Paroxysmal Supraventricular Tachycardia with Unusual Induction*

Concealed Reentry or Automaticity?

Kazumasa Hiejima, M.D.; Shutaro Satake, M.D.; Yuki Sakamoto, M.D.; Fumio Suzuki, M.D.; and Toyomi Sano, M.D.

In a patient with documented paroxysmal supraventricular tachycardia, earlier atrial extrastimuli consistently induced His-ventricle (H-V) block and "atrioventricular junctional" beats, which were always followed by an echo or paroxysmal supraventricular tachycardia. In "atrioventricular junctional" beats, a V wave was preceded by an H deflection with the same interval as that of the sinus beat. As for the underlying mechanism of paroxysmal supraventricular tachycardia, two possibilities were considered: (1) concealed atrioventricular nodal reentry, and (2) "triggered automaticity."

Recent electrophysiologic studies have demonstrated that a common mechanism for the initiation of paroxysmal supraventricular tachycardia in man is atrioventricular nodal reentry. It has been suggested, on the other hand, that "triggered activity" may cause the types of cardiac arrhythmias that usually are attributed to reentry.

This report describes electrophysiologic studies in a woman with a history of paroxysmal supraventricular tachycardia, as the underlying mechanism of which two possibilities were considered: (1) concealed atrioventricular nodal reentry, and (2) triggered automaticity.

METHODS

The patient was a 37-year-old woman with documented paroxysmal supraventricular tachycardia. She had atrial and ventricular septal defects (the former, ostium secundum defect and the latter, Roger's type). The defects were closed on Jan 8, 1974. The electrocardiogram of this patient at rest showed normal P-R intervals and an incomplete right bundle-branch block.

The patient was studied in a supine position and was in a postabsorptive nonsedated state. She was not receiving medications at the time of the examination.

Electrophysiologic studies were performed, utilizing incremental and extrastimulus techniques with simultaneous electrocardiographic and His bundle recordings. Stimuli were delivered by a programmable digital pulse generator with a stimulation isolation unit (San-Ei Sokki 3F-36).

The atrial, His bundle, and ventricular electrograms of the basic driving impulse were represented by A1, H1, and V1, respectively. The atrial, His bundle, and ventricular electrograms of the atrial test impulse were represented by A2, H2, and V2, respectively.

RESULTS

Electrophysiologic study during sinus rhythm showed P-A, atrio-His (A-H), and His-ventricle (H-V) intervals of 20, 90, and 36 msec, respectively. Rapid atrial pacing revealed progressive prolongation of the A-H interval (90 to 150 msec), resulting in Wenckebach's A-H block beyond a rate of 150 impulses per minute. The H-V interval remained constant.

Atrial extrastimuli were coupled at the driving cycle length of 780 msec. As A1-A2 intervals were decreased from 600 to 340 msec, A2-H2 intervals increased from 105 to 156 msec. At an A1-A2 interval of 330 msec, H-V block occurred with an A2-H2 interval of 160 msec. The breadth of the H2 deflection was slightly enlarged. At an A1-A2 interval of 320 msec, H-V block also occurred with an A2-H2 interval of 175 msec. In addition, a beat, which probably originated somewhere in the atrioventricular junction (ie, an "atrioventricular junctional" beat), appeared in early diastole (Fig 1). In this beat, H deflection (H*) preceded the V wave with the interval of 36 msec, identical with that of sinus beats. An H2-H* interval was 450 msec (A2-H*, 625 msec). This beat was followed by two sinus beats; however, the P wave of the first sinus beat was considered to be atrial fusion because the high right atrial potential almost coincided in position with the low right atrial potential which was recorded on the His bundle electrogram. In consequence, retrograde conduction from the "atrioventricular junctional" beat was considered, the time interval between H* and the low right atrial potential (atrial fusion) being measured as 370 msec. At an A1-A2 interval of 300 msec, paroxysmal supraventricular-tachycardia occurred, as seen in Figure 2. Similarly, H-V block occurred when the A2-H2 interval was 195 msec, and the "atrioventricular junctional" beat consistently appeared when the H2-H* interval was 480 msec (A2-H*, 635 msec). The P wave following this beat was of sinus origin because a high right atrial potential preceded a low right atrial potential by the same interval as...
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FIGURE 1. Occurrence of H-V block by atrial test stimulus. Cardiac cycle length is 780 msec. S1, A1, H1, and V1 signify stimulus and atrial, His bundle, and ventricular electrograms, respectively, of basic driving impulse. S2, A2, H2, and V2 signify similar electrograms of atrial test impulse. Numbers in each tracing are in milliseconds. At A1-A2 interval of 320 msec, H-V block occurs in spite of prolongation of A2-H2 interval and is followed by "atrioventricular junctional" beat (H*). Note that H-V interval remains constant. Af indicates atrial fusion. Time lines are 250 msec, and paper speed is 100 mm/sec. Tracings are high right atrial electrogram (HRA), His bundle electrogram (HBE), and electrocardiographic lead V1 and lead 2 (II).

