Long-term control of reciprocating paroxysmal tachycardia by ventricular pacing in a case of Wolff-Parkinson-White syndrome

G NILSSON, I RINGQVIST

From the Departments of Medicine and Clinical Physiology, Central Hospital, Västerås, Sweden

SUMMARY A patient is described with a complex electrocardiographic pattern, including pre-excitation, grade I atrioventricular block, reciprocal rhythm of atrial origin, and frequent attacks of reciprocating tachycardia at a rate of about 135 beats per minute. Long-term control of these attacks was obtained by synchronous ventricular pacing at 80 beats a minute, which was below the rate during the attacks of tachycardia and above the spontaneous heart rate between the attacks.

Pacemaker treatment has been used for different purposes in cases of paroxysmal tachycardia. Firstly, magnetically induced atrial or ventricular asynchronous pacing has been successfully used in terminating attacks of paroxysmal tachycardia of both ventricular and supraventricular origin. Secondly, prophylactic pacing has been used in order to avoid severe sinus bradycardia and atrioventricular block when large doses of antiarrhythmic drugs are required to prevent such attacks. Thirdly, long-term atrial or ventricular pacing at a rate above the spontaneous heart rate but below the rate of the tachycardia has been described. This technique has been used mainly for ventricular tachycardia but also in a few cases of supraventricular tachycardia.^{1,2} The present report concerns a patient successfully treated for paroxysmal reciprocating tachycardia of atrial origin by means of ventricular pacing below the rate of the tachycardia. Furthermore, this patient had alternating atrioventricular block and pre-excitation, suggesting a complex defect in the atrioventricular connection.

Case report

The patient was a 32 year old man previously healthy apart from his heart disease. His father had had a myocardial infarction when he was 55 years old. His mother suffered from diabetes, but was otherwise well. He had one brother who was healthy. The patient had a physically light office job and performed mild exercise in the holidays without any particular shortness of breath. He had never had chest pain.

Since puberty he had suffered from troublesome daily attacks of palpitation. After fainting at 16 years of age, auscultation disclosed an irregular heart rhythm, with paroxysmal attacks of tachycardia at a rate of about 140, but no other abnormality. The chest x-ray film showed moderate cardiomegaly without enlargement of any specific heart chamber. The blood pressure was 115/70 mmHg. The electrocardiogram was similar to those recorded later and is described below. A coronary angiogram was normal. On exercise testing the patient managed 900 kpm for four minutes and then gave up because of shortness of breath. The attacks of reciprocating tachycardia increased in duration and frequency during exercise. He was first treated with verapamil 80 mg three times a day which was later replaced by digitoxin 0.1 mg once a day without improvement.

When first seen at our hospital at the age of 32 years his condition had been unchanged for many years. The clinical, electrocardiographic and exercise findings were unaltered. Coronary arteriography was not repeated. Echocardiography showed moderate enlargement of the left atrium and left ventricle. There was reduced movement of the walls of the latter, which were of normal thickness. These findings suggested some form of cardiomyopathy. There were no valvular lesions. The electrocardiogram showed a complex variable picture.

(1) Sinus rhythm with a prolonged PR interval (0.22-0.28 s) (Fig. 1).

(2) Pre-excitation with a PR interval of 0.10 s, a widened QRS complex, and slurred delta waves (Fig. 2).

Fig. 1 Reciprocating tachycardia. Rate 135/minute. On the right of the figure there is bradycardia, at 54/minute, because of reciprocal rhythm of atrial origin. The reciprocal atrial activation discharges and resets the sinus node. PR prolonged at 0.28 s (trace recorded at 25 mm/s).

Fig. 2 (a) Shows pre-excitation with a short PR interval and widened QRS complex with slurred delta waves (trace recorded at 50 mm/s). (b) Illustrates escape capture bigeminy (trace recorded at 25 mm/s).

(3) Reciprocal rhythm of atrial origin with a P-QRS-P' configuration (Fig. 1). The occurrence of P' was strongly positively correlated with the length of the preceding PR interval. Retrograde conduction to the atria required a PR interval of at least 0.26 s. The RP' interval was fixed at 0.17 s and P' was superimposed upon the ST segment. The retrogradely conducted impulse P', which had a frontal plane vector of about -90° , discharged the sinoatrial node and thereby postponed the ensuing cycle. This resulted in long periods of bradycardia at a rate of about 50 beats a minute.

(4) The bradycardia described above was sometimes replaced by escape capture bigeminy because of the escape of an idionodal or high ventricular pacemaker (Fig. 2). This escape beat was occasionally preceded by a non-conducted sinus impulse.

(5) Attacks of reciprocating paroxysmal tachycardia at about 135/minute (Fig. 1 and 3) which always began with a sinus P wave after a long RR interval (1.5 to 1.7 s), caused by the discharge and resetting of the sinus pacemaker by reciprocal retrograde atrial activation. The sinus P wave beginning the paroxysm was conducted with first degree atrioventricular block (PR

Fig. 3 (a) *Reciprocating tachycardia*. Note right bundle-branch block caused by phasic aberrant conduction of the second beat in the last paroxysm of tachycardia (trace recorded at 50 mm/s). (b) *Ventricular pacing at a rate of 80*. Note retrograde atrial activation (trace recorded at 25 mm/s).

interval 0.26 to 0.28 s). The electrocardiogram was consistent with a re-entry tachycardia probably using an abnormal conduction pathway between atria and ventricles. The existence of such a condition can be deduced both from the pre-excitation as well as from the retrograde atrial activation previously described. During tachycardia the RP' interval was 0.17 s and P'R 0.28 s. All attacks of paroxysmal tachycardia were terminated by a P', indicating retrograde atrial activation.

