'Incessant' tachycardias in Wolff-Parkinson-White syndrome¹

II: Role of atypical cycle length dependency and nodal-His escape beats in initiating reciprocating tachycardias

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Descriptions of patients with the Wolff-Parkinson-White (WPW) syndrome and reciprocating tachycardia in whom the initiation of the arrhythmia depended neither on the occurrence of premature beats nor on antecedent cycle-length shortening are given. In 5 the occurrence of escape beats in the bundle of His, usually in the presence of sinoatrial disease, activated the tachycardia circuit, but in the other 2 there were unusual mechanisms related to bradycardia-dependent block in the anomalous pathway, and delayed response to shortening of the atrial cycle length, respectively. Careful assessment of such mechanisms is essential for the correct choice of antiarrhythmic prophylactic therapy.

In a previous article we have described a group of patients with the WPW syndrome in whom the induction of incessant reciprocating tachycardia depended not on extrasystoles but on critical shortening of the sinus cycle length, without antecedent PR lengthening (Krikler *et al.*, 1976a). However, not all incessant tachycardias can be explained either on this basis or because of premature beats; we have recognized 2 slightly different additional mechanisms and report 7 cases that illustrate these aspects. The methods of study have already been outlined (Krikler *et al.*, 1976a).

Case reports

Case 1

A 35-year-old woman had suffered from progressively more frequent attacks of tachycardia (2 to 10 a month) for 5 years. The surface electrocardiogram showed the WPW syndrome type B (Fig. 1). Electrophysiological study, however, indicated that

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the accessory pathway was probably not right-sided, as endocardial mapping showed that the earliest ventricular depolarization occurred in the interventricular septum; furthermore, the induction of functional right bundle-branch block during tachycardia did not slow its rate, as it should have done had a right-sided bypass been implicated in the circuit (Slama, Coumel, and Bouvrain, 1973; Coumel and Attuel, 1974; Spurrell, Krikler, and Sowton, 1974a). Pre-excitation was suppressed by ajmaline (Fig. 1, lower panel); though two ventricular stimuli were induced in order to stop the tachycardia, the first (beat 2) would probably have sufficed as it was not followed by a retrograde P'. There was no prolongation of the PR interval in beat 10, when the tachycardia was initiated.

The onset of all bouts of tachycardia was exactly like that seen in Fig. 1, initiation being by beats identical to beat 10. As can be seen in the lower panel of Fig. 1, the first RR interval (11 to 12) is slightly longer than succeeding ones, which is against the presence of an initial common pathway. The only satisfactory explanation for the occurrence of tachycardia after beat 10 is the prolongation of the PP interval from 580 ms (beats 8 to 9) to

FIG. 1 Case 1: the upper panel comprises a 12 lead electrocardiogram showing type B pre-excitation. The lower panel consists of simultaneous recordings of leads I, III, unipolar and bipolar right atrial (RA) leads, and a His bundle electrogram (HBE), with tracings taken after the administration of 50 mg ajmaline intravenously. Beat 1 is the last complex of the tachycardia, which was terminated by two ventricular stimuli (beats 2 and 3). Beats 4 to 10 are of sinus origin, all having the same, normal, PR interval (167 ms). Tachycardia is reinitiated by the lengthening of PP to 600 ms before beat 10. (In intracardiac leads P and A are used interchangeably for atrial deflections.)

FIG. 2 Case 2: simultaneous recording of surface leads I and II and His bundle and right atrial electrograms. Beats 1 and 2 are His bundle escape beats, at an interval of 1070 ms. Right atrial stimulation at a constant cycle length of 640 ms was started at beat 3, this beat representing fusion between the resultant atrial impulse involving the anomalous pathway and a third His bundle escape beat, H3, occurring precisely 1070 ms after H2. The fourth and fifth beats show pre-excitation with prolonged stimulus-H (Ps-H) interval of 220 ms; tachycardia is initiated by beat 6.

FIG. 3 Case 3: simultaneous recordings of leads I and II, right atrial electrogram (RA) and a His bundle electrogram (HBE). The first five beats are caused by pacing at a constant rate of 167 beats a minute (stimulus-stimulus [Ps-Ps] interval 360 ms). The first two beats show clear evidence of pre-excitation, the H deflection following the beginning of QRS. The stimulus-delta (Ps-delta) interval is 120 ms, and the St-H (Ps-H) 140. In beats 1 to 5, Ps-H remains constant, and the pre-excitation pattern disappeared in beats 3, 4, and 5. Stimulation was stopped after beat 5, and re-entry at once occurred.

600 ms (beats 9 to 10). It is assumed that concealed anterograde conduction was present within the accessory pathway in beats 4 to 9, which prevented re-entry from occurring, and that this concealed conduction had been dissipated by beat 10 because of the lengthening PP interval, a phenomenon thus most probably related to paradoxical, bradycardia-induced, block in the accessory pathway.

