His bundle recordings in diagnosis of impulse formation in Kent and Mahaim tracts

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His bundle electrograms were recorded in 2 patients with ectopic beats arising in accessory atrioventricular tracts. Case 1 had Wolff-Parkinson-White (WPW) type A and a left-sided Kent tract with a short effective refractory period. Though ectopic impulse formation most probably occurred within the Kent tract itself, a vulnerability-related origin in the ventricular muscle close to the distal end of the Kent tract could not be excluded. In Case 2, with a Mahaim tract extending from His bundle to ventricles, there were three types of QRS morphologies resulting from: (a) atrioventricular conduction exclusively through the normal pathway; (b) atrioventricular conduction through both, normal pathway, and Mahaim tract; and (c) ectopic impulse formation in the Mahaim tract. Specialized electrophysiological studies were essential to diagnose these unusual arrhythmias.

There have been several reports postulating ectopic impulse formation in accessory atrioventricular tracts (Bix, 1953; Katz and Pick, 1956; Soffer, 1962; Scherf and Cohen, 1964; Bermudez and Childers, 1970; Mustakallio and Sarkkonen, 1973; James and Puech, 1974; Wyndham and Rosen, 1976). However, this assumption could not be proven because His bundle recordings were not obtained in these cases. This justifies the presentation of two possible examples of ectopic impulse formation within Kent and Mahaim tracts.

Subjects and methods

His bundle recordings were obtained in 2 patients referred for electrophysiological studies after the procedure had been explained and consent obtained (Castillo and Castellanos, 1970; Castellanos *et al.*, 1973b). For the purpose of the presentation a Kent tract was considered as an accessory atrioventricular connexion not attached to this normal AV node-His Purkinje system. A Mahaim tract was a His-ventricular connexion.

The AH interval was measured from onset of atrial (A) to onset of His bundle (H) deflections in

the His bundle electrographic (HBE) leads. The HV interval was measured from the onset of the H deflection to the beginning of ventricular depolarization in whichever lead (intracardiac or surface) it occurred first.

Case 1

Additional information from this patient with Wolff-Parkinson-White (WPW) type A, including the morphology of sinus and driven beats, the functional properties of the Kent tract, and the mechanisms of repetitive arrhythmias has been presented elsewhere (Castellanos *et al.*, 1973b).

Sinus impulses were conducted exclusively through the AV node-His Purkinje system with a complete right bundle-branch block morphology (first two beats in Fig. 1 and first beat in Fig. 2). The AH and HV intervals measured 85 and 55 ms, respectively. Premature atrial impulses (third beat in Fig. 1 and second beat in Fig. 2) were conducted to the ventricles almost exclusively through the Kent tract with WPW type A morphology. The PR interval became shorter and the H deflection appeared at, or slightly after, the onset of a wide QRS complex which occurred before the end of the antecedent T wave.

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FIG. 1 (Case 1) Intermittent WPW syndrome type A (third QRS complex) with an ectopic beat (1) probably arising in a left side Kent tract. A=low right atrial electrogram. HBE=His (H) bundle electrographic lead. St=pacemaker stimulus artifact.

In addition, Fig. 1 shows an ectopic (last) QRS complex almost identical to the previous (third) ventricular complex which in this patient represented exclusive Kent conduction. This ectopic beat (1) was not preceded by a P wave nor an H deflection. Though it most probably arose in the Kent tract itself a 'true' ventricular extrasystole originating close to the lower end of the tract could not be excluded completely.

FIG. 2 (Case 1) Ectopic beat (2) arising in a left-sided Kent tract or in the ordinary ventricular muscle close to its distal end.

In contrast, the last QRS complex in Fig. 2 was not identical to that resulting from exclusive accessory AV pathway conduction (second ventricular complex) because: (a) the QRS axis changed to a closer to $\pm 180^\circ$ position; (b) lead II became predominantly negative; and (c) in V1 the height of the R wave increased with the notch being displaced downward. This ectopic beat (2), which was followed by a P wave with a low-to-high right atrium sequence, could have arisen in: (a) the ventricular muscle (close to the end of the Kent tract) or (b) within the latter itself, the different contour being the result of irregular propagation through incompletely recovered muscle. The latter possibility was supported by its coupling interval (360 ms) which was shorter than that of the ectopic beat in Fig. 1 (400 ms), both being preceded by similar RR (or VV) intervals.

To summarize, in this patient impulse formation probably occurred in a left-sided Kent tract. Yet, vulnerability-related ventricular ectopic beats could not be completely excluded.

