Atrial parasystole

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Two cases of atrial parasystole showing the various manifestations of the arrhythmia are presented. Analysis of the underlying mechanisms shows that atrial parasystolic bigeminy with 'reversed' coupling is a form of escape-capture bigeminy, sinus escapes being followed by an ectopic capture of the atria. Reasons are given for the rarity of atrial fusion beats. The similarities and differences between atrial and ventricular parasystole are explored. It is suggested that an atrial parasystolic pacemaker may lie within a major atrial preferential conducting pathway, and may consist of a congenitally ectopic fragment of sinus nodal tissue. The clinical significance of the arrhythmia is discussed; the associated diseases apparently represent a cross-section of medical ward experience.

Parasystole is a dual rhythm in which the parasystolic pacemaker is protected from the effects of the dominant, usually faster pacemaker: this protection, which is the essence of the arrhythmia, is situated within the immediate vicinity of the parasystolic focus, and is operative throughout its entire cycle (Schamroth, 1964). When a parasystolic ventricular and a sinus pacemaker coexist, a characteristic arrhythmia appears. The ventricular beats bear no constant relation to the preceding QRS complexes (i.e. the coupling intervals vary) and indeed may well occur so late in diastole that they fuse with the ensuing sinus QRS: the ectopic beats do, however, bear a consistent relation to each other, so that the interectopic intervals are in simple multiples of the basic ectopic cycle length. In those rare instances in which the

parasystolic focus is in the atria, and thus in the same bi-atrial chamber¹ as the sinus pacemaker, the resulting disturbance of rhythm is modified, though the essential characteristics of the arrhythmia are not changed. In particular, coupling intervals vary less in atrial parasystole: a fixed bigeminy, closely resembling an extrasystolic bigeminy with nearly constant coupling, may occur. Atrial fusion beats are rare. The manifestations of atrial parasystole will be presented in this paper and the underlying mechanisms analysed.

Case reports

Case 1 The electrocardiogram (Fig. 1, a continuous recording of standard lead II) was obtained from a 75-year-old man with severe obstructive lung disease. The arrhythmia was

¹ Electrophysiologically, both atria usually behave as a single chamber, and will be referred to as the bi-atrial chamber.

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FIG. 1 Case 1. A continuous strip of standard lead II. Premature atrial beats, marked with a black dot, occur with varying coupling. The interectopic intervals are simple multiples of 120–124, and thus the rhythm is atrial parasystole.

FIG. 2 Case 1. Strips of standard lead III and aVF. Atrial premature beats occur in bigeminy. The second last P wave in standard lead III arises from AV junctional focus. The last P wave in this lead occurs with a different coupling interval, but maintains a constant interectopic interval, thus revealing the rhythm to be atrial parasystolic bigeminy with reversed coupling. (See text for discussion.)

present during two weeks only, when a small dose of digitalis was being given. The basic sinus rhythm is punctuated by atrial premature beats. The ectopic P' waves resemble the sinus P waves, but begin more sharply, and rise more steeply to a higher peak. The premature P' waves are not accurately coupled to their preceding sinus P waves, the coupling intervals varying from 42¹ to 70. The intervals between the P' waves – the interectopic intervals – are all in simple multiples of 120 to 124, thereby indicating that these beats are related to each other. This combination of inconstant coupling and simply related interectopic intervals is the hallmark of parasystole.

Fig. 2 was recorded two days later, and consists of strips of standard lead III and lead aVF. A bigeminy is present, every second beat being an atrial premature beat. As the coupling intervals are nearly constant, this closely resembles the commoner extrasystolic atrial bigeminy. In

¹ All time intervals are expressed in hundredths of a second, i.e., 22 = 0.22 second.

an extrasystolic bigeminy with a regular dominant rhythm and fixed coupling intervals, the interectopic intervals will also be constant. This is not parasystole, but due simply to the constancy of the post-extrasystolic pause and the constant coupling intervals. In Fig. 2, the true situation is revealed by the last pair of P waves in standard lead III. The first P wave of this pair is inverted, arising from an AV junctional focus, which anticipates the sinus impulse. The second P wave of this pair arises from the same atrial focus as the earlier ones, but now the coupling is conspicuously different – 54 instead of 32 to 40. The interectopic interval, however, is constant at 116. It is clear that the atrial focus was not discharged by the AV junctional impulse, and is parasystolic. It is noteworthy that the parasystolic focus is discharged neither by the sinus impulse nor by the lower junctional impulse, and is thus protected from below as well as from above.

