Evaluation of intracardiac recordings in diagnosis of impulse formation and concealed conduction in atrioventricular nodal bypass tracts

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Invasive electrophysiological studies were performed in 2 symptomatic patients with recurrent arrhythmias in which impulse formation presumably occurred within atrioventricular nodal bypass tracts. Case 1 had ectopic beats arising within, or close to, the upper end of a left-sided atrioventricular nodal bypass tract of the type described by Brechenmacher. In addition, this conduction was 'concealed' during sinus rhythm and right atrial pacing because the relatively prolonged right-to-left atrial conduction time allowed right atrial impulses to reach the His bundle via the atrioventricular node before they could do so through the atrioventricular nodal bypass tract. Case 2 had ectopic beats arising in a right-sided atrioventricular nodal bypass tract which did not conduct in either forward or retrograde directions, its presence being detected only when initiating impulses. However, it could not be determined whether this tract was an 'abnormal' atrio-His connection or a 'normal' transitional (atrio-atrioventricular nodal) tract. Though intracardiac studies complement body surface recordings, they should be interpreted with knowledge of their inherent limitations.

Most studies dealing with automaticity of accessory pathways have referred to ectopic beats arising in Kent tracts as reported in recent reviews on this subject (Przybylski et al., 1975; Castellanos et al., 1976). It seems appropriate, therefore, to discuss two possible examples of impulse formation in atrioventricular nodal bypass tracts.

Subjects and methods

Specialised intracardiac studies were performed in two symptomatic patients referred for investigation. The procedure was explained and informed consent obtained. As outlined in previous communications from our department, one catheter electrode was used to pace, alternatively, the atria and ventricles (Castellanos et al., 1971, 1973, 1977; Agha et al., 1976). Additional catheters were introduced to record the electrical activity of the high right atrium (HRA), the left atrium (LA) via the proximal coronary sinus, the area of the His bundle (HBE), and the right ventricular apex (RVA).

The following intervals were measured during sinus rhythm (number in parentheses indicate normal values in our laboratory):

(a) HRA—septal low right atrium (LRA): from onset of HRA electrogram to beginning of atrial electrogram recorded by the HBE lead (20 to 45 ms) = conduction time from close to the sinus node to septal LRA in the vicinity of the atrioventricular node.

(b) LRA–LA interval: from onset of the LRA electrogram to the onset of the left atrial electrogram recorded by the catheter electrodes in the proximal coronary sinus (> 20 ms). This interval does not represent linear conduction time from low right atrium to left atrium but differences in arrival of excitation at the recording sites.

(c) LRA–H interval: from the beginning of the atrial to the onset of the His bundle electrograms in the HBE lead (55–120 ms) = atrioventricular nodal conduction time.

(d) HV interval: from the beginning of the His bundle electrogram to the onset of ventricular depolarisation in whichever lead (surface or intracardiac) it occurred first (35–55 ms) = conduction time from His bundle to the earliest site of (left or right) ventricular activation.

(e) H–RVA interval: from beginning of the His bundle electrogram (H) to that of the electrogram recorded by the catheter in the right
ventricular apex (RVA) (55–75 ms) = conduction time through His bundle, right bundle-branch and that part of the ordinary ventricular muscle located between the site of exit (from the right bundle-branch) and the recording electrodes (Castellanos et al., 1973). During RVA pacing at the right ventricular apex retrograde activation of the atria by the impulse traversing the atrioventricular node (from the His bundle) occurs in an LRA-LA-HRA sequence (Agha et al., 1976; Amat-y-Leon et al., 1976; Gallagher et al., 1976). When the H-LRA intervals exceed 55 ms (thus excluding retrograde conduction via atrioventricular nodal bypass tract) the corresponding values are: LRA-LA: 20–50 ms; LRA-HRA: 35–75 ms (Agha et al., 1976).

It should also be noted that a decrease in St1–St2 intervals produces a progressive and proportional increase of the St2-LRA2, St2-LA2 and St2-HRA2 intervals with H2 (when and if recorded) appearing in front of LRA2 at similar (or longer; but definitely not shorter) intervals (Castillo and Castellanos, 1970).

