Two Cases of Supraventricular Parasystole

By NANCY C. FLOWERS, M.D., G. DANIEL COPELAND, M.D.,
and DANIEL A. BRODY, M.D.

PARASYSTOLE is a mechanism resulting from rhythmic formation of impulses from two foci in the heart. In the case of atrial parasystole, one focus is usually in the sinoatrial node and the other somewhere in the atrial myocardium. The focus with the faster impulse formation serves as a dominant pacemaker for the heart. The parasystolic focus characteristically forms impulses with remarkable regularity and seems invulnerable to the effects of the sinus impulse. On some occasions the atrial depolarization wave is not visible with each fired parasystolic impulse due to refractoriness of the surrounding atrial muscle from a recently conducted sinus impulse. The parasystolic pacemaker, however, continues to operate in an almost perfectly rhythmic fashion as evidenced by the fact that each atrial depolarization wave of the ectopic form occurs at the time of a multiple of the expected interval if not at the expected interval. Thus, the idea of an abnormal protection or entrance block was proposed to explain the inability of the sinus impulse to discharge and render refractory the parasystolic focus.\(^1\) Alternatively, Katz and Pick suggested the possibility that fibers in the adjacent area to the parasystolic focus might normally have the property of unidirectional conduction and provide a preferential pathway allowing impulses to leave but not to enter the area.\(^1\) We readily admit that we do not have the final answer, nor do we know of any conclusive experimental evidence to explain the phenomenon. We do think, however, that it can be diagnosed by the criteria suggested by Katz and Pick,\(^2\) which include (1) variation in coupling of the ectopic beats and (2) intervals between successive parasystolic P waves or between fusion and parasystolic P waves that are either equal to or a close multiple of a common denominator.

The following two cases illustrate the relatively rarely reported supraventricular parasystole. Though it is readily conceded that this phenomenon occurs with less frequency than its counterpart, ventricular parasystole, we believe that at least some of the responsibility for its apparent rarity lies in the fact that it is often overlooked. It would be difficult indeed to understand this if all cases were as obvious on the standard electrocardiogram as the first case we present. Without the aid of esophageal electrocardiography, however, our second case would doubtless have been overlooked.

Case Reports

Case 1

A 58-year-old white coal miner was followed at Beckley Memorial Hospital and the ambulatory-patient clinic from October 19, 1959, until he succumbed on June 10, 1962. He had been found to have advanced pulmonary emphysema and fibrosis, recurrent bouts of pneumonia, bronchiectasis, pulmonary hypertension, and possibly arteriosclerotic heart disease, as evidenced by electrocardiographic findings of a possible old anterior myocardial infarction. He was intermittently on digitalis leaf but it was generally conceded that most of his respiratory symptoms were initially, at least, on the basis of pulmonary rather than myocardial disease. Later in his course cor pulmonale became a prominent feature.

His final admission was between June 4, 1962, and June 10, 1962, for increasing dyspnea, irrational behavior, and restlessness. At the time of this admission he was cyanotic about the lips, mucous membranes, and nailbeds. The anteroposterior diameter of his chest was increased, retraction of the intercostal spaces was evident on inspiration, and his use of accessory muscles of inspiration in the neck was prominent. His chest...
was hyperresonant to percussion. Fine rales were present in both lung bases. A systolic gallop rhythm was present. The liver was two finger-breadths below the right costal margin and was tender. Moderate lower extremity edema was noted.

The patient’s carbon dioxide content was 44 mEq./L. and later increased to 51 mEq./L. The nonprotein nitrogen was 31 mg. per cent. The electrocardiogram on admission showed sinus rhythm with borderline first-degree atrioventricular block. Failure of the R waves to progress across the precordium was considered to represent an old anteroseptal myocardial infarction but equally likely was the possibility that this pattern was due to marked pulmonary disease. On June 8, 1962, an electrocardiogram showed a paraystolic focus in lead aVF that was presumed to be either quite low in the atrium or possibly in the atrioventricular node. Premature atrial systoles from other sites were also present.

The patient was maintained on 0.1 Gm. of digitals leaf daily and started on dichlorphenamide, a carbonic anhydrase inhibitor. About 48 hours after admission he had lost 8 pounds of weight, possibly related to diuresis. On June 9, 1962, his electrocardiogram showed a more intense pattern of right heart strain, developing deeper S waves in V5 and V6 and more right axis deviation. On June 10, 1962, he suddenly died. Autopsy findings included diffuse severe pulmonary emphysema, pulmonary sclerosis, organized thrombi of small branches of the pulmonary artery, fibrous pleural adhesions, and right heart dilatation and hypertrophy.

**Case 2**

An 85-year-old Negro man was admitted to the urology service of the John Gaston Hospital on July 12, 1958, for urinary symptoms associated with benign prostatic hypertrophy. He had experienced exertional dyspnea for 3 to 4 months and slight ankle edema since a stroke 3 to 4 years previously. Two weeks prior to admission he had had an amputation of his right second toe for what was thought to be arteriosclerotic gangrene.

Physical examination revealed a blood pressure of 180/90 mm. Hg, peripheral pulse of 64, respiratory rate of 20, and temperature of 99°F. The chest was clear to auscultation, and percussion of the breath sounds was distant. No rales were heard. A grade-II/III systolic murmur was noted over the precordium. The presence of a diastolic murmur was questioned by one examiner. This was not described. An irregular cardiac rhythm was noted. The prostate was considerably enlarged, smooth, non-nodular, and quite firm. The entire right foot was black, thought to represent ischemic gangrene, though dorsalis pedis pulses were felt bifurlarly. At this time the electrocardiogram was interpreted as showing an incomplete atrioventricular block of the Wenckebach type with premature ventricular systoles. The patient’s arrhythmia was thought possibly to be due to coronary artery disease, since he had received no digitals and had peripheral arteriosclerosis of a severe degree, benign prostatic hypertrophy with urinary obstruction, and evidences of an old cerebrovascular accident with residual left hemiparesis. The blood urea nitrogen was 16 mg. per 100 ml. *Pseudomonas aeruginosa* was

### Table 1

**Coupling Intervals in Seconds of Parasytolic P to Preceding P**

<table>
<thead>
<tr>
<th>Case 1 (fig. 1)</th>
<th>Case 2 (fig. 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.36 0.36</td>
<td>0.40</td>
</tr>
<tr>
<td>0.24 0.24</td>
<td>0.40</td>
</tr>
<tr>
<td>0.32 0.26</td>
<td>0.46</td>
</tr>
<tr>
<td>0.36 0.36</td>
<td>0.56</td>
</tr>
<tr>
<td>0.32 0.40</td>
<td>0.62</td>
</tr>
<tr>
<td>0.32 0.28</td>
<td>0.30</td>
</tr>
<tr>
<td>0.28 0.30</td>
<td></td>
</tr>
<tr>
<td>0.32 0.28</td>
<td></td>
</tr>
<tr>
<td>0.46 0.32</td>
<td></td>
</tr>
<tr>
<td>0.30 0.42</td>
<td></td>
</tr>
<tr>
<td>0.32</td>
<td></td>
</tr>
</tbody>
</table>

*In case 1 the coupling intervals varied from 0.24 to 0.46 second. In case 2 the coupling intervals varied from 0.30 to 0.62 second.

### Table 2

**Parasytolic Intervals in Seconds**

<table>
