Re-entrant tachycardia using two bypass tracts and excluding AV node in short PR interval, normal QRS syndrome

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SUMMARY In patients with the short PR interval, normal QRS complex syndrome, paroxysmal tachycardias are usually the result of circus movement involving the AV node and a partial or complete AV nodal bypass. We report 2 patients with this syndrome who suffered distressing rapid paroxysms of tachycardia but in whom there was evidence of a concealed direct VA connection. In both patients, tachycardia was initiated with critical AV prolongation distal to the His bundle, in response to programmed atrial premature stimuli. The constancy of the timing of the atrial echo from the onset of the QRS complex in the presence of a varying HV interval is evidence for involvement of the ventricles in the re-entry pathway. In addition, in both patients the appearance of left bundle-branch block during tachycardia was associated with appropriate prolongation of tachycardia cycle length consistent with the presence of a direct VA connection. The short AH interval during tachycardia and the absence of critical AH prolongation suggests the participation of a rapidly conducting pathway in the anterograde limb of the tachycardia circuit.

The syndrome of short PR interval and normal QRS complex associated with paroxysmal rapid heart action was described by Lown *et al.* (1952). The advent of techniques of intracardiac recording and stimulation has allowed analysis of the syndrome in great detail. It is now generally accepted that the short PR interval is the result of conduction in a partial or complete AV nodal bypass (Durrer *et al.*, 1970; Castellanos *et al.*, 1971; Mandel *et al.*, 1971). Intracardiac recordings in patients with this syndrome show a short AH interval as a result of conduction in the accessory pathway. Alternative explanations of the short PR interval include enhanced AV nodal conduction and have been enumerated by Caracta *et al.* (1973). The mechanism of tachycardia in the short PR interval, normal QRS complex syndrome has not been clearly elucidated. Intracardiac electrophysiological studies (Mandel *et al.*, 1971; Denes *et al.*, 1974) suggest that the most common mechanism is associated with block in the rapidly conducting pathway and critical prolongation of the AH interval allowing re-entry towards the atrium in the fast extranodal pathway. In the 2 patients described in this report a different mechanism was evident.

Patients and methods

A 63-year-old man (case 1) was admitted to hospital in March 1977 for investigation of paroxysmal palpitation which he had experienced for 12 years. Acute attacks had failed to respond reliably to intravenous propranolol or verapamil and in recent months he had required DC conversion to restore sinus rhythm. Oral therapy which had included propranolol and disopyramide reduced the frequency of attacks. Physical examination and routine investigations were normal. The surface electrocardiogram showed a PR interval of 0.11 s during sinus rhythm and a normal QRS complex.

The second patient (case 2), a 26-year-old man suffered rapid paroxysms of palpitation since the age of 12 years. Various medications including digoxin, procainamide, propranolol, and verapamil had failed to control attacks. In the 2 years before investigation he had had several attacks requiring DC conversion to sinus rhythm. On other occasions intravenous verapamil was administered and successfully terminated the tachycardia only once. The patient was admitted for investigation in June 1977. Physical examination and routine investigations were normal. The surface electrocardiogram revealed a PR interval of 0.12 s and normal QRS complexes.

Both patients were studied in the postabsorptive non-sedated state. All medications were stopped 4 days before study. Four pacing electrodes were introduced into the right femoral vein under local anaesthetic and were positioned fluoroscopically in the apex of the right ventricle, against the septal leaflet of the tricuspid valve and in the high right atrium. In case 1 an additional electrode catheter was introduced via the left basilic vein and positioned in the coronary sinus. All intracardiac signals were filtered between 50 and 700 Hz and recorded simultaneously with surface leads I, aVF, and V1 on an Elema Mingograph at 100 mm/s, with time markings at 1-second intervals.

Stimulation was performed using a Devices 4270 isolated stimulator with 1.5 ms square wave pulses delivered at approximately twice the diastolic threshold.

DEFINITIONS

The following abbreviations, terms, and definitions are used throughout the text—

- A_1, H_1, V_1 the atrial, His bundle, and ventricular electrogram during basic atrial pacing.
- A_2, H_2, V_2 the atrial, His bundle, and ventricular electrograms produced by premature atrial stimulation.
- AH the interval between the intrinsic deflection of the atrial electrogram and the intrinsic deflection of the His bundle electrogram on the His bundle electrode.

