Mahaim conduction producing left axis deviation and normal QRS

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SUMMARY An unusual patient is described in whom electrophysiological studies strongly suggest the occurrence of Mahaim conduction. The patient whose electrocardiogram previously showed a left anterior hemiblock pattern then developed advanced atrioventricular (AV) block (AH block). Beats conducted through the atrioventricular node always had a short HV interval (20 ms) and QRS complexes of left anterior hemiblock pattern. Junctional escape beats always had a normal HV interval (50 ms) with normal intraventricular conduction. His bundle pacing showed the StV interval and QRS contour of escape beats. These findings suggest the existence of an accessory pathway (Mahaim fibres) passing from the area of block, presumably the uppermost portion of the His bundle, to the posteroinferior division of the left bundle-branch. The surface electrocardiogram did not show the characteristic delta wave of the Wolff-Parkinson-White syndrome. Our observations suggest that patients in whom there is conduction along Mahaim fibres may show only the pattern of intraventricular conduction defect without a delta wave.

Although anatomical studies in cases of the Wolff-Parkinson-White syndrome have occasionally shown the presence of Mahaim fibres (Lev *et al.*, 1975), actual conduction through Mahaim fibres leading to the ventricular pre-excitation is reportedly very rare (Lev *et al.*, 1966; Castillo and Castellanos, 1970; Massumi, 1970; Rosen *et al.*, 1971; Coumel *et al.*, 1972; Castellanos *et al.*, 1975; Lev *et al.*, 1975; Tonkin *et al.*, 1975; Touboul *et al.*, 1975).

Clues to conduction by Mahaim fibres have been classically said to include the presence of a normal or prolonged PR interval with a delta wave (Wolff and White, 1948; Pick and Katz, 1955), and the persistence of pre-excitation in beats of atrioventricular junctional origin (Pick and Katz, 1955; Lev *et al.*, 1966). Recently, His bundle recording with atrial or His bundle pacing has made it possible to recognise Mahaim conduction even in the presence of other accessory pathways such as Kent or James fibres (Coumel *et al.*, 1972; Tonkin *et al.*, 1975).

In the present case, electrophysiological study after the development of advanced atrioventricular

block strongly suggested the existence of Mahaim fibres. Mahaim conduction was associated only with a left anterior hemiblock pattern without a delta wave.

Case report

A 48-year-old woman was admitted to Kyushu University Hospitals on 12 April 1976. She had had several episodes of palpitation in October 1974, lasting for only 10 seconds or less. An electrocardiogram at that time (Fig. 1, A) showed sinus rhythm with normal PR interval (0.18 s). The QRS complexes were of normal duration (0.08 s) without delta wave, but there was left axis deviation (-35°) with q in aVL and S in lead II and III, compatible with left anterior hemiblock. Six months before admission, she became short of breath on exertion. An electrocardiogram then showed advanced atrioventricular block. She had no syncopal attacks.

On admission, electrocardiograms showed advanced atrioventricular block (Fig. 1, B). The second QRS complex, a conducted beat, shows left axis deviation with QRS duration 0.08 s, similar to that seen in October 1974. Other QRS complexes are junctional escape beats and, in contrast to the

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Fig. 1 (A) Electrocardiogram on 9 October 1974. Normal sinus rhythm (67/min) with QRS complexes of left anterior hemiblock pattern. (B) Electrocardiogram on admission, 13 April 1976. Advanced atrioventricular block is noted. The second QRS complex, a conducted beat, is similar to those in the upper panel. Other QRS complexes are junctional escape beats and show normal intraventricular conduction. (C, D) Parts of a 24-hour recording of the electrocardiogram (lead CM 5). (C) Mobitz type II block with 3:2 atrioventricular conduction. The PR interval of conducted beats was constant. (D) Junctional escape rhythm at 28 beats a minute with normal intraventricular conduction in advanced atrioventricular block.

conducted beat, show normal axis ($+30^\circ$) and the QRS duration 0.06 s. A 24-hour recording of the electrocardiogram (Fig. 1: lower panel) showed advanced atrioventricular block with occasional atrioventricular conduction, mainly 2:1, rarely 3:2 (Fig. 1, C) or 1:1. The junctional escape rhythm varied from 28 to 44 a minute (Fig. 1, D). When 3:2 atrioventricular conduction was present, the PR interval of the conducted beat was constant, suggesting Mobitz type II block. Every conducted beat showed the left anterior hemiblock pattern, and the same PR interval and QRS duration as in October 1974. During her hospital stay, a repeat 24-hour recording of the electrocardiogram did not show any QRS complex with the delta wave of the classical Wolff-Parkinson-White syndrome.