The electrocardiogram in Fig. 3 is part of the same long tracing of standard lead II shown in

FIG. 3 Case 1. Part of the same tracing as Fig. 1. The sixth P wave in the upper strip, marked F, is an atrial fusion beat. (See text.) P' in the lower strip is interpolated (see Fig. 7).

FIG. 4 Case 2. Strips of standard leads II and III. Ectopic P waves with varying coupling but constant interectopic intervals occur, showing atrial parasystole. Some of the parasystolic P waves are not conducted to the ventricles, some are conducted with aberrant ventricular conduction, and some are conducted normally.

Fig. 1. The parasystolic cycle ranges from 120 to 124 (mean 123). The first, third, and eighth P waves are parasystolic in origin. The beginning of these P waves rises abruptly, forming a sharp angle with the baseline. The beginning of the sixth P wave, marked F, is smoothly rounded, as is that of the sinus P waves. However, this P wave is a little taller than the sinus P waves, and has a slightly steeper descending slope. It is thus intermediate in form between the sinus and the ectopic P waves. Furthermore, this P wave begins 0.06 sec. before the ectopic P' wave is due (as indicated by the half open circle). Therefore, this P wave results from a fusion between the atrial parasystolic and the sinus impulses. The second P' wave in the lower strip is interpolated.

Case 2 The electrocardiogram (Fig. 4) was recorded during a period of mental confusion from a 74-year-old man with a history of ischaemic heart disease. The arrhythmia disappeared with rest and sedation, but recurred temporarily after he broke his femur some months later. At no time did he have angina or take digitalis. For the most part, sinus P waves and ectopic P' waves alternate in an atrial bigeminy with conspicuous variation of the coupling intervals, which range from 36 to 60. The interectopic intervals range from 115 to 120; the intersinus intervals range from 114 to 130. In standard lead III, a supraventricular premature beat from a different focus (arrow) interrupts the sinus rhythm, but does not affect the regularity of the P' series. Some of the P' waves fall so early as to fail to pass the AV junction, others are conducted with varying degrees of ventricular aberration, and others are conducted normally. Though the ectopic cycle is a little variable, the interectopic intervals vary much less than do the coupling intervals; and all the hallmarks of atrial parasystole are present.

The electrocardiogram in Fig. 5 was recorded from Case 2 four months later, shortly after he

had suffered a fracture of the femur. All strips were recorded at the same session; the bottom two strips are continuous. P waves from many different supraventricular foci are present. Some of these P waves (marked with a dot) closely resemble the parasystolic P' waves shown in Fig. 4.

In the three upper strips, the interectopic intervals are a simple multiple of 98 (range 94 to 100). In the bottom strip, the interectopic intervals are 145, 148, and 144. These are not a simple multiple of 98. All the figures, however, are simple multiples of 49 ± 1 . An atrial parasystole is therefore present, the basic cycle length of which is 49 ± 1 , and which is complicated by an exit block. If every second impulse reaches the atrial muscle to inscribe a P' wave, the apparent cycle length is 98 ± 2 , and 2:1 exit block is present. If every third impulse reaches the atria and is manifest, the apparent cycle length is 147 ± 3 , and 3:1 exit block is present.

Mechanisms

(a) Parasystolic bigeminy: 'reversed' coupling.