HV

the interval between the intrinsic deflection of the His bundle electrogram and the earliest ventricular activation in any lead.

VA'

the interval between the earliest ventricular activation in any lead and the retrograde atrial electrogram in the high right atrial electrode.

Results

CASE 1

At electrophysiological study during sinus rhythm the AH interval was 60 ms with an HV interval of 35 ms. During incremental atrial pacing the AH interval increased from 60 ms at a cycle length of 610 ms to 80 ms at a cycle length of 375 ms. Programmed atrial premature stimuli were delivered throughout electrical diastole. Fig. 1a shows the anterograde conduction curve constructed by plotting the H_1H_2 and V_1V_2 intervals against the coupling interval of the premature stimulus, A_1A_2 . A_1A_2 values between 610 and 385 ms produced a small prolongation of the H_1H_2 interval. As the coupling interval was shortened from 385 ms to 310 ms prolongation of the H_2V_2 interval occurred and left bundle-branch block appeared. Within this range of coupling intervals the A_2H_2 interval increased by only 10 ms to 90 ms.

Fig. 2a shows initiation of tachycardia at a coupling interval of 310 ms. The H_2V_2 interval increased to 85 ms at this coupling interval. At all

Fig. 1 Anterograde conduction curves in cases 1 and 2.

Fig. 2 (a) Initiation of tachycardia by premature atrial stimulation (S_2). (b) Tachycardia with normal QRS complexes and a cycle length of 290 ms compared with tachycardia with left bundle-branch block and cycle length of 310 ms. HRAE, high right atrial electrogram; DCSE, distal coronary sinus electrogram; MCSE, mid-coronary sinus electrogram; PCSE, proximal coronary sinus electrogram; HBE, His bundle electrogram; I, aVF, V1, surface electrocardiographic leads.

intervals of A_1A_2 which resulted in left bundle-branch block and an atrial echo or sustained tachycardia the V_2A' interval remained between 165 and 170 ms despite variations in the A_2H_2 and H_2V_2 intervals. The same V_2A' interval was observed during tachycardia. Narrow complex tachycardia was not initiated by a single atrial premature stimulus. During tachycardia earliest activation of the atria occurred on the proximal coronary sinus electrogram. A single ventricular stimulus during tachycardia 15 ms before the onset of the His bundle potential was conducted retrogradely, resetting the atrial electrogram without disturbing the His bundle depolarisation or altering the atrial activation sequence. This is shown in Fig. 3. Appropriately timed ventricular stimuli during tachycardia resulted in conversion of the left bundle-branch block tachycardia to narrow complex tachycardia. When this occurred the cycle length of tachycardia was shortened by an amount corresponding to the shortening of the VA' interval (Fig. 2b). Fig. 4a shows narrow complex tachycardia with a short $A'H$ interval of 55 ms and normal HV interval. Termination of both narrow and broad complex tachycardia was reliably achieved by rapid atrial pacing or double ventricular stimuli. Programmed ventricular premature stimuli after a regular pacing sequence resulted in constancy of retrograde conduction time with earliest activation of the atria on the proximal coronary sinus electrogram.

CASE 2

During sinus rhythm the AH interval was 65 ms and HV 35 ms. Incremental atrial pacing prolonged the AH interval from 65 ms at a cycle length of 610 ms to 90 ms at a paced cycle length of 375 ms. The HV interval remained unchanged. The H_1H_2 and V_1V_2 intervals resulting from progressively shortened A_1A_2 values are shown in Fig. 1b.

Fig. 3 Premature ventricular stimulation during tachycardia resulting in exact atrial capture. S, stimulus artefact.

Fig. 4 Tachycardia in case 1 (a) and case 2 (b) demonstrating short AH interval.

Values of A_1A_2 between 610 and 360 ms caused minimal prolongation of the A_2H_2 intervals. Earlier premature stimuli resulted in gradual prolongation of the H_2V_2 interval. Fig. 5 compares a premature stimulus at 440 ms with a stimulus at 320 ms which resulted in prolongation of the H_2V_2 interval to 105 ms, right bundle-branch block, and initiation of tachycardia. Shorter coupling intervals resulted in similar responses. At all coupling intervals which produced atrial echo beats or sustained tachycardia the V_2A' interval remained constant at 210 to 215 ms, though the H_2V_2 interval varied from 105 to 135 ms and the A_2H_2 interval varied minimally.