Case 2

Other tracings from this 23-year-old man with intermittent conduction through a Mahaim tract have been discussed previously (Castillo and Castellanos, 1970). However, the duration of the pertinent intervals in Fig. 3-5 was determined as currently performed in our department. This re-

FIG. 3 (Case 2) Sinus rhythm with exclusive conduction through the normal atrioventricular pathway (left) and through both normal atrioventricular pathway and Mahaim tract extending from His bundle to ventricles (right).

sulted in slightly different values from those previously reported (Castillo and Castellanos, 1970).

During sinus rhythm with exclusive conduction through the AV node-His Purkinje system the PR, AH, HV intervals and QRS complexes measured 140, 85, 45 and 80 ms, respectively (Fig. 3, left). In contrast, sinus beats propagating through both normal atrioventricular pathway and Mahaim tract had PR, AH, and HV intervals measuring 130, 85 and 35 ms, respectively (Fig. 3, right). In this beat the HV interval represented conduction time from His bundle to ventricles through the Mahaim tract. The QRS complex showed an initial slurring (delta wave) and a duration of 90 ms.

During atrial stimulation the AH interval increased while the HV interval and QRS complex remained unchanged (Fig. 4). This response to atrial pacing excluded Kent conduction and it is in keeping with a functional electrophysiological Mahaim tract extending from His bundle to ventricles (Castillo and Castellanos, 1970).

An escape rhythm also occurred intermittently in this patient (Fig. 5). Though the QRS complexes were wider (130 ms) and the HV intervals shorter (15 to 20 ms) than those of sinus beats conducted through both normal pathway and Mahaim tract (Fig. 3, right), the direction of the initial slurring (delta wave) was the same.

These ectopic beats probably did not originate in the His bundle with conduction to the ventricles

via both His-Purkinje and Mahaim pathways, since the HV interval and QRS morphology would have been similar to those in Fig. 3, right. Neither did they arise in the proximal bundle-branches or fascicles, since, in absence of bundle-branch disease (Fig. 3, left), the QRS complexes should have shown an 'incomplete' (not a 'complete' as in Fig. 5) bundle-branch block pattern with an H deflection appearing *after* the onset of ventricular depolariza-

FIG. 4 (Case 2) Atrial stimulation showing (by comparison with Fig. 3, right) an increase in AH interval without any change in HV interval or QRS morphology.

tion (Rosenbaum *et al.*, 1970; Castellanos *et al.*, 1972).

The possibility that the deflection in Fig. 5 was a right bundle electrogram preceded by the 'missed' (the result of technical reasons) H deflection of a His bundle escape rhythm cannot be excluded completely. However, for this to occur the His bundle beats must have had bradycardia-dependent (phase 4) bundle-branch block, which appears unlikely in a young individual without heart disease (proven by cardiac catheterization) who electrophysiologically and clinically had pre-excitation syndrome and was taking no medicines.

In summary, this patient had an escape rhythm possibly arising within a Mahaim tract extending from His bundle to ventricles.

Discussion

Although several authors have suggested that impulse formation could occur within the accessory atrioventricular tracts themselves (Bix, 1953; Katz and Pick, 1956; Soffer, 1962; Scherf and Cohen, 1964; Bermudez and Childers, 1970; Mustakallio and Saikonen, 1973; James and Puech, 1974; Wyndham and Rosen, 1976), as far as we know this is the third report in which specialized intracardiac recordings have been used to support the validity of the assumptions (Coumel *et al.*, 1973).

Puech and Grolleau (1972) discussed the tracings of a patient with WPW type B who also had late extrasystoles interpreted as originating in a low atrial focus. The latter, however, could have arisen in the Kent tract, the corresponding P wave (which preceded the wide QRS complex) resulting from simultaneous depolarization of the atria by both sinus and ectopic wavefronts.

The differential diagnosis between impulse formation in Kent and Mahaim tracts is difficult to make from the surface electrocardiogram alone. Identification of the ectopic beats requires prior knowledge of the type(s) of pre-excitation present. Since this cannot always be done from the analysis of surface leads only, specialized intracardiac studies are essential (Castillo and Castellanos, 1970).

For example, in patients with Kent tracts, atrial stimulation from the appropriate sites and cycle lengths will eventually show that the His bundle deflection appears after the onset of ventricular depolarization (Castillo and Castellanos, 1970). If an ectopic beat has a QRS morphology identical to that resulting from exclusive Kent conduction (Fig. 1) impulse formation in the latter can indeed be postulated. However, extrasystoles arising in the ventricular muscle close to the lower end of the

tract will show a similar morphology. Our first patient had a Kent tract with a short effective refractory period (Castellanos *et al.*, 1973b). Therefore, premature atrial impulses were able to reach the ventricles early enough to produce a QRS complex appearing on top of the preceding T wave (vulnerable period). For this reason repetitive firing in the ventricular muscle close to the end of the Kent tract could have been the mechanism of the ectopic beats in Fig. 1 and 2.

