Wolff-Parkinson-White syndrome *Circus movement tachycardia dependent on procainamide*

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A patient with Wolff-Parkinson-White syndrome was disabled by rapid ventricular rates during atrial arrhythmia and by periods of asystole. Circus tachycardia was induced by atrial stimulation only after the administration of procainamide. Nevertheless, treatment with this drug controlled the ventricular rate and spontaneous circus tachycardia did not occur.

The effects of antiarrhythmic drugs on conduction through accessory atrioventricular pathways and on the induction of tachycardia by atrial or ventricular stimulation have recently been recorded (Wellens and Durrer, 1974; Mandel *et al.*, 1975). This report concerns a patient with pre-excitation and life-threatening atrial arrhythmia. Circus movement tachycardia could be provoked by atrial stimulation only after the administration of procainamide.

Case report

A 57-year-old labourer was seen in January 1969, because of rapid palpitation. There were no significant clinical findings. The electrocardiogram showed sinus rhythm with type B ventricular pre-excitation. The PR interval was 0.08 s and the PS interval 0.22 s. Subsequently the attacks of palpitation were infrequent. Digoxin, 0.25 mg daily, and practolol, 50 mg t.i.d., were started at another hospital in 1973. He then had occasional syncopal attacks: loss of consciousness was brief and often incomplete.

In March 1975, he was admitted as an emergency after an attack. The electrocardiogram showed atrial flutter, rate 270 per minute, usually with 2:1 atrio-ventricular block and wide QRS complexes. There were frequent brief episodes of 1:1 conduction and it was suspected that prolonged periods with a ventricular rate of 270 were responsible for the syncope. However, monitoring indicated that the spontaneous cessation of paroxysmal atrial flutter or, at times, fibrillation was followed by asystole which was often of sufficient duration to cause loss of consciousness. That digitalis was a factor in causing sinus node dysfunction was proved by withdrawal and subsequent rechallenge with digoxin. Verapamil also delayed recovery (Fig. 1). During sinus rhythm pre-excitation persisted but the PR and PS intervals had increased to 0.13 and 0.24 s.

ELECTROPHYSIOLOGICAL STUDY

Forty-eight hours after the discontinuation of digoxin and practolol, His bundle activity was recorded using a tripolar electrode introduced percutaneously into the left femoral vein. An ink-jet

FIG. 1 Prolonged asystolic pause following cessation of atrial flutter. Treatment: oral verapamil 40 mg t.i.d., a further dose of 6 mg had been administered intravenously 15 minutes before the record.

recorder was employed and records were made at a paper speed of 100 mm/s. A modified lead II electrocardiogram was recorded simultaneously. Two poles of a multipolar electrode, previously introduced for control of the asystolic pauses, which were in close proximity to the lower part of the lateral wall of the right atrium, were used to introduce bipolar test stimuli at a rate of 40 per minute.

During sinus rhythm, rate 65 per minute, pre-excitation was evident on the body surface lead (Fig. 2A). The PR interval was 130 ms. His bundle deflections were present in the intracardiac lead with a PH interval of 110 ms and an HV interval of 20 ms. Stimuli following 345 ms or longer after the spontaneous P wave were invariably conducted via the accessory pathway, as were three out of three stimuli with intervals of 300 to 305 ms. No stimulus with an interval shorter than 300 ms produced atrial depolarization. Five stimuli following the P wave at intervals of 320 to 340 ms were recorded. Four of the five were conducted to the ventricles entirely via the atrioventricular node. The beat induced by the most premature of these stimuli was followed by a close coupled beat conducted via the atrioventricular node (Fig. 2A). Pure atrioventricular nodal conduction was associated with an HV interval of 35 ms.

Procainamide, 140 mg (2.4 mg/kg), was injected intravenously over a 6-minute period. Right atrial stimulation now frequently caused prolonged episodes of tachycardia (Fig. 2B). During sinus rhythm (rate 65 to 70/min) ventricular aberration was reduced (PR 180 ms, PH 130, and HV 50 ms). Stimuli less than 370 ms after the P wave did not produce atrial depolarization. All 6 stimuli recorded with intervals of 370 to 440 ms induced sustained tachycardia, conducted entirely via the atrioventricular node, at a rate of 120 per minute. The HV interval for beats conducted exclusively via the atrioventricular node was now 60 ms. Tachycardia was repeatedly terminated by single premature stimuli or by trains of stimuli at 220/min (Fig. 2B).

Most test stimuli 500 ms or longer after the spontaneous P wave resulted in aberrantly conducted beats, but on five occasions stimuli at intervals of 500 to 685 ms were conducted entirely by the atrioventricular node, indicating depression of conduction in the accessory pathway throughout the cardiac cycle. None of these stimuli induced tachycardia.

MANAGEMENT

Although procainamide had been associated with the appearance of pacing-induced tachycardia, the

clinical problem was one of an uncontrollable ventricular rate during atrial arrhythmia. Treatment with slow release procainamide was started. Procainamide, 4 g per day, was required to control the ventricular rate during attacks (plasma level $3.0 \mu\text{mol/l}$ ($8.1 \mu\text{g/ml}$)). On this dosage exclusive atrioventricular nodal conduction during atrial arrhythmia was often apparent, the average heart rate was 130 per minute, and very short cycles were absent. Regular narrow complex tachycardia similar to that induced by pacing did not occur. During the 10-month period of follow-up he has been well and has not noticed palpitation.

