Electrophysiological Diagnosis of Participation of Accessory Pathway in Patients with Paroxysmal Supraventricular Tachycardia

MEICHI ITO, M.D., SUSUMU SHINODA, M.D., MICHIO NAGASHIMA, M.D.
KENZO CHIMORI, M.D., YASUMICHI KINOSHITA, M.D.
AND HIROYUKI SUZUKI, M.D.

Electrophysiological studies were performed on 34 patients whose reentrant circuit of paroxysmal supraventricular tachycardia (PSVT) involved normal atrioventricular (AV) conduction system as the antegrade limb and either overt (25 patients) or concealed (9 patients) accessory AV pathway as the retrograde limb. The diagnosis of this mechanism was made by one or more of the following: 1) eccentric retrograde atrial activation sequence; 2) effect of bundle branch block on ventriculotrial (VA) conduction time; 3) paradoxically premature atrial capture; 4) atrial capture by premature ventricular stimulation when His bundle was refractory during PSVT; 5) no significant prolongation of VA conduction time with retrograde atrial activation sequence identical to that of PSVT during incremental and premature ventricular stimulation; 6) shortening of cycle length with constant H-V and V-A intervals after atropine administration.

The participation of accessory pathway in PSVT circuit should be decided by as many of the above-mentioned findings or procedures as possible for optimal therapy. Electrophysiological studies have led to the recognition that accessory pathway, functionally silent during antegrade conduction, is responsible for some patients with PSVT. If the mechanisms of PSVT were more carefully analyzed, the recognition of PSVT cases involving accessory pathway would increase.

Paroxysmal supraventricular tachycardia (PSVT), the most commonly observed tachyarrhythmia in patients with Wolff-Parkinson-White (WPW) syndrome, is occasionally difficult to treat. Although various mechanisms of PSVT have been clarified in recent years using intracardiac recording and programmed cardiac stimulation techniques, a more complete understanding of the underlying mechanisms by which PSVT develops is essential for optimal therapy.

In patients with WPW syndrome, the most frequent form of PSVT encountered is a reentrant PSVT in which atrioventricular (AV) conduction occurs through the normal AV conduction system and ventriculotrial (VA) conduction occurs via Kent bundle. In some patients with WPW syndrome, however, PSVT does not involve Kent bundle. The absence of electrocardiographic evidence of ventricular preexcitation, on
Fig. 1. Positions of electrode catheters during electrophysiological study. Two bipolar catheters through femoral vein are positioned across the tricuspid valve for His bundle recording and in the low lateral right atrium for recording of low lateral right atrial electrogram. Hexapolar catheter for right atrial and ventricular stimulation and recording of high right atrial electrogram, and coronary sinus bipolar catheter for left atrial stimulation or recording are introduced via antecubital veins.

On the other hand, recent reports have suggested that an accessory pathway functions only in the retrograde direction during PSVT.

The effectiveness of modern antiarrhythmic agents and the progress in the surgical treatments of refractory PSVT have increased the clinical importance of determining whether the accessory pathway participates in the reentrant circuit of PSVT. The purpose of the present paper is to describe the electrophysiological study for determining the participation of accessory pathway in PSVT.

The term "supraventricular", used here to describe the appearance of the QRS morphology on electrocardiogram during PSVT, is a misnomer from a mechanistic viewpoint since the reentrant circuit involves ventricles as well as atria.

MATERIALS AND METHODS

Electrophysiological studies were performed on 72 patients with either documented or suspected PSVT in postabsorptive and nonsedated state. Of the 72 patients, 34 patients (18 males and 16 females) ranging in age from 15 to 68 (mean 40) years were the subjects of the present study. The 34 cases included ten cases of WPW syndrome type A and 15 of type B. The remain-

Fig. 2. WPW syndrome type B. Simultaneous recording of surface electrocardiograms and intracardiac electrograms. In this and subsequent figures: LA=left atrial electrogram; HBE=His bundle electrogram; HRA=high right atrial electrogram; LLRA=low lateral right atrial electrogram; A=low septal right atrial potential; V=ventricular depolarization; S=stimulus. During premature (a) and incremental ventricular pacing (b) retrograde conduction time (S-LA, S-HRA) remains unchanged. Note the sequence of V3-A2-H2 deflections in HBE during ventricular extrastimulation (a). Retrograde atrial activation sequence during PSVT (c) is identical to those of ventricular pacing.

