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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Aided by the Sam and Sarah Ehrlich Memorial Fund for Cardiovascular Research and the Michael Reese Research Foundation.
of ventricular parasystole and re-entry is seen in the same tracing, and the association of parasystole with an incomplete A-V dissocia-
lar or ventricular premature beats of more or less aberrant contour depending on the site of ectopic impulses formation. Contrary to the

![Image](http://circ.ahajournals.org/)

**Fig. 1. Ventricular parasystole with simple interference.** The conventions in the diagram below the tracing are as follows: The lower horizontal line represents the ectopic ventricular focus. Its manifest discharge (corresponding to the premature beats in the electrocardiogram) is indicated by the upwards directed vertical arrows, its ineffective (latent) discharge by the dots. The simultaneous short vertical arrows pointing towards each other (at the end of the top strip) represent simultaneous activation by a sinus node and an ectopic ventricular impulse (a ventricular fusion beat). The values within the horizontal arrows (upper line) indicate the temporal sequence of the ectopic impulses; the longer intervals are also given as multiples of the short ones. The values just to the left of the vertical arrows (lower curve) are the time intervals between premature beats and the last preceding sinus beat (manifest coupling); the values in parentheses just to the left of the dots are similar time intervals in the case of the latent discharges of the ventricular focus (latent coupling). All time values represent the number of hundredths of a second.

The two tracings are successive portions taken from a long strip of lead II. The slightly irregular sequence of sinus beats (51 to 70 per minute) is disturbed by premature ventricular complexes. With the exception of the last beat in the upper strip, which is intermediate in contour between the normal and ectopic beats, all have the same bizarre contour with QRS prolonged, revealing their ventricular origin. As evident from the diagram, the coupling of the premature beats to the preceding sinus beat varies considerably (0.52 to 0.96 second), resulting in interpolation of the beats with the shorter coupling, and a ventricular fusion beat on occasion of the longer coupling. The long intervals between two ectopic beats are multiples of the shortest ones. All this indicates the presence of a ventricular parasystolic focus operating at a rate of 38 to 40 per minute. As determined in the entire tracing, and exemplified in the two short strips shown, this ventricular pacemaker becomes manifest in the form of a premature beat whenever its discharge follows activation of the ventricles by a sinus impulse by 0.52 second or more, and its discharge remains latent whenever this interval is 0.48 second or less. Thus, in this case, the only factor dominating the manifestation of the slower impulses of the ventricular pacemaker is simple interference which results from the refractory phase of the ventricles after each stimulation by the more rapid impulses from the sinus node. The duration of ventricular unresponsiveness (0.48 to 0.52 second) is considerably longer than that of the Q-T interval (0.40 second) of the sinus beats.

Comment

**The Diagnosis of Parasystole**

Typically, parasystole is manifested in the electrocardiogram by the appearance of auriculo-

![Image](http://circ.ahajournals.org/)

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,\textsuperscript{34} 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\textsuperscript{3, 9, 24, 23, 31} or temporarily depressed\textsuperscript{21, 26, 28} then the
parasystolic cycle may be measured directly by
the time of appearance of two or more successive ectopic beats (fig. 4).

\textbf{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.

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).

\textbf{The Localization of the Parasystolic Focus}

While it has been stated\textsuperscript{23} that supraventricular parasystolic rhythm, especially in the A-V
node, is rare, several such cases are on record,\textsuperscript{9, 20-22, 24} and one is illustrated in figure 4. Unlike
ventricular parasystole, the nodal type seems
PARASYSTOLE

to occur in cases without clinical evidence of heart disease.

As in paroxysmal tachycardia, supraventricular origin could be postulated in any case of parasystole if the assumption is made that there is aberrant spread of the ectopic impulse through the ventricular myocardium. How-
ventricular 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 pre-

![Diagram](https://example.com/diagram.png)

**Fig. 3. Ventricular parasystole with demonstration of protection block, both in the left bundle branch.** The conventions in the diagram are the same as in figures 1 and 2.

Regular sinus beats (rate 88 per minute) have widened QRS complexes (0.14 second) and, in V2, a contour usually seen with left bundle branch system block. This was confirmed by the appearance in the other leads of the entire tracing, which included a long strip of lead II. Premature beats with the appearance characteristically seen in right bundle branch system block (see V3) but with less prolonged QRS complexes (0.10 second) occur with coupling to preceding sinus beats. The long interval separating two premature beats in the first portion of lead II is a multiple of the remaining shorter ones shown. Thus, a parasystolic pacemaker discharging at a regular rate of 98 per minute appears to be operating in the left ventricle distal to a blocking lesion in the left bundle branch. Its impulses remain latent when they occur less than 0.40 second after a sinus beat. The fourth ventricular complex from the end in lead V2 is a sinus beat. Its QRS begins only 0.04 second before the expected parasystolic impulse; yet the ventricular fusion beat anticipated in the presence of left bundle branch block under these circumstances (25) does not occur. Evidently, here, the short time of 0.04 second permitted the sinus impulse to reach the left ventricle via the interventricular septum and to prevent the activation by the parasystolic pacemaker.

A fusion beat in parasystole may fail to occur at the expected time if the sinus and ectopic impulses share a common pathway during activation of the particular ventricle which is the seat of the ectopic pacemaker. This consideration together with that of the contour and QRS duration of the sinus and ectopic beats suggests that the parasystolic pacemaker in this instance is located (a) in, or close to, the main left bundle branch and (b) in a region which is “protected” from the faster sinus impulses by an area of depressed unidirectional conduction.

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 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,
Three types of ventricular activation are seen: (1) impulses arising in the sinus node at a rate varying between 50 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 after 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. 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 idioventricular 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.
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 tissue, 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. This latter factor will become manifest in the form of varying A-V conduction times, when the parasystolic focus is located in the A-V node, as in figure 4.

The Determination of the Refractory Period

It is ordinarily impossible to determine exactly the refractory period of the human heart. Parasystole offers the unique opportunity of determining, the duration of the unresponsiveness following stimulation of tissues surrounding the ectopic pacemaker, provided that manifest and latent spacing of ectopic impulses remains regular over a long period of observation. 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. In the instance illustrated in part in figure 1, the duration of ventricular unresponsiveness could be defined within the limits of 0.05 second. It proved to be considerably longer than the normal Q-T interval which usually is identified with the duration of ventricular refractoriness. While both ventricular refractory period 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 repolarization does not necessarily coincide temporally 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 dissociation 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 stimulus 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 conditions 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 dissociation, under the common heading of "pararhythms." Occasionally, the two types of disturbance of rhythm may occur in association as shown in this report (fig. 5) and previously. 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 dissociation.

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 parasystole, the origin of the ectopic rhythm in the interventricular septum was suggested by comparison 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 impaired conduction. In this instance, therefore, the theoretic concept of protection block appeared to be substantiated. However, alternative mechanisms in other instances are possible and their relative role may vary from case to case.

3. The presence of a parasystolic center provides 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 parasystole, 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 effected 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.

ACKNOWLEDGMENTS

The author is indebted to Drs. L. N. Katz and R. Langendorf for valuable criticisms.

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 o 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 refractoria 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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Circulation. 1953;8:243-252
doi: 10.1161/01.CIR.8.2.243

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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