Temporary, Catheter-Induced Block in Accessory Pathways

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SUMMARY Catheter-induced conduction delay or block in the accessory atrioventricular pathway of four patients with the preexcitation syndrome was observed. Block in the accessory pathway occurred during routine catheter placement and lasted from 90 seconds–14 hours.

All of the pathways were in locations readily accessible to catheter trauma, located in the right anterior septal/paraseptal area in three patients, and in the posterior septum near the orifice of the coronary sinus in the fourth. The location of the accessory pathway was confirmed at surgery by endocardial mapping in three of the four patients. The fourth patient did not undergo surgery.

These findings suggest the need for caution in performing and interpreting electrode catheter studies which are undertaken to document the presence of accessory pathways.

CATHETER-INDUCED CONDUCTION DELAY or block in the bundle of His\(^2\) and right bundle branch\(^3\) have been previously observed during right heart catheterization. The purpose of this study is to extend this observation to the case of accessory atrioventricular (AV) pathways underlying the preexcitation syndrome. Block of the accessory pathway occurred during routine catheter placement in four patients, and lasted from 90 seconds–14 hours.

Methods

The four patients were among those referred to Duke University Medical Center for evaluation of symptomatic tachyarrhythmias related to preexcitation syndromes. The method of study in the Clinical Electrophysiology Laboratory has been previously published.\(^9\)\^-\(^10\) These studies were done using multiple electrode catheters\(^4\) to simultaneously record from several regions of the heart:

1) A specially designed \#7F bipolar electrode catheter was percutaneously introduced via the right femoral vein and advanced to the right atrium for mapping the right atrial activation sequence. The lumen of this catheter accepted a 0.032-inch diameter spring-guide wire or a blunt trocar designed to resemble a Brockenbrough needle.

2) A \#6F quadripolar electrode catheter was per-
cutaneously introduced via the left subclavian or left antecubital vein and advanced to the coronary sinus to record and stimulate the left atrium.

3) A #6F tripolar electrode catheter was percutaneously introduced via the right femoral vein and used to record the bundle of His electrogram.

4) A #6F quadripolar electrode catheter was percutaneously introduced via the right femoral vein and advanced to the right ventricle for recording and stimulation.

All antiarrhythmic therapy was discontinued 48 hours before study. Anticoagulation was achieved during the study with intravenous heparin 100 IU/kg. Electrograms and surface electrocardiographic leads were recorded simultaneously and permanently stored on magnetic tape at 3/4 in/sec. Graphic records were made on-line by a Mingograf-800 ink jet recorder at a paper speed of 200 mm/sec.

Case Reports

Case 1

VR, a 23-year-old black female with a history of paroxysmal tachycardia since the age of 15, has previously been reported in part.11 She had been treated with multiple antiarrhythmic drugs without control of her tachycardia and had required cardioversion several times. The cardiovascular system was normal by physical examination, and an ECG showed normal sinus rhythm and a normal QRS complex. The retrograde sequence of atrial activation initially demonstrated early atrial activity on the His bundle catheter. Premature ventricular depolarizations, introduced after inscription of the His deflection preexcited the atrium, suggested the presence of an accessory ventriculoatrial connection.12 A quadripolar catheter in the right atrium was exchanged for the atrial mapping catheter, and mapping of the right atrium was begun. The tachycardia was terminated by block of retrograde conduction as the modified Brockenbrough electrode catheter with trocar was positioned in the foramen ovale (fig. 1). Multiple attempts were made to initiate reciprocating tachycardia again, and all were unsuccessful. Right ventricular pacing resulted in complete ventriculoatrial dissociation (fig. 2). One hour later, no retrograde conduction had returned, and the catheters in the femoral vein were removed. Fourteen hours after retrograde block was first observed, reciprocating tachycardia spontaneously occurred. Since this patient was a surgical candidate with a life-threatening arrhythmia which was uncontrolled by antiarrhythmic drugs, she was returned to the laboratory where all catheters were introduced again and the study was completed in order to precisely define the location of the accessory pathway before an attempt at surgical division. The sequence of retrograde atrial activation recorded during reciprocating tachycardia demonstrated that the accessory pathway was located in the septum. On the 14th day of hospitalization, the patient was taken to the operating room, where epicardial and endocardial mapping were performed. The maps made in the operating room confirmed the septal location of the accessory pathway.11 The accessory pathway was permanently ablated by cryosurgery. The patient has been free of arrhythmias for 22 months.