An electrophysiological study was performed on 21 April 1976. After the study, the patient was treated with oral isoprenaline (15 mg q.d.) which restored normal (1:1) atrioventricular conduction. The QRS complexes were similar to those in October 1974, showing left axis deviation.

Electrophysiological study

The techniques for obtaining His bundle electrograms in our laboratory have been described previously (Takeshita *et al.*, 1974). The best atrioventricular conduction seen during the study was 3:1 conduction with junctional escape beats, as illustrated in Fig. 2. The second and fourth QRS complexes are conducted beats and preceded by the A wave and His bundle deflection. The AH and HV intervals are 130 ms and 20 ms, respectively. These QRS complexes show left anterior hemiblock pattern with QRS duration 80 ms. The third A wave is not followed by a His bundle deflection

Fig. 2 Recording of the His bundle electrogram (HBE), high right atrial electrogram (HRA), and lead III. The arrow indicates the His deflection. There is 3:1 atrioventricular conduction with junctional escape beats. Conducted beats have normal AH, short HV interval (20 ms), and QRS complex of the left anterior hemiblock pattern. Junctional escape beats have normal HV interval (50 ms) and QRS complex shows normal intraventricular conduction. The third A wave is not followed by the His deflection, indicating AH block.

Fig. 3 His bundle stimulation (St) (indicated by arrow) showing that the StV interval and QRS complex were similar to the HV interval and QRS complex of junctional escape beats shown in Fig. 2.

indicating AH block. The first and third QRS complexes are preceded only by the His bundle deflection with HV interval 50 ms, indicating the origin of these impulses above the bifurcation of the His bundle. The electrical axis of these escape beats is normal and the QRS duration is 60 ms. The conducted beats show a short HV interval (the normal HV interval in our laboratory is 35 to 55 ms) but, in contrast, escape beats have a normal HV interval. The QRS duration of conducted beats is longer than that of the junctional escape beats. During the study numerous unsuccessful attempts were made to record a split or other His bundle deflection by varying the position of the His bundle catheter.

Pacing the His bundle (Fig. 3) at a cycle length of 860 ms (slightly shorter than the spontaneous atrial cycle length) was associated with the stimulus

Fig. 4 2:1 atrioventricular conduction after intravenous atropine (1 mg), with short HV interval and QRS complex of left anterior hemiblock pattern. The absence of junctional escape beats indicates that the cycle length (1730 ms) of junctional escape beats cannot be less than 1420 ms (twice the atrial cycle length). The arrow indicates the His deflection.

to V wave (St V) interval of 50 ms and 1:1 conduction. The QRS complex with pacing the His bundle was similar to that of escape beats shown in Fig. 2, with electrical axis of $+30^\circ$ and the QRS duration 60 ms. The pacing interval was varied between 860 and 660 ms, but the StV interval and the QRS configuration did not change. These findings suggest that intraventricular conduction distal to the His bundle was normal.

Intravenous administration of atropine (1 mg) improved atrioventricular conduction to 2:1 AH conduction (Fig. 4). Conducted beats showed the QRS complex of left anterior hemiblock pattern with QRS duration 80 ms. As junctional escape beats were not seen after atropine, the junctional escape interval could not have been less than 1420 ms (twice the atrial cycle length).

The slow basic rate of the junctional escape rhythm (28 to 44 per minute) unaffected by atropine indicates that the location of escape pacemaker is in the upper portion of His bundle rather than in

Fig. 5 Schematic illustration of ventricular excitation. The left-hand panel shows a ventricular complex which is composed by fusion of conduction by the normal His-Purkinje system (HPS) and that through Mahaim fibres (M) from the uppermost portion of the His bundle to the posteroinferior divisions of the left bundle-branch (LBB). Pre-excitation of posterobasal endocardial surface of the left ventricle produces a QRS complex of left anterior hemiblock pattern with short HV interval (20 ms). The right-hand panel shows normal intraventricular conduction of a junctional escape beat with normal HV interval (50 ms), when atrioventricular (AV) block is present. The dotted area indicates simultaneous block both in the normal pathway and Mahaim fibres.

the atrioventricular node (Narula and Samet, 1971; Scherlag *et al.*, 1973). The presence of Mobitz type II block with 3:2 atrioventricular conduction (Fig. 1, C) indicates that AH block was in the uppermost portion of the His bundle (Narula *et al.*, 1971).