The phenomenon persisted for 10 minutes, as long as the ajmaline continued to be active, with spontaneous resumption of tachycardia in the identical fashion.

The tachycardia has subsequently been effectively prevented with oral amiodarone (Coumel and Bouvain, 1973).

Case 2

A 45-year-old man presented with frequent attacks of tachycardia, occurring especially at rest; his electrocardiogram showed the WPW syndrome type B.

Electrophysiological study showed the initiation of reciprocating tachycardia following the introduction of atrial pacing at a constant slow rate (Fig. 2). Both in His bundle escape beats and in tachycardia, the retrograde conduction time up the anomalous pathway was 130 ms (beats 1, 2, 6, 7, 8, and 9). The stimulus-H (Ps-H) interval is prolonged to 220 ms in beats 4 and 5, and there is no further prolongation in beat 6 where tachycardia is initiated because of the development of anterograde block in the accessory pathway. No drugs were given during the study; the refractory period of the bypass was spontaneously long.

Case 3

A 43-year-old man with infrequent attacks of paroxysmal tachycardia was found to have the WPW syndrome type A complicated by left bundle-branch block (Krikler *et al.*, 1976b). It was possible to initiate tachycardia by regular right atrial pacing (Fig. 3); the stage had obviously been set for re-entry at beat 3 but this was prevented by the continued pacing for another two beats. Spontaneous initiation of the tachycardia is shown in the upper panel of Fig. 4; the His bundle escape beat that followed termination of the tachycardia immediately re-established a reciprocating mechanism, as it occurred within the circuit itself. In the lower panel (Fig. 4) tachycardia started immediately after atrial stimulation had been discontinued, the His bundle escape beat after the ninth atrial stimulus immediately establishing tachycardia. Atrial driving had prevented the start of tachycardia, probably because it produced concealed anterograde conduction into the accessory pathway, thus rendering it unavailable for retrograde conduction. The His escape 2750 ms after the termination of tachycardia (Fig. 4) suggests concomitant sinoatrial disease (Mandel *et al.*, 1971).

Case 4

A 75-year-old woman presented with alternating bradycardia and tachycardia, without overt pre-excitation before the study. Spontaneous intermittent pre-excitation occurred during the investigation (Fig. 5, upper left panel); His bundle escape beats (Fig. 5, upper right panel) do not always initiate tachycardia if for example sinus P waves, not conducted through the AV junction to the ventricles, prevent retrograde conduction from the His bundle escape beat and thus retrograde atrial activation. Validation of the location of the stimulus applied to the His bundle shown in the seventh beat in the middle panel of Fig. 5 is obtained from the HR interval of 50 ms, identical to that seen

FIG. 4 Case 3: simultaneous recordings of leads III and V1. In the upper panel, reciprocating tachycardia is apparent with retrograde P' waves; this is stopped by paired atrial stimuli (PsPs). A subsequent pause is terminated by a His bundle escape beat which reinitiates the tachycardia. In the lower panel the first nine impulses are the result of atrial stimulation at a fixed rate (Ps); the first beat after their cessation is a His bundle escape, which initiates reciprocating tachycardia.

in the spontaneous sinus beat and the His bundle escape beat in the bottom panel in this figure. In this patient pre-excitation was always latent, except briefly during this study, being represented only in the limb leads, so the praecordial pattern is not known. Because the stimulus was applied high in the right atrium, the Ps-R and Ps-delta intervals are longer than during low right atrial stimulation (middle panel).

Case 5

A 60-year-old woman with a history of alternating attacks of tachycardia and bradycardia had been considered to have reciprocating AV nodal tachycardia. The first run of tachycardia (Fig. 6) showed alternating cycle lengths. Apparent sinus bradycardia may in reality reflect 2:1 sinoatrial block; so the apparently premature His bundle beat, which would have been early in relation to sinus bradycardia, could reflect a genuine escape beat in relation to sinoatrial block. One might be tempted to diagnose reciprocating AV nodal tachycardia, but the alternating cycle lengths suggested an extranodal bypass (Spurrell, Krikler, and Sowton, 1974b; Curry and Krikler, 1976), and the fact that the tachycardia was slower in the presence of left bundle-branch block than during its absence

(171 as opposed to 182 beats a minute) pointed to the presence of an otherwise concealed left-sided anomalous pathway (Slama *et al.*, 1973; Spurrell *et al.*, 1974a).

