

Intracardiac Recording by Catheter Electrode of Accessory Pathway Depolarization

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At electrophysiologic study in a patient with the Wolff-Parkinson-White syndrome, intracardiac catheter recordings demonstrated a deflection that occurred 30 ms before ventricular activation. The rapid deflection was present during ventricular preexcitation but not during normal atrioventricular conduction. All QRS complexes

were preexcited to varying degrees during atrial fibrillation, yet the deflection consistently preceded ventricular activation by 30 ms. This deflection most likely represents the rare recording of a Kent bundle depolarization with an intracardiac electrode catheter.

In patients with the Wolff-Parkinson-White syndrome, ventricular preexcitation results from fusion of atrial impulses activating the ventricle over the accessory pathway and over the normal atrioventricular (AV) conduction system (1,2). Because the accessory pathway represents a discrete anatomic structure connecting atrium and ventricle (3,4), it seems reasonable to assume that if an electrode is positioned very close to the Kent bundle, one might record electrical activity from the accessory pathway. Indeed, Kent bundle depolarization has been recorded during epicardial mapping at surgery (5,6). If depolarization from these accessory fibers can be recorded by an appropriately placed hand-held electrode at surgery, it should be possible to accomplish the same result in selected patients using a catheter electrode. The purpose of this study is to report the recording of Kent bundle depolarization with an intracardiac electrode catheter at the time of electrophysiologic examination.

Electrophysiologic Study

The patient is a 30 year old man referred for evaluation of recurrent syncope. The 12 lead scalar electrocardiogram (Fig. 1)

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shows ventricular preexcitation and the initial 40 ms QRS vector suggests that the accessory pathway is located in the right anterior paraseptal area (2). Electrode catheters were inserted percutaneously in the femoral and subclavian veins and positioned at various areas of the heart. Electrophysiologic examination was performed as previously reported (7). The electrogram recorded during sinus rhythm with the catheter positioned at the His bundle area demonstrates two distinct deflections inscribed before the onset of the broad ventricular deflection (Fig. 2). The first rapid deflection (A) is due to local atrial activation and the second deflection (AP) most likely represents accessory pathway depolarization. In the His bundle tracing, activation time from atrium to accessory pathway is 30 ms; from the accessory pathway to ventricular (V) activation the time is 30 ms. Both of these intervals remained constant during incremental atrial pacing and atrial premature stimulation. The accessory pathway deflection was always present before each preexcited ventricular complex throughout the study. Note that ventricular activation in the His bundle lead occurs almost simultaneously with the onset of the delta wave, a finding consistent with conduction over a right anterior paraseptal pathway.

Other possible causes of the Kent bundle electrogram. Three other possibilities that could explain the sharp spike between the atrial and ventricular deflections are the recording of atrial, ventricular or His bundle activation. The effective refractory period of the Kent bundle was less than or equal to the effective refractory period of the AV node. However, when a premature atrial complex resulted in no conduction over the accessory pathway, there was also absence of the Kent bundle electrogram (Fig. 3). Additionally, during atrial fibrillation (see later), the rapid deflection remained unchanged. Thus, it is very unlikely that the deflection occurring 30 ms after the onset of the atrial deflection was a second or split atrial depolarization (Fig. 2). Ventricular activation is an unlikely explanation because the spike was recorded 30 ms before the onset of ventricular activation recorded in the surface leads.

Two findings militate against the explanation that the Kent bundle electrogram was a His depolarization with rapid activation

