency catheter ablation of the SP were performed in 12 patients (40%) who complained of prominent symptoms and requested a curative procedure. The indications to proceed with ablation of the SP were firmly ascertained by an unequivocal correlation between the symptoms reported by the patients and the periodic and sudden prolongation of the PR interval, which was confirmed by 24-h ambulatory electrocardiographic monitoring or by remote telemonitoring.

The electrophysiologic studies were performed in the post-absorptive and nonsedated state after a signed informed consent was obtained from each patient. Quadrupolar, 4-F electrode catheters (St. Jude Medical, St. Paul, Minnesota) were introduced percutaneously from the left femoral vein and positioned in the high right atrium, the His bundle region, and at the right ventricular apex. A 6-F decapolar electrode catheter (St. Jude Medical) was advanced from the left subclavian vein into the coronary sinus. Surface electrocardiographic leads I, aVF, and V₅, and all intracardiac electrograms were continuously recorded and stored on a computer-based digital amplifier/recorder (PrukaCardioLab IT System, GE Healthcare, Milwaukee, Wisconsin). Bipolar electrograms were recorded at a bandpass between 30 and 500 Hz. Stimuli, which were 2 ms in duration, were delivered at twice the end-diastolic threshold by a programmable digital stimulator (Bloom DTU 215, Fisher Medical Technologies, Denver, Colorado). Anterograde and retrograde AV nodal conduction was ascertained by incremental pacing and by the extrastimulation technique. The conduction properties and refractory periods, as defined previously (6), were measured, and AV nodal re-entrant supraventricular tachycardia was diagnosed by standard criteria (7).

For the ablation procedures, a 7-F, large-tip, 4-mm long, deflectable, quadrupolar electrode catheter, with 2-mm interelectrode distance (Bard Electrophysiology, Lowell, Massachusetts) was introduced percutaneously from the right femoral vein and advanced to the right atrium. Radiofrequency energy was delivered as a continuous, modulated, sinusoidal waveform at 500 KHz in unipolar mode, between the distal tip of the ablation catheter and a large, posterior skin patch electrode, using a radiofrequency energy generator (Stockert EP Shuttle, Stockert, Freiburg, Germany). The energy was delivered in steps to ablate the SP from the right posterior and inferior aspect of the interatrial septum (8). Radiofrequency energy was delivered at least twice at 50 W, for 50 s, with the temperature limited to 50°C during SSPC, if possible. An ablation attempt was classified as successful when SSPC or anterograde dual AV node conduction was eliminated.

**LONG-TERM FOLLOW-UP.** The 12 patients who underwent SP ablation were followed in our ambulatory department at 1 month after discharge and every 6 months thereafter. They were interviewed, and 12-lead and 24-h ambulatory electrocardiograms were recorded at each visit and analyzed by an investigator.

**STATISTICAL ANALYSIS.** The values are expressed as mean ± SD, median (interquartile range), or count.
(percentage). The short and long PR intervals, and the electrophysiologic measurements made before and after SP ablation were analyzed using SPSS version 18.0 for Windows (IBM, Armonk, New York).

**RESULTS**

**PATIENT CHARACTERISTICS.** Among the 30 patients, 21 (70%) were symptomatic, of whom 14 (47%) presented initially with mild to moderate chest fluttering, chest pressure, or both, and 7 reported similar symptoms during detailed interrogation. These symptoms were closely correlated with intermittent SSPC during ambulatory electrocardiographic monitoring, whereas 9 patients were free from symptoms related to SSPC. All patients denied experiencing episodes of paroxysmal palpitation consistent with AV nodal re-entrant tachycardia. An abnormal electrocardiogram was the cause of referral of 16 (53%) patients. Associated cardiovascular disorders included vasovagal syncope in 5 patients, paroxysmal atrial fibrillation in 2 patients, and hypertension in 2 patients (Table 1).

**ELECTROCARDIOGRAPHIC OBSERVATIONS.** Episodes of SP conduction occurred frequently throughout the day, particularly during sleep, and lasted a few seconds, or was sustained and stable for up to 30 min. The mean, stable, short and long PR intervals measured 202 ± 42 ms and 472 ± 110 ms, respectively. The mean (Δ) between short and long PR intervals was 270 ± 101 ms and was ≥200 ms in 26 of the 30 patients, although the sinus rates corresponding to each PR interval (69 ± 10 beats/min vs. 74 ± 15 beats/min) were similar. PR intervals >400 ms produced the P-on-T phenomenon in 24 patients (80%), and P-on-R was observed in 4 patients with >600-ms PR intervals (Figure 1).

