

Case reports

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Atrioventricular reciprocal rhythm and chronic reciprocating tachycardia in a newborn infant with concealed Wolff-Parkinson-White syndrome

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A case of atrioventricular reciprocal rhythm and chronic reciprocating tachycardia in a newborn infant is presented. Electrophysiological studies suggest that these rhythm disturbances are related to the presence of a right-sided atrioventricular accessory pathway capable only of retrograde conduction (concealed Wolff-Parkinson-White syndrome). The technique of recording the sequence of atrial activation during the tachycardia is described and its clinical importance emphasised.

An impulse originating in the sinus node or in the atria may, in its course to the ventricles, retrogradely reactivate the atria. This phenomenon, when perpetuated, may produce a run of reciprocating tachycardia (Kistin, 1965). The presence of dual pathways or the development of functional longitudinal dissociation within the atrioventricular node is known to favour its occurrence (Mendez and Moe, 1966; Denes *et al.*, 1973). Experimental studies have shown that such a phenomenon can also occur in the sinus node (Han *et al.*, 1968) and in atrial tissue (Allessie *et al.*, 1973). An additional analogous situation is the existence of an accessory atrioventricular pathway in the Wolff-Parkinson-White syndrome which permits rapid conduction in a ventriculoatrial direction (Wolff, 1959; Roelandt and Van der Hauwaert, 1968). This paper relates the occurrence of atrioventricular reciprocal rhythm and chronic reciprocating tachycardia in a newborn infant with 'concealed' WPW syndrome in which an atrioventricular accessory pathway is capable only of retrograde conduction (Zipes *et al.*, 1974; Neuss *et al.*, 1975; Wellens and Durrer, 1975). The technique of recording the sequence of atrial activation during tachycardia is described and its clinical importance emphasised.

Case report

A 23-year-old black woman was diagnosed as having a breech position of the fetus at the 38th week of gestation. Simultaneously, fetal distress was clinic-

ally suspected because of the finding of a 'cardiac arrhythmia' on auscultation of the fetal heart sounds. Subsequently, a caesarian section was performed. The birthweight of the baby boy was 4024 g (7 lb 3 oz), and the baby appeared normal. However, frequent episodes of abrupt onset and termination of a tachycardia at a rate of 250/minute (Fig. 1) were noted immediately after birth. Since neither gross cardiac anomaly nor cardiac decompensation was present, the newborn was observed for 10 days and was discharged without any medication.

Two weeks later, the baby was admitted with a history of anorexia and vomiting of 4 days' duration. On admission, now 24 days old, the baby was afebrile, acyanotic, but pale and tachypnoeic, with a respiration rate of 80 per minute. Blood pressure was 70/40 mmHg in the arms and the apical heart rate was 250/minute and regular. No murmurs were audible. There were moist râles at both lung bases. The liver was palpable 3.5 cm below the right costal margin. Other findings were unremarkable. The chest x-ray film indicated slight cardiac enlargement and a moderate degree of pulmonary venous engorgement. Laboratory data and clinical observations indicated no evidence of thyroid dysfunction or myocarditis. On the electrocardiogram, there was right ventricular predominance compatible with the age, and the rhythm disturbance was the same as had been observed since birth (Fig. 1). The mechanism by which this rhythm disturbance occurred was interpreted as an

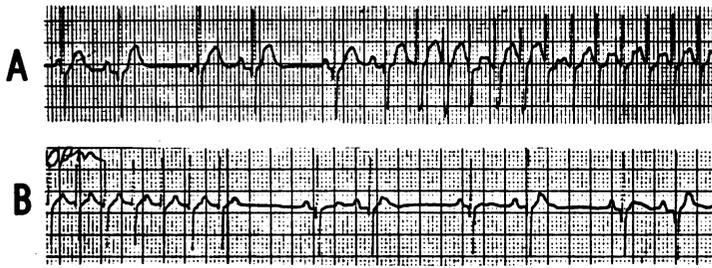


Fig. 1 Abrupt onset and termination of tachycardia. The distorted T waves corresponding to the 2nd and 4th QRS complexes in the upper panel and the 9th, 11th, and 13th QRS complexes in the lower panel were interpreted as non-conducted 'premature atrial beats' with resetting of the cardiac cycle length. The tachycardia was considered as a form of 'ectopic atrial tachycardia'. Panels A and B were not continuous.

