Parasystole

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Five instances of parasystolic rhythm are presented in which detailed analysis revealed some particular physiologic aspect pertinent to the understanding of this type of disturbance of rhythm. In four cases the ectopic parasystolic focus was localized in the ventricles and in one in or near to the A-V junction; in two of the former instances the site of the ectopic focus appeared to be the interventricular septum. The mechanism of protection of the parasystolic center, while not identical in every case, might be effected by an area of true block, as exemplified by one instance. Parasystole offers the rare opportunity to determine the refractory phase of the A-V junction or of the ventricular myocardium in the human heart. Occasionally parasystolic rhythm may appear in association with other types of manifest multiple cardiac pacemakers and produce very complex arrhythmias, which can be resolved by application of known physiologic principles.

Normally, rhythmic contraction of the entire heart is initiated and maintained by a single pacemaker, the sinus node. It dominates all other potential pacemakers by virtue of its relatively faster rate of cyclic discharge. Simultaneous activity of two cardiac pacemakers occurs in A-V dissociation; here, each pair of chambers, the auricles and ventricles, is governed entirely, or for the most part, by its own pacemaker. Rarely, two rhythmic and completely independent pacemakers operating at different rates can be seen to be in competition for the activation of the ventricles or of the auricles. Such a condition is termed parasystole.

Fleming who conceived this type of disturbance of cardiac rhythm while analyzing polygraphic curves was aware of the basic problem involved, namely, to account for the undisturbed activity of the slower cardiac center in a chamber which, at the same time, responds to stimuli from another faster center. Since then, based on the electrocardiogram, a number of clinical instances of parasystolic rhythm has been reported as well as similar observations in the experimental animal. Temporary or permanent "immunity" of a subsidiary pacemaker to impulses of the dominant rhythm has been accepted as a fact although its explanation remains controversial.

The present report deals with some observations bearing on the mechanism of parasystole. Five electrocardiograms which satisfy criteria postulated for the diagnosis of parasystolic rhythm were analyzed. The methods used for analysis and the conclusions which were derived are described in the legends of figures 1 to 5 and this approach can be followed by studying the corresponding diagrams. Each case illustrates a particular physiologic aspect of parasystolic rhythm.

Thus, figure 1 presents an example of ventricular parasystole in its "purest" form with its implications concerning the operation of the refractory period and of interference. In figures 2 and 3 the site of a ventricular parasystolic focus appears to be revealed by a consideration of the contour of ectopic and dominant beats; moreover, figure 3 seems to provide some clue as to the mechanism of "protection" of such a parasystolic ectopic focus. In figure 4, a parasystolic focus in, or close to, the A-V node appears to be shielded from other impulses passing the A-V junction in both directions. Factors responsible for the apparent irregularities of the manifestation of this parasystolic pacemaker are revealed by detailed analysis, and the duration of the refractory phase of A-V junctional tissues can be estimated closely. Finally, in figure 5, the unusual combination

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of ventricular parasystole and re-entry is seen in the same tracing, and the association of parasystole with an incomplete A-V dissociation aptly illustrates the fundamental difference in the two apparently similar mechanisms.

**Comment**

**The Diagnosis of Parasystole**

Typically, parasystole is manifested in the electrocardiogram by the appearance of auricular or ventricular premature beats of more or less aberrant contour depending on the site of ectopic impulses formation. Contrary to the common variety of premature beats, their coupling with the preceding dominant beats varies widely and leads to interference of dominant and ectopic impulses at various levels. Thus, compensatory pauses, interpolation and fusion beats can usually be seen in the same tracing (figs. 1 and 2). This, however, as has been
shown in this department, may occur also in the absence of parasystole. In order to prove the latter, the presence of an autonomous ectopic rhythm, different in rate from that of the dominant pacemaker, must be demonstrated. If the rate of the normal beats is slower or temporarily depressed then the parasystolic cycle may be measured directly by the time of appearance of two or more successive ectopic beats (fig. 4).

This is done when (a) the intervals separating ectopic beats can be reduced to a least common denominator corresponding to the cycle length of the parasystolic ectopic rhythm and (b) all ectopic impulses which occur after the end of the refractory phase following dominant beats become manifest in the record (figs. 1 to 5).