Case descriptions

CASE 1

Electrocardiograms from this 23-year-old man with palpitations since childhood showed, almost consistently, an automatic rhythm characterised by negative P waves in leads I, II, III, aVF (bottom strip), and V2–V6 showing short PR intervals and normal QRS complexes. The surface electrocardiographic pattern is that which has been attributed to coronary sinus, low left atrial, or atrioventricular junctional rhythms.

Fig. 1 Case 1. Automatic ectopic rhythm with negative P waves in leads I, II, III, aVF (bottom strip), and V2–V6 showing short PR intervals and normal QRS complexes. The surface electrocardiographic pattern is that which has been attributed to coronary sinus, low left atrial, or atrioventricular junctional rhythms.

Fig. 2 Case 1. Sinus beats conducted through the atrioventricular node (left) and ectopic beats presumably originating in the upper end of a left-sided atrioventricular nodal bypass tract. HRA, high right atrium; HBE, His bundle electrographic lead; LA, left atrium; LRA, low (septal) right atrium; H, His bundle; V, onset of ventricular depolarisation. In this Fig. all intervals are expressed in ms from the moment of inscription of the first recorded atrial electrogram (0).
interval elapsing between inscription of LRA and H did not represent linear conduction time from LRA to His bundle, but the difference in the time of arrival of excitation at the corresponding sites. The different configuration of the atrial deflections in the HBE and HRA leads in the ectopic, as compared with sinus beats, supports these assumptions. Moreover, during left atrial pacing (Fig. 3, right) the St(LA)–H interval had more or less the same value (55 ms) as the LA–H interval of the ectopic beats shown in Fig. 2, right. On the other hand, mid right atrial stimulation (Fig. 3, left) resulted in LRA–H intervals (70 ms) similar to those of sinus beats. The corresponding HV intervals and QRS complexes were normal when either right or left atrium was paced (Fig. 3).

Pacing studies showed that 1:1 atrioventricular conduction occurred up to rates of 225 and 175/min from the left atrium and mid right atrium, respectively (Fig. 4).

During ventricular pacing with the extrastimulus technique the retrograde atrial activation sequence was similar to that of the ectopic beats in Fig. 2 since the LA2–LRA2 and LA2–HRA2 intervals measured

![Fig. 3](Image)

**Fig. 3** Case 1. Mid right atrial (MRA), and left atrial (LA) pacing at a basic cycle length of 600 ms showing conduction to the His bundle through the atrioventricular node bypass tract from the left atrium and through the atrioventricular node from MRA. All values in ms.

![Fig. 4](Image)

**Fig. 4** Mid right atrial (MRA) and left atrial (LA) pacing at the maximal rate capable of producing 1:1 atrioventricular conduction, namely, 175/min from MRA and 225/min from left atrium.

![Fig. 5](Image)

**Fig. 5** Case 1. Retrograde atrial activation pattern during ventricular pacing with the extrastimulus method. Note that the premature (St2) impulse activated the His bundle with considerable infra-His delay, thereafter reaching the left atrium (through the atrioventricular node bypass tract) ahead of the low right atrium. The retrograde atrial arrival of activation sequence was similar to that of ectopic beats in Fig. 2, right.
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20 and 90 ms, respectively (Fig. 5). This sequence was maintained as the St1–St2 intervals were decreased in spite of the fact that the St2–LA2, St2–LRA2, and St2–HRA2 showed a concomitant (and proportional) increase presumably because of infra-His bundle delay since, when (at the shorter St1–St2 intervals) retrograde activation of the His bundle occurred, the St2–H2 increased but the H2–LA2 kept a fixed value of 50 ms (Fig. 5).

To summarise, the findings in this case are compatible with a left-sided atrioventricular nodal bypass tract (Brechenmacher, 1976) conducting in a forward direction (preferentially from the left atrium) as well as in a retrograde direction. Spontaneous impulse formation producing the ectopic beats could have occurred at, or close to, the atrial end of the atrioventricular nodal bypass tract.

Case 2
This 56-year-old man with old inferior wall myocardial infarction and dizzy spells had ectopic beats and short runs of supraventricular tachycardia. During sinus rhythm the atria were activated in the normal fashion (Fig. 6). Right atrial pacing was possible with 1:1 atrioventricular response and progressive increments of AH until a rate of 155/min was reached at which moment the atria failed to respond. Thus, the exact rate at which atrioventricular Wenckebach occurred could not be determined.

