Atrioventricular dissociation with accrochage

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In atrioventricular dissociation the sinus and nodal pacemakers may interact with each other independently of the conducting tissues so that the slower pacemaker accelerates and passes into phase with the ventricular pacemaker, the resultant disturbance of rhythm being atrioventricular dissociation with accrochage, i.e. hooking together of P waves and QRS complexes by active physiological processes, both mechanical and electrical. Illustrations of various ratios, 1:1, 2:1, 2:3 accrochage are demonstrated from 7 patients. It is suggested that accrochage may have clinical importance as it may protect the patient with heart block from syncope by acceleration of the ventricular rhythm and protection of the ventricular pacemaker from interference. The cardiographic features of this spontaneous disturbance of rhythm are discussed with particular reference to the frequencies of the pacemakers and the hooking together of atrial and ventricular systole.

In orthograde atrioventricular block the atrial rhythm may be disturbed by retrograde conduction of idioventricular impulses (Cohn and Fraser, 1914; Scherf and Schott, 1953) or by sinus allorhythmia (Erlanger and Blackman, 1910; Scherf, 1945). Though the mechanisms vary in each instance, the result is a closer linking together of atrial and ventricular impulses (Fletcher and Brennan, 1959; Fletcher and Morton, 1968). There is, however, a third mechanism less well documented in the English published reports, which links together atrial and ventricular systole in atrioventricular dissociation so that the P waves and QRS complexes synchronize with each other or almost so. It appears to depend upon active physiological factors which can be shown experimentally, and which therefore constitute a disturbance of rhythm *sui generis*. As will be apparent from the cases described in this paper, it may have clinical importance. Before the analysis of cardiograms a brief résumé of the principles of interpretation may be helpful.

When two independent foci of impulse formation exist side by side in a biological medium, they may interact together under certain circumstances in such a way that the focus with the lower frequency accelerates to approximate that of the higher frequency, and, in addition, the two foci tend to beat in

phase, i.e. synchronism of impulse occurs (Segers, 1946). Parallel conditions occur naturally in the human heart when the sinus node functions with an independent ventricular pacemaker, as in atrioventricular block, and in certain of the pararrhythmias, such as interference dissociation. The first clinical case was reported by Segers, Lequime, and Denolin (1947), who based their interpretations on experimental data in the frog's heart (Segers, 1946). Since the characteristic finding is a 'hooking together' of the P waves and QRS complexes, the term atrioventricular dissociation with accrochage (*accrocher*, to hook together) is used to describe the disturbance of rhythm as suggested by Stock (1969) to distinguish it from fortuitous synchronization of P waves and QRS complexes which may occur in atrioventricular dissociation over a few cycles. Accrochage of impulses may occur in various ratios, 1:1, 2:1, and more rarely 3:1, 4:1, or even 3:2 (Segers, 1946). It is not known how common atrioventricular dissociation with accrochage is in clinical practice. Published records are few (Segers *et al.*, 1947; Marriott, 1956, 1957; Stock, 1969). In the 1:1 ratio it appears to correspond with 'isorhythmic dissociation' of French authors (Veil and Codina-Altès, 1928). Greater awareness may reveal a higher incidence. We have records of seven cases which have been well documented over a 12-year period. They were all men of 57 to 61 years, who had cardiographic confirmation of ischaemic heart disease. As in all disturbances

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TABLE Atrioventricular dissociation with accrochage

Case No.	Sex	Age (yr)	Disease*	Accrochage				
				Ratio	Atrial rate per min	Vent. rate per min	RP hundredth sec	PR hundredth sec
1	M	61	CHD	1:1	65-79	65-79	16-0	2-4
2	M	60	CHD	1:1	70-85	70-85	2	2
3	M	59	CHD	1:1	66-80	66-80	4	2
4	M	58	CHD	1:1	70-80	70-80	10	4
5	M	60	CHD	2:1	100	50	0	2-9
6	M	57	CHD	2:1	90	45	0	0-9
7	M	57	CHD	3:2	88	110	6	8-12

* CHD, coronary heart disease.

of rhythm, long tracings are required for meaningful analysis. The figures below are representative tracings of atrioventricular dissociation with accrochage with various ratios. The important data of the cases are summarized in the Table. Four of the patients had 1:1 accrochage, two 2:1 accrochage, and one had accrochage rendered 'out of phase' by conducted beats in interference dissociation. For convenience, time intervals are given in hundredths of a second.