**Fig. 2. Parasystole originating in the interventricular septum.** The conventions in the diagram are the same as in figure 1. In addition, the values in parentheses between interrupted horizontal arrows (upper line) are the temporal sequence calculated for latent discharges of the ventricular focus.

There is a regular sinus rhythm (76 per minute) with occasional premature beats. The QRS duration of the sinus beats is 0.12 second and its contour in all the leads (including precordial) suggests a minor left sided intraventricular conduction defect. By contrast, the QRS duration of the premature beats is normal (0.09 second). The latter appear (a) at varying intervals—the long one in lead II, for example, is three times as long as the short ones in this and in other leads; (b) with varying coupling to the preceding sinus beat (0.56 to 0.80 second)—the longest ones result in ventricular fusion beats. Thus, a parasystolic pacemaker with a discharge rate of 45 to 46 per minute is present. The refractory phase of the ventricles, as far as it can be determined in these three short strips, is somewhere between 0.32 second (the longest coupling of a latent discharge) and 0.50 second (the shortest coupling of a manifest discharge). The shorter QRS duration of the ectopic beats compared with that of the sinus beats suggests that the parasystolic focus is located distal to a lesion blocking intraventricular conduction, at a point approximately equidistant from both bundle branches, most likely in the interventricular septum.

**The Localization of the Parasystolic Focus**

While it has been stated that supraventricular parasystolic rhythm, especially in the A-V node, is rare, several such cases are on record, and one is illustrated in figure 4. Unlike ventricular parasystole, the nodal type seems
However, in most instances, the constant contour of the ectopic ventricular complexes, despite marked variations of their coupling to the preceding beats, is against such an interpretation. On the other hand, the absence of ventricular fusion beats, as in figure 3, does not necessarily prove supraventricular origin of the ectopic rhythm.

Premature beats in the presence of an intraventricular conduction defect may occasionally exhibit a more normal contour, and/or QRS complexes shorter in duration, than the normal beats (figs. 2 and 3). Such observations have been subject to various interpretations. Persistence of the phenomenon in the face of marked variations of the coupling of the premature beats renders supernormal phase of intraventricular conduction a less likely explanation than localization of the ectopic focus to the interventricular septum as proposed in this report and previously by us and by others.

Retrograde conduction of the parasystolic impulse to the auricles could be used to favor its localization within the A-V node. However,
Fig. 4. *Supraventricular (nodal) parasystole with demonstrated bidirectional protection.* The convention in the diagrams are as follows: The vertical lines at the level of A indicate auricular activation; the solid vertical lines at the level of V, activation of the ventricles by a supraventricular impulse; the interrupted vertical lines at the level of V, activation by an idioven tricular impulse. Figures between lines at the level of A and V represent the time sequence between neighboring auricular and ventricular beats respectively. Supraventricular impulses originating in or near the A-V junction are indicated by dots at the level of A-V, and intervals separating them are indicated by figures within horizontal arrows. The oblique lines at the level of A-V stand for conduction of impulses from auricles to ventricles or vice versa; the varying of inclination of these lines indicates varying speeds of conduction of the impulses through the A-V junctional tissues; short lines at right angles to them, blockage of the impulse. A figure to the left of a dot (at the level of A-V) gives the calculated time interval between a nodal impulse and the last preceding sinus impulse crossing the A-V junction. The time values are as in figure 1.