9 cases showed recurrent episodes of PSVT with no electrocardiographic evidence of ventricular preexcitation.

Two to four catheters with two, three, four or six electrodes, 10 mm apart, were introduced percutaneously under local anesthesia into femoral and antecubital veins for intracardiac recording and/or electrical cardiac stimulation (Fig. 1). His bundle and high right atrial electrograms in all patients, low lateral right atrial electrogram in 19, and left atrial electrogram via coronary sinus catheter in 12 and through atrial septal defect in one were recorded as previously described.

PSVT was initiated using the right and/or left atrial extrastimulus technique. Right ventricular pacing was performed in 30 patients. One mg of atropine sulfate was given intravenously in 5 patients. PSVT was induced both before and after the drug administration, and the A-H, H-V and V-A intervals from the onset of the rapid deflection were measured and compared. These intracardiac recordings were made simultaneously with two to five leads of surface electrocar-
Fig. 3. Same case as shown in Fig. 2. a. Paradoxically premature atrial capture is seen during PSVT with right bundle branch block pattern initiated with atrial extrastimulation (S₁-S₂=800, S₁-S₂=300 msec). QRS complex marked with an asterisk is fusion beat. V-A interval (95 msec) following the QRS is shorter than the others (130 msec). Consequently, the atrial cycle length (A-A=285 msec) is shorter than the ventricular cycle length (R-R=300 msec). b. Termination of PSVT following atrial capture by premature ventricular stimulation when His bundle is refractory.

Fig. 4. Retrograde atrial activation sequence during PSVT in patients with left-sided (a), right-sided (b) and septal Kent bundle (c). The earliest retrograde atrial activity is recorded in left atrial electrogram (a), low lateral right atrial electrogram (b) and atrial deflection on His bundle electrogram (c).

Fig. 5. Effect of bundle branch block on retrograde conduction time. V-A intervals prolong following left (a) and right bundle branch block (b) marked with asterisk.

gram on photographic recorder at a paper speed of 100 mm/sec. Care was taken to ground all equipments. All procedures were completed without any complications.

RESULTS

In all 34 patients the reentrant circuit of PSVT involved the normal AV conduction system as the antegrade limb and either the overt (25 patients) or concealed (9 patients) accessory AV pathway as the retrograde limb. The participation of retrograde accessory pathways during PSVT was verified by demonstration of one or more of the following: 1) no significant prolongation of VA conduction time by less than 20 msec with retrograde atrial activation sequence identical to that of PSVT during incremental and/or premature ventricular stimulation in 30 patients (Fig. 2); 2) ability to capture the atria during PSVT with critically timed ventricular extrastimulus delivered when His bundle is refractory in 14 patients (Fig. 3); 3) paradoxically premature atrial capture in 6 patients (Fig. 3); 4) eccentric retrograde atrial activation pattern during PSVT in 13 patients (Fig. 4); 5) increase

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Fig. 6. Effect of atropine on PSVT. Following the administration of atropine, A-H and R-R intervals shorten, while H-V and V-A intervals and retrograde atrial activation sequence remain unchanged.

Fig. 7. Time relation of atrial to ventricular activation during PSVT. V-A/V-V ratio, obtained from His bundle electrogram as illustrated in the upper right, is plotted on the abscissa and shows the differences among mechanisms of PSVT. In this figure and Fig. 8: AVN-K (34) = thirty-four patients with reentrant circuit involving the normal AV conduction system as antegrade pathway and Kent bundle as retrograde pathway; K-K (1) = a case in whom reentrant circuit is composed of two different Kent bundles.

Fig. 8. Heart rate during PSVT.

V-A/V-V ratio was obtained from His bundle electrogram. The ratios were less than 0.5 in all 34 patients. In 48 patients in whom PSVT mechanisms were elucidated, V-A/V-V ratios (Fig. 7) and heart rate (Fig. 8) were evaluated. The PSVT of the group with reentrance utilizing Kent bundle revealed a lower V-A/V-V ratio than the groups with AV nodal reentry (p < 0.001) and atrial focus or reentry groups (p < 0.01), and a higher heart rate than the group with PSVT due to atrial focus or reentry (p < 0.01).