Appropriately timed single ventricular stimuli during right bundle-branch block tachycardia resulted in conversion to narrow complex tachycardia without change in cycle length. On one occasion tachycardia with left bundle-branch block was precipitated by a non-programmed stimulus. The VA' interval increased by 30 ms with a corresponding increase in cycle length. Fig. 6 shows all 3 varieties of tachycardia. Right bundle-branch block and normal complex tachycardia both had a cycle length of 290 ms with VA' intervals of 210 ms, whereas left bundle-branch block tachycardia had a cycle length of 320 ms and VA' of 240 ms. Exact capture of the atria by ventricular stimulation could not be achieved presumably because right ventricular apical stimuli were too remote from the tachycardia circuit. Fig. 4b shows a short A'H interval of 70 ms and HV of 40 ms during narrow complex tachycardia. Termination of tachycardia was reliably achieved by rapid atrial pacing or appropriately timed double ventricular stimuli. Programmed

ventricular premature stimuli resulted in constancy of retrograde conduction time.

Fig. 5 Atrial premature stimuli (S_2) in case 2 at coupling intervals of 440 ms (upper panel) and 320 ms (lower panel). RVAE, right ventricular apical electrogram.

Fig. 6 Three forms of tachycardia in case 2.

Discussion

The presence of a short PR interval and narrow QRS complex on the surface electrocardiogram is frequently associated with paroxysmal tachycardias (Lown *et al.*, 1952; Castellanos *et al.*, 1971). In recent years electrophysiological studies have shown abnormal responses to atrial pacing in many of these patients. In normal subjects, incremental atrial pacing or programmed atrial premature stimuli at progressively shorter coupling intervals results in smooth prolongation of AH conduction (Wit *et al.*, 1970). In patients with the short PR interval, normal QRS complex syndrome this prolongation of the AH interval may be attenuated or absent (Castellanos *et al.*, 1971; Bisset *et al.*, 1973; Caracta *et al.*, 1973; Denes *et al.*, 1974; Neuss *et al.*, 1975a).

This response has been attributed to complete or partial bypass of the AV node by accessory tracts (James, 1961) inserting into the lower AV junction. The existence of a bypass tract has been proven histologically in one patient with this syndrome (Brechenmacher *et al.*, 1974).

In both patients described here the AH interval increased minimally in response to incremental atrial pacing and programmed atrial premature stimuli, suggesting that conduction to the ventricles was not occurring in the normal delaying pathways. Conduction in a 'fast pathway' with short functional refractoriness such as an atrionodal bypass, at earlier coupling intervals of atrial premature stimuli results in preactivation of the AV junction. The short AH functional refractoriness (Bisset *et al.*, 1975) may result in prolongation of the H₂V₂ interval and bundle-branch block at early coupling intervals (Coumel *et al.*, 1972; Seipel *et al.*, 1976). Fig. 7 is a diagrammatic representation of this mechanism.

Dual intranodal pathways have been demonstrated in animals and in patients with intra-AV nodal re-

entry (Moe and Mendez, 1966; Goldreyer and Damato, 1971; Denes *et al.*, 1973). A similar phenomenon may be seen in patients with the short PR interval, normal QRS complex syndrome and is thought to represent block in the fast intranodal or extranodal pathway (Denes *et al.*, 1974). In patients with duality of intranodal conduction onset of tachycardia during programmed atrial stimulation is associated with critical AH delay. In the patients described here, no sudden prolongation of the AH interval occurred and the major delay occurred distal to the site of His bundle recording. These observations suggest continued anterograde conduction in the fast pathway and the lack of effect of verapamil on tachycardia cycle length and AH interval, lends credence to this hypothesis. With critical AV conduction delay atrial echo beats or sustained tachycardia is initiated with return to the atria either via the AV node or a concealed direct VA connection.

The participation of a concealed direct VA connection (Spurrell *et al.*, 1974a; Tonkin *et al.*, 1975)

Fig. 7 Diagram of mechanism of AV junctional pre-excitation by atrial premature stimulus (star). AVN, atrioventricular node; ANP, atrionodal pathway; HIS, His bundle; A, atrium; V, ventricles.