Atrioventricular dissociation with 1:1 and 4:1 accrochage

Case 1 A man aged 61 years had a previous history of myocardial infarction. He was observed over a three-year period during which 30 serial cardiograms were studied, each containing a long strip of lead II of not less than one hundred beats. Oesophageal leads were recorded at intervals to define the atrial rhythm more precisely. The records showed two disturbances of rhythm which appeared to depend on the sinus rate. The commonest rhythm which was dominant in 25 of the serial tracings occurred when the sinus rate was between 65 to 79 beats a minute. Fig. 1 is a representative tracing selected from 2500 similar cardiac cycles. It demonstrates atrioventricular dissociation with 1:1 accrochage. Two pacemakers are functioning independently, the sinus node and the atrioventricular node. The important features are that (1) the automaticity of the atrioventricular node is enhanced to what may be considered as the normal sinus rate at rest, 65-69 beats a minute. (2) the P waves and the QRS complexes are 'hooked together' in phase. When the sinus rate accelerated to over 80 beats a minute, the P waves passed 'out of phase' with the QRS complexes, revealing that orthograde atrioventricular conduction was intact, though impaired (Fig. 2). In Fig. 2 the atrial rate is 100 a minute, partial atrioventricular block is present with varying PR intervals and blocked P waves of the Wenckebach type. There are nodal escape beats with retrograde block as shown in the oesophageal leads which synchronize with sinus

P waves which may be significant in view of the development of 1:1 accrochage at slower sinus rates. Long tracings of Fig. 2 of 100 beats or more suggested that an automatic nodal pacemaker was present at a rate of 25 beats a minute. Comparison of Fig. 1 and 2 suggests that with rapid sinus rates orthograde atrioventricular conduction discharges the nodal pacemaker, but when it fails to occur the nodal pacemaker activates the ventricles and synchronizes with the sinus pacemaker, i.e. 4:1 accrochage is the mechanism involved. When atrial and ventricular systole are synchronized, the autonomy of the two pacemakers results from 'mutual extinction of orthograde and retrograde conduction in the bundle of His' (Dressler, 1929). Thus the polarity of the notched P waves in Fig. 1 excludes nodal rhythm with retrograde conduction as an alternate mechanism. In Fig. 1(A) the P wave characteristically 'moves through' the QRS complex with gradual shortening of the RP interval from 16 to 0 to precede it with a PR interval of 2-4, which resembles the behaviour of the P wave described in the first case published by Segers *et al.* (1947) as a basis for diagnosis of accrochage, though in the opposite direction. When the sinus rate exceeded 71 a minute the P wave preceded the QRS complex with a short PR interval (Fig. 1B). Graphically the rhythm may then resemble accelerated conduction through paraspecific pathways (Mahaim, 1947), but this mechanism is excluded as the P wave passes through the QRS complex. Disturbance of the ventricular rhythm by exit block of a ventricular impulse (Fig. 1A) or by abnormal ventricular beats (Fig. 1B) did not disturb accrochage in the succeeding beats, though it temporarily reversed the sequence of P waves and QRS complexes.

In summary therefore, there was intact but impaired atrioventricular conduction with an independent nodal pacemaker with retrograde block whose rate was adjusted to the sinus rate and became in phase with it over prolonged periods within a wide range of 65 to 79 beats a minute, with distinctive cardiographic features, i.e. 1:1 accrochage. Cases 2, 3, and 4 (Table) resembled Case 1 with relatively rapid ventricular rates between 66 to 80 beats a minute due to 1:1 accrochage. As in Case 1, accrochage was abolished

FIG. 1 Atrioventricular dissociation with 1:1 accrochage. Case 1 (Table): time intervals in hundredths of a second. (A) 1-4 is continuous, and shows sinus arrhythmia, atrial cycle lengths, 52-90, rate 67 a minute. The P waves and QRS complexes remain 'hooked together'. The notched sinus P waves are positive and move through the QRS complexes with diminishing RP intervals from 16 to 0 to precede them with a PR interval of 2-4. In B the atrial rate has increased to 79 a minute and the P waves precede the QRS complexes, PR = 2. The ventricular pacemaker is nodal. Exit block of ventricular beats (BP) and ventricular ectopic beats (E) did not abolish accrochage but reversed the sequence of P waves and QRS complexes.

by sinus rates exceeding 80 beats a minute which revealed various degrees of partial heart block.