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**Figure 1.** Catheter-induced trauma to an accessory pathway during reciprocating tachycardia. The tracings from the top down are standard ECG leads V1 and I, a right atrial electrogram recorded on the modified Brockenbrough mapping catheter positioned near the foramen ovale (RA-BROCK), and bipolar electrograms recorded from the right ventricle (RV), the region of the His bundle (HBE), and the coronary sinus (CS). A = atrium; H = His bundle; V = ventricle. During reciprocating tachycardia at a cycle length of 265 msec, manipulation of the Brockenbrough electrode catheter near the foramen ovale terminated the tachycardia (arrow) with retrograde block. See text for discussion.
Comment, Case 1

This patient had an accessory pathway which conducted exclusively in the retrograde direction, a condition found in approximately 40% of patients referred to this institution for paroxysmal supraventricular tachycardia in whom a delta wave is neither present in sinus rhythm nor inducible by atrial pacing. Atrial preexcitation by premature ventricular depolarizations, induced when the His bundle was refractory, confirmed the participation of this accessory pathway in the tachycardia. Complete retrograde conduction block was observed when the mapping catheter and trocar were positioned in the foramen ovale. That complete block was present is shown by the ventriculoatrial dissociation which occurred during ventricular pacing (fig. 2) at a cycle length of 300 msec. Since 300 msec was longer than the cycle length of the tachycardia (265 msec), retrograde conduction should have occurred. The study was continued 1 hour after block was first observed, and conduction did not resume. Since tachycardia was next recorded 14 hours later, the exact time required for return of conduction was between 1–14 hours. At operation, the location of an accessory pathway in the atrial septum just anterior to the bundle of His was confirmed, a site easily traumatized by the mapping procedure.

Case 2

JJ was a 25-year-old white female with a history of reciprocating tachycardia for 12 years, premature ventricular beats for five years and recent onset of atrial fibrillation on two occasions one year before admission. A routine ECG demonstrated preexcitation.

On the second hospital day, an electrophysiologic study was performed. During reciprocating tachycardia, the earliest area of atrial activation was found near the orifice of the coronary sinus and was associated with a ventriculoatrial conduction time in this area of 80 msec. Sudden prolongation of the ventriculoatrial conduction time was observed each time the atrial mapping catheter was rotated near this location. Figure 3 is a continuous strip recorded 1 minute, 46 seconds after a sudden prolongation had occurred. The strip, recorded over 8 seconds, shows a gradual decrease of the conduction time toward its original value (80 msec). Figure 4 demonstrates an example of prolongation of the ventriculoatrial conduction time induced by manipulation of the catheter. In this example, the initial “control” ventriculoatrial conduction time was 110 msec (in contrast to the 80 msec control value shown in fig. 3) due to incomplete recovery of the accessory pathway from catheter manipulation occurring less than 1 minute, previously.

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**Figure 2.** Demonstration of ventriculoatrial dissociation during ventricular pacing following catheter trauma of an accessory pathway. Recording and legend are identical to figure 1. Following the event shown in figure 1, right ventricular pacing at a cycle length of 300 msec demonstrated complete ventriculoatrial dissociation. RA-BROCK = electrogram recorded on the modified Brockenbrough catheter positioned near the foramen ovale; RV = right ventricle; HBE = His bundle; CS = coronary sinus; S = pacing artifact; A = atrium.