Three types of ventricular activation are seen: (1) impulses arising in the sinus node at a rate varying between 59 and 65 per minute and with a P-R interval of 0.18 second; (2) impulses arising in or near the A-V junction activating not only the ventricles but also, in retrograde fashion, the auricles as evidenced by inverted P waves in leads II and III preceding normal QRS complexes at varying intervals; (3) premature ventricular complexes of bizarre configuration originating in the ventricles, likewise transmitted in retrograde direction to the auricles. While the coupling of the ventricular ectopic impulses to the preceding beats is fixed (0.48 second) that of the supraventricular ectopic impulses is not. This is due to (a) premature appearance of some of the latter, and (b) delay in their forward and retrograde conduction. The rate of discharge of the ectopic nodal pacemaker (55 per minute) can be determined by measuring the intervals between nodal beats having equal P-R intervals—for example the interval between the seventh and eighth beat in lead I (1.08 second) and between the fourth and eighth beat in lead II (3.20 second or 3 × 1.07 second). The latter is a multiple of the former. On the assumption that sinus as well as nodal impulses that follow long pauses are conducted through the A-V junction at the same rate of speed, the time of discharge of additional nodal impulses can be calculated. Thus, for example (see diagram), the time of nodal discharge responsible for the sixth complex in lead I is defined by the interval between the QRS before and that after this beat (0.46 plus 1.04 second) minus 1.08 second, the cycle between nodal impulses. This makes the time interval separating this nodal discharge from the time at which the preceding sinus impulse traverses the A-V junction 0.42 second. Using the cycle length 1.08 second as that of the regular parasystolic nodal pacemaker, permits location of its several discharges in this lead (indicated by dots in the diagram). A similar method can be employed in leads II and III. In lead II a cycle length of 1.07 second is used for the parasystolic nodal pacemaker (see diagram).

Nodal impulses occurring 0.41 second, or later, following passage of a sinus impulse through the A-V junction are transmitted to both auricles and ventricles, whereas those occurring 0.36 second, or less, after a sinus impulse passage are not conducted and remain latent. Thus the refractory phase of the A-V junctional tissues can be determined within the narrow range of 0.05 second. Furthermore analysis shows that neither the impulses transmitted from the sinus node nor the retrograde ones initiated by the premature ventricular beats disturb the regular action of the nodal parasystolic pacemaker. There is thus protection of the nodal parasystolic pacemaker from impulses from above and from below while it itself can transmit impulses in both directions (except for the refractory period).
Fig. 5. Ventricular parasystole (with retrograde conduction and a reciprocal beat) associated with incomplete A-V dissociation due to nodal tachycardia. In the diagram below each lead, the conventions in the upper part (labeled A, A-V and V) are the same as used in figure 4, while those in the lower part (labeled P) are the same as in figs. 1 to 3. Three types of ventricular activation are seen in leads I and II and four types in lead III. Impulses are present which arise in a ventricular parasystolic pacemaker with a discharge rate of 49 to 51 per minute, but only those coming 0.48 to 0.51 second or more following a supraventricular impulse become manifest in the form of more or less bizarre appearing beats (best seen in lead III). The tendency for retrograde conduction of these parasystolic idioventricular impulses to occur is evidenced by an instance (fourth and fifth complex combination in lead III) where such an impulse not only reaches and activates the auricles in retrograde fashion but also re-enters the A-V junction and causes a reciprocal beat. In addition, this retrograde impulse causes a disturbance of the regular discharge of the sinus node represented by the succession of upright P waves occurring at a rate of 65 to 68 per minute. In fact, this is the only interruption of the sinus rhythm found.

Most of the sinus impulses are prevented from activating the ventricles by interference from a third and faster pacemaker (67 to 71 per minute) arising in the A-V node. Only those auricular impulses reach and activate the ventricles which arrive at the A-V junction after a sufficiently long period to allow for recovery from a preceding impulse, whether conducted in forward or retrograde direction. This latter they do with varying P-R intervals, and with varying degrees of aberrant intraventricular conduction. This last mechanism is, of course, A-V dissociation with ventricular captures.

Penetration into the A-V junction (concealed conduction) of some of the “nonconducted” auricular impulses (first P wave in lead I and seventh P wave in lead III) is suggested by the failure of subsequent idioventricular parasystolic impulse to complete its retrograde conduction into the auricles.

Both the sinus and retrograde ventricular impulses discharge the nodal pacemaker prematurely when they reach it, but the regular action of the parasystolic ventricular focus remains undisturbed owing to some protection mechanism.

At times, for example, the sixth beat in lead I and the second beat from the end in lead III, all three pacemakers—the sinus node, the A-V node and the parasystolic ventricular focus—are discharging almost simultaneously. In such instances only two of these impulses become manifest, while the third (nodal or ventricular) remains latent due to the refractoriness of the junctional tissues or of the ventricular myocardium established by the others.

