Multifocal ventricular parasystolic tachycardia

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Two cases of spontaneous multifocal ventricular parasystolic rhythm are described. One case showed double and the other fivefold parasystole. All seven foci had an enhanced rate of discharge, ranging from 57 to 102/min and at least three of them showed exit block. Though beats from three foci displayed very short coupling intervals, occasionally interrupting the terminal part of the preceding T wave, in none of the cases was repetitive firing or ventricular fibrillation seen. Both patients had organic heart disease and both are still alive six months after the arrhythmia was first recorded.

Problems in the diagnosis of multiple parasystole and some mechanisms which may be responsible for irregular interectopic intervals are discussed. It is concluded that multiple parasystole is probably not a very rare arrhythmia if long strips of simultaneously recorded multiple leads are available.

Parasystole is characterized by a regular ectopic rhythm coexisting with and independent of the dominant rhythm. The maintenance of regular rhythmlicity, despite depolarization of the compartment in which the ectopic focus is situated by the impulses of the dominant rhythm, points to a protective mechanism which is thought to be located within or in the immediate vicinity of the ectopic centre (Schamroth, 1964). These characteristics are manifested electrocardiographically by varying coupling intervals1 between the beats of the two rhythms and by a simple numerical relation between the interectopic intervals (Katz and Pick, 1956; Chung, 1968; Scherf and Schott, 1973). Fusion beats, if present, support the diagnosis (Scherf and Schott, 1973), but their absence is by no means an argument against it (Marriott, Schwartz, and Bix, 1962). Single ventricular parasystole is not an uncommon arrhythmia. It has been found to occur in 0-08 per cent (1 in 1200) of routine electrocardiograms in a general hospital by Scherf and Schott (1973) and in 0-11 per cent (1 in 900) by Chung, Walsh, and Massie (1964). On the other hand, only very few cases of spontaneous multifocal parasystole are on record (Chung et al., 1964; Chung, 1968; Roelandt, Pool, and Schamroth, 1972; Dolgin, 1973; El-Sherif and Samet, 1975).

It is the purpose of this communication to report and discuss two new cases of multifocal ventricular parasystole.

Case histories

Case 1
A 45-year-old woman with hypertensive heart disease complained of palpitation. She was not on digitalis therapy. On physical examination, she had an irregular heart beat and hypertension, and her electrocardiogram and chest x-ray film showed evidence of left ventricular hypertrophy. A long continuous rhythm strip (Fig. 1a and b) showed double ventricular parasystole in conjunction with multifocal ventricular extrasystoles.

Case 2
A 59-year-old man with an old inferior and anterior wall infarction had advanced left and right heart failure. At the time of recording of the electrocardiograms shown in Fig. 2a–e he was on digitalis therapy with a serum digoxin level of 3.0 ng/ml (upper limit of therapeutic range 2.5 ng/ml). Five different ventricular parasystolic rhythms and several ventricular ectopic beats of possible multifocal origin could be identified. One parasystolic rhythm (to be discussed as group 2A) continued for several weeks despite discontinuance of digitalis therapy.

Analysis of electrocardiograms

Fig. 1 (Case 1) is composed of continuous strips taken from a long, simultaneous recording of leads...
FIG. 1 Case 1. Continuous strips (a and b) taken from a simultaneous recording of leads III and V3R. Parasystolic beats of group A and B are indicated by closed circles and squares, respectively. Ectopic beats marked with asterisks, arrows, and triangles show different but constant coupling intervals. Note that those marked with arrows only follow the parasystolic beats of group A (closed circles). F=fusion beat. \( F? \)=possible fusion beat. See text for further discussion.
Multifocal parasystole

![Image of electrocardiograms](https://example.com/figure2.png)