**APPEARANCE AND DISAPPEARANCE OF SSPC DURING AMBULATORY ELECTROCARDIOGRAPHIC MONITORING.** The spontaneous onset during sinus rhythm and disappearance of SSPC in the absence of premature atrial and ventricular complexes or sino-atrial block were observed in 28 patients. In 2 patients, no episode of SSPC was observed during ambulatory electrocardiography or telemetry monitoring. Most of the patients underwent exercise testing without induction of the long PR interval during exercise. Isoproterenol was used in patients unable to exercise or who underwent the head-up tilt table test. In 1 patient, SSPC developed 5 min after the discontinuation of isoproterenol infusion during the head-up tilt table test. A long PR interval developed abruptly in all patients (Figure 1A), preceded by a sudden, albeit minimal, lengthening of the sinus cycle length (CL), whereas the termination of SSPC coincided with a minimal, although sudden, shortening of the sinus CL (Figure 1B). The long PR interval occasionally ended with a blocked P-wave as the sinus CL lengthened further. The blocked P-wave was followed by a few sinus cycles with a short, fixed PR interval associated with a minimally shortened sinus cycle or atypical Wenckebach period, followed by SSPC with a minimal increase in the sinus CL (Figure 1C). This termination was often observed during sinus bradycardia, during sleep or early in the morning. A similar mode of termination could be reproduced by carotid sinus massage during SSPC (Figure 1D).

**ELECTROPHYSIOLOGIC OBSERVATIONS.** The electrophysiologic characteristics of dual AV nodal pathways before and after SP ablation are summarized in Table 2. Anterograde dual AV node conduction and SSPC were elicited by programmed electrical stimulation in all 12 patients. However, AV nodal re-entrant tachycardia, whether typical or atypical, was never induced, including during isoproterenol infusion. Frequent, isolated AV nodal echoes were observed during SSPC in Patient #5 only (Table 2). Intermittent, spontaneous SSPC was easily induced during sinus rhythm in 3 patients and by atrial pacing in 9 patients. The modes of induction were spontaneous deceleration of the sinus rate, atrial premature events, single atrial extrastimulation, and right atrial pacing.

### Table 1: Baseline Characteristics of 30 Patients With Sustained Slow Pathway Conduction

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Count (Percentage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>19 (63%)</td>
</tr>
<tr>
<td>Age, yrs, median</td>
<td>31 (22-48)</td>
</tr>
<tr>
<td>Patients reporting symptoms</td>
<td>21 (70%)</td>
</tr>
<tr>
<td>Associated cardiovascular disorders</td>
<td>9 (30%)</td>
</tr>
<tr>
<td>Vasovagal syncope</td>
<td>5</td>
</tr>
<tr>
<td>Paroxysmal atrial fibrillation</td>
<td>2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>2</td>
</tr>
<tr>
<td>Echocardiographic measurements</td>
<td></td>
</tr>
<tr>
<td>Left ventricular</td>
<td></td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>66 ± 5</td>
</tr>
<tr>
<td>End-diastolic diameter, mm</td>
<td>46 ± 3</td>
</tr>
<tr>
<td>Left atrial anteroposterior diameter, mm</td>
<td>32 ± 5</td>
</tr>
<tr>
<td>Causes of patient referral</td>
<td></td>
</tr>
<tr>
<td>Chest fluttering and discomfort</td>
<td>14 (47%)</td>
</tr>
<tr>
<td>Abnormal electrocardiogram</td>
<td>16 (53%)</td>
</tr>
<tr>
<td>2 different PR intervals</td>
<td>13</td>
</tr>
<tr>
<td>First-degree AV block</td>
<td>10</td>
</tr>
<tr>
<td>Type I second-degree AV block</td>
<td>3</td>
</tr>
<tr>
<td>Transient third-degree AV block</td>
<td>1</td>
</tr>
</tbody>
</table>

Values are n (%) of observations or mean ± SD. AV = atrioventricular.
Programmed ventricular stimulation did not initiate SSPC, but it did terminate SSPC. The AH interval of the FP ranged between 80 and 190 ms, and that of the SP ranged between 380 and 480 ms. FP conduction was depressed with marked prolongation of the anterograde AV block CL, ranging from 480 to 1,000 ms (mean: 735 ± 148 ms) and a sinus CL ranging between 650 and 980 ms. The effective refractory period (ERP) of the FP was also markedly prolonged, from 400 to 710 ms (mean: 593 ± 116 ms). The conduction properties of the SP were also markedly depressed, although the AV block CL (mean: 622 ± 114 ms) and ERP (mean: 425 ± 160 ms) were shorter than those of the FP. Ventricular pacing revealed ventriculoatrial (VA) dissociation in 10 patients, and in 2 patients (Patients #5 and #10 in Table 2), it revealed prolonged VA block CLs of 460 and 750 ms, respectively. Patient #10 had a prolonged VA conduction time via a retrograde SP, which was eliminated by radiofrequency catheter ablation.