These findings may be summarised schematically as shown in Fig. 5. Conducted beats were always associated with a short HV interval and with a QRS configuration of left anterior hemiblock pattern. Escape beats or His bundle pacing at various rates showed a normal HV or StV interval and normal electrical axis, indicating normal intraventricular conduction below the His bundle. The degree of shortening of the HV interval in conducted beats was similar to the prolongation of the QRS duration. Though the short HV interval with left axis deviation in conducted beats could conceivably be caused by congenitally short posteroinferior divisions of the left bundle-branch, as postulated in some patients with incomplete endocardial cushion defect (Goodman *et al.*, 1974), this possibility is ruled out by normalisation of the HV interval and QRS axis in escape beats or during His bundle pacing. Our observations suggest conduction over an accessory pathway in conducted beats, causing pre-excitation of the posterobasal portion of the left ventricle. The atrioventricular block appears to be in the uppermost portion of the His bundle. The observations that conduction in the accessory pathway occurred at the same time as block in the normal atrioventricular conduction pathway, and that retrograde conduction to the accessory pathway did not occur with escape beats or His bundle pacing, suggest that the accessory pathway arises in the region of AH block. The absence of a delta wave on the surface electrocardiogram may be explained by insertion of the accessory pathway into the posteroinferior divisions of the left bundle-branch rather than into the ventricular myocardium as was suggested in the case reported by Tonkin *et al.* (1975).

Discussion

The present case is analogous to those reported by Touboul *et al.* (1975) in which HV intervals were abnormally short at rest, but lengthened suddenly to normal, with disappearance of the pre-excitation pattern during atrial pacing at increasing rates. Normalisation of the HV interval and QRS pattern was thought to result from functional exclusion of accessory Mahaim conduction. In the present case the observations that conducted beats were associated with a short HV interval (20 ms), and that escape beats and His bundle paced beats were

associated with a normal HV interval (50 ms), can best be explained by intermittent block of Mahaim conduction together with block of the ordinary atrioventricular conduction system in the uppermost part of the His bundle.

The presence of congenitally short posteroinferior divisions of the left bundle-branch as an alternative explanation for the short HV interval with left axis deviation in conducted beats is ruled out by normalisation of the HV interval and QRS axis with junctional escape beats or by His bundle pacing. Another possible explanation for the findings in the present case could be the presence of an area of intermittent block in the His bundle with a localised area of permanent impairment of conduction including those fibres constituting the left anterior division of the left bundle-branch. If this were the case, in order to explain the short HV interval, one would have to postulate that His bundle deflections observed during normal atrioventricular conduction were recorded from the site of delay in the fibres of the left anterior fascicle. However, the failure to record a different His deflection with normal HV interval when attempts were made to do so by varying the position of the His bundle catheter makes this possibility unlikely.

In the present series, Mahaim conduction was associated only with a left anterior hemiblock pattern. Classical Wolff-Parkinson-White complexes were not noted even during repeated 24 hour recordings of the electrocardiogram. Previous reports suggest that Mahaim conduction may be associated with various degrees of the ventricular pre-excitation (Lev *et al.*, 1966; Castillo and Castellanos, 1970; Massumi, 1970; Rosen *et al.*, 1971; Coumel *et al.*, 1972; Castellanos *et al.*, 1975; Lev *et al.*, 1975; Tonkin *et al.*, 1975; Touboul *et al.*, 1975). The classical Wolff-Parkinson-White pattern with the delta wave was seen in cases reported by Lev *et al.* (1966, 1975), Massumi (1970), Coumel *et al.* (1972), and Touboul *et al.* (1975). In some cases, Mahaim conduction may produce slight initial slurring of the QRS complex (Castillo and Castellanos, 1970; Rosen *et al.*, 1971) or only bundle-branch block patterns (Castellanos *et al.*, 1975; Tonkin *et al.*, 1975). It has been suggested that if Mahaim fibres end in the bundle-branch system or close to the Purkinje network, pre-excitation may be insufficient to produce a definite delta wave (Castellanos *et al.*, 1975; Tonkin *et al.*, 1975). The left anterior hemiblock pattern seen in conducted beats in the present case may be explained by Mahaim fibres ending in the posteroinferior divisions of the left bundle-branch. This condition may be analogous to that seen in some patients with incomplete endocardial cushion

defect, in whom a short HV interval and left anterior hemiblock pattern have been attributed to congenitally short posteroinferior divisions of the left bundle-branch (Goodman *et al.*, 1974). The absence of a right bundle-branch block pattern in the present case is not unexpected; many of the patients with incomplete endocardial cushion defect reported by Goodman *et al.* had no right bundle-branch block pattern after operation, despite congenitally short posteroinferior divisions of the left bundle-branch and resulting short HV interval and left axis deviation. Our case is the first to be reported in which the left anterior hemiblock pattern appears to be produced by conduction over Mahaim fibres.

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