This type of analysis, therefore, makes an otherwise inexplicable record fit into an orderly scheme.
retrograde spread of ventricular premature impulses is more common than generally assumed, and is demonstrated by figure 5. This figure reveals that even re-entry of the retrograde impulse may be initiated by a parasystolic ventricular focus. Retrograde activation of the auricles may temporarily depress the discharge rate of the sinus node and thus result in spontaneous temporary manifestation of the parasystolic interval. For this purpose, carotid sinus pressure and/or application of cholinergic drugs have been suggested as diagnostic procedures in parasystole.

The “Protection” of the Ectopic Focus

Undisturbed simultaneous activity of two rhythmic centers of different rates, side by side as it were, has been recorded in isolated fibers of the specific muscular system of the heart. While this observation provides strong support in favor of the concept of parasystolic rhythm, the mechanisms by which one rhythmic center is protected from the impulses of others has been a matter of speculation. The idea of an area of unidirectional block surrounding an ectopic focus has been proposed, with allowance for temporary alteration of conditions of entrance and exit of impulses, to account for occasional irregularities of the parasystolic center (see below). Scherf, however, rejects the concept of protection block. As a result of extensive experimental and clinical studies on ectopic impulse formation he concludes that guarding of an ectopic center from extraneous impulses can be accounted for by the inherent, manifest or latent rapid rate of its discharge. He states that the main factor determining the type of manifestation of an ectopic center is its excitability in relation to the strength of impulses dominating the rhythm of the heart.

If the interpretation of figure 3 of this report is correct, it can be used in support of the block theory of protection. A depressed area in the left bundle branch, evidenced by the contour of the sinus beats, appears to prevent passage of impulse to, but not from, a slow ventricular pacemaker. Thus, true block not only seems to protect continuous formation of impulses in an ectopic focus but may actually be the factor permitting the original initiation of its activity. In this case Scherf’s concept is not necessary.

However, the mechanisms effecting protection of an ectopic center from extraneous impulses may not be the same in every case. In figure 4, which is an instance of nodal parasystole, impulses by-passing a rhythmic pacemaker in two directions are conducted through the A-V junction at normal speed. Hence, an area of regional depression protecting the parasystolic center from discharge appears less likely. Under such circumstances, any ectopic focus could be conceived as operating apart from the normal pathways of forward and retrograde conduction, and as being connected to adjacent structures by fibers with unidirectional conduction. Such a dissociation of functions of the junctional tissues, while hypothetic in our present stage of knowledge, does not seem impossible considering the complex histologic architecture of the A-V node. The same mechanism may also prevail in other parts of the heart and give rise to a parasystolic pacemaker.

The Regularity of the Parasystolic Pacemaker

Regularity of discharge of the ectopic pacemaker is the accepted cardinal sign of parasystole. Thus in figures 1 to 5, the cycles, both those actually measured and those calculated, do not vary by more than 0.05 second, which is within the limits of errors of measurement. If criteria used for the differential diagnosis of paroxysmal tachycardias were applied, much greater variations could be allowed in the case of a ventricular focus, but this would render the diagnosis of parasystole very difficult. As a matter of fact, parasystole has been recognized in the face of an apparent irregularity of its pacemaker by assuming either a temporary “break through” of the impulses of the extraneous dominant pacemaker past the barrier of protection of the ectopic focus (intermittent parasystole) or a temporary release of an “exit block” which had kept many of its impulses confined exclusively to the immediate region of the parasystolic pacemaker. Furthermore, variations in the manifest cycle of the parasystolic beats can be expected with varying degrees of delayed con-
duction of impulses from the ectopic center to
the surrounding myocardium such as occurs in
second degree A-V block.\textsuperscript{1, 7, 13, 14, 15} This latter factor will become manifest in the form of
varying A-V conduction times, when the para-
systolic focus is located in the A-V node, as in
figure 4.