**Figure 4.** Catheter-induced trauma to the accessory pathway during reciprocating tachycardia. The recordings and legends are identical to those shown in figure 3. Note that the relative sequence of atrial activation is the same as in figure 3. The ventriculoatrial (VA) conduction time is initially 110 msec (compared to the patient’s usual VA interval of 80 msec as shown in fig. 3) due to incomplete recovery from an episode of catheter trauma immediately preceding this recording. See text for discussion. RV = right ventricle; SEPTAL RA = coronary sinus orifice; HBE = His Bundle; PROX. CS = proximal coronary sinus; DIST. CS = distal coronary sinus.
Recovery of an accessory pathway from catheter trauma during persistent reciprocating tachycardia. Recordings from the top down are standard ECG lead V1, with bipolar recordings from the right ventricle (RV), the right atrial septum near the coronary sinus orifice (SEPTAL RA), the region of the His bundle (HBE), the proximal (PROX. CS) and distal (DIST. CS) coronary sinus, and time lines (10 msec). The ventriculoatrial (VA) conduction time is indicated in milliseconds as measured from the onset of ventricular activation to the most rapid atrial deflection recorded from the proximal coronary sinus. This continuous 8-second recording illustrates recovery of accessory pathway conduction from first degree block. The VA conduction time decreases from 165 to 80 msec. The VA conduction time of 80 msec persisted until further manipulation of the coronary sinus catheter.
Alternate explanations for the variations in the ventriculoatrial conduction intervals observed in this case included the possibility that two accessory pathways with different conduction times were present or that the patient had dual AV nodal pathways in addition to the accessory AV pathway.

If two accessory AV pathways were present, catheter manipulation could have caused complete block of the more rapidly conducting pathway and "unmasked" the more slowly conducting pathway. However, the gradual return of conduction to control values as shown in figure 3 would make this explanation unlikely. Moreover, if two accessory AV pathways were present, they would necessarily have been almost contiguous because the sequence of atrial activation did not change as the conduction time changed.

The presence of dual AV nodal pathways could furnish another explanation for the observed phenomenon. Thus, before the catheter manipulation occurred, antegrade conduction could have occurred over the slow AV nodal pathway and retrograde conduction over the accessory pathway. During catheter manipulation, complete block could have occurred in the accessory pathway resulting in a change in retrograde conduction from the accessory pathway to the fast (but possibly diseased) AV nodal pathway explaining the sudden jump of ventriculoatrial conduction from 110-165 msec. However, antegrade refractory periods of the AV node (A1>A2 vs H1H2) performed at two cycle lengths (i.e., 600 and 500 msec) failed to demonstrate discontinuous curves before or after surgery. Furthermore, complete ventriculoatrial dissociation was observed during right ventricular pacing performed during the postoperative electrophysiologic restudy.

Comment, Case 2

This patient had an accessory pathway which conducted in both the antegrade and retrograde direction. Manipulation of the atrial mapping catheter with its stiff trocar near the orifice of the coronary sinus apparently caused first degree retrograde conduction block, as shown by the sudden prolongation of the ventriculoatrial conduction time. The effect that this event had on antegrade conduction could not be assessed because the tachycardia was not terminated and sinus rhythm was not observed. At operation, the location of the accessory pathway in the posterior interatrial septum in the region of the coronary sinus was confirmed. The accessory pathway was successfully divided without injury to the normal conduction system and the patient has remained free of arrhythmia for a period of 22 months follow-up.

Case 3

RH was a 4-year-old white male with a three-month history of paroxysmal tachycardia. A routine ECG confirmed the presence of preexcitation. During electrophysiologic study, the delta wave suddenly dis- appeared during manipulation of the His bundle catheter and a normal QRS complex was observed. Despite the fact that tachycardia had been induced at the initiation of the study with premature atrial beats, tachycardia could no longer be induced. Complete ventriculoatrial dissociation was documented during pacing of the right ventricle as well as the left ventricle (paced via a catheter advanced to the distal coronary sinus). A delta wave was not demonstrated by pacing the right atrium, the coronary sinus, or during induced atrial fibrillation. The catheters were removed and the patient was returned to the ward. Several hours later, the delta wave was again recorded and the tachycardia resumed.

Comment, Case 3

This patient had an accessory pathway which conducted in both the antegrade and retrograde direction. The exact location of the accessory pathway cannot be stated because endocardial mapping was not performed. An analysis of the delta wave based on our previous cases would suggest that the accessory pathway was located in the right anterior paraseptal region, a location which could have been easily injured during placement of the tripolar catheter across the tricuspid valve.

Case 4

JK was a 28-year-old white male with a history of paroxysmal tachycardia from the age of 13. Preexcitation was first documented at the age of 18 (fig. 5), but ECGs recorded after the age of 23 failed to show a delta wave. Reciprocating tachycardias persisted, however, and he was referred for evaluation.