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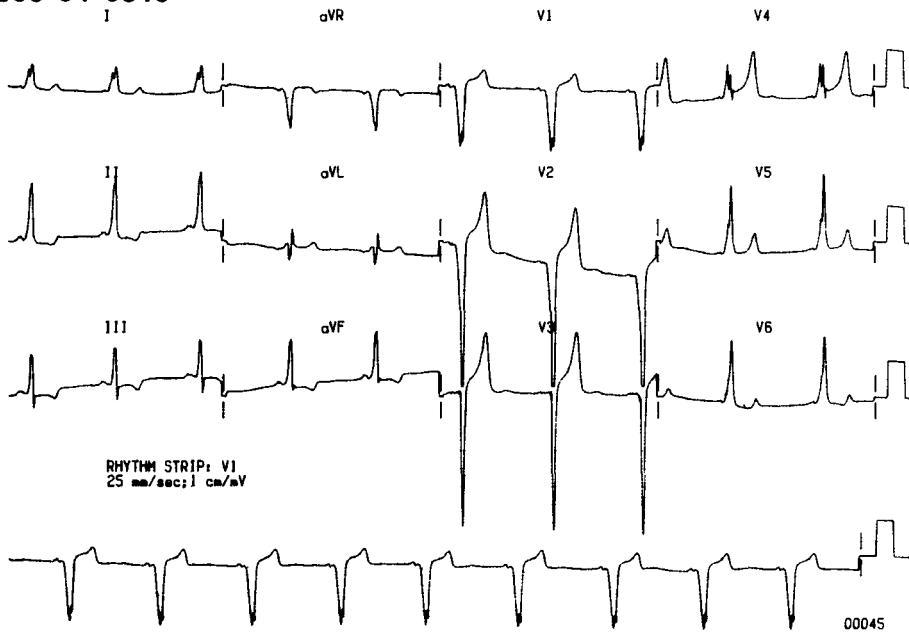
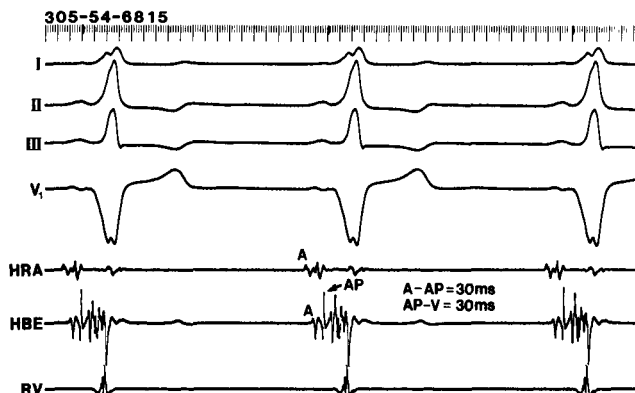


Figure 1. Twelve lead electrocardiogram during preexcitation.

time (30 ms) from the atrium to the His bundle. First, inexplicably at one moment in the study (possibly owing to catheter trauma of the accessory pathway [8]), there was a sudden loss of preexcitation during sinus rhythm and the resultant AH interval was 90 ms and the HV interval was 45 ms (Fig. 4). The rapid deflection that always occurred 30 ms after the local atrial depolarization during preexcitation disappeared. Second, during atrial fibrillation all QRS complexes were preexcited, but to varying degrees, and the rapid depolarization consistently preceded each ventricular electrogram by 30 ms (Fig. 5). Because the HV interval would vary as the degree of ventricular preexcitation varied, but the interval between activation of accessory pathway and ventricle would not, this suggests that the electrogram preceding the ventricular depolarization was related to depolarization of the accessory pathway and not the His bundle.

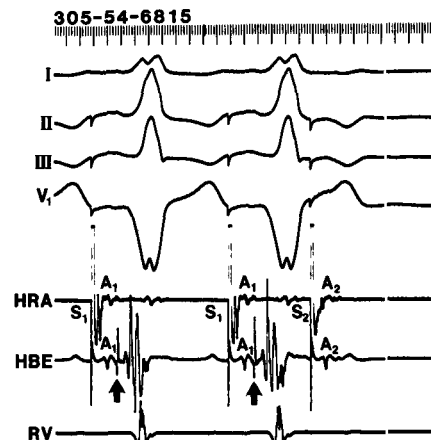
Figure 2. Accessory pathway electrogram recorded during normal sinus rhythm. The top four tracings are electrocardiographic leads I, II, III and V₁ and the bottom three tracings are intracardiac electrograms recorded from the high right atrium (HRA), His bundle area (HBE) and right ventricle (RV). A = atrial deflection; AP = accessory pathway electrogram, time lines = 50 ms for each major division. See text for details.