As in case 1, values for the retrograde activation intervals were determined (during ventricular pacing with the extrastimulus technique) from the wavefronts elicited by St2 since the corresponding electrograms were best seen in premature beats delivered at short coupling intervals (Fig. 7, left). That the impulse reaching the His bundle (from the ventricles) activated the atria retrogradely through the atrioventricular node was suggested by the normal H–LRA interval of 70 ms and by inscription of the LRA electrogram 40 and 70 ms, respectively, before that of LA and HRA. Moreover, the St2–LRA2, St2–LA2 and St2–HRA2 showed a concomitant increase (while the H2L–RA2 interval remained constant) as the St1–St2 intervals were decreased.

This patient also had His bundle beats (Fig. 7, right) during which the surface P wave was buried within the ventricular complexes. Because the HV and H–RVA intervals, as well as QRS morphology,
were similar to those of sinus beats (Fig. 6) it was inferred that the impulse originating in the His bundle reached the ventricles through the His-Purkinje system in a normal fashion. That the H–LRA, LRA–LA, and LRA–HRA intervals had the same duration as those recorded during retrograde (VA) conduction of premature ventricular beats (Fig. 7, left) indicates that the atria were also depolarised retrogradely by the impulse emerging from the atrioventricular node.

In addition, there were other ectopic beats in which the LRA electrogram preceded H by an interval of only 20 ms (Fig. 8). The P wave (appearing at a short interval in front of QRS) seemed to have been slightly negative in all three standard leads. The normal duration of the HV and H–RVA intervals as well as of the QRS complexes indicated that the impulse reaching the His bundle activated the ventricles through the His-Purkinje system as during sinus rhythm (Fig. 6).

These ectopic beats did not originate in the His bundle (or ventricles) because the LRA was activated before the His bundle (and also before the left atrium). Moreover, the different duration (in respect to those of the beats shown in Fig. 7) of the LRA–LA and LRA–HRA intervals (70 and 90 ms, respectively) indicates that the atria were not activated by the impulse emerging from the atrioventricular node.

To summarise, the events occurring in Fig. 7 suggest that these beats arose in a right-sided atrioventricular nodal bypass tract. From its origin the impulse spread toward right atrium and His bundle reaching the former slightly before the latter. Thus, the (short) LRA–H did not represent linear conduction time from one site to another but differences of arrival of excitation at the corresponding sites.

**Discussion**

Case 1 probably had an abnormal left-sided atrio-His tract of the type described by Brechenmacher in 1976 showing preferential (exclusively forward) conduction from the left atrium: the atrioventricular nodal bypass tract conduction was ‘concealed’ during sinus rhythm and right atrial pacing. This phenomenon was the result of the relatively longer right atrium to left atrium conduction time (as compared with the right atrium to low right atrium conduction time) which allowed the right atrial impulses to reach the His bundle through the atrioventricular node before they had time to do so through the atrioventricular nodal bypass tract (Fig. 2).

Brechenmacher (1976) reported on the electrophysiological and pathological findings in 2 patients with atrioventricular nodal bypass tract which differed from the so-called James fibres in three aspects: (1) the atrio-His tracts were located on the left side of the His bundle whereas the James fibres were located on the right side of the septum; (2) atrio-His tracts penetrated the His bundle, whereas the fibres described by James penetrated the atrioventricular node and (3) the histological structure of these tracts was that of ordinary atrial myocardium, whereas James described tracts predominantly composed of Purkinje fibres.

In case 1 (Fig. 5) retrograde conduction occurred only through the left-sided atrioventricular nodal bypass tract and not via the atrioventricular node or a left-sided Kent tract (Gallagher et al., 1975; Agha et al., 1976; Amat-y-Leon et al., 1976). The former was excluded because the low right atrium was not inscribed ahead of the left atrium.

During ventriculo-atrial conduction through a left-sided Kent tract, the left atrium also occurs before the low right atrium. However, in these cases the St2–L2 and St2–LRA2 intervals do not show a progressive and significant increase with shortening of the St1–St2 intervals, and H2, when seen, is not inscribed at a fixed interval ahead of LA2.
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LRA2. In fact, it might even appear after LRA2 (Castillo and Castellanos, 1970).