During electrophysiologic study, considerable difficulties were encountered during attempts to catheterize the coronary sinus. After much manipulation, a catheter was successfully passed into the coronary sinus. The remainder of the catheters were then easily positioned in the routine manner. During ventricular pacing, 2:1 retrograde block was unexpectedly found at cycle lengths near sinus cycle, and shorter cycle lengths of ventricular pacing demonstrated complete ventriculoatrial dissociation (fig. 6). Antegrade pacing and refractory period determinations failed to initiate tachycardia or to elicit preexcitation. Atrial flutter was induced and again failed to demonstrate preexcitation.

Three days later, the electrophysiologic study was repeated in order to locate the pathway before surgery. An electrode catheter was cautiously advanced into the right ventricle and the atrial activity was recorded using an esophageal lead. Retrograde conduction at this time was demonstrated during right ventricular pacing to cycle lengths as short as 260 msec. Figure 7 demonstrates intact ventriculoatrial conduction at a cycle length of 320 msec. Catheters were advanced to their usual positions in the right atrium and across the tricuspid valve. The sequence of retrograde atrial activation determined during right
ventricular pacing demonstrated initiation of activation in the septal area. Reciprocating tachycardia was induced during which introduction of premature ventricular depolarizations in the right ventricle confirmed the presence of an accessory pathway (fig. 8).

The endocardial map and the previously recorded ECG with preexcitation were both consistent with an accessory pathway located in the right anterior paraseptal region. This location was subsequently verified by epicardial and endocardial mapping at the

![ECG images](chart.png)

**Figure 5.** ECG of case 4 demonstrating antegrade preexcitation four years before electrophysiologic study. The 12-lead ECG demonstrates positive delta waves in I-III, aV_{1}, and V_{4}-V_{6}; negative delta waves are present in aV_{6} and V_{1}-V_{3}.

![Pacing tracing](chart2.png)

**Figure 6.** Complete ventriculoatrial dissociation during right ventricular pacing following catheter trauma in case 4. The time interval measured from the right ventricular stimulus to the most rapid atrial deflection recorded on the high right atrial catheter (S-A) is indicated in milliseconds on the right atrial (RA) tracing. This tracing was recorded during ventricular pacing after difficulty had been encountered manipulating the coronary sinus catheter into the coronary sinus. Ventriculoatrial dissociation is evident. Atrial activation proceeds from right atrium to left atrium (the latter recorded via the coronary sinus). The arrow indicates a fusion beat. RV = right ventricle. Prox. CS = proximal coronary sinus; Dist. CS = distal coronary sinus.
time of successful division of this accessory pathway at surgery. He has remained free of arrhythmia in five months of followup.

Comment, Case 4

The accessory pathway in this patient had bidirectional conduction properties until age 23, at which time antegrade conduction was no longer apparent. Acquired unidirectional block in an accessory pathway has been previously reported. Retrograde block was induced by catheter manipulation and probably occurred during a prolonged attempt to catheterize the coronary sinus. Partial conduction returned within 2 hours, and intact retrograde conduction returned three days later when the study was repeated. Endocardial mapping data suggested earliest activation in the right anterior septal location consistent with the previously recorded antegrade preexcitation, and this location was confirmed at surgery.

Figure 9 is a composite illustration locating the accessory pathways found susceptible to catheter trauma.

Discussion

Recognition of the presence of an accessory pathway as the underlying mechanism of recurrent tachycardia has important implications in determining optimal medical, pacemaker, or surgical therapy. The use of electrode catheter techniques to diagnose the presence of an accessory pathway has recently assumed even more importance with the recognition that accessory pathways may be the underlying mechanism of reciprocating tachycardia, even in the absence of documented antegrade preexcitation. Our finding that electrode catheter studies may obscure the demonstration of accessory pathways suggests a need for caution in the execution and interpretation of electrophysiologic studies.

Case 3 demonstrated that third degree (complete) antegrade block of conduction in an accessory pathway can be caused by catheter manipulation. First degree retrograde block was observed in case 2, while third degree retrograde block was shown in cases 1, 3 and 4. In all patients, block was temporary and conduction resumed minutes or hours later.

Cases 1 and 4 were patients in whom antegrade conduction block was already present at the time of study. The explanation for this unique physiology is unknown but an experimental model has been proposed. It is possible that accessory pathways with exclusive retrograde conduction may be more easily blocked by trauma, because of geometric considerations inherent in the presence of antegrade block. It is of interest that two of the four patients reported had accessory pathways capable of only retrograde conduction.