AV conduction curves revealed smooth patterns in all 14 patients, 7 cases with concealed WPW syndrome and 7 with WPW syndrome in which His bundle deflections could be discernible because of the normalization of QRS complexes during electrophysiological studies (Fig. 9).

The configuration of P wave in electrocardiogram lead I during PSVT was evaluated in 43 patients whose lead I was available and the PSVT...
VA conduction time prolongs with incremental or premature ventricular pacing.9,10 This response is similar to AV conduction mode during atrial pacing, indicating the conduction via AV node rather than accessory AV connection. In contrast, no significant change in VA conduction time is seen following ventricular pacing with an increasing rate or prematurity in patients with WPW syndrome (Fig. 2). When the retrograde atrial activation sequence during ventricular pacing is identical to that of PSVT, it is concluded an accessory pathway is utilized for the retrograde route of PSVT circuit.9

In some cases with accessory pathway, however, VA conduction time may increase due to: 1) an increase in conduction delay from the site of stimulation to the recording site of atrial electrograms;11 2) a change of VA conduction pattern through the normal AV conduction system and Kent bundle as the former shows rate-related conduction delay or block;9 3) the presence of multiple Kent bundles with varying locations, conduction times and refractory periods.12

It is also important to note the retrograde His
bundle deflection during ventricular extrastimulation. The demonstration of His bundle deflection following atrial deflection in His bundle electrogram (V$_2$-A$_2$-H$_2$ sequence as shown in Fig. 2a) indicates that His bundle is not utilized during VA conduction, and is diagnostic of the conduction through Kent bundle. On the other hand, AV nodal reentrance is suggested when VA conduction time increases gradually with the appearance of His bundle deflection after ventricular extrastimulation.

2. Premature ventricular depolarization during PSVT

Induction of the premature ventricular depolarization at the progressively shorter coupling interval during PSVT results in atrial capture. When His bundle deflection occurs almost simultaneously with ventricular pacing stimulus and the A-H interval is identical to those of PSVT (Figs. 3b and 11a), it is impossible that the impulse proceeds to atria retrogradely via His bundle because of the refractoriness. From this observation, it must be concluded that the retrograde impulse transmission occurs via an accessory pathway other than the normal AV conduction system.

The interval between atrial activations that interpose ventricular premature depolarization during PSVT may be less than the ventricular cycle length preceding the premature beat (R-R > P-P in Fig. 12a). This phenomenon, referred to as paradoxically premature atrial capture, provides convincing proof that reentry involves Kent bundle. If premature ventricular stimulation is delivered before the anticipated arrival activation from PSVT, a paradoxically premature atrial capture may occur because of shortening of the reentrant circuit of PSVT (Fig. 12b). Hence the closer the stimulation site is to the ventricular end of the Kent bundle the more likely this finding is apt to appear. In a case illustrated in Fig. 11, paradoxically premature atrial capture was not induced by right ventricular apical pacing but by high right ventricular septal stimulation utilizing His bundle catheter. This paradoxical capture is produced more easily when the bundle branch block occurs in the ventricle where the Kent bundle is connected (Figs. 3a and 12c).

We have experienced 72 patients with PSVT. In 16 of 20 patients (80%) without ventricular pacing examinations, the presence or absence of an accessory AV pathway participation in PSVT could not be determined by analyzing only the antegrade conduction. On the other hand, the mechanisms of PSVT could be determined in 44 of 52 patients (85%) undergoing the retrograde
stimulation studies. These results suggest the usefulness of ventricular pacing studies.

3. Eccentric retrograde atrial activation

Simultaneous recording of atrial activities from multiple sites provides an important information. The location of the earliest atrial electrogram is thought to be the site nearest the atrial end of accessory pathway. When the retrograde atrial activation occurs through the AV node during ventricular pacing or PSVT due to AV nodal reentry, the low septal right atrial activity recorded by the His bundle catheter appears earlier than any other atrial activity. Such a retrograde atrial activation sequence is also observed in patients with septal accessory pathway for the retrograde route of the reentrant circuit of PSVT (Fig. 4).