Discussion

Drugs which block conduction in the accessory pathway, such as procainamide, may prevent the induction of tachycardia in patients with the Wolff-Parkinson-White syndrome. Thus the initiation of tachycardia by premature stimuli was prevented in 6 of 10 patients given procainamide, quinidine, or ajmaline, studied by Wellens and Durrer (1974) and in 6 of 9 patients given procainamide reported by Mandel *et al.* (1975). However, Wellens and Durrer predicted that drugs that selectively prolong the refractory period of the accessory pathway may also widen the range of premature beat intervals at which tachycardia can be initiated. Furthermore, they described 3 patients in whom tachycardia could be induced by critically timed stimulation only after administration of a drug with this effect. In 2 of these patients only ventricular stimulation resulted in tachycardia, and in the third quinidine appeared to permit the initiation of circus movement tachycardia during atrial stimulation by preventing an atrial re-entrant beat from blocking the atrial component of the tachycardia pathway. Four patients in whom tachycardia could not be provoked by the extrastimulus technique were included in the study by Mandel *et al.* (1975). In none of these was the initiation of tachycardia after procainamide administration reported.

In this patient, procainamide administration permitted the initiation of tachycardia by premature atrial stimulation. Though re-entry was possible before procainamide was given, the drug widened the range of premature stimulation intervals at which re-entry could be elicited, in association with an increase in the refractory period of the accessory pathway relative to that of the atrioventricular node.

Re-entry involving the accessory pathway is the likely mechanism of the coupled premature beat and episodes of tachycardia after atrial stimulation, since atrioventricular conduction of the initiating and subsequent beats was invariably via the atrioven-

FIG. 2 His bundle electrograms. (A) Asynchronous atrial pacing at 40/min. Paced beats in upper panel and on left of lower panel are conducted via the accessory pathway and H deflections do not precede ventricular activation. The stimulus on the right of the lower panel (P-to-stimulus 320 ms) induces a beat conducted via the His bundle which is followed by a single, similarly conducted beat. (B) After procainamide 140 mg intravenously. The delta wave is smaller. The stimulus in the upper panel initiates tachycardia during which the ventricles are activated via the normal pathway. Tachycardia was terminated by a series of stimuli at 220/min (lower panel). S=right atrial stimuli; C=stimuli capturing the atria during rapid pacing.

tricular node. It seems impossible to attribute the different effects of stimulation before and after procainamide to chance variations in the timing of the stimuli. Sustained tachycardia did not occur during several minutes of stimulation at the beginning of the study, yet recurred only seconds after the end of each episode after procainamide administration.

Since the 4 normally conducted beats induced during the control period resulted from stimuli with intervals comparable to those of other beats that were conducted via the accessory pathway, concealed anterograde accessory pathway conduction following these 4 stimuli may have hindered subsequent retrograde conduction, explaining the difficulty in initiating re-entry. In support of the possibility that procainamide abolished concealed anterograde conduction, the ventriculoatrial interval before the first re-entrant atrial complex of induced episodes of tachycardia (265 ms) was shorter than the corresponding interval before the single re-entrant beat elicited before procainamide administration (280 ms), consistent with indirect enhancement of retrograde conduction by the drug.

A low amplitude deflection is apparent on the His bundle lead 155 ms after the onset of the V deflection of the single re-entrant beat (Fig. 2A). Similar deflections occurred following 2 of the 3 other normally conducted beats that did not initiate circus movement tachycardia. If these low amplitude deflections were atrial in origin and resulted from early retrograde conduction in the accessory pathway, failure to establish tachycardia could have been associated with subsequent anterograde block of these premature atrial beats in the atrioventricular node. Slowing of retrograde conduction by procainamide may have delayed the arrival of the impulse at the atrioventricular node until after the end of its refractory period, permitting completion of the tachycardia circuit.

The ease with which the effect of procainamide in facilitating re-entry during atrial stimulation was shown may be explained by the low dose of the drug employed. The patients in the other reported studies received 10 mg/kg compared with 2.4 mg/kg administered to this patient. Higher doses might have prevented tachycardia by blocking retrograde conduction in the accessory pathway.

The contrast between the result of atrial stimula-

tion and that of spontaneous atrial premature beats, and the absence of spontaneous circus movement tachycardia during procainamide therapy require comment. The prematurity of atrial stimuli which induced circus movement tachycardia after procainamide but never induced atrial flutter or fibrillation was comparable to that of spontaneous ectopics which precipitated atrial arrhythmias but never circus tachycardia. The paced region low in the right atrium was probably quite close to the accessory pathway, since regular pacing at a rate slightly faster than the sinus rate was associated with a reduction in the PR interval to 0.08 s. The rapid conduction of a premature stimulus to the adjacent accessory pathway would tend to favour atrioventricular nodal conduction if the bypass were still refractory, and the induction of tachycardia. Spontaneous atrial ectopics may have arisen proximal to a potential atrial re-entry site (e.g. the sinoatrial node) and initiated atrial arrhythmia.

Prolongation of the PR interval with negligible increase in the PS interval occurred during the 6-year period of observation and correlated with the appearance of severe arrhythmias. Disease of the accessory pathway or its atrial connexions may have developed. The appearance of drug-induced sinus node dysfunction supports the latter possibility. Spontaneous arrhythmia is more likely to have been the result of pathological changes in the atria than in the accessory pathway. Though H deflections preceded the delta wave during sinus rhythm, this was not the case during atrial stimulation (Fig. 2A). The accessory pathway, is, therefore, truly atrioventricular, rather than His-ventricular.

The patient was clearly at considerable risk of sudden death. Therapy with procainamide reduced the risk.

References

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