In atrial parasystole, both the protected atrial pacemaker and the unprotected sinus pacemaker are situated in the same bi-atrial chamber. As a result, the sinus node is vulnerable to, and will frequently be prematurely discharged by, the ectopic impulse. The sinus cycle will be consequently reset, while the parasystolic cycle continues undisturbed. This is illustrated in Fig. 6, in which the 'build-up' of the sinus impulse toward a threshold is pictured above a representation of events at the atrial level. In Diagram I of Fig. 6, the parasystolic impulse E₁ reaches the sinus node and aborts the immature impulse there at point Sd₁, interrupting and resetting the

FIG. 5 Case 2. Strips of lead aVF and standard lead II. The bottom two strips are continuous. P' waves are indicated by a black dot. The interectopic intervals are at first in multiples of 98 ± 2 , and change abruptly to 147 ± 3 , indicating atrial parasystole with varying exit-block. (See text for discussion.)

sinus cycle. The recharge of the sinus impulse now begins anew, which on reaching threshold, discharges spontaneously at point S₂, after the expiry of the sinus cycle (sc). Now, if the duration of the ectopic cycle (ec) is a little greater than the sinus cycle plus the refractory period of the sinus pacemaker (rp), the next ectopic impulse (E₂) will again discharge the sinus pacemaker (at point S_{d2}) and the sinus cycle is again reset. In this manner, the sinus discharge is causally related and linked to the protected ectopic discharge, and the bigeminy is perpetuated (see also Fig. 8B). This is a form of escape-capture bigeminy (Bradley and Marriott, 1958; Schamroth and Dubb, 1965), but one in which a sinus escape is followed by an ectopic capture of the atria.

The time of appearance of the sinus P waves is thus dependent on, and coupled to, the parasystolic P' waves. This is the reverse of the situation in an extrasystolic arrhythmia, in which the ectopic complexes are dependent on, and coupled to, the dominant beats. A similar 'reversed' coupling may occasionally appear during ventricular parasystole. This

occurs if the ventricular impulse is completely conducted retrogradely through the AV junction, atria, and SA junction to discharge the sinus node. Such complete retrograde conduction is the exception. The relatively slowly conducting AV junction will usually protect the sinus node and effectively prevent the maintenance of a bigeminy.

Atrial parasystolic bigeminy thus depends upon a fortuitous arithmetical relation between the rates of the two centres of impulse formation, of which one is protected and the other is vulnerable. Once begun, the bigeminy tends to persist. The bigeminy may be interrupted and the true nature of the arrhythmia revealed under the following circumstances. (1) If there is sufficient sinus arrhythmia to make the ectopic impulse so early that it falls during the absolute refractory period of the atria induced by a sinus impulse (see also *Interpolation* below); (2) if a different ectopic impulse discharges the sinus node and further dislocates its rhythm (see Fig. 2); (3) if the parasystolic impulse suffers exit-block and fails to leave its focus and invade the atria.

FIG. 6 I. A diagram to show the mechanism of atrial parasystolic bigeminy. The upper saw-tooth line represents the 'build-up' of the sinus impulse towards threshold. A=atrial level; S₁, S₂, and S₃ are sinus impulses; E₁, E₂, and E₃ are ectopic impulses; rp=the refractory period of the sinus node; sc=the duration of the sinus cycle; ec=the ectopic cycle. (See text.)

II. Interpolation of an atrial premature beat. The conventions are the same as in I. Ectopic impulse E₂ invades the atria before the end of the refractory period of the sinus node, so that the sinus cycle is not reset. S₃ then occurs when anticipated, and an ectopic beat is sandwiched between two sinus beats. Note: The AV junctional and ventricular levels have not been drawn. Conduction delay across the sino-atrial junction is shown in Fig. 7.

FIG. 7 A diagram to show the characteristic allorhythmia of an interpolated atrial premature beat. P₁, P', P₂, and P₃ refer to the P waves so marked in the lower strip of Fig. 3 (see text). S=sinus node; S-A=sino-atrial junction; A=atrial level. AV junctional and ventricular levels have not been drawn.