The sequence of retrograde atrial activation is considered eccentric when the earliest atrial activity during PSVT or ventricular pacing does not occur either in His bundle lead or in the proximal coronary sinus close to its ostium. The demonstration of eccentric retrograde atrial activation is characteristic and diagnostic of PSVT incorporating Kent bundle as the retrograde pathway of the reentrant circuit. The earliest retrograde atrial activation occurs in the left atrial electrogram during ventricular pacing and PSVT, in patients with accessory AV connection between the left atrium and left ventricle (Figs. 4 and 6). With an accessory pathway between low lateral right atrium and right ventricle, on the other hand, the activation of the low lateral right atrium precedes low septal right atrial activation during ventricular pacing and PSVT (Fig. 4).

When the atrial activity recorded from His bundle catheter is earlier than any other atrial activity during ventricular pacing and PSVT, James fiber as well as septal Kent bundle is also speculated as the retrograde pathway of PSVT circuit. In such a case the participation of James fiber can be ruled out, if retrograde His bundle potential appears following atrial activity during ventricular extrastimulation with the shortening of coupling interval (Fig. 2a). Since retrograde atrial activation over James fiber is preceded by His bundle activation, atrial deflection in His bundle lead is not followed by His bundle deflection.

These eccentric retrograde atrial activation sequences cannot deny PSVT due to an intraatrial reentry or an ectopic firing of atrial origin. These two mechanisms can be excluded, however, if the eccentric atrial excitation pattern during ventricular pacing is identical to that of PSVT.

Left atrial recording and stimulation are more difficult. The left atrium may be approached...
directly across the atrial septum through an atrial septal defect or patent foramen ovale. In patients without those natural routes the information concerning the site of accessory pathway becomes apparent when a coronary sinus catheter is successfully positioned. In patients with left-sided Kent bundle, this procedure is worthless unless the left atrial electrogrogram is obtained.

4. Bundle branch block during PSVT

Another valuable diagnostic clue may be obtained by analyzing the effect of functional bundle branch block on the VA conduction time during PSVT. When block occurs in the bundle branch running to the ventricle in which an accessory AV pathway inserts, the reentrant impulse must travel down the opposite bundle branch and through the ventricular septum before activating the ventricular end of accessory pathway (Fig. 13C and b). This finding implicates the participation of Kent bundle on the same side as the bundle branch block, but the absence of this finding does not exclude the presence of septal Kent bundle.

The functional bundle branch block does not necessarily occur. The occurrence of left bundle branch block seems to be incidental, especially in patients with left-sided Kent bundle because the functional block is prone to occur in right rather than left bundle branch.

In patients with AV nodal reentrant PSVT, the initiating premature beat is usually associated with marked A-H prolongation, reflecting antegrade conduction via a slow AV nodal pathway (Fig. 14). In patients with reentrance utilizing Kent bundle, on the other hand, the initiating premature beat may be associated with a relatively short AV nodal conduction time. Consequently the H1-H2 interval is shorter than the refractory period of the bundle branch, and causes the bundle branch block.

Denes et al. described that the incidence of functional bundle branch block was higher during the electrically induced PSVT than during spontaneous PSVT, since PSVT episodes are more frequently recorded in the former than the latter. The functional bundle branch block is apt to appear during the initiation of PSVT with atrial extrastimulus technique as shown in Fig. 5. Therefore, one should observe carefully the relation between QRS complexes and VA conduction times in the first several beats following the induction of PSVT.

5. Effect of atropine on PSVT

Recent electrophysiological studies have elucidated that atropine facilitates both antegrade and retrograde AV nodal conduction and exerts no effect on conduction through His-Purkinje system. In five patients, PSVT initiated after atropine showed the shortening of A-H interval with fixed H-V interval compared with PSVT in the control study (Fig. 6). These results coincide with those observed in patients without ventricular preexcitation, indicating that the normal AV conduction system is responsible for the antegrade AV conduction.

During PSVT due to AV nodal reentry, A-H and H-A intervals are approximate measurements of the conduction time through antegrade slow and retrograde fast AV pathways, respectively. Wu et al. described that atropine decreased both A-H and H-A intervals during AV nodal reentrant PSVT. No anatomical fractionation of AV node has been documented. Therefore, it is less likely that PSVT is due to dual AV nodal pathways, when atropine exerts effect on only antegrade limb of PSVT circuit.