The potential to traumatize accessory pathways is related to the type of catheter being used and the location of the accessory pathway. The atrial mapping catheter with its trocar in position presents a rigid object to the endocardial surface. This type of catheter accounted for two of four cases. Theoretically, any accessory pathway which traverses the tricuspid anulus (septal or parietal) may be traumatized by a catheter introduced into the right atrium; in our series, only pathways in the vicinity of the septum were traumatized.

Based on our experience, some recommendations appear justified. When a delta wave is present in sinus rhythm, it should be constantly observed during any catheter manipulation. If no delta wave is present, but a concealed accessory pathway is suspected, we
FIGURE 8. Demonstration of atrial preexcitation during reciprocating tachycardia after return of function in the accessory pathway. Recordings from the top down are standard ECG leads V₁, I, and bipolar electrograms from the right ventricle (RV), the lateral right atrium (RA), the region of the His bundle (HBE), and the esophagus. The atrial electrogram on the HBE recording occurs at the end of the ventricular electrogram. The time interval between successive His deflections remains constant at 305 msec. After the third beat of tachycardia, a stimulus (indicated by the arrow) is introduced into the right ventricle simultaneous with the activation of the His deflection. Despite the fact that the His deflection is not disturbed, the next atrial response occurs prematurely, shortening the atrial cycle length to 280 msec. See text for discussion.

FIGURE 9. Approximate location of accessory pathways traumatized during catheter electrode study. A schematic representation of the heart in cross section is shown at the level of the atrioventricular rings. The numerals refer to the cases described. Cases 1, 3 and 4 were situated in the anterior septal/paraseptal region just anterior to the region of the recorded His deflection, while case 2 was located more posteriorly near the orifice of the coronary sinus.

attempt to place a pacing catheter in the right ventricle with a single pass; the remaining catheters are then positioned during ventricular pacing while monitoring the ventriculotriatral interval recorded by an esophageal lead or an atrial recording from the high right atrium. Hopefully, these safeguards will lead to prompt recognition of catheter trauma during electrophysiologic study of manifest or concealed preexcitation syndromes and will help to limit the extent of such trauma.

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Electrophysiologic Demonstration of Concealed Conduction in the Human Atrium

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SUMMARY In the course of electrophysiologic evaluation of six patients with sick sinus syndrome, two patients with chronic conduction system disease and four patients with paroxysmal supraventricular tachycardia or atrial flutter-fibrillation, the phenomenon of concealed conduction within the atrium (At) was repeatedly observed. One pair of intracardiac electrodes was used to deliver electrical stimulus (St) to the high right At and two additional pairs of electrodes were utilized to record high right and low septal right atrial electrograms. The interelectrode distances were 10 mm apart. In all 12 patients, high right atrial capture could be accomplished at a pacing rate of $\geq 200$ beats/min. Concealed intra-atrial conduction was evident when the intra-atrial conduction time of the propagated St during 2:1 St-At block was more than 40 msec longer than that during 1:1 St-At conduction at half the St frequency. This indicated that the nonpropagated St during 2:1 St-At block partially penetrated the At and in turn, delayed conduction of the subsequently propagated St. Further observations revealed that a gradual increase in the St frequency resulted in a progressive prolongation of the intra-atrial conduction time and a shifting of 2:1 to 3:1 St-At block (alternating Wenckebach periodicity in the At) in five patients. These findings clearly demonstrate the occurrence of concealed conduction in the human At.

CONCEALED CONDUCTION is defined as the effect of a non-propagated impulse upon conduction of a subsequently propagated impulse. This electrophysiologic phenomenon can be validated in animal experiments with programmed electrical stimulation as well as microelectrode recordings of action potentials from various levels of the conduction system.

In the human heart, Langendorf et al. and Carleton and Graettinger have experimentally induced concealed atrioventricular (AV) conduction with atrial stimulation. Damato and coworkers have subsequently used electrode catheter recordings of intracardiac electrograms to localize the sites of concealment. As observed in animal experiments, concealed conduction in the human heart usually occurs in the AV node or His-Purkinje system or both and to our knowledge, has not been previously demonstrated in the human atrium. In the present study, we have repeatedly observed its occurrence in the human atrium, using the techniques of intra-atrial stimulation and recordings.
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