Case Reports

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Reversed reciprocal rhythm

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An example of reversed reciprocal rhythm is presented with associated 2:1 heart block and other abnormalities of conduction. The electrocardiograms have been analysed and compared with an earlier electrocardiogram in which sinus rhythm was present. Despite dramatic electrocardiographic abnormalities, evidence is offered which suggests that there may have been only a small alteration in activity of the already damaged cardiac conducting tissue.

Analysis of this patient's electrocardiograms may be of value in understanding the behaviour of abnormal cardiac conducting tissue.

The term *reciprocal rhythm* was introduced by Dock (1928) for the situation where impulses arising in the AV node or ventricle are conducted to the atrium, with return of conduction to the ventricles (Fig. 1a). Examples of reciprocal rhythm show the electrocardiographic sequence of a QRS complex, abnormal P wave, then a second QRS complex. An example of reciprocal conduction with origin in the AV node was described as early as 1915 by White.

Reversed reciprocal rhythm is exceedingly rare. It appears on the electrocardiogram as a P wave followed by a QRS complex, then a second P wave of different shape. This sequence is believed to represent conduction from the atrium to the ventricle, with return of conduction to the atrium from somewhere along the AV pathway (Fig. 1b).

Both these arrhythmias require that re-entry occur. This consists of return of the impulse via part of the route already taken. As pointed out by Schmitt and Erlanger (1928), re-entry can be explained by assuming there is unidirectional block in that part of the conducting pathway which conducts retrogradely. This mechanism was also invoked by Wolferth and McMillan (1929) to explain their Case 2 which was the first recorded example of reversed reciprocal rhythm. The returning impulse also has to arrive at any common part of the pathway when this has become non-refractory. As suggested by Mines (1913), presumably sufficient time must

elapse between conduction in the two directions for excitability to recover.

Schamroth (1960) reviewed the 12 cases of reversed reciprocal rhythm reported by 1960, and added one of his own. In his example there were prolonged episodes of paroxysmal tachycardia, consisting of reciprocating rhythm with electrocardiographic evidence of alternating retrograde atrial depolarization, and anterograde ventricular depolarization. Mines (1913) showed that reciprocating rhythm readily occurs experimentally, but such sequences are rarely seen in man.

An example of reversed reciprocal rhythm is described here with an analysis of the

FIG. 1 (a) Pathway of conduction in reciprocal rhythm; (b) pathway of conduction in reversed reciprocal rhythm.

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associated arrhythmias. Further evidence for the retrograde nature of the abnormal P waves has been obtained by using an oesophageal lead.

Case history

The patient aged 65 years presented in 1962 with mild diabetes mellitus and a duodenal ulcer, blood pressure 140/80 mm. Hg, heart rate 75 a minute, and a normal heart clinically and radiologically. The electrocardiogram showed sinus rhythm, right bundle-branch block, and first degree heart block.

He improved on antacids, then remained well on a reduced carbohydrate diet until 1966. His dyspepsia then recurred, and his heart rate was found to be only 36 a minute (Fig. 2b) and blood pressure 180/70 mm. Hg.

Left heart failure has been controlled with thiazides since its onset in 1967, and he has suffered minimal glycosuria and intermittent dyspepsia.

He has had no ischaemic symptomatology other than trophic skin changes in his legs where the peripheral pulses are absent.

Electrocardiograms The initial electrocardiogram of 1966 (Fig. 2b) shows that the ventricular rate has fallen to half that of the sinus rhythm of 1962 (Fig. 2a), yet there is still a regular (sinus) P to QRS relation and the right bundle-branch block pattern persists. Anterograde conduction therefore follows the same route. However, P waves of the type found in retrograde atrial conduction (Winternitz and Langendorff, 1944) follow each QRS complex. Oesophageal leads (Fig. 3) confirm that they spread retrogradely through the atria. This sequence of sinus P wave, QRS complex, then retrograde P wave is the pattern of *reversed reciprocal rhythm*.

The usual rhythm is interrupted occasionally by episodes of reciprocating rhythm as seen in Fig. 2c (fourth and fifth QRS complexes). When this happens the next sinus P wave is followed by another, at a PP interval half that seen in most of this first basic rhythm. This suggests that retrograde atrial conduction causes *sinu-atrial* block in this rhythm.