Case 6

A 68-year-old woman had suffered from progressively more troublesome tachycardia for 25 years, and was found to have the WPW syndrome type A (Fig. 7) complicated by left bundle-branch block (Krikler *et al.*, 1976b) and sinoatrial disease; in tachycardia the QRS complexes show an LBBB pattern (Fig. 8). She showed initiation of tachycardia by two mechanisms; (a) with progressively faster right atrial pacing (Fig. 7); and (b) following His bundle escapes, provided that their retrograde conduction up the anomalous pathway was not blocked by sinus impulses (Fig. 8), analogous to the situation seen in Case 4 (Fig. 5). Continued atrial stimulation prevented further attacks during the study by suppressing the occurrence of escape beats.

Case 7

A 67-year-old man had suffered from recurrent supraventricular tachycardia for more than 50 years; numerous electrocardiograms had all failed to show pre-excitation except transiently im-

FIG. 5 Case 4: the upper two panels were recorded at a paper speed of 25 mm/s, the middle and bottom at 50 mm/s. Left upper panel shows the three standard leads recorded simultaneously, with regular right atrial pacing: alternating pre-excitation is seen. In the right upper panel a right atrial electrogram is shown simultaneously with V6: the first five QRS complexes represent reciprocating tachycardia, which is terminated by a spontaneous atrial premature beat (*); the three beats thereafter reflect escape in the His bundle, the third one restarting the reciprocating tachycardia.

Middle panel: the first four stimuli were applied low in the right atrium and the QRS complexes show alternating pre-excitation; the fifth stimulus produces a fusion beat caused by activation of atrium and His bundle and probably the uppermost part of the interventricular septum. The sixth stimulus was applied to the interventricular septum, and the seventh to the bundle of His, with a Ps-R interval of 50 ms; this resulted in the initiation of the tachycardia (the eighth, ninth, and tenth stimuli were ineffective). Only the seventh stimulus triggered tachycardia because it was the only one in which the His bundle alone was stimulated, without any atrial or ventricular activation.

Bottom panel shows simultaneous recordings of leads III and HBE: the first complex is a spontaneous sinus beat and the second a His bundle escape beat. Note that in each case the HR interval is 50 ms.

mediately after conversion to sinus rhythm by verapamil (Krikler and Curry, 1976). Electrophysiological study confirmed the presence of type A pre-excitation (Fig. 9A); the bypass refractory period in the anterograde direction was 230 ms. In Fig. 9B, after he had received intravenous ajmaline (0.75 mg/kg body weight), each termination of tachycardia by induced single ventricular premature beats was followed by a His bundle escape beat which invariably reinitiated the tachycardia.

Discussion

It is generally agreed that three conditions are

necessary for the establishment of a re-entry tachycardia: (a) the existence of a potential circuit; (b) unidirectional block in the circuit; (c) slowing of conduction so that the travelling impulse does not encounter a refractory area at any point during its circuit. Even if the first prerequisite is present, the latter two are often obscured at the initiation of a paroxysm though in reality one or other may be present, albeit potentially rather than overtly. In the previous article we have shown that, for example, in the WPW syndrome, slowing of conduction (requirement (c)) need not implicate the anterograde pathway; there was no lengthening of the PR interval at the start of the tachycardia (Krikler *et al.*, 1976a). The initiation of the paroxysm was linked to

the development of unidirectional block in the accessory pathway, perhaps seen only as the disappearance of concealed anterograde conduction along that route.

A variety of these aspects is illustrated in the 7 cases now presented. *Block in the accessory pathway*, however produced, led to tachycardia in all our cases; this had been produced pharmacologically (Case 7); by phase 3 block caused by an increase in the rate of the stimulation (Case 6); or by phase 3

block undoubtedly the result of a fatigue phenomenon, the rate of stimulation not having been increased (Cases 2 and 3). The situation in Case 1 was highly unusual, in that the block was paradoxical, because of prolongation of the atrial cycle length, in association with concealed as opposed to overt anterograde conduction in the accessory pathway.

Lengthening of the PR (or AH) interval at the start of tachycardia has been extensively discussed

FIG. 6 Case 5: continuous tracing of V2 (the ladder diagram refers to the bottom strip). In the upper panel, tachycardia is present at the start of the tracing, with alternating cycle lengths; after spontaneous cessation, sinus bradycardia is noted for five beats, followed by a premature His bundle beat that restarted the tachycardia. The first six beats of tachycardia showed left bundle-branch block, with a heart rate of 171 beats a minute; this was followed by normalized conduction at a rate of 182 beats a minute.

FIG. 7 Case 6: continuous simultaneous tracings of leads V1 and V6, showing the effects of gradually increasing right atrial pacing. There is no good evidence of pre-excitation at slow pacing rates, but this becomes increasingly evident as the pacing rate exceeds 120 beats a minute. At a pacing rate of 167 beats a minute anterograde bypass block is induced; at 171 beats a minute, when pacing was stopped, reciprocating tachycardia showing a left bundle-branch block pattern was established at a rate of 187.