The Determination of the Refractory Period

It is ordinarily impossible to determine ex-
actly the refractory period of the human heart. Para-
systole offers the unique opportunity of
determining, the duration of the unresponsive-
ness following stimulation of tissues surround-
ing the ectopic pacemaker, provided that mani-
fest and latent spacing of ectopic impulses
remains regular over a long period of observa-
tion. The refractory phase is defined, on one
hand, by the longest calculated coupling to the
previous beat of a latent ectopic discharge, and,
on the other hand, by the shortest of such
coupling resulting in a manifest ectopic beat.\textsuperscript{4, 16, 14} In the instance illustrated in part in figure
1, the duration of ventricular unresponsiveness
could be defined within the limits of 0.05 sec-
ond. It proved to be considerably longer than
the normal Q-T interval which usually is iden-
tified with the duration of ventricular refrac-
toriness. While both ventricular refractory pe-
riod and Q-T duration may vary to a similar
degree with variations in rate and, therefore,
from case to case (figs. 1, 2, 3, 5), it becomes
obvious that the electrical phenomenon of re-
polarization does not necessarily coincide tem-
porally with a functional phenomenon related
to the heart’s excitability.

An approximate measurement of the normal
and abnormal refractory period of A-V nodal
tissues can be obtained in incomplete A-V dis-
sociation by correlating the incidence, and the
P-R intervals, of “ventricular captures” with the
respective R-P distances. Such calculations,
however, have only relative value since neither
the exact time of arrival of the auricular stimu-
lus at the A-V junction, nor the point of its
interference with the nodal impulse, is known.
The procedure indicated in the diagrams of
figure 4 represents an attempt to define the
refractory period of nodal tissues by a method
similar to that used in the case of ventricular
parasystole, in the belief that it may yield more
exact information in the future, when applied
to a larger amount of material.

The Definition of Parasystole

There is no general agreement to which con-
ditions of disturbed cardiac rhythm the term
parasystole should be applied. Thus, instances
exemplified in this report have been classified
by some, together with instances of A-V dis-
sociation, under the common heading of “para-
rhythms.”\textsuperscript{47, 48} Occasionally, the two types of
disturbance of rhythm may occur in association
as shown in this report (fig. 5) and previously.\textsuperscript{24}
Under such circumstances, the need becomes
obvious to define and to distinguish sharply
these two apparently similar conditions: One,
the “protected” simultaneous activity of two
cardiac pacemakers which is parasystole; the
other, its “unprotected” counterpart appearing
as complete and incomplete A-V dissocia-
tion.

Summary and Conclusions

1. The principles of parasystolic rhythm and
their implications are discussed on the basis of
five selected examples.

2. In two instances of ventricular parasys-
tole, the origin of the ectopic rhythm in the
interventricular septum was suggested by com-
parison of the contour of the ectopic beats with
that of the dominant beats. In one of them, the
ectopic center appeared localized in, or close to,
the left bundle branch and in a region of im-
paired conduction. In this instance, therefore,
the theoretic concept of protection block ap-
ppeared to be substantiated. However, alterna-
tive mechanisms in other instances are possible
and their relative role may vary from case to
case.

3. The presence of a parasystolic center pro-
vides the unique possibility of determining in
the human heart the duration of the refractory
phase in tissues which surround the ectopic
pacemaker. In one case of ventricular para-
systole, it was found to outlast the electrical
activity of the ventricles as determined by the
Q-T interval.

4. The occasional association of parasystole
with other complex disturbances of cardiac
rhythm requires a sharp distinction between the various types of arrhythmias affected by simultaneous activity of more than a single cardiac pacemaker.

5. The term parasystole refers to those instances of double rhythm of the auricles or of the ventricles in which one pacemaker is "protected" from the impulses of the other.

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SUMARIO ESPAÑOL

Se presentan cinco casos de ritmo parasistólico con análisis detallado que revela algún aspecto particular fisiológico pertinente al entendimiento de este tipo de disturbio en ritmo. En cuatro de los casos el foco ectópico parasistólico se localizó en los ventrículos y en uno, en cerca de la juntura A-V; en dos de los primeros casos la localización del foco ectópico fué en el septo interventricular. El mecanismo de protección del centro parasistólico, aunque no idéntico en cada caso, puede ser afectado por un área de bloqueo, como se demostró en un caso. El parasistole ofrece la rara oportunidad de determinar la fase refractiva de la juntura A-V o del miocardio ventricular en el corazón humano. Ocasionalmente el ritmo parasistólico puede aparecer en asociación con otros tipos de pacificadores cardíacos múltiples manifiestos y produce unas arritmias muy complejas que pueden ser analizadas mediante la aplicación de principios fisiológicos ya conocidos.

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