**FIG. 2** Case 2. Representative strips taken from a simultaneous recording of leads I, II, III., and ensiform lead. (a) Three different parasystolic rhythms are indicated by closed circles (group A), squares (group B), and triangles (group C). Numbers in the upper part of the figure indicate coupling intervals of beats of group C (triangles); note the extremely short coupling interval of the first beat of this group. Numbers in the lower part of the electrocardiogram indicate interectopic intervals measured from supposed fusion beats (F) to the interrupted vertical lines, each of which indicates the onset of a pure parasystolic beat. The asterisk marks an ectopic beat of undetermined origin. (b) Continuous with Fig. 2a. The parasystolic cycle length of group B (squares) appears at the end of the figure. Group A parasystolic rhythm (closed circles) displays an exit block at the end of the strip. The ectopic beat marked with an asterisk is of undetermined origin. (c) This strip is continuous with Fig. 2d but not with 2b. The ectopic beat marked with a vertical bar belongs to group D. Numbers in the upper part of the record indicate interectopic intervals. Note the shortest manifest interval of Group C (triangles) in the left-hand part of the figure (compare with Fig. 2a). (d) Continuous with Fig. 2c, but not with 2e. Beats are numbered in the upper part of the record. Note the constant QRS configuration and varying coupling intervals of the ectopic beats which have been marked with an asterisk. Only two of these ectopic beats were recorded in the whole strip. Also note the short episode of bi-directional parasystolic tachycardia with fixed coupling between groups A and D at the end of the strip. See text for further discussion. (e) Representative strip taken from the same record as Fig. 2a–d. Beats arising from the fifth parasystolic centre (group E) are indicated by open circles. In this strip the asterisks indicate ectopic beats with fixed coupling. Other symbols as in Fig. 2a–d. Numbers in the upper part of the record indicate coupling interval of group E beats.
III and V3R. The record shows sinus rhythm interrupted by multiform ventricular ectopic beats. Those marked with arrows, asterisks, and triangles, show fixed but different coupling intervals. They probably represent ventricular extrasystoles of multifocal origin. Two groups of ectopic beats have distinct and constant QRS patterns and both show variable coupling intervals. They are indicated by closed circles and squares in the diagrams and are discussed as group I A and I B, respectively.

Group I A beats are readily identified by the tail R waves in V3R and the notched R waves in lead III. Their coupling intervals vary from 48 to 108. On several occasions the ectopic cycle length (Scherf and Schott, 1973) is directly measurable (Fig. 1b) and constant at 88 to 90, corresponding to a rate of 66-6 to 68/min. Longer interectopic intervals are near multiples of the ectopic cycle length, but the variation in the calculated intervals (from 80 to 96) is large. The ectopic intervals, however, seem to lengthen slightly from Fig. 1a to 1b, corresponding to a slight decrease in rate. Such slowing of the ectopic centre might, in this case, afford a possible explanation for the fact that the interval of 288 (Fig. 1b) is slightly longer than three times the cycle length, which is in contrast to the usual observation in parasystole (Chung, 1968; Scherf and Schott, 1973). Alternatively, lengthening of a parasystolic interval could also be the result of one or more of the following: changes in impulse formation or conduction resulting from concealed re-entry (Singer et al., 1974), concealed conduction of a preceding blocked impulse in the ectopic-ventricular junction, or relative refractoriness of the myocardium following a preceding depolarization (Vedoya, quoted by Scherf and Schott, 1973).

In support of the parasystolic character of the rhythm is the finding of fusion beats, marked with an F in the diagram. The true nature of the nineteenth QRS complex (F?) in Fig. 1b could not be established with certainty. It may represent a fusion beat between group I A and group I B, or between group I A or I B and one of the extrasystolic beats, or it may represent the activity of another ectopic centre.

The ectopic beats of group I B are characterized by R waves in V3R and QS deflections in lead III. Their coupling intervals vary from 52 to 92 and their interectopic intervals are multiples of 56 to 61 (mean 58-8) corresponding to a rate of about 102/min.

In group I A and in group I B an exit block must be postulated to account for the occasional absence of these beats outside the ventricular refractory period (Scherf and Bornemann, 1961; Pick, 1966; Pick, Langendorf, and Jedlicka, 1973).

Fig. 2a-e is composed of representative strips taken from a long simultaneous recording of leads I, II, III, and the ensiform lead (Ve) of Case 2. The basic rhythm is atrial fibrillation with a fast ventricular response. The conducted supraventricular beats have a notched QRS complex in leads II and III, consistent with old inferior wall infarction. In all the strips several multiform ventricular ectopic beats can be identified, five groups of which show a remarkably constant QRS configuration in all the represented leads, irrespective of their widely varying coupling intervals. These are discussed below as groups 2A, B, C, D, and E. Furthermore, it is to be noted that the supraventricular beats do not differ significantly in QRS configuration, even when their degree of prematurity and length of the preceding RR interval are comparable to those of beats of groups A–E. Thus, aberrancy as the cause of the abnormal QRS configurations of groups A–E can almost certainly be excluded (Marriott and Sandler, 1966; Singer and Ten Eick, 1971).

