Studies of intra-atrial conduction with bipolar atrial and His electrograms

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With the use of His bundle electrograms, intra-atrial conduction delay has been found to be a cause of first-degree heart block. Twenty-one patients with evidence of atrioventricular conduction defects or dysrhythmias were studied with His bundle electrograms and 2 were found to have delayed intra-atrial conduction while in sinus rhythm. A further 3 patients had normal PA intervals during sinus rhythm but prolonged StA intervals during atrial pacing. The significance of this is discussed.

In view of the difficulty in timing the onset of atrial depolarization from the surface electrocardiogram, a bipolar electrogram from the high right atrium recorded simultaneously with the His electrogram was used to time intra-atrial conduction in 11 patients. This interval was found to be significantly greater than the PA interval measured from the surface electrocardiogram. It is suggested that this be done routinely during recording of His bundle electrograms.

Since the introduction of His bundle electrocardiography in man (Scherlag *et al.*, 1969), more detailed assessment of intracardiac conduction has become possible. Attention has been focused mainly on defects in the atrioventricular (AV) node and His-Purkinje system. Recently it has been shown that conduction defects also occur in the specialized conducting system of the atrium (Narula *et al.*, 1970, 1971; James and Scherf, 1971; Castellanos, Castillo, and Agha, 1971a). This finding has increased the importance of accurate measurement of the intra-atrial conduction time. This interval is usually measured as the time taken from the onset of the P wave on the surface electrocardiogram to the onset of the A deflection on the His electrogram, both traces being recorded simultaneously (PA time). Difficulty in timing the onset of the P wave on the surface electrocardiogram, particularly at high recording speeds, makes accurate assessment of the PA interval difficult and can lead to considerable errors. To reduce these errors, a bipolar atrial

electrogram was recorded from the superior vena caval-right atrial junction simultaneously with the His electrogram. This measurement of intra-atrial conduction is termed the P₁A interval and is compared with the standard method of measuring the PA interval using a surface electrocardiogram. The use of the high atrial electrogram also facilitates localization of supraventricular ectopic beats and the demonstration of retrograde conduction.

Patients and methods

Using the technique described by Scherlag *et al.* (1969), His bundle electrograms were recorded from 21 patients (aged 23 to 72 years) with evidence of atrioventricular conduction defects or dysrhythmias. Details of the findings in the individual patients are shown in Table 1.

The procedures used were explained in detail to all the patients studied and informed consent was obtained.

A bipolar electrode catheter was introduced percutaneously into the right femoral vein and positioned across the tricuspid valve. The electrogram was monitored as the tip of the catheter was slowly withdrawn until the characteristic His bundle deflection appeared between the atrial and ventricular deflections. A filter setting of 40-400 Hz was used on the electrocardiogram preamplifier, and recordings were made on a Cambridge multichannel oscilloscopic photographic recorder at a paper speed of 100 mm/sec. In 12 patients a separate bipolar electrode catheter was passed to the junction of the superior vena cava and the right atrium. This was

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TABLE 1 *His electrograms in 21 patients*

Case No.	Age and sex	P ₁ A (msec)	PA (msec)	AH (msec)	HV (msec)	Remarks
1	62 M	30		80	60	Left anterior hemiblock + right bundle-branch block; anterior myocardial infarction
2	58 F	50		75	65	Intermittent left bundle-branch block
3	58 F	40		110	70	Left bundle-branch block
4	62 M	45		150	50	First-degree heart block
5	60 F	40		80	40	Left bundle-branch block
6	23 M	20		100	40	Ventricular ectopics
7	54 M	45		80	35	Left anterior hemiblock + right bundle-branch block; anteroseptal myocardial infarction
8	49 M	40		70	80	Left posterior hemiblock + right bundle-branch block; anterolateral myocardial infarction
9	62 F	30		85	85	Narrow QRS; block below His bundle; pacemaker implanted
10	55 M	50		85	45	—
11	38 F	40		85	45	Junctional ectopics
12	66 F	50-70		70	50	Atrial dysrhythmias; sinus node disease
13	40 M		20	70	80	Left bundle-branch block
14	64 F		40	115	35	Sinoatrial block; atrial dysrhythmias
15	48 M		30	125	55	Left anterior hemiblock + right bundle-branch block; inferolateral myocardial infarction
16	63 F		30	125	65	Left anterior hemiblock; sinoatrial block; inferior myocardial infarction
17	65 M		80	120	40	Intermittent left bundle-branch block; inferior myocardial infarction
18	72 M		40	110	70	Left bundle-branch block; intermittent complete heart block; pacemaker implanted
19	61 F		45	85	40	Left anterior hemiblock; paroxysmal atrial flutter; anterior myocardial infarction
20	60 M		30	40	50	Paroxysmal ventricular tachycardias; anterior myocardial infarction
21	49 M		40	70	90	Left bundle-branch block