'ectopic atrial tachycardia' and frequent 'non-conducted premature atrial beats'. The boy was given diuretic therapy and was accordingly digitalised (digoxin 0.14 mg I.V. in 24 hours followed by 0.09 mg orally per day). Intermittent episodes of the tachycardia persisted despite gradual increase in oral digoxin dosage (serum concentration up to 3.5 ng/ml). Digoxin was then discontinued and propranolol instituted (0.095 mg I.V. followed by 0.025 mg orally every 6 hours). The dosage of propranolol was also gradually increased but failed to control the arrhythmia. After discontinuance of propranolol for 2 days, cardiac catheterisation was performed in a post-absorptive basal state after pethidine and promethazine sedation. Only a patent foramen ovale was found.

ELECTROPHYSIOLOGY

After completion of the above study, a tripolar electrode catheter was introduced from the right femoral vein, and under fluoroscopy, positioned across the septal leaflet of the tricuspid valve to

obtain His bundle electrograms. As usual, low septal right atrial activity was recorded from the same electrode catheter as well. Through the same femoral vein, an additional tetrapolar electrode catheter was placed in the atria for the purpose of recording atrial electrograms and pacing the corresponding atrium. During sinus rhythm, the PA, AH, and HV intervals were 25, 80 and 30 ms, respectively (Fig. 2). These values were normal for the age (Roberts and Olley, 1972). Of note, there were frequent P-QRS-P' complexes interrupting the cardiac cycle. The electrocardiographic characteristics of these P-QRS-P' complexes fulfilled the diagnostic criteria of a 'reciprocal rhythm' (Kistin, 1965; Roelandt and Van der Hauwert, 1968) in which the P' waves were related to retrograde activation of the atria (the low septal right atrium in the vicinity of the atrioventricular node was activated 20 ms before the high right atrium) (Fig. 2). The question arises whether the P' waves were not, in fact, premature beats from an atrioventricular nodal or low atrial focus. The

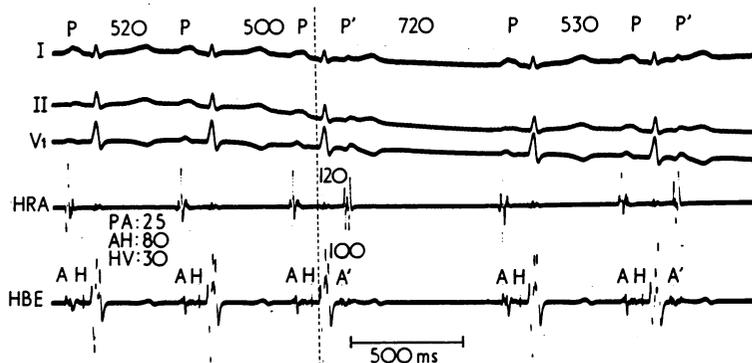


Fig. 2 Atrial reciprocal rhythm. Initiation of atrial reciprocal (echo) beats, P-QRS-P' complexes (the 3rd, and 5th QRS complexes), related to spontaneous shortening of the sinus cycle length. The PA, AH, and HV intervals (25, 80, and 30 ms, respectively) were normal for age. Using the onset of ventricular depolarisation (dash line) as a reference, the low septal right atrium (A') in the His bundle electrocardiographic lead (HBE) was activated 20 ms before the high right atrium (HRA) during the P' waves (V-LRA=100 ms; V-HRA=120 ms).

following prominent features negate this possibility. First, the occurrence of P' waves was invariably preceded by shortening of the sinus cycle length. As shown in Fig. 2, spontaneous shortening of the sinus cycle length from 520 to 500 and from 720 to 530 ms, respectively, induced a P' wave. Secondly, high right atrial pacing at a cycle length of 500 ms with only minimal increase in AH conduction time (5 ms increment) constantly induced 1:1 reciprocal rhythm (P-QRS-P' complex) (Fig. 3). Thirdly, the QRS-P' interval was constant in every P-QRS-P' complex. These selective links, i.e. cycle length dependency and constant QRS-P' interval, would be too fortuitous for atrioventricular nodal or atrial premature beats to occur. Furthermore, the reciprocal rhythm, when perpetuated, initiated a run of reciprocating tachycardia with a rate of 250/minute (cycle length 240 ms) (Fig. 4A). During the tachycardia, the sequence of atrial activation was the same as those of P' waves during the reciprocal rhythm—the low septal right atrium being activated 20 ms before the high right atrium. More than 20 spontaneous bouts of this tachycardia were observed during the study. Without exception, in each instance, a gradual decrease in the sinus cycle length was a requisite condition for initiating tachycardia.