observed in the sinus beat; and in addition, the high right atrial potential and low right atrial potential were identical to those of the sinus beat in configuration. Nevertheless, paroxysmal supraventricular tachycardia

FIGURE 2. Sustained paroxysmal supraventricular tachycardia. At A1-A2 interval of 300 msec, "atrioventricular junctional" beat is followed by paroxysmal supraventricular tachycardia. A' signifies retrogradely depolarized A wave. Tracings are high right atrial electrogram (HRA), His bundle electrogram (HBE), and electrocardiographic lead V1 and lead 2 (II). Numbers in each tracing are in milliseconds. Time lines are 250 msec, and paper speed is 100 mm/sec. S1, A1, H1, and V1 indicate stimulus and atrial, His bundle, and ventricular electrograms, respectively, of basic driving impulse. S2, A2, and H2 are similar electrograms of atrial test impulse. H*, "Atrioventricular junctional" beat. Numbers in tracings are in milliseconds.

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His-ventricle block, “atrioventricular junctional” beat, and echo or paroxysmal supraventricular tachycardia were consistently observed between A\textsubscript{1}-A\textsubscript{2} intervals from 320 to 280 msec (echo zone).

At an A\textsubscript{1}-A\textsubscript{2} interval of 270 msec, the atrial refractory period was reached; and at the shorter A\textsubscript{1}-A\textsubscript{2} intervals, A\textsubscript{2} and the “atrioventricular junctional” beat never appeared. The functional and effective refractory periods of the atrioventricular node were 390 and 280 msec, respectively. The functional and effective refractory periods of the His-Purkinje system were 430 and 410 msec, respectively.

Ventricular stimuli also induced paroxysmal supraventricular tachycardia. Figure 3 shows a representative strip. It seems that various stimuli (S\textsubscript{2}, S\textsubscript{3}, S\textsubscript{4}, and S\textsubscript{5}; Fig 3) reflected a period of atrioventricular dissociation, although the fourth high right atrial potential appeared slightly prematurely. Ventricular reentry was induced by the S\textsubscript{1}, (labelled Vr on Fig 3). The S\textsubscript{3} and S\textsubscript{4} were ineffective. The mechanism for initiation of paroxysmal supraventricular tachycardia by S\textsubscript{1} will be discussed later.

**DISCUSSION**

Unusual features of this case consisted of occurrences of H-V block, apparent atrioventricular junctional beat, and echo or paroxysmal supraventricular tachycardia at the critical A\textsubscript{1}-A\textsubscript{2} intervals. The second H deflection (H\textsuperscript{*}) appearing usually after the H-V block is an unusual phenomenon because of a long interval between H\textsubscript{2} and H\textsuperscript{*}. As to the mechanism of this H deflection, the following possibilities were entertained: (1) premature atrioventricular junctional beat; (2) splitting of the His potential; (3) escaped atrioventricular junctional beat; (4) the second depolarization of the His bundle by conduction of the same impulse (S\textsubscript{1}) through a different route; (5) complete concealed atrioventricular nodal reentry; and (6) “triggered automaticity.” The second and fourth possibilities are very unlikely because of the long intervals between H\textsubscript{2} and H\textsuperscript{*}. Furthermore, these intervals between H\textsubscript{2} and H\textsuperscript{*} are too short for the third possibility, since the escape interval was found to be 880 msec on pacing off.