On the other hand, a *very premature* impulse arising in the Kent tract could propagate very irregularly throughout the ventricles because of relative refractoriness (Castellanos *et al.*, 1973a), thus resulting in a QRS morphology (Fig. 2) different from that of beats arising later in the cycle (Fig. 1).

There have been few reports (using His bundle recordings) describing the features of supraventricular beats conducted to the ventricles simultaneously through the AV node-His-Purkinje system and a Mahaim tract extending from His bundle to ventricles (Castillo and Castellanos, 1970). This was postulated in Case 2 by the alternation of narrow and wide QRS complexes during sinus rhythm (Fig. 3) and by assessment of the effects of atrial stimulation on QRS duration and HV intervals (Fig. 4). It should be stressed that we are not referring to those cases in which an electrophysiological AV nodal bypass has coexisted with a Mahaim tract (Coumel *et al.*, 1972; Castellanos *et al.*, 1975), neither to the cases in which the latter has extended from the AV node to ventricles (Tonkin *et al.*, 1975) or bundle-branches to ventricles (Castellanos *et al.*, 1975).

Coumel *et al.* (1973) reported a case in which there was a Kent tract (or a Mahaim tract associated with a James tract) coexisting with complete AV block resulting from 'congenital absence of the bundle of His'. Because of the latter an H deflection was not recorded in this patient. Escape beats were occasionally seen, but it could not be determined whether they arose in a Mahaim or in a Kent tract.

The Mahaim escapes depicted in Fig. 5 were not preceded by P waves. That the impulses reached the ventricles exclusively through the accessory tract was suggested by the similarities of the initial portions of the QRS complexes (delta wave) of fusion and escape beats, as well as by the increase in total ventricular activation time and decrease in H-V intervals in the latter (Fig. 5) as compared with the former (Fig. 3, right).

It should be stressed that in Fig. 5, the H-V interval did not represent linear conduction time from His bundle to ventricles through the Mahaim tract but differences in arrival of excitation at these

FIG. 5 (Case 2) Mahaim escapes showing (in reference to Fig. 3, right) shorter H-V intervals and wider QRS complexes. The ventricles were depolarized exclusively through the Mahaim tract. H⁻ indicates retrograde activation of His bundle from the site of origin (see Fig. 6, left)

sites. Thus in Fig. 5 the His bundle was activated retrogradely from the Mahaim tract whereas in Fig. 3, right, and in Fig. 4 it was depolarized antegradely from the atrioventricular node.

The differential diagnosis of the type of Mahaim escapes postulated in this communication includes impulse formation in: (a) His bundle (atrioventricular junction) with conduction to the ventricles via both His-Purkinje system and Mahaim tract (Pick, 1956); (b) His bundle (atrioventricular junction) with conduction exclusively via the Mahaim tract (Pick, 1956); and (c) bundle-branches, or fascicles of the left bundle-branch (Rosenbaum *et al.*, 1970) (Fig. 6).

The electrophysiological evaluation of pre-excitation syndromes with multiple catheter electrodes (which are needed in most patients with reciprocating atrioventricular tachycardias) requires a thorough understanding of iatrogenic factors which can lead to errors in interpretation. For example, mechanical stimulation of the ordinary muscle at the right ventricular inflow tract by the catheter electrode recording His bundle activity can stimulate impulse formation in a right septal Kent tract (WPW type B). Likewise, mechanical stimulation by a catheter introduced in the distal coronary sinus or great cardiac vein produces pseudo (left-sided) Kent extrasystoles in patients with WPW type A (Castellanos *et al.*, 1973c). In these beats the corresponding QRS morphologies are different from those resulting from exclusive Kent conduction. Moreover, the onset of the ven-

FIG. 6 (Case 3) Differential diagnosis of Mahaim (M) escapes (left-sided schematic). H-V intervals and QRS morphologies are similar to those of beats shown in Fig. 5. Impulse formation in His bundle with conduction through both normal atrioventricular pathway and Mahaim tract (second schematic) results in HV intervals and QRS morphologies similar to those of beats in Fig. 3, right. Impulse formation in His bundle with conduction exclusively through Mahaim tract (third schematic) results in HV intervals as in Fig. 3, right, and QRS morphologies as in Fig. 5. Fascicular beats (last schematic) have short H-V or (VH⁻) intervals with QRS morphologies depending on the site or origin: right bundle-branch (RBB), left bundle-branch (LBB) or fascicles of the left bundle-branch.

tricular electrogram recorded by the corresponding catheter electrodes can occur as early as 20 ms before the onset of QRS in the surface leads (Castellanos *et al.*, 1973c).

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