FIG. 8 Case 6: leads V1, V6, and RA, showing spontaneous cessation of tachycardia with LBBB pattern; the subsequent beat reflects fusion between a sinus impulse with anterograde bypass conduction, and a His bundle escape; the following beat is a His bundle escape that reinitiates tachycardia.

(Goldreyer and Damato, 1971; Rosen, 1973), but the precise importance of this phenomenon needs to be reassessed. It need not be present (Case 1), as we have also shown in the preceding paper (Krikler *et al.*, 1976a). Even if PR prolongation is present, as in Cases 2, 3, and 6, tachycardia started without additional prolongation, the accessory pathway being blocked in Case 2; the onset was seen in Cases 3 and 6 when atrial stimulation was interrupted. In addition, these latter two cases show that PR lengthening is by no means essential, since the tachycardia could be initiated by His bundle escape beats, without prolongation of the conduction time.

Escape beats within the re-entry circuit constitute an important mechanism for the initiation of attacks (Cases 3, 5, and 7), but this still conforms with the requirements for re-entry mentioned above. Thus, in Case 2 the escape beats failed to provoke tachycardia in the presence of concealed retrograde conduction into the normal pathways; similarly, as in Cases 4 and 6, concealed anterograde conduction of the P waves also prevented the onset of tachycardia. On the other hand, lack of atrial activation, as indicated by the absence of a P wave, could enable an incessant tachycardia to start (Case 7). We have been able to show the presence of the WPW syndrome in these cases, but this does not mean that this phenomenon cannot occur in nodal re-entry; it does however appear to us much less likely because under those circumstances the escape occurs *outside* the circuit.

Thus, while in patients with reciprocating tachycardias associated with pre-excitation, attacks are usually initiated by extrasystoles (Durrer *et al.*, 1967) or sometimes by shortening of the sinus cycle length (Krikler *et al.*, 1976a), a different group of

mechanisms can be shown to be of importance. That nodal-His bundle escape beats, originating as they do within the substrate for the circuit, are able to induce tachycardias with ease is evident in 5 of our patients, often in the context of sinoatrial disease, itself an important mechanism for the occurrence of paroxysmal tachycardia. This has indeed been recognized in the Wolff-Parkinson-White syndrome complicated by atrial fibrillation (Dreifus, Kimbiris, and Wellens, 1973) and reciprocating AV tachycardia (Harper *et al.*, 1974). Sinoatrial disease was present or considered highly likely in three of our patients (Cases 3, 5, and 6) but His bundle escape beats also occurred when it was not possible to affirm its presence (Cases 4 and 7); in Case 7 the escapes were associated with the administration of ajmaline. It is self-evident that for tachycardia to occur, His bundle escape beats must be conducted, anterogradely better than retrogradely, thus leaving the AV node ready for the circus wave once it has reached the atrium via the bypass.

These phenomena are of interest in that they may explain the occurrence of tachycardias complicating pre-excitation in the absence of premature beats, and without immediately antecedent conduction delay in the blocked pathway. Nodal-His escape beats are potentially of great importance but other mechanisms are more complex and each atypical case needs the most careful scrutiny in order to define possible causes for tachycardia. That this is of therapeutic as well as of academic significance is shown by Case 6, in whom drug therapy proved unhelpful by producing further depression of the SA node, leading to syncope, as well as a consequent increased tendency to tachycardia induced

FIG. 9 Case 7: (A) RA, coronary sinus electrogram (CSE), HBE, and leads I, III, V1, and V6 bringing out the pre-excitation as the pacing rate was progressively increased (intervals between stimuli decreased from 390 to 330 ms). (B) Continuous tracings (HRA, CSE, HBE, and lead I), showing termination of tachycardia by ventricular stimuli (St) and immediate reinitiation by His bundle escape beats.

by the junctional escape beats (Krikler *et al.*, 1976b). That more than one mechanism may apply in a patient is well shown by the same case, who had tachycardia initiated both by progressive atrial pacing and by the junctional escape beats.

While these atypical types of tachycardia are unusual, they are by no means rare, and may apply particularly to older subjects in whom the junctional escape beats, perhaps originating in the bundle of His, occur more frequently as a consequence of disease affecting proximal physiological pacemaking cells, e.g. sinoatrial disease. Successful therapy, both for the attacks themselves and for their prophylaxis,

thus requires careful understanding of these mechanisms, the nature of which should be defined in all patients who present with apparently atypical onset of tachycardia. It is important to remember that depressant antiarrhythmic drugs may paradoxically induce incessant tachycardias in this way; elucidation of apparent paradoxical response of this sort may be of practical importance as well as indicating the reason for the apparent failure of treatment. Finally, it is in cases like this that long-term pacing may be particularly helpful in the prevention of attacks, an aspect that we are currently exploring.

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