Atrioventricular dissociation with 2:1 accrochage

Case 5 A man aged 60 years had a history of angina pectoris for five years. Twenty-one serial cardiograms each containing a long strip of lead II with not less than 100 beats were recorded over a period of 18 months. Fifteen of the tracings showed atrioventricular block, of which Fig. 3 is a

representative sample. Six of the tracings showed sinus rhythm with right bundle-branch block, rate 60 to 70 a minute (Table), the QRS complexes resembling those in Fig. 3. Fig. 3 illustrates atrioventricular block with 2:1 accrochage which closely resembles the case of Segers *et al.* (1947). The sinus rate is 100 a minute, and the ventricular rate 50 a minute. It is likely that the idioventricular centre is above the bifurcation of the main bundle of His in the atrioventricular node, as intraventricular block was present in sinus rhythm. Alternate P waves are 'hooked' to ventricular complexes. The PR intervals of the synchronized beats successively shorten from 9 to 2, which makes it unlikely that the basic mechanism is 2:1 atrioventricular block. A more satisfactory explanation is that the nodal pacemaker has synchronized with alternate sinus beats during complete atrioventricular block, i.e. the sinus pacemaker has interacted with the nodal pacemaker to bring it into phase in a 2:1 accrochage, but without unduly increasing the automaticity of the atrioventricular node. A 2:1 ratio was usually produced in experimental records when the atrioventricular conducting tissues were destroyed (Segers, 1946). In Case 6 (Table), the ventricular rate was maintained at 45 beats a minute by 2:1 accrochage as the dominant rhythm, the PR interval varying from 0-9.

FIG. 2 Sinus rhythm with partial atrioventricular block and synchronized beats. Case 1 (Table): Time intervals in hundredths of a second. E, oesophageal lead, 40 cm, from which cycle lengths are measured. Atrial rate, 100 a minute, with Wenckebach block. Note synchronization of fourth P wave with nodal beat (S) 4:; accrochage (see text).

Atrioventricular dissociation with interference with 2:3 accrochage

Case 7 A man aged 57 years had a history of myocardial infarction after which he was observed over a four-year period. Twenty serial cardiograms were recorded, in which sinus rhythm was dominant in 12 tracings with a rate range of 60 to 75 a minute. In the remaining 8 tracings dissociation with interference was the dominant rhythm over prolonged periods of not less than 100 beats. Fig. 4 is a selected sample to demonstrate the mechanism of the pararrhythmia, with oesophageal leads to define the atrial rhythm more precisely. For this disturbance of rhythm the atrial rate is relatively fast, 88 beats a minute, as sinoatrial block is the usual mechanism of this pararrhythmia (Scherf and Schott, 1953). The ventricular rhythm, which appears to be nodal in origin, has a rate of 110 beats a minute. When the sinus impulse is not conducted to the ventricles to disturb the ventricular rhythm (Fig. 4,D), it synchronizes with the nodal impulse in a consistent fashion. After each conducted sinus beat the P wave preceded and synchronized with the next QRS complex with a PR interval of 6, and in the succeeding beat the P wave and QRS complexes are also synchronized but the P wave follows the QRS complex with an RP interval of 8–12, i.e. one atrial cycle length contains one ventricular cycle length. The third P wave is 'out of phase' with the QRS complex and its impulse is conducted in the ventricles. In the tracing the faster nodal pacemaker appears to enhance the slower sinus pacemaker so as to synchronize with it a ratio of 2 synchronized beats out of 3. An alternative but less likely interpretation of Fig. 4 may be mentioned. According to Marriott (1956), the PR interval in accrochage may at times exceed the RR interval, i.e. the atrial impulse may synchronize with the ventricular impulse of the preceding cycle. In Fig. 4 the first P wave may activate the second ventricular complex with a PR interval of 56 which is longer than the first ventricular cycle of 52, i.e. the second P wave synchronizes with the QRS complex of the first P wave. But this mechanism is not followed through in succeeding cycles, and dissociation with interference with 2:3 accrochage is probably the more acceptable explanation. Moreover, it has already been noted that synchronization tends to extinguish conduction in the bundle of His in either direction (Dressler, 1929).