In case 2, the right-sided atrioventricular nodal bypass tract was ‘concealed’ in the sense that it did not conduct in either forward or retrograde directions. The presence of this atrioventricular nodal bypass tract was detected only because it initiated impulses, as shown by the retrograde atrial activation pattern (Fig. 8) which was different from that of impulses emerging from the atrioventricular node (Fig. 7). However, it could not be determined whether the ectopic beats arose in an abnormal atrio-His bundle tract or in one of the multiple transitional fibres which normally bypass part of the compact atrioventricular node (Anderson et al., 1975).

It should be stated that inscription of low right atrium ahead of left atrium by itself is not diagnostic of retrograde atrioventricular nodal conduction. This sequence can also occur when a right-sided Kent bundle is present (Gallaher et al., 1975). In these cases the St2-LRA2 and St2-LA2 intervals do not show a progressive increase as the St1-St2 interval is shortened and H2, when seen, is not inscribed at a fixed interval ahead of LRA2.

The ectopic beats in Fig. 1, 2, and 8 had a surface electrocardiographic pattern characterised by a short PR interval with negative P waves in all three standard leads and normal QRS complexes. The genesis of this pattern has been a subject of considerable debate and speculation since it has been attributed to many different mechanisms, namely: atrioventricular nodal rhythm, coronary sinus rhythm, low atrial rhythm, etc. The most extensive review on this subject was made by Scherf and Schott (1973) (specifically see pages 116, 117, and 129 to 131).

We agree with these authors, as well as with Watson (quoted by Scherf and Schott, 1973, page 551) who stated that, ‘in patients with spontaneously occurring arrhythmias it is not possible to differentiate left atrial, from A–V junction, or coronary sinus, rhythms with any degree of certainty, by means of scalar electrocardiography and perhaps vectorcardiography.’

Intracardiac electrograms add another dimension to body surface recordings. The findings in cases 1 and 2 suggest that the pattern of ‘atrioventricular junctional rhythm with preceding activation of the atria can also result from impulse formation within an atrioventricular nodal bypass tract. This assumption cannot be proven conclusively but is supported by the reports of Sherf and James (1966, 1969) who first suggested that ectopic beats could arise in the tracts that normally bypass part of the atrioventricular node.

Although specialised intracardiac studies did show that conduction through an atrioventricular nodal bypass tract could be ‘concealed’, they also have limitations. Foremost among these is that the exact site of impulse formation cannot be determined (using catheter electrodes) with the same degree of precision as if one were to be using roving endocardial electrodes for mapping purposes with the atria exposed during cardiopulmonary bypass (Kupersmith et al., 1974). Thus, in case 1 a coronary sinus, or low left atrial, origin with preferential conduction to His bundle via a closely located left-sided atrioventricular nodal bypass tract (though less likely) could not be totally excluded.

Moreover, the degree of penetration into the coronary sinus required to record left atrial potentials cannot be determined exactly. Right atrial potentials are recorded immediately before entrance to the coronary sinus, as well as from its ‘os’ and within a nondetermined distance within the coronary sinus itself. Because the interatrial septum lies at an angle of approximately 45° to the frontal plane and the left atrium lies as much behind it as it does the right atrium (Walmsley and Watson, 1966a, b) one cannot use, exclusively, the spatial (right or left) relation between the catheter electrode intended to be placed into the coronary sinus and the catheter located over the septal surface of the right atrium to determine when the former starts to record left atrial potentials.

Recording several bipolar electrograms from the distal and proximal coronary sinus as stressed by Gallacher et al. (1975, 1976) is more selective in this respect. In previous reports from our department we considered that catheter electrodes were positioned behind the left atrium if pacing through them produced negative P waves in all three standard leads (Agha et al., 1972, 1976).

The limitations of the catheter techniques, just described, do not invalidate the assumption made in this study. On the contrary, they should be recognised as useful guidelines for future studies oriented toward the analysis of the mechanism of spontaneous ectopic impulse formation in normal atrioventricular junctional tissues or in abnormal atrio-His tracts.

We are indebted to Dr. Claude Brechenmacher for bringing to our attention before publication, his findings on the first two patients in whom the presence of left-sided atrio-His tracts was first detected by electrophysiological and anatomical studies.
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