**POST-ABLATION ELECTROPHYSIOLOGIC STUDIES.** We used a standard posterior approach for the SP ablations, most of which were ablated at P2 and M1 sites. After successful ablation of the SP, anterograde dual AV nodal conduction was eliminated in 11 patients. Dual AV nodal conduction persisted in a single patient (Patient #2 in Table 2). However, after the ablation procedure, SSPC was no longer inducible with programmed electrical stimulation in any patient. In the 11 patients who underwent successful SP ablation (Figure 2A), the AV block CL of the FP was markedly shortened from 735 ± 148 ms to 438 ± 81 ms. The ERP of the FP was also significantly shortened (Figure 2B), except in 1 patient (Patient #2 in Table 2) in whom dual AV nodal conduction persisted, without
shortening of the ERP of the FP. In 4 patients in whom SSPC was maintained by right atrial pacing, the AH interval over the SP shortened gradually while radiofrequency energy was being delivered to the SP area (Figure 3). In 6 patients who presented with first-degree AV block, the mean PR interval decreased from 257 ± 42 ms before ablation to 233 ± 48 ms after ablation of the SP (Table 2). No AV block was observed from injury to the FP. In-hospital telemetry and 24-h ambulatory electrocardiograms revealed no further SSPC, and no Wenckebach periodicity or transient complete AV block in any patient.

Over a median follow-up of 2 years (interquartile range: 1 to 3 years), no recurrence of SSPC was observed on 24-h ambulatory electrocardiography, and all patients remained free from symptoms attributable to an arrhythmia. A single patient developed asymptomatic, type I second-degree AV block. In patients whose pre-ablation recordings revealed the presence of first, or transient type I

### TABLE 2  Electrophysiologic Characteristics Before and After Slow Pathway Ablation in 12 Patients

<table>
<thead>
<tr>
<th>Patient #</th>
<th>Sinus CL Before</th>
<th>AV Block CL</th>
<th>AV Nodal ERP</th>
<th>Short PR Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>After</td>
<td>FP</td>
<td>SP</td>
<td>Before</td>
</tr>
<tr>
<td>1</td>
<td>650</td>
<td>640</td>
<td>480</td>
<td>440</td>
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<td>2</td>
<td>740</td>
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<td>3</td>
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</tr>
<tr>
<td>4</td>
<td>920</td>
<td>630</td>
<td>800</td>
<td>360</td>
</tr>
<tr>
<td>5</td>
<td>980</td>
<td>650</td>
<td>&gt;980</td>
<td>420</td>
</tr>
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<td>6</td>
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<td>920</td>
<td>780</td>
<td>750</td>
<td>420</td>
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<td>8</td>
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<td>750</td>
<td>550</td>
</tr>
<tr>
<td>9</td>
<td>860</td>
<td>860</td>
<td>&gt;860</td>
<td>500</td>
</tr>
<tr>
<td>10</td>
<td>840</td>
<td>880</td>
<td>700</td>
<td>550</td>
</tr>
<tr>
<td>11</td>
<td>720</td>
<td>650</td>
<td>500</td>
<td>420</td>
</tr>
<tr>
<td>12</td>
<td>780</td>
<td>580</td>
<td>770</td>
<td>360</td>
</tr>
</tbody>
</table>

Numerical values are milliseconds. *460 ms. †240 ms. ‡750 ms. Patient #2: the SP was only modified. Patient #5: the ERP of the FP was not measured because of frequent induction of single AV nodal echoes. Patients #9 and #12: the ERP of the FP was not measured because of incessant onset of SSPC during sinus rhythm. Patient #10: at baseline, VA conduction was exclusively via the SP.

CL = cycle length; ERP = effective refractory period; FP = fast pathway; SP = slow pathway; VABCL = ventriculoatrial block cycle length; VAD = ventriculoatrial dissociation.

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**FIGURE 2** Shortening of the Atrioventricular Block Cycle Length of the Fast Pathway and Effective Refractory Period Between Before and After Slow Pathway Ablation

![Figure 2](image-url)

The atrioventricular (AV) block cycle length (CL) of the fast pathway was markedly shortened (A) and the effective refractory period (ERP) was also significantly shortened after slow pathway ablation (B). Values are mean ± SD.
second- or third-degree AV block, repeat serial 24-h ambulatory electrocardiograms confirmed the elimination of the previously observed AV block.

**DISCUSSION**

Intermittent SSPC during sinus rhythm is a rare electrocardiographic manifestation of dual AV nodal physiology (9). Previous reports have described various electrocardiographic observations of SP conduction associated with minor variations in sinus CL and atrial and ventricular premature complexes (1,10). Detailed analyses of the beginning and end of SSPC suggest that an unusually prolonged refractoriness of the FP, repetitive concealed conduction between the SP and the FP, and autonomic influences on the AV node during sinus rhythm are involved in the periodic variations of the PR interval (10).