In order to record the sequence of atrial activation for the localisation of the pathway of ventriculoatrial conduction during the tachycardia (Gallagher *et al.*, 1975; Wellens and Durrer, 1975), the tetrapolar catheter was manipulated during the tachy-

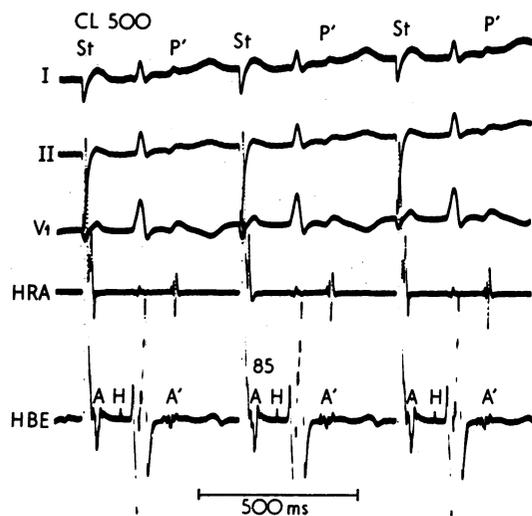


Fig. 3 High right atrial (HRA) pacing at cycle length (CL) of 500 ms induced 1:1 reciprocal rhythm (P-QRS-P' complex).

cardia. Through the interatrial communication, the electrode catheter was placed in the left atrium, and it was noted that the left atrium was activated 15 ms after the low septal right atrium in the vicinity of the atrioventricular node during the tachycardia (Fig. 4B). The electrode catheter was then positioned in the low lateral right atrium at its junction with the inferior vena cava to record the low lateral right atrial electrogram, and this revealed that low lateral right atrial activation preceded low septal right atrial activation by 20 ms during the tachycardia (Fig. 4C). Thus the sequence of retrograde atrial

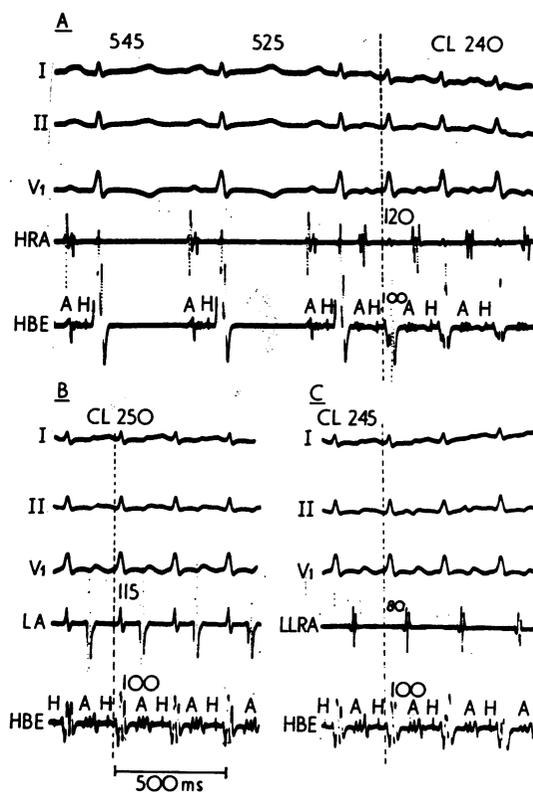


Fig. 4 (A) Spontaneous onset of a run of reciprocating tachycardia preceded by a shortening of the sinus cycle length (CL) from 545 to 525 ms. During tachycardia (CL=240 ms), the low septal right atrium (in HBE) is activated 20 ms before the high right atrium (HRA) (V-LRA=100 ms; V-HRA=120 ms). The dash line demarcates the onset of ventricular depolarisation. (B) During reciprocating tachycardia, the left atrium (LA) was activated 15 ms after the low septal right atrium (in HBE) (V-LA=115 ms; V-LRA=100 ms). (C) During reciprocating tachycardia, the low lateral right atrium (LLRA) was activated 20 ms before the low septal right atrium (in HBE) (V-LLRA=80 ms; V-LRA=100 ms).

activation during tachycardia in this newborn started at low lateral right atrium, followed by low septal right atrium and then left atrium and high right atrium, suggesting the presence of a right-sided accessory atrioventricular pathway conducting in a ventriculoatrial direction (Gallagher *et al.*, 1975; Wellens and Durrer, 1975).

Electrical stimulation was performed in both atria. In no instance did ventricular pre-excitation ever become evident (anterograde conduction block in the accessory pathway, thus concealed on the surface electrocardiogram). Premature stimulation of either atrium could easily provoke and terminate a run of reciprocating tachycardia. Of note was the observation that late premature atrial beats which induced no or only minimal atrioventricular nodal conduction delay could trigger the onset of a run of tachycardia similar to what have been recently described in patients with 'concealed' WPW syndrome (Zipes *et al.*, 1974; Neuss *et al.*, 1975; Sung *et al.*, 1976).