Discussion

Requisites for diagnosis of depolarization of accessory pathway. This patient had a rapid deflection recorded on the His bundle electrogram that was positioned temporally between the atrial and ventricular electrograms during ventricular preexcitation. If this deflection was due to depolarization of the accessory pathway, the following requisites should be fulfilled: 1) the electrogram must be present during preexcitation but not during normal AV conduction; 2) the electrogram must be recorded only on the electrode close to the presumed site of the pathway; 3) the activation time

Figure 3. Premature atrial stimulation. Atrial pacing (A₁) at 500 ms is associated with an accessory pathway depolarization (arrow) that occurs 30 ms before ventricular activation. A premature atrial complex (A₂) does not conduct over the accessory pathway or normal conduction system, and the accessory pathway electrogram is not present. S = stimulus, other abbreviations as before.



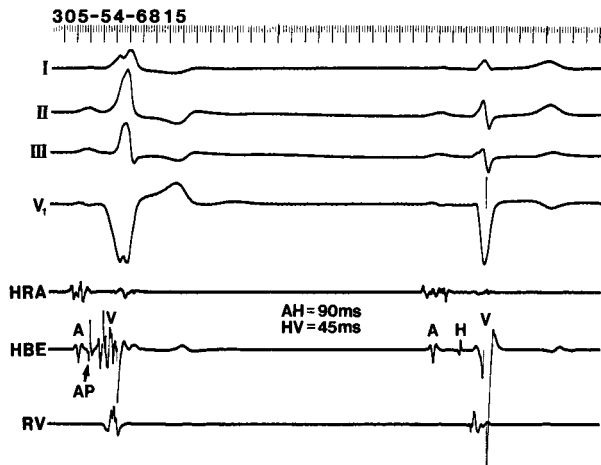
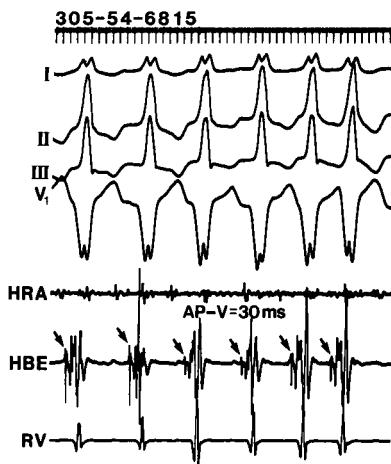


Figure 4. Preexcitation (left) and normal (right) atrioventricular conduction. The accessory pathway deflection is absent during normal atrioventricular conduction and the AH interval is 90 ms, 60 ms longer than the atrium to accessory pathway activation time (see Fig. 2).

from the accessory pathway to the ventricle must be consistent with published data (5) from direct measurements made in the operating room (38 ms); 4) accessory pathway to ventricular activation time should remain fixed during incremental atrial pacing and premature atrial stimulation; and 5) the activation time from the accessory pathway to the ventricle must remain the same during atrial fibrillation. The last point is very important because one would not expect a constant relation between His bundle depolarization and QRS complexes preexcited to varying degrees during atrial fibrillation. This condition also excludes the presence of an atrio-Hisian pathway (9) with a short AH interval.

Figure 5. Preexcitation during atrial fibrillation. Note that an accessory pathway deflection occurs 30 ms before each ventricular depolarization.



The identification of a distinct accessory pathway electrogram at the same time that the His bundle electrogram was recorded would have been further proof that the first of the two electrograms was related to Kent bundle depolarization. The AH interval was 90 ms and consequently the His deflection was temporally located in, and obscured by, the broad ventricular electrogram (Fig. 4). Although a rapid deflection was recorded in the His bundle ventricular electrogram 90 ms after local atrial depolarization, the exact origin of this deflection cannot be determined.

All of the preceding requisites are fulfilled in this patient. Accessory pathway depolarization is rarely recorded in the clinical electrophysiology laboratory, partly because the pathway must be located very close to the recording catheter. Importantly, in this study the electrode catheter that recorded accessory pathway depolarization was positioned close to the Kent bundle. The accessory pathway was in the right anterior paraseptal area and the Kent bundle depolarization was recorded only on the His bundle lead. Further, the sudden loss of preexcitation (Fig. 4) was most likely caused by catheter trauma, indirectly supporting the proximity of the His bundle catheter to the accessory pathway. It is worthwhile to note that the right anterior septal/paraseptal area was the location of three of the four accessory pathways that had temporary catheter-induced block observed in a previous report (8).

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