**DETERMINANTS OF SSPC.** We identified several electrophysiologic factors that contributed to the development of SSPC during sinus rhythm. The anterograde and retrograde conductive properties of the FP were markedly abnormal, with very long AV block CL and ERP, and with VA dissociation. The anterograde conduction properties of the SP were also very fragile, although the anterograde AV block CL and ERP were shorter than those of the FP. Because these conduction abnormalities persisted over a range of sinus CLs, SSPC was easily initiated and maintained by the block in the FP and a shift of conduction to the SP, which was associated with sudden, although minimal, lengthening of the preceding sinus CL. This was probably due to transiently increased parasympathetic nervous activity (Figure 1A). Sustained SP conduction also disappeared without a blocked P wave when a decrease in vagal sympathetic activity or an increase in sympathetic activity promoted conduction over the FP and accelerated the sinus rate (Figure 1B). This coupled change in the PR interval and sinus CL seemed to be due to a differential sensitivity to vagal activity of a depressed FP, compared with the SP, at a time when the sinus CL was critically near the block CL of the FP. Another mode of termination of SSPC by a blocked P wave can be explained by a surge in vagal activity, which influences SP conduction and causes simultaneous conduction block over both pathways (Figure 1C). This
type of termination was usually observed while pa-

patients were asleep or early in the morning (when vagal tone is increased); this was a mode of termi-

nation we could reproduce by carotid sinus massage
during SSPC (Figure 1D). Therefore, the autonomic

nervous system plays a critical role in the appearance
and disappearance of SSPC besides the unique elect-

rophysiologic characteristics of dual AV nodal phys-

iology in susceptible patients.

EFFECTS OF SP ABLATION ON THE CONDUCTION

PROPERTIES OF THE FP. The abnormal conduction

properties of the FP present before the ablation pro-

cedure might have raised concerns with respect to

the risk of worsening AV conduction by ablation of the SP

(11,12). In this study, catheter ablation of the SP

improved the conduction properties of the FP. In

patients with typical AV nodal re-entrant tachycardia,

previous investigators observed that successful abla-

tion of the SP shortens the refractoriness of the FP

(12–14). They suggested that this might be caused by a

loss of electrotonic interaction between the 2 path-

ways after the SP ablation.

CLINICAL IMPLICATIONS OF SSPC. Reports of

intermittent episodes of SSPC have been rare since

the first report by Schamroth and Perlman in 1973

(9), probably because of unfamiliarity with SSPC and

its complex electrocardiographic interpretation,

rather than its low prevalence. Mild to moderate

symptoms were reported by two-thirds of our pa-

tients. The P-on-R (or on T) phenomenon due to

SSPC causes AV dysynchrony, a sudden increase in

atrial pressure, and a vagally mediated reflex similar
to the pacemaker syndrome (15). Sustained SP

caracteristics during sinus rhythm is not simply of

academic interest, and its electrocardiographic

expression may not be as rare as suspected. As

observed in our patients, SP conduction often sim-

ulates an accelerated junctional rhythm, with long

PR intervals and P waves superimposed on the peak

of the T-wave, typical or atypical Wenckebach block,

and transient AV dissociation due to the emergence

during SSPC. In particular, the progressive shortening of

the AH interval during radiofrequency energy delivery to

the SP, while maintaining SSPC with right atrial pac-
ing (Figure 3), is not well understood. It might be

explained by a progressive attenuation of the aniso-

tropic conduction properties of the SP by the

anatomic modifications of the posterior Koch triangle

(16). A fundamental understanding of dual AV nodal

cardiac properties should be correlated with

anatomy and pathology by various experimental

techniques (17,18). Nevertheless, this study is

contributory, because it is the first to: 1) illustrate the

unique electrophysiologic characteristics of the FP

and SP in patients presenting with SSPC that develops
during sinus rhythm; and 2) treat symptomatic pa-

tients with this disorder.

CONCLUSIONS

SSPC during sinus rhythm was characterized by 2
distinct PR intervals on 12-lead and 24-h ambulatory

electrocardiograms. Its main determinants were:

1) markedly abnormal, bidirectional conduction

properties of the FP; and 2) the influence of the para-

sympathetic system on the AV node. Catheter ablation

of the SP normalized the anterograde conduction

properties of the FP. Ablation of the SP safely and
effectively eliminated SSPC in symptomatic patients.

ACKNOWLEDGMENT The authors thank Rodolphe

Ruffy, MD, for reviewing our manuscript.

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KEY WORDS atrioventricular node, catheter ablation, dual atrioventricular nodal pathway, fast atrioventricular nodal pathway, slow atrioventricular nodal pathway.