(b) Interpolation If the ectopic impulse falls early in the sinus cycle, at a time when the atria have recovered excitability but when the sinus node is still refractory, the ectopic impulse will fail to reach and reset the sinus pacemaker. The spontaneous discharge of the sinus node then occurs when anticipated. Interpolation of the ectopic impulse between two sinus impulses results. This is illustrated in Diagram II of Fig. 6, where impulse E₂ is interpolated between S₂ and S₃. This phenomenon is evident electrocardiographically in the lower strip of Fig. 3. Incomplete penetration of the S-A junction (the junctional delay area between the S-A node and the surrounding atrial myocardium) by the ectopic impulse may render it partially refractory to the next sinus impulse. This produces a characteristic disturbance of PP intervals (Langendorf *et al.*, 1962): the interval between a pair of sinus P waves enclosing an interpolated atrial ectopic beat is longer than the intersinus interval. The subsequent PP interval is foreshortened. This is illustrated in Fig. 7, a diagram of the conduction sequence of the interpolated parasystolic impulse in the lower strip of Fig. 3. Impulses S₁, S₂, and S₃ are regular consecutive sinus impulses resulting in P waves P₁, P₂, and P₃, respectively. P' is the parasystolic impulse which penetrates the S-A junction but fails to reach and reset the sinus pacemaker. This renders the junction partially refractory. Conduction of S₂ through the SA junction is therefore prolonged, and the inscription of P₂ is delayed. But for the effect of P', P₂ would have been inscribed as indicated by the dotted line. The next sinus impulse S₃ - P₃ is not so delayed. P₂ is thus shifted towards P₃, producing the characteristic allorhythmia of interpolation. A strictly analogous variation of RR intervals occurs with an interpolated ventricular parasystolic or extrasystolic beat (Schamroth, 1967b).

(c) Atrial fusion Ventricular fusion beats have certain characteristics which lead to easy recognition (Marriott, Schwartz, and Bix, 1962). Fusion is suspected if an ectopic QRS is due near the time of a sinus QRS, and an intermediate QRS is seen. Changes in QRS configuration are easily detected because of the finely inscribed, detailed, and complex QRS deflections. Thus, very small degrees of fusion may be deduced from subtle changes in the direction of the initial or terminal QRS vectors, or in the T wave. Detection is further facilitated by the fact that the two complexes concerned are usually

conspicuously different; and because the relatively slowly conducting AV junction effectively isolates the two rhythms, coupling intervals vary greatly, and end-diastolic ectopic beats are common.

Several factors militate against the occurrence and the recognition of atrial fusion beats during atrial parasystole.

1) *Lack of P wave detail* The P wave is less detailed, less complex, and coarser than the QRS complex. Subtle changes are therefore more difficult to detect. Furthermore, the P waves from the sinus and the atrial pacemaker are not usually conspicuously different, so that the recognition of fusion by changes in shape alone is very difficult. Nor are atrial repolarization waves - T_a waves - sufficiently distinct to help. In Fig. 3, atrial fusion is postulated because the P wave concerned has an initial vector typical of the sinus P waves in that lead, and is of intermediate height and terminal slope, and because the timing is correct.

2) *Limited duration of opportunity for fusion* In the sixth beat of Fig. 3, probable atrial fusion is recognized when the ectopic P' wave was due 0.06 sec. after the onset of the sinus P wave. Yet, in the ninth beat of the upper strip of Fig. 1, fusion did not occur though the ectopic P' wave was due 0.08 sec. after the start of the P wave. This suggests that within 0.08 sec., the sinus impulse has spread to the tissues surrounding the atrial parasystolic focus, i.e. this is the conduction time from the sinus to the ectopic focus. Therefore, in this case, if the sinus fires first, the time available for possible fusion is not greater than 0.07 sec. Should the ectopic focus discharge first, a similar limit for the duration of opportunity for fusion exists. While this is not known, it should also be about 0.07 sec., but may well be less. In both these cases, and in many of the others reported, the sinus and parasystolic P waves are similar, with some differences in their initial contour, but with little difference in their terminal contour. Thus, once the ectopic impulse has inscribed the initial part of the P wave, the contribution of the sinus impulse to the shape of this wave may not be recognizable. Moreover, the time of appearance of the more variable sinus impulse may not be known. Though a sinus impulse may fuse in the atria with an earlier appearing ectopic impulse, this fusion may well not be diagnosable.