Ectopic beats of group 2A are indicated by closed circles in the diagrams. Their QRS complex is characterized by an rS’ deflection in lead I and a QR in leads II and III. They represent the most frequently occurring ectopic beats and have coupling intervals ranging from 24 (e.g. second group 2A beat in Fig. 2c) to 58. Short manifest intervals range from 94 to 104, though the majority are remarkably constant at 96 to 100, denoting a rate of 60 to 62/min. Longer interectopic intervals are multiples of 101 to 107. Furthermore, the eleventh QRS complex in Fig. 2a is a fusion beat with a conducted supraventricular impulse. From these observations there seems to be no doubt that group 2A represents the enhanced activity of a parasystolic centre.

The intervals of 224 and 76 which follow each other in Fig. 2d are of interest, since they differ from the presumed ectopic cycle length. However, their sum equals 300, or 3 x 100. Two explanations seem possible. The first one invokes regular firing at the presumed cycle length of 100 with delayed conduction during the exit of impulse 24 in Fig. 2d. This causes a corresponding shortening of the subsequent interval. This explanation seems very likely, since beat 24 occurs during the relative refractory phase at the end of the preceding T wave. A similar mechanism has been advanced by Vedoya (quoted by Scherf and Schott, 1973) to explain interectopic intervals which are slightly longer than a whole number multiple of the ectopic

\(^1\)All intervals are expressed in hundredths of a second, unless otherwise specified.
cycle length. Delayed conduction during the exit of the impulse has also been shown during fixed rate artificial pacing (Scherf, 1970; Pick et al., 1973). The second explanation assumes sudden dissipation of an exit block (Scherf and Bornemann, 1961), pointing to a smaller common denominator of about 25. Thus the actual discharge rate would be approximately 240/min. It should be pointed out, however, that even if we assume the slower discharge rate of about 60/min, an exit block (Pick, 1966; Pick et al., 1973) must occasionally be postulated, e.g. at the end of the strip in Fig. 2b.

Group 2B beats are characterized by broad QS deflections in leads I, II, and III. They are represented by squares (Fig. 2a and b) and have coupling intervals of 50 to 64. The ectopic cycle length (end of Fig. 2b) is 66, corresponding to a discharge rate of 91/min. Longer intervals, which in this case can adequately be explained by ventricular refractoriness during the Q-aU interval (Pick, 1953; Watanabe, 1971), are multiples of 65 to 70. The fourteenth QRS complex (F) in Fig. 2a is a fusion beat between a group 2B and a conducted supraventricular impulse.

The most conspicuous morphological features of group 2C beats are the narrow peaked R wave in lead I and the notched, relatively small QS deflection in lead III. The ectopic beats of this group are represented by triangles in Fig. 2a and 2c. Coupling intervals vary from 28 to 44. The shortest manifest interval (Fig. 2c) is 76. Longer intervals are multiples of 72 to 78, mean 74-7, corresponding to a rate of about 80/min. Only few, occasionally isolated, beats of group 2C were recorded. The possibility should, therefore, be considered that group 2C might represent ventricular extrasystoles with a conduction disturbance during their exit (Langendorf and Pick, 1955). However, the finding that longer interectopic intervals in the presence of an irregular dominant rhythm are an approximate multiple of the short one, favors a parasystolic mechanism. The occurrence of only few beats of group 2C must then either be the result of the combination of ventricular refractoriness and an increase in exit block, or of temporary loss of automatic activity (Scharloth and Marriott, 1961).

Group 2D beats are indicated by vertical bars (Fig. 2d) and characterized by broad QS deflections in leads I and II and, unlike group 2B, a notched R in lead III. Coupling intervals vary from 32 to 64. Short manifest intervals are constant at 96 to 98 (rate 61/min) and the long interval between beats 9 and 21 (Fig. 2d) is a multiple of 97. The subsequent interval of 178, however, is not: the two explanations which have been suggested above for group 2A to account for the intervals between beats 19 to 24 and 24 to 26 (Fig. 2d) are again possible; a third possibility, namely intermittent parasystole caused by temporary loss of protection (Scharloth and Marriott, 1961; Steffens, 1971; Watanabe, 1971; Cohen, Langendorf, and Pick, 1973; Kinoshita, 1974) and depolarization by beat 22, seems unlikely in view of the fact that the preceding longer interectopic interval was not disturbed and remained a simple multiple of the presumed ectopic cycle length. A short episode of bidirectional ventricular parasystolic tachycardia results from the activity of groups 2A and D at the end of Fig. 2d. There is also a short episode of fixed coupling between the two rhythms in the same part of the record, caused by the equal rates of these two parasystolic rhythms (Langendorf and Pick, 1967).