Discussion

The interaction between the atria and ventricles in atrioventricular dissociation independent of the function of the conducting tissues may depend upon both mechanical and electrical factors. Both were proven to play a part independent of each other in synchronization of impulses in the frog's heart, and both mechanisms were independent of nerve tissue (Segers, 1946). The mechanical contraction of the human ventricles may

FIG. 3 Atrioventricular dissociation with 2:1 accrochage. Case 5 (Table): Time intervals in hundredths of a second. Alternate sinus P waves were hooked to QRS complexes. The PR intervals successively diminish from 9–2. Atrial cycle lengths vary from 52–62, rate 104 a minute. The QRS complexes measure 120 due to right bundle-branch block. Ventricular rate, 50 a minute.

draw down the atria, and it was believed at one time that it influenced impulse formation in the atria (Cohn and Fraser, 1914). Such a mechanism appears unlikely in the same cases reported above for the primary determinant of accrochage was the sinus rate with the exception of Case 7 (Table). The sinus rate appeared actively to 'boost up' the nodal pacemaker. The electrical properties of the volume conductor medium rather than the effects of relatively weak atrial contractions on the atrioventricular node appear to be more important. In clinical conditions it may be that the harmonics of the pacemakers invading the same electrical field may interact to produce a common frequency in favour of the faster rhythm. According to Segers *et al.* (1947), synchronization remains constant when the rates do not differ by more than 10 per cent of the average rate. The electrical theory of accrochage is more acceptable in Case 7 (Table) in which the rapid nodal pacemaker enhances the sinus rate to produce an unusual interference dissociation with a rapid atrial rhythm.

All the cases reported in this paper had

coronary heart disease. None of them had syncopal attacks. Accrochage in atrioventricular dissociation may act as a protective mechanism in two ways. First it tends to augment the ventricular rate in heart block which may increase the cardiac output, and secondly it 'extinguishes' conducting in the bundle of His, and allows the ventricles to be dominated by a regular nodal pacemaker. In the case described by Segers *et al.* (1947), unconsciousness was related to exercise presumably due to acceleration of the atrial rhythm so that synchronization became 'out of phase' and accrochage abolished. One of Marriott's patients (1956) had rheumatic heart disease, and Stock (1969) reported a case of accrochage after electrical cardioversion. When 1:1 accrochage was present the atrial rate was 65–80 beats a minute (Table), the ventricular beats adapting themselves to synchronize with constantly varying atrial cycle lengths. When the P waves and QRS complexes occur almost simultaneously, accrochage may be obscured and mistaken for nodal rhythm or slow ventricular tachycardia if the P waves distort the QRS complexes. This difficulty was noted by Marriott (1956) and is illustrated in Fig. 5 (Table, Case 2). The unusually rapid nodal rate may suggest 1:1 accrochage with a sinus pacemaker, but the diagnosis may only be apparent in serial tracings after exercise with an accelerated atrial rate out of phase with a slower nodal pacemaker. With more rapid atrial rates as in Cases 5 and 6 (Table), 2:1 accrochage is more likely, though Stock (1969) reported 1:1 accrochage at a rate of 125 a minute after cardioversion. More complex ratios of accrochage may also occur as in Fig. 4 (Table, Case 7) and in the case reported by Marriott (1956) in which every third P wave synchronized with every other QRS complex which the author describes as a 3:2 synchronization.

Accrochage may be regarded as a physiological means of placing a slower pacemaker 'on demand' to a faster maker. The slower pacemaker must have the capacity to respond, and in the series in this paper and as far as can be ascertained from published records accrochage has not been reported with centres lower in the ventricles than the atrioventricular node. When 1:1 accrochage develops the ventricular rate may be within the normal range. It is not unlikely to be disturbed by exit block of the nodal pacemaker or by ventricular ectopic beats. In dissociation with interference it may be the mechanism involved in producing a rapid sinus rate which is otherwise unusual in this pararrhythmia. Finally, accrochage is disturbed by placing the

FIG. 4 Atrioventricular dissociation with interference with 2:3 accrochage. Case 7 (Table): Time intervals in hundredths of a second. E 40 – oesophageal lead, used for measurements in A. B, 1–3 is continuous. Ventricular rate, 110 a minute is faster than atrial rate which is 88 a minute. The dominant ventricular beats occur in groups of 3, the first 2 synchronize with P waves which enclose on the RR interval. The third beat is out of phase, the sinus impulse being conducted prematurely to disturb the ventricular rhythm (D).

atrial rhythm 'out of phase', usually by acceleration which makes orthograde conduction manifest and interrupts the nodal pacemaker.

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FIG. 5 Atrioventricular dissociation with 1:1 accrochage. Case 2 (Table): Continuous strip, lead II. The P waves and QRS complexes are synchronized. The P waves are positive and move through the QRS complexes with apparent widening. The tracing resembles nodal rhythm or a slow ventricular rhythm, rate 75 a minute.

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