On the other hand, VA conduction time over Kent bundle is not altered by autonomic influences or such drugs as propranolol and atropine. We previously reported that the conduction time through Kent bundle remained unaffected not only antegrade but also retrogradely, irrespective of the atropine administration. In our cases with PSVT showing the retrograde conduction via Kent bundle, the effect of atropine was negative on VA conduction time during PSVT (Fig. 6). Thus, the administration of atropine serves as an aid to differentiate the mechanisms of PSVT.

Procaine amide, ajmaline and quinidine are known to suppress the retrograde conduction over Kent bundle as well as the antegrade conduction through His-Purkinje system. In contrast, atropine exerts no effect on these parts of the reentrant circuit of PSVT and facilitates only the antegrade AV nodal conduction. Therefore, the pharmacological effect of atropine in the dosage used is more simple in analyzing the mechanisms of PSVT, in comparison with antiarrhythmic agents mentioned above. Furthermore, this method does not necessitate a special apparatus.

Recently Farré et al. reported that the intravenous injection of 0.5 mg of atropine shortened VA conduction time during PSVT with an accessory pathway as the retrograde limb of PSVT.
circuit. Their result conflicts with ours. In their case, VA conduction over the accessory pathway showed progressive prolongation and is followed by block in the accessory pathway during ventricular pacing at increasing rates. Their case is considered to be an exception.

6. Relation between atrial and ventricular activations during PSVT

The atria and ventricles are requisite components of the reentrant circuit responsible for PSVT using Kent bundle. Therefore, continuation of PSVT in the presence of AV or VA block, regardless of site, rules out the participation of Kent bundle in PSVT. In patients with AV nodal reentrance, PSVT persisted although infranodal block was present (Fig. 14), demonstrating the lack of ventricular participation in this arrhythmia. None of the 34 patients showed the persistence of PSVT in spite of AV block.

Timing of atrial relative to ventricular activation during PSVT is also helpful in analyzing the mechanism of PSVT as shown in Fig. 7. During PSVT with the reentrance utilizing Kent bundle functioning in retrograde manner, ventricular depolarization must be followed by the atrial activation. In our all cases with such a PSVT mechanism, the retrograde atrial activation was found with V-A interval less than one half the V-V interval. In patients with AV nodal reentranse, on the other hand, the onset of atrial activation appeared prior to or at the onset of the ventricular activation (Fig. 14), consequently V-A/V-V ratio approximating to 1.0. This pattern excludes the possibility of concealed Kent bundle.

7. AV conduction curve

It is also important to note the initiation mode of PSVT, with particular attention to the site of conduction delay which appears to be requisite for the development of PSVT. Utilizing His bundle recording and atrial extrastimulus technique, the H₁-H₂ or A₂-H₂ responses are plotted against A₁-A₂ coupling intervals (Fig. 9). Sudden jump of H₁-H₂ or A₂-H₂ interval occurs at a critical A₁-A₂ coupling interval, suggesting failure of a fast AV nodal pathway and antegrade conduction through a slow pathway. Slow pathway conduction allows the fast pathway to recover for retrograde conduction, and sustained AV nodal reentrance results (Fig. 14). These two AV conduction curves are characteristic of "dual pathways" cases.

Total or partial AV nodal bypass fibers with longer effective refractory period than AV node may develop the block at the critical A₁-A₂ interval and be used for the retrograde pathway of the PSVT reentrant circuit. The AV conduction curve may show "dual pathways" pattern. Narula described that the retrograde limb of PSVT circuit was completed by the concealed atrio-His bypass fibers which functioned in only the retrograde direction in a significant number of patients labeled to have PSVT due to dual AV nodal pathways. Gallagher et al also observed a discontinuous curve in patients with PSVT incorporating accessory pathway. Therefore, the finding of discontinuous AV conduction curve in an investigation of PSVT should not be considered pathognomonic of a reentrant mechanism using dual AV nodal pathways.

On the other hand, "reflection" case characterized by continuous AV conduction curve may result from the following situations: 1) two AV nodal pathways with different refractory periods and similar conduction times; 2) fast AV nodal pathway with shorter refractory period for antegrade conduction and slow pathway with longer refractory period for retrograde conduction. Recently the presence of smooth AV conduction curve in patients with PSVT has been interpreted as suggesting the utilization of Kent bundle? Our studies revealed the same result.