FIG. 2 (a) Electrocardiogram 1962: right bundle-branch block and first degree heart block; (b) continuous rhythm strip (lead II) of first new basic rhythm since 1966; (c) continuous rhythm strip (lead II) showing spontaneous alteration of the rhythm of (b) with reciprocating rhythm sequences, revealing the true sinus rate, and including some 'nodal' complexes.

FIG. 3 Unipolar oesophageal lead patterns of reversed reciprocal rhythm in this patient.

When QRS complexes are not followed by 'retrograde' P waves (Fig. 2c, upright QRS complexes No. 8 and No. 9), the RP interval is greater than would occur with the PR intervals in most of the tracing. There is therefore also 2:1 heart block.

A second basic rhythm occurring since 1966 (Fig. 4) is analysed (Fig. 5) using measurements from the strip shown, and from a subsequent similar length. The QRS complexes followed by a 'retrograde' P wave (Fig. 5a) occur earlier than those not followed by one. The PR intervals of these two groups fall in different ranges, and where there is no following 'retrograde' P wave the QRS complexes each follow the preceding one by virtually the same interval (1.74 to 1.79 seconds). Anterogradely conducted atrial impulses therefore initiate reversed reciprocal conduction, while 'nodal' impulses (presumably arising between upper AV node and division of the left bundle) are not conducted to the atria.

As the sinus PP interval here is shorter than the minimum interval between ventricular complexes, 2:1 heart block is again present. Atrial impulses could therefore be conducted at half the sinus rate (i.e. at 29 a minute). The 'nodal' rhythm is faster than this (approximately 34 a minute), and there is the expected AV dissociation. The nodal rate here is the same as occurs in the first basic rhythm (Fig. 2c). As the sinus rate is slower (58 a minute) than when revealed in the first basic rhythm (72 a minute) the AV dissociation has

FIG. 4 Second basic rhythm since 1966, with ladder diagrams representing atrial and ventricular conduction (lead II).

FIG. 5 (a) Graph relating PR and RR intervals preceding any QRS complex; (b) graph relating PR interval to the interval between the preceding R wave and the subsequent sinus P wave (RP interval). In both (a) and (b), each closed circle represents a QRS complex not followed by a 'retrograde' P wave.

arisen by default rather than by usurpation (Miller and Sharrett, 1957).

'RP/PR reciprocity' seen in Fig. 5b is consistent with the arrival of conducted atrial impulses during the relatively refractory period of the AV node (Mendez, Gruhzt, and Moe, 1956; Moe, Preston, and Burlington, 1956). Atrial impulses not conducted when in the shorter RP range fall in the 'critical phase'.

Since RP/PR reciprocity is obeyed in the same way after both conducted and 'nodal' complexes, the 'nodal' impulses must influence the site of delay of AV conduction. But the PP interval of the *paired consecutive capture beats* seen in Fig. 4 (QRS complexes 5 and 6; 14 and 15) is greater than the 'nodal' interval. As pointed out by Schamroth and Friedberg (1965), this means that the AV delay must occur above the site of origin of 'nodal' impulses. Therefore, there is retrograde conduction from the site of origin of 'nodal' impulses in this patient, and this influences the upper AV node in the same way as occurs after reversed reciprocal rhythm sequences. The similarity of the delay in subsequent anterograde conduction in both cases seems likely to have arisen as a result of retrograde conduction in each case. This refractoriness will have a delayed onset, and this may be responsible for the 2:1 heart block. A further contributory factor is that retrograde conduc-

tion also always blocks the next sinus impulse so that this does not arrive at the AV node after the shortest cycle lengths when the refractory period would normally be at its minimum (Mendez *et al.*, 1956). It can be seen that whatever gave rise to retrograde conduction in the first place may have caused the whole new complex of arrhythmias.

In the present patient there has been little change in these rhythms since their onset in 1966. The responsible process since then has presumably been chronic and may have been so before these arrhythmias began. Damage to part of the AV node may have gradually reached the stage when conduction would only occur retrogradely. It is therefore possible that further AV nodal damage, or a depressant of AV conduction, would give rise to sinus rhythm by blocking retrograde conduction.

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