3) *Resetting of sinus node* As shown above, the presence of two centres of impulse formation in the same bi-atrial chamber facilitates

FIG. 8 Relating the possibility of atrial fusion to the resetting of the sinus cycle. Impulse formation in the S-A node (S) and the ectopic focus (e) is shown by the black dots. The refractory period of the sinus node is represented by the stippled area. The sinus impulse does not penetrate into the parasystolic focus. The rates of the two pacemakers are assumed to be moderately steady. (A) The parasystolic cycle is a little longer than the sinus cycle; occasional parasystolic beats appear. (B) The parasystolic cycle exceeds the sum of the sinus cycle and the sinus refractory period; parasystolic escape-capture bigeminy results. (C) The parasystolic cycle equals two sinus cycles less the last coupling interval; atrial fusion results (marked f). (D) The parasystolic cycle equals the sinus cycle; repetitive fusion results.

the resetting of the unprotected sinus node. The frequency of this resetting and the resulting arrhythmic disturbance is dependent on the relation of the two cycle lengths. This is diagrammatically illustrated in Fig. 8. This theoretical analysis is based on the assumption that the two rates remain moderately steady.

a) If the parasystolic cycle exceeds the sinus cycle but is less than the sinus cycle plus the refractory period of the sinus node (Diagram A of Fig. 8), there will be occasional 'captures' of the atria by the ectopic impulse.

b) If the parasystolic cycle exceeds the sum of the sinus cycle plus the ensuing refractory period of the sinus node (Diagram B of Fig. 8), parasystolic bigeminy –

escape-capture bigeminy – of the atria will ensue.

c) If the parasystolic cycle is shorter than the sinus cycle (not illustrated), the ectopic impulses anticipate every sinus discharge and thus usurp complete control of the atria, resulting in an uncomplicated ectopic atrial rhythm – an atrial parasystolic tachycardia.

The aforementioned relation with the resetting of the sinus cycle militates against the occurrence of atrial fusion complexes. These will only occur under the following rare circumstances.

a) If the parasystolic cycle equals two, or any integral number of sinus cycles, less the last coupling interval, both impulses will simul-

taneously invade the atria and fuse (Diagram C of Fig. 8).

b) If the parasystolic cycle equals the sinus cycle, or any integral number of sinus cycles, fusion will recur regularly (Diagram D of Fig. 8).

The aforementioned principles may be modified under the following special circumstances: (a) in the presence of sinus arrhythmia; (b) in the presence of exit-block; (c) in the presence of a variable ectopic cycle; and (d) if the sinus cycle is reset by another – different – ectopic impulse.

Fusion between atrial parasystolic and sinus impulses then is rare because (1) it is difficult to recognize, (2) the coupling intervals vary little, (3) there is only a short period of opportunity for fusion, and (4) because the mathematical relations between a protected and an unprotected centre of impulse formation in the same chamber militate against fusion except in unusual circumstances. Scherf, Yildiz and De Armas (1959) commented that such fusion is rare. The only clear cases are those of Vedoya (1944, Case 5) and of Katz, Eschelbacher, and Strauss (1937).

(d) Exit-block The parasystolic discharge will activate the surrounding myocardium whenever the myocardium is not refractory after activation by the other pacemaker. Occasionally, however, calculation reveals that, though the myocardium is responsive at the time of the parasystolic discharge, activation does not occur. This has been explained on the basis of exit-block – a phenomenon in which an impulse is confined to its focus of generation by a 'block' at the ectopic-myocardial junction. Exit-block is also present if the calculated ectopic cycle length is less than the cycle of the dominant pacemaker, or if it is less than the duration of any post-ectopic pause; for without exit-block an ectopic tachycardia would be present.