In the reentrance utilizing concealed Kent bundle for the retrograde pathway, PSVT is occasionally induced by premature atrial depolarization in the presence of little or no AV conduction delay. The mechanism for this phenomenon is assumed to be 1) rate-related decrease in the retrograde refractory period of Kent bundle, 2) block at the atrial junction with Kent bundle resulting in the absence of the antegrade concealed conduction into atrial end of Kent bundle that may have prevented retrograde conduction through Kent bundle, and/or 3) rate-related decrease in atrial refractoriness that allows impulse to reactivate the atrium retrogradely over Kent bundle. When PSVT was induced without the sudden prolongation of A-H interval, the participation of an accessory pathway in PSVT should be considered.

8. Electrocardiographic findings during PSVT

The rate of PSVT reflects the geometry and the function of tissues involved. According to Wu et al and Farshidi et al the heart rate of PSVT incorporating a concealed accessory path-
way was faster than that of AV nodal reentrant PSVT despite the longer reentrant circuit in the former than the latter. We observed no difference between these two mechanisms (Fig. 8).

P wave configuration in lead I during PSVT provides another helpful diagnostic clue. Negative P wave in lead I is considered diagnostic of left atrial origin of atrial depolarization, whereas other have questioned the reliability of this criterion. As pointed out by previous authors, negative P wave in lead I during PSVT, if discernible, is diagnostic of left-sided Kent bundle as the retrograde pathway of PSVT reentrant circuit (Fig. 10).

9. Concealed WPW syndrome

The typical electrocardiogram of WPW syndrome represents a fusion beat resulting from ventricular depolarization through normal AV conduction system and Kent bundle. The relative contribution of these two sources of activation varies depending on 1) location of Kent bundle, 2) intraatrial conduction time, 3) conduction time through Kent bundle and 4) conduction time via normal AV conduction system. Therefore, the electrocardiographic finding of WPW syndrome may not be present because of the late input to the ventricle via Kent bundle due to 1) distant location of Kent bundle, 2) intraatrial conduction delay, 3) long conduction time over Kent bundle and 4) short conduction time through normal AV conduction system. In some cases atrial stimulation in proximity to the atrial end of Kent bundle may produce preexcitation pattern due to an abolition of intraatrial conduction delay associated with distant pacing site.

In 6 of 9 patients with concealed WPW syndrome the site of accessory pathway could be determined by eccentric retrograde atrial activation sequence and/or appearance of functional bundle branch block. All 6 cases revealed left-sided Kent bundle as the retrograde pathway of PSVT reentrant circuit. In all 12 (Farshidi et al.), all 9 (Wu et al.), 10 of 12 (Sung et al.) and 6 of 9 patients (Gillette) concealed AV accessory pathways were also left-sided. The distance between sanoatrial node and accessory pathway may have a correlation with the appearance of concealed WPW syndrome.

The antegrade refractory period of Kent bundle during atrial extrastimulus technique is generally longer than the retrograde refractory period during ventricular pacing. When this discrepancy is extreme the unidirectional block within Kent bundle may occur even during normal sinus rhythm, and the prolongation of the cardiac cycle length may produce an antegrade conduction through an accessory pathway. Waxman and Wald reported a concealed WPW syndrome patient in whom the application of carotid sinus massage produced a WPW syndrome pattern with a long pause.

Reentry could be localized to several sites including the sinoatrial node, atrium, AV node or a macroreentrant circuit involving the normal AV conduction system in antegrade manner and a concealed Kent bundle in retrograde manner. In the absence of electrocardiographic evidence of ventricular preexcitation, however, most of PSVT has been thought to be due to reentry within AV node. Most reports in literature concerning the AV nodal reentry have not excluded other mechanisms. Inadequate electrophysiological studies may be responsible for exaggerating the reported incidences.

The elucidation of concealed WPW syndrome by recent electrophysiological studies has been attracting attention from the therapeutic viewpoint since these patients can be treated by surgical incision of the accessory pathway. In patients with PSVT and no electrocardiographic evidence of ventricular preexcitation, the incidence of concealed Kent bundle in various reports ranges from 13 to 67%. The difference in the incidence may be explained by the differences in the scale of the series and the period during which data were collected. We made a diagnosis of concealed WPW syndrome in 9 of 39 patients (23%) without ventricular preexcitation. With the diffusion of electrophysiological studies, gradual increase in the reported incidence of concealed WPW syndrome is expected.

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