Fig. 8 An atrial premature stimulus (star) may result in anterograde conduction delay sufficient to allow re-entry to the atrium (AE). Such re-entry may involve an atrionodal pathway (ANP) or a direct ventriculoatrial pathway (VAP). Critical conduction delay above the His bundle associated with a constant H-AE interval suggests re-entry within the AV node or via an atrionodal pathway. Delay below the site of the His bundle with a constant V-AE interval suggests re-entry via a direct VA pathway.

in the tachycardia circuit is suggested by the timing of the atrial echo beat. The constancy of the V_2A' interval in the presence of a varying H_2V_2 interval makes it unlikely that the re-entrant pathway is located entirely above the His bundle and implies the participation of the ventricles in the tachycardia circuit. Intra-AV nodal re-entry or re-entry in an atrionodal pathway results in nearly constant H_2A' intervals. If, however, the H_2V_2 is constant no conclusion can be drawn about the site of re-entry. Fig. 8 summarises these concepts.

The appearance of atrial echoes with critical HV conduction delay has been described previously (Lozano *et al.*, 1973; Varghese *et al.*, 1974; Neuss *et al.*, 1975b). Both Lozano *et al.* (1973) and Varghese *et al.* (1974) interpreted their findings as indicative of a combination of intraventricular and intra AV nodal re-entry. However, a concealed, direct, VA connection could not be excluded in their patients. The short AH in response to programmed atrial premature beats and the lack of AH prolongation with increasing prematurity of atrial stimulation suggests the presence of a fast anterograde pathway in the patient reported by Lozano *et al.* (1973). Neuss *et al.* (1975b) report a patient with similar findings to the patients described here. They also

observed constancy of the V_2A' interval in the presence of an increasing H_2V_2 interval in response to premature atrial stimuli delivered during sinus rhythm. This observation was regarded as proof of involvement of the ventricles within the re-entrant circuit. No comment was made concerning the mechanism of critical conduction delay. The absence of atrial echo beats or tachycardia at later coupling intervals of A_2 is probably related to limiting atrial refractoriness at the time of retrograde activation of the VA pathway (Pritchett *et al.*, 1977), or alternatively to concealed anterograde invasion of the VA pathway during atrial stimulation (Zipes *et al.*, 1974).

Additional evidence for an accessory concealed VA connection in our patients was provided by the following observations.

(1) In both patients the appearance of left bundle-branch block during tachycardias was associated with an increase in VA' conduction and tachycardia cycle length (Coumel and Attuel, 1974; Pritchett *et al.*, 1976; Spurrell *et al.*, 1974b).

(2) In case 1 coronary sinus recordings revealed early activation of the low left atrium near the proximal coronary sinus during tachycardia. This does not prove that retrograde activation is via a direct AV pathway since retrograde conduction in an atrionodal pathway may produce similar eccentric atrial activation. However, a ventricular premature stimulus, delivered 15 ms before the His bundle potential, conducted retrogradely with exact atrial capture and identical atrial activation sequence. Retrograde conduction in an atrionodal pathway would be impossible in these conditions and the retrograde limb of the re-entrant circuit, is therefore, a direct VA pathway (Sellers *et al.*, 1976).

The coexistence of anomalous atrionodal and atrioventricular pathways has been described previously (Coumel *et al.*, 1971, 1972; Neuss *et al.*, 1975b; Zipes *et al.*, 1974; Castellanos *et al.*, 1975) and unusual re-entry mechanisms may result from these combinations. The mechanism of re-entry in our patients is dependent on an interaction between two functionally abnormal pathways. Conduction delay at infra-Hisian level occurs as a result of pre-activation of the AV junction and retrograde conduction occurs when critical delay is sufficient to allow re-entry in the VA pathway and atrial myocardium.

In the patients described by Lozano and his colleagues (1973) tachycardia was initiated in the presence of a short AH interval which persisted during sustained tachycardia. These observations suggest the incorporation of a partial or complete AV nodal bypass tract in the anterograde limb of the re-entrant circuit. Re-entrant circuits utilising an

anterograde fast pathway bypassing the AV node and a retrograde direct VA pathway may result in rapid, distressing tachycardias unresponsive to therapy directed towards the AV node itself, and in these cases verapamil was not effective. Detailed electrophysiological studies are required to define the tachycardia mechanism in these patients.

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