There is a further circumstance in which exit-block may be diagnosed. Should a constant 2:1 exit-block change abruptly to one with a 3:1 ratio, the apparent common denominator of the interectopic intervals will change to one and a half times itself. Similarly, a change from a 3:1 to a 4:1 conduction ratio will result in an increase of one-third in the apparent ectopic cycle length. That is to say, if the largest common denominator in a continuous recording changes abruptly to one with which it has a simple *fractional* relation, exit-block with a change in conduction ratio may be diagnosed.

Thus, in Fig. 4, an abrupt increase in the common denominator from 98 to 147 (i.e., $1\frac{1}{2}$ times 98) allows the diagnosis of a basic ectopic cycle length of 49 with 2:1 exit-block changing abruptly to 3:1 exit-block.

Discussion

Incidence Well-documented cases of atrial parasystole are rare. In a search of the published material, Eliakim (1965) could find only 15 reported instances and added one more. To these may be added that of Attinger (1940), two of Holzmann (1960), two of Mouloupoulos and Sideris (1967), and Deshpande (1968). The two reported here make a total of 24 cases. This is, then, a very much rarer arrhythmia than either ventricular or junctional parasystole. Exit-block has been present in the cases of Katz *et al.* (1937), Holzmann (1960), Deshpande (1968), and in one of the two in this paper, a total of four cases. Though figures for the frequency of exit-block in other forms of parasystole are not available, exit-block does not appear to be less common in atrial parasystole.

Clinical significance Most cases of atrial parasystole have been reported as curiosities, and a review of well-substantiated cases does not reveal any particular clinical association. It has been stressed that parasystole is a sign of a diseased heart and is associated with digitalis therapy (Faltitschek and Scherf, 1932; Scherf *et al.*, 1959). On the other hand, Jervell (1932), Vedoya (1944), and Eliakim (1965) reported cases of atrial parasystole without heart disease. Certainly, patients without heart disease are much less likely to have long electrocardiographic strips taken and have atrial parasystole diagnosed. The association of parasystole and heart disease must be regarded as not definitely proven. Digitalis is often incriminated, but in no reported case is the appearance and regression of the arrhythmia so clearly related to the drug as in Case 1 above. Case 2 exemplifies the apparent provocation of the arrhythmia by an acute psychotic episode and by an acute injury, in the absence of digitalis and not necessarily related to ischaemic symptoms.

Site of the parasystolic pacemaker Of the reported cases in which the parasystolic focus is clearly atrial (as opposed to junctional, coronary sinus, or atrio-nodal), the P' waves of many bear a remarkable resemblance to one another. These include those reported by Jervell (1932), Attinger (1940), Scherf *et al.* (1959), Chung, Walsh, and

Massie (1964, Cases 1, 3, and 4), Mouloupoulos and Sideris (1967), and Deshpande (1968), and the two reported here. In all these cases the P' wave arises more abruptly to a higher peak than the sinus P wave, while the later portions of the two waves are similar. Further, the P-R and the P'-R intervals are equal or nearly so.

Now, there is considerable evidence that intra-atrial conduction and internodal conduction occur via definite pathways, and are not radial, as was formerly thought (James, 1966; Merideth and Titus, 1968). It seems possible that the atrial parasystolic focus lies within a major atrial conducting pathway, and is close to the sinus node. The 'family resemblance' and the rarity of atrial parasystole suggest that in these patients the focus is a portion of sinus tissue a little distance from the main node itself. This 'ectopic sinus tissue' may be an embryonal rest. Interestingly, even the sinus node itself may be parasystolically protected (Schamroth, 1967a). If an atrial parasystole does reflect a congenital anatomical variant, then the clinical features of this condition would reflect a cross-section of hospital practice, which does seem to be the case.

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