TABLE 2 *Comparison of intra-atrial conduction as measured from high atrial electrogram (P₁A) and surface P wave (PA)*

Case No.	Age and sex	Atrial electrocardiogram P ₁ A (msec)	Surface electrocardiogram PA (msec)	P ₁ A-PA
1	62 M	45	30	15
2	58 F	50	40	10
3	58 F	40	30	10
4	62 M	40	30	10
5	60 F	40	30	10
6	23 M	20	0	20
7	54 M	45	30	15
8	49 M	40	30	10
9	62 F	30	20	10
10	55 M	50	35	15
11	38 F	40	20	20
Mean	52.8	40.0	26.8	13.2
Standard deviation		± 9.4	± 10.1	P < 0.01

used to record a bipolar electrogram simultaneously with the His electrogram. The same catheter was used to pace the atrium at rates of up to 187 a minute, using a modified Devices external battery-powered pacemaker. Precautions were taken to isolate all equipment from leakage currents.

The first rapid deflection of the atrial electrogram (P_1) was used to determine the onset of atrial depolarization and the interval to the atrial (A) deflection of the His electrogram was measured. This was compared with the PA interval measured from the beginning of the P wave of the surface electrocardiogram (P). During atrial pacing the time from the pacing stimulus to the A deflection (StA interval) was measured. The normal PA time is 20 to 40 msec; AH is 50 to 120 msec and HV is 35 to 55 msec (Narula *et al.*, 1971; Castellanos *et al.*, 1971a).

In Cases 1 to 11 (Table 2), comparisons were made between the P_1A and PA intervals. Case 12 was not included in the comparison because her PA interval varied from 50 to 70 msec. In 4 of the 11 patients (Cases 8, 9, 10, and 11), simultaneous surface (lead II), high atrial, and His electrograms were obtained. In the remaining 7 patients the surface and His electrograms and the atrial and His electrograms were recorded consecutively. The heart rate varied by no more than 6 beats a minute between recordings in all patients except one (Case 5), in whom the heart rate was 68 a minute during the surface and His recording and 85 a minute during the atrial and His recording.

Results

Patients with abnormal intra-atrial conduction Of the 21 patients studied, 2 (Cases 12 and 17) had a prolonged intra-atrial conduction time when in sinus rhythm and in neither was the defect greatly increased by atrial pacing. Case 12 was a 66-year-old woman with sinus node disease who presented with several episodes of supraventricular tachycardia and atrial fibrillation. At the time of the His electrogram, sinus bradycardia was present with extreme variation in sinus cycle length. A prolonged sinus recovery time of 3.8 sec on cessation of atrial pacing was found. The P_1A interval varied from 50 to 70 msec (mean 60) with no apparent change in focus as judged from the P wave configuration on the atrial electrogram. The StA time at a rate of 162 a minute ranged from 50 to 70 msec.

Case 17 showed intermittent left bundle-branch block and a prolonged PR interval (240 msec). The AH (120 msec) and HV intervals were normal (40 msec) but the PA was prolonged to 80 msec (Fig. 1). During atrial pacing the StA interval was 100 msec at 85 a minute. In 3 patients (Cases 14, 16, and 18) the PA intervals were normal but on atrial pacing StA intervals were prolonged (80 msec or more).

FIG. 1 Case 17. First-degree heart block due entirely to intra-atrial conduction delay.

FIG. 2 Case 8. Surface electrocardiogram (lead II), high atrial electrogram (HAE), and His electrogram (HBE), showing earlier atrial depolarization in HAE.