The consistent appearance of H\textsuperscript{*} following the blocked H\textsubscript{2} is noted, suggesting that H\textsuperscript{*} was coupled with A\textsubscript{2} or H\textsubscript{2}. At the A\textsubscript{1}-A\textsubscript{2} interval of 330 msec, H-V block occurred without the appearance of H\textsuperscript{*}. This finding with further prolongation of A\textsubscript{1}-H\textsubscript{2} intervals (Fig 1) suggests strongly the possibility that complete concealed reentry might have occurred in the atrioventricular node with this critical interval. Namely, S\textsubscript{1} was conducted to the His bundle and depolarized it (H\textsubscript{2}). In addition, the impulse turned at a certain portion of the atrioventricular node, proceeded in a retrograde direction, and reached a connecting bridge. Without discharging the atrium then, it returned antegrade to the His bundle, resulting in H\textsuperscript{*}. There may have been a tissue, probably the atrioventricular node, which had a long effective refractory period for retrograde conduction, and because of this tissue the retrograde conduction, in the case of the A\textsubscript{2}-H\textsubscript{2} interval between 175 and 205 msec, failed to depolarize the atrium, whereas the retrograde conduction with the A\textsubscript{2}-H\textsubscript{2} interval of 240 msec depolarized it (not shown).

On the other hand, Wit and Cranefield\textsuperscript{6} found that in the cardiac muscular fibers of the simian mitral valve, the amplitude of the after-depolarization increased as the length of the stimulus cycle was decreased or increased after premature stimulation and, as a result, can reach threshold to yield nondriven, sustained rhythmic
activity, which they termed "triggered activity." These investigators concluded that such activity might cause the types of arrhythmias that usually were attributed to reentry. According to this concept, junctional automaticity might be triggered at critical H-H* intervals in our case.

The induction of paroxysmal supraventricular tachycardia in Figure 3 might also be explained by two alternative mechanisms. If this was reentry, retrograde block in the fast pathway and interference in the slow pathway might occur during S1, and the following sinus beat could conduct via the fast pathway with an A-H interval of 140 msec and then return via the slow pathway with occurrence of paroxysmal supraventricular tachycardia. If this was a triggered automaticity, S1 could conceal the His bundle, and conduction of the subsequent sinus beat to the His bundle could achieve the critical H-H interval and trigger the tachycardia.

REFERENCES
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Suicidal Hanging*

An Association with the Adult Respiratory Distress Syndrome

Charles M. Fischman, M.D.;** Michael S. Goldstein, M.D.;† and Laurence B. Gardner, M.D.†

*From the Division of General Medicine, University of Miami School of Medicine, and Jackson Memorial Hospital, Miami, Fla.
**Medical Resident.
†Assistant Professor of Medicine.

Reprint requests: Dr. Fischman, Jackson Memorial Hospital, 1611 NW 12th Avenue, Miami 33136

CASE REPORTS

CASE 1

A 22-year-old man was found hanging in his cell at a local jail. Guards claimed he was cut down within one minute after hanging himself and immediately brought to Jackson Memorial Hospital. His rectal temperature was 39.6°C (103.3°F), the pulse rate was 100 beats per minute, blood pressure was 140/80 mm Hg, and the respiratory rate was 40/min. Bilateral coarse rhonchi were heard. The patient was comatose and unresponsive, and was aspirating vomitus. He was intubated and placed on a volume respirator, and penicillin and hydrocortisone were administered. Results of blood gas analysis on a sample of arterial blood drawn while the patient was receiving 100-percent oxygen revealed an arterial-to-venous shunt of 32 percent. Estimated central venous pressure was 12 cm H2O. The findings from a complete blood cell count were normal. A chest roentgenogram showed generalized, fluffy alveolar densities in both pulmonary fields (Fig 1). High-volume ventilation with positive end-expiratory pressure (PEEP) was begun, and therapy with dexamethasone sodium phosphate and clindamycin phosphate was added to the patient’s regimen. He improved over the next 48 hours, and a repeat chest x-ray film showed marked clearing of the infiltrates (Fig 2). After four weeks, the patient evidenced marked pulmonary improvement and had normal

This study of two victims of suicidal hanging describes a previously unknown association between near-fatal hanging and the adult respiratory distress syndrome. We report on the pathophysiologic results of this pulmonary complication and the implications of this finding regarding the treatment of these patients.

In suicidal hanging, death often occurs within minutes. If rescued, most victims later succumb to respiratory failure. The cases in this report suggest that the adult respiratory distress syndrome is a frequent complication of near-fatal hanging and that therapy directed toward this complication may result in improvement.

FIGURE 1. Chest roentgenogram on admission, showing floc­cular nodular infiltrates scattered throughout both pulmonary fields (case 1).