Since the natural history of 'concealed' WPW syndrome is unknown at this young age, the newborn was discharged on a combination of digoxin (0.15 mg daily) and propranolol (7.5 mg every 6 hours) in addition to diuretic therapy. In a follow-up period of 6 months, episodes of atrioventricular reciprocal rhythm and reciprocating tachycardia persisted. However, the patient had remained clinically stable.

Discussion

That atrioventricular reciprocal rhythm and reciprocating tachycardia could be related to the existence of an accessory pathway capable only of retrograde conduction (concealed WPW syndrome) was first postulated by Codina-Altés and Pijoan de Beristain (1950) and later assumed by Eldridge (1958) and Schamroth (1960). The presentation of this case has thus confirmed this hypothesis. The following observations suggest that a right-sided accessory atrioventricular pathway (Kent bundle, type B) was responsible for the rhythm disturbances in this newborn infant.

(1) Abnormal sequence of retrograde atrial activation during the reciprocating tachycardia, i.e. the low lateral right atrium was activated 20, 35, and 40 ms before the low septal right atrium, the left atrium, and the high right atrium, respectively, indicating the presence of a right-sided accessory pathway conducting in a ventriculoatrial direction (Gallagher *et al.*, 1975; Wellens and Durrer, 1975).

(2) Anterograde conduction block in the accessory pathway, since ventricular pre-excitation was not evident during sinus rhythm, nor reciprocating

tachycardia, and could not be induced by pacing either atrium.

As we have previously pointed out (Sung *et al.*, 1976), the initiation of a reciprocating tachycardia in patients with 'concealed' WPW syndrome may not require a triggering mechanism provided by a premature atrial beat owing to the presence of pre-existing anterograde unidirectional block in the accessory pathway. Under these circumstances, shortening of the sinus cycle length appears to be one of the factors favouring spontaneous onset of a reciprocating tachycardia. This is probably related to decrease in the refractory period of the atria and the accessory pathway, or a decrease in the depth of anterograde penetration of the accessory pathway, allowing the impulse to reactivate the atrium retrogradely; consequently, an atrioventricular reciprocal rhythm and/or a reciprocating tachycardia ensues. Spontaneous onset of a reciprocating tachycardia without an antecedent extrasystole can also be observed in patients with electrocardiographically manifest WPW syndrome (Krikler *et al.*, 1976) and in patients with dual atrioventricular nodal pathways (Coumel, 1975) when the sinus cycle length shortens to a critical point, at which anterograde unidirectional block develops in the accessory atrioventricular pathway and in one of the two intranodal pathways, respectively.

Management of chronic reciprocating tachycardia in patients with concealed WPW syndrome may present a therapeutic dilemma. Since anterograde unidirectional block is already present, the initiation of reciprocating tachycardia depends on the impulse arrival time at the ventricular end of the accessory pathway relative to the refractory periods of the corresponding atrium and the accessory pathway (Sung *et al.*, 1976). Digitalis and propranolol tend to prolong conduction time in the atrioventricular node which is a part of the tachycardia circuit. Therefore, both drugs can be used to prevent or interrupt the reciprocating tachycardia. However, prolongation of the atrioventricular nodal conduction time delays the impulse arrival at the ventricular end of the accessory pathway. Besides, digitalis may shorten the refractory periods of the atrium and the accessory pathway (Wellens and Durrer, 1973), and thus facilitate an onset of reciprocating tachycardia in these patients. Further studies on the efficacy of various pharmacological agents on the reciprocating tachycardias in patients with concealed WPW syndrome are needed.

In a long-term follow-up study on 62 infants and children with the WPW syndrome, Giardina *et al.* (1972) noted that the episodes of paroxysmal supraventricular tachycardia usually responded

promptly to digitalis in the usual therapeutic dosage, and 'the prognosis into adult life' was quite good. Others (Lubbers *et al.*, 1972; Nadas *et al.*, 1952) had drawn similar conclusions. Nevertheless, these studies probably did not include patients with concealed WPW syndrome, since electrocardiographic evidence was the only criterion for inclusion. Thus, the true incidence of concealed WPW syndrome may be much greater than is generally realised. The report of this newborn with the concealed WPW syndrome which probably began in fetal life, and later, the follow-up will certainly add to the knowledge of the natural history of patients with the WPW syndrome.

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