FIG. 3 Case 9. Electrocardiogram (lead II), high atrial electrogram (HAE), and His electrogram (HBE), showing difficulty in determining onset of atrial depolarization from surface P wave. The second complex is an atrial premature beat (APB).

Case 14 was a 64-year-old woman who presented with palpitations. Her surface electrocardiogram showed normal P waves on occasions. At other times she had inverted P waves in leads II, III, and aVF, frequent supraventricular ectopics, and occasional 3:2 sinoatrial block. Her PA was normal (40 msec) but during atrial pacing StA was prolonged to 85 msec at a rate of 85 a minute.

Case 16, a 63-year-old woman with a previous inferior myocardial infarction, prolonged PR interval, interpolated ventricular ectopic beats, sinus bradycardia, and sinoatrial block presented with an 18-month history of blackouts. The His electrogram in sinus rhythm showed a PA interval of 30 msec, AH of 125 msec, and HV of 65 msec. Atrial pacing at 92 a minute resulted in an StA of 80 msec which increased to 90 msec at 133 a minute with 2:1 AV block proximal to the His bundle. The sinus recovery time after atrial pacing was slightly prolonged at 1.6 seconds. The findings suggest disease of the sinus node and delay in conduction involving the atrium, AV node, and His-Purkinje system.

Case 18 was a 72-year-old man who presented with Adams-Stokes attacks. His electrocardiogram showed sinus rhythm with a PR interval of 0.22 sec and complete left bundle-branch block. Subsequent recordings showed intermittent complete heart block. When in sinus rhythm, the His electrogram showed a PA interval of 40 msec, AH of 110 msec, and HV of 70 msec. Atrial pacing at a rate of only 80 a minute resulted in an StA of 140 msec. This rose to 150 msec at a rate of 160 a minute. A permanent demand pacemaker was subsequently inserted.

PA vs P₁A The results in the 11 patients studied are shown in Table 2. The P₁A interval (measured from the atrial electrogram) ranged from 20 to 50 msec (mean 40) whereas the PA interval measured from the surface lead ranged from 0–40 msec (mean 26.8). The difference between the two measurements was significant ($P < 0.01$). In Case 6, the PA interval appeared to be 0 when measured from the surface P wave but the P₁A interval was 20 msec. The P₁A interval always exceeded the PA interval by at least 10 msec. An example is shown in Fig. 2 of simultaneously recorded surface, high atrial (HAE), and His (HBE) electrograms from Case 8. Fig. 3 from Case 9 illustrates the difficulty that may occur in timing the onset of surface P wave with consequent variation in the PA interval, in this case between 10 and 20 msec.

Discussion

The use of His electrography has led to the recognition of conduction disturbances in the

specialized conducting system of the atrium, as well as to the clarification of the more common atrioventricular blocks. Narula and his colleagues (1971) reported a patient who had a prolonged PR interval due entirely to prolongation of intra-atrial conduction; the PA time being 110 msec (normal 20–45 msec). Another patient reported by the same group also had a prolonged intra-atrial conduction time (PA of 100 msec) in association with impaired conduction in the AV junction (AH time 225 msec), His-Purkinje system (HV time 70 msec) and sinus bradycardia (Narula and Samet, 1971). These cases show that defects in intra-atrial conduction may occur alone or in association with disease in other parts of the conducting system. In this study we have found two patients with prolonged intra-atrial conduction. In Case 12, the association of an intra-atrial conduction defect, sinus node disease, paroxysmal supraventricular tachycardia, and atrial fibrillation suggests that atrial disease was responsible for the development of the dysrhythmias. It is of interest that her intra-atrial conduction time varied from 50 to 70 msec even in beats which appeared to be arising from the same atrial focus, as judged by the configuration of the intra-atrial electrogram. The variation in the PA interval could be explained either by differential conduction through the atrium from two separate but adjacent atrial foci or by intermittent preferential conduction from the same focus (Narula *et al.*, 1971). The variation was not related to RR or PP intervals which varied considerably (from 830 to 1320 msec).