(6) The second beat in the paroxysms often had a right bundle-branch block configuration, reflecting phasic aberrant ventricular conduction because of the long preceding RR interval (Fig. 3).

(7) A few unifocal ventricular premature beats were found only after long preceding RR intervals, indicating secondary ventricular extrasystoles (Fig. 3).

The electrocardiographic findings suggested a trial of pacing might be effective. The long RR interval invariably preceding the attacks of tachycardia could be suspected of being of pathogenetic significance for these attacks, in which case pacing would suppress them. Furthermore, antiarrhythmic drugs in high doses could be used more safely during pacing. Because of the intermittent atrioventricular block we preferred ventricular rather than atrial pacing and placed a temporary pacemaker electrode in the right ventricle. Before pacing, a 24 hour recording of the electrocardiogram showed that the paroxysms of reciprocating tachycardia were present 10% of the time and the patient experienced palpitation. At a pac-

ing rate of 70/minute the frequency and duration of the paroxysms diminished conspicuously and at a rate of 80 they disappeared totally at rest. This was confirmed with 16 hours of continuous monitoring. The heart rate of 80 was not accompanied by palpitation and the patient felt well. The electrocardiogram showed pacemaker-induced ventricular activation with retrograde atrial activation (Fig. 3). During exertion the paroxysms occasionally returned. When the pacing rate was increased their frequency fell but the patient then noted palpitation at rest.

When the pacing impulse by asynchronous pacing appeared in a critical phase of the re-entry circuit in reciprocating tachycardia it should have stopped the tachycardia, but this did not happen and so we therefore ultimately fitted the patient with a permanent synchronous (QRS inhibited) pacemaker (Microlith), with a basic rate of 80/minute. The patient was also treated with metoprolol (100 mg) twice a day and digitoxin 0.25 mg daily, and he remained well. Four hours ambulatory electrocardiographic recording disclosed only paced rhythm. During the first 30 Watt load of an exercise test paced rhythm persisted; at 60 Watts there was alternation between paced and sinus rhythm; at 90 and 120 Watts there was sinus rhythm only with a maximum rate of 150/minute.

Discussion

The combination of pre-excitation, reciprocal rhythm of atrial origin, and reciprocating tachycardia in the

same patient fits well with the assumption of an anomalous atrioventricular communication. The combination is rare and we have found only one previously described patient with the combination.³ This patient had an electrocardiographic picture very similar to our patient and was subjected by Schamroth to a detailed electrocardiographic analysis.³ Similar cases have also been described by Krikler *et al.*^{4,5} The reciprocal rhythm of atrial origin and reciprocating tachycardia are readily explained by a differential refractoriness between the normal and the anomalous pathway. It is thus known that the anomalous atrioventricular bypass in the Wolff-Parkinson-White syndrome usually has a longer refractory period but a faster conduction rate than the normal atrioventricular communication.⁶ A critically timed impulse may therefore penetrate the normal communication and return to the atria through the abnormal communication, thereby creating a circus movement. The relation between a long PR interval and reciprocal atrial activation in our patient is best explained by an upper common pathway requiring a relatively long reciprocal time to recover. On the other hand, the presence of grade I atrioventricular block and pre-excitation in the same electrocardiographic tracing points to alternative pathways within the atrioventricular node. One pathway may have normal conduction velocity and connections with the abnormal atrioventricular communication, thereby creating the typical fusion ventricular activation complex of pre-excitation. Another pathway may have a slowed conduction and no connection with the abnormal atrioventricular communication, thereby creating grade I atrioventricular block with a narrow QRS complex. The complex labyrinthine structure of the atrioventricular node⁷ may form the anatomico-physiological basis for such different pathways.

The paroxysmal re-entry tachycardia in the Wolff-Parkinson-White syndrome is seldom so troublesome that permanent pacing is justified. A similar case was described with successful dual demand pacemaking designed to switch into the fixed rate mode (at 70 beats a minute) when the cardiac rate exceeded 150.⁵ The findings in our patient indicate that synchronous ventricular pacing at a relatively high basic rate (80 beats a minute) may be valuable in preventing paroxysms of re-entry tachycardia. The retrograde P' wave during pacing has an identical appearance to the P' during reciprocal rhythm of atrial origin. This is a

very strong argument that P' is not an ectopic premature atrial beat. The prevention of paroxysmal supraventricular tachycardia by pacing at a rate below that of the tachycardia but above that of the heart rate between the paroxysms has previously been described by Moss and Rivers² and Sowton *et al.*⁸ and is of considerable theoretical and practical interest. The electrophysiological explanation of the good results in our case seems to be, at least partly, the use of the anomalous atrioventricular bypass in retrograde conduction of the pacing impulse. The non-refractory period of this pathway is probably too short to allow its participation in the re-entry circus movement of the reciprocating tachycardia. From a theoretical point of view such occupation of the anomalous pathway by retrogradely conducted pacing impulses indicates that artificial pacing may be of value in preventing attacks of tachycardia in patients with the Wolff-Parkinson-White syndrome.

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Requests for reprints to Dr Göran Nilsson, Medical Department, Central Hospital, S-721 89 Västerås, Sweden.