Finally, the ectopic beats of group 2E show tall R waves in lead I and QS deflections in leads II and III. Isolated beats were recorded on several occasions. Four of them are seen in Fig. 2e and marked with open circles. Coupling intervals range from 42 to 58 and the interectopic intervals are multiples of 100 to 106, corresponding to a rate of 57 to 60/min. As in group 2C, ventricular refractoriness and exit block or temporary dissipation of automatic activity must be postulated to account for the brevity of this episode of manifest group 2E parasystolic activity.

Discussion

Several authors have focused attention on electrophysiological mechanisms which may be responsible for sudden and at times rather wide variations in the manifest or calculated shortest interectopic intervals in parasystole (Scharloth and Marriott, 1961; Scherf and Bornemann, 1961; Steffens, 1971; Cohen et al., 1973; Scherf and Schott, 1973; Kinoshita, 1974; Singer et al., 1974). In such cases, the diagnosis of parasystole must be based on the demonstration of variable coupling and constant interectopic intervals during at least one, but preferably several episodes of the ectopic rhythm. But even then, there is controversy concerning the acceptable variability in what are to be considered constant interectopic intervals. It has been pointed out that, when longer rhythm strips are analysed, some degree of irregularity is almost invariably present (Chung, 1968; El-Sherif and Samet, 1975). It is also evident that when interectopic intervals are measured to or from a fusion beat, they may differ from those measured between two pure ectopic beats, since with a fusion beat one sometimes does not know exactly at what moment the ectopic
depolarization starts to contribute to the morphology of the mixed beat.

With these considerations in mind, we feel that in Case 1 double, and in Case 2 quintuple ventricular parasystole can be diagnosed. During the analysis of the electrocardiogram of Case 2 we have already indicated that aberrancy of supraventricular impulses can be excluded because of the constant QRS configuration of each group in multiple leads, irrespective of their degree of prematurity and length of the preceding RR interval (Marriott and Sandler, 1966; Singer and Ten Eick, 1971). For the same reasons variable intraventricular conduction during a single ectopic ventricular tachycardia with an irregular discharge and varying degrees of exit block can be excluded.

Our cases show that the diagnosis of multifocal parasystole is greatly facilitated and can sometimes only be established when long, multiple, and simultaneously recorded leads are available. In a single lead, complexes arising from different parasystolic foci may display near-identical configurations, e.g. Case 2, lead I, QRS configuration of groups B and D. Furthermore, when such complexes are interspersed in a single lead, the apparent irregularity of interectopic intervals may even lead to rejection of the diagnosis of parasystole.

With the availability of multiple simultaneously recorded leads multifocal parasystole may well turn out not to be as rare an arrhythmia as would be inferred from the rarity of published reports. From an electrophysiological point of view one would also expect that the primary derangement which leads to regular impulse formation within and protection around an ectopic focus, will exert the same type of influence on other groups of pacemaking cells in the atria or ventricles; though the sensitivity of these cells to a particular environmental change may differ (Hoffman and Cranefield, 1964), identical cell types, such as Purkinje fibres, may be expected to react in the same way.

It is also interesting to find that in our cases all the seven parasystolic centres had an enhanced rate of discharge, ranging from 57 to 102/min. In three of these a exit block could be shown. Also unusual is the observation that beats arising from three of the ectopic centres (Case 2, groups A, C, and D) at times had extremely short coupling intervals, leading to interruption of the terminal part of the T wave of the preceding beat. In none of these cases was repetitive firing or ventricular fibrillation seen.

As in all other published cases (Chung et al., 1964; Chung, 1968; Roelandt et al., 1962; Dolgin, 1973; El-Sherif and Samet, 1975), our patients had organic heart disease. They are, however, still alive six and nine months after the arrhythmia was first detected.

References

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