In Case 17, the prolonged PR interval and left bundle-branch block suggest the presence of trifascicular block. However, the His electrogram showed that the prolongation of the PR (240 msec) was due to prolonged intra-atrial conduction (PA 80 msec) and the AH (120 msec) and HV (40 msec) times were normal. Thus, delayed intra-atrial conduction was responsible for apparent atrioventricular block. James and Scherf (1971) have suggested that complete AV dissociation may occur with failure of the sinus impulse to be conducted to the AV node. They quoted three cases of AV dissociation in which the block was associated with conspicuous changes in the P waves suggesting altered intra-atrial conduction. Unfortunately, His bundle electrograms were not available in their cases to localize the site of the block but this case confirms their hypothesis.

The recognition of the clinical importance of intra-atrial conduction has made accurate measurement of the PA interval more significant. The PA time, as measured from the earliest deflection of the P wave on the surface electrocardiogram to the A

deflection of the His bundle electrogram, has been accepted as a measure of the conduction time from the SA node to the AV node (Narula *et al.*, 1971). The surface electrocardiogram is, however, a poor indicator of the earliest atrial depolarization as there is commonly an isoelectric period before the P wave of 10 to 25 msec during which the atrium is already depolarizing (Waldo *et al.*, 1970). Further difficulty in measurement results from the low amplitude and slow rise of the surface P wave. This is aggravated by the high recording speeds necessary to measure conduction times in the distal conducting system. Thus, errors of up to 20 msec in the PA time might occur when the surface electrocardiogram is used. This error is reduced by measurement from an intra-atrial electrocardiogram if the electrode is situated in the region of the SA node because the tracing obtained from the bipolar electrode at the SVC-RA junction shows a sharp deflection, the onset of which is easy to determine even at fast paper speed. In all 11 cases in which the comparison was made, the intra-atrial conduction time exceeded that measured from the surface electrocardiogram. In Case 6, the onset of the surface P wave appeared to coincide with the A deflection of the His electrogram but the P₁A interval measured from the high atrial electrogram was 20 msec. The mean difference between the intervals measured by the two methods was 13.2 msec and was significant at the 1 per cent level.

A further advantage of recording simultaneous intra-atrial and His electrograms is the more complete analysis of dysrhythmias with retrograde conduction. Retrograde P waves, often invisible on the surface electrocardiogram, are usually seen on the intra-atrial record and retrograde conduction can be timed. Further complex refinements have been described in man (Castellanos, Castillo, and Myerburg, 1971b) and in the dog (Damato *et al.*, 1970), but the use of a single intra-atrial electrode can provide useful information during more routine studies.

The significance of an StA interval during atrial pacing which is much longer than the PA interval during sinus rhythm is not clear. It has been claimed that atrial pacing at moderate rates may occasionally unmask intra-atrial conduction disturbances (Castellanos *et al.*, 1971a, Fig. 2). These authors report a patient with complete right bundle-branch block and left anterior hemiblock in whom the P₁A interval was 30 msec while in sinus rhythm, but the StA time during atrial pacing was 115 msec. Case 18 in our series is very similar, in that the PA time while in sinus rhythm was 40 msec but the StA time at an atrial pacing rate of only 80/minute was 140 msec. A lesser defect was unmasked in Cases 14 and 16 in whom pacing showed StA times of 85

and 80 msec with normal PA times. Each had evidence of sinoatrial node disease and suffered recurrent atrial dysrhythmias. The findings support the suggestion of Castellanos *et al.* (1971a) that a prolonged StA interval may represent a latent defect in atrial conduction.

Cases 16, 17, and 18 are also of considerable interest because of the association of sinoatrial nodal and atrial disease with disease in the AV node and His-Purkinje system. The fact that three instances were found in 21 patients suggests that this combination may be more frequent than previously realized.

Atrial pacing is employed routinely during His electrography to stress the conducting system and assess sinus node function (Damato *et al.*, 1969a, b; Narula *et al.*, 1971; Castellanos *et al.*, 1971a). The use of the pacing catheter to record a high intra-atrial electrogram simultaneously with the His bundle electrogram does not involve any additional procedure. This allows more accurate assessment of the intra-atrial conduction time and is recommended for routine use in patients who are having conduction system studies. Atrial pacing may be of value in exposing latent defects in intra-atrial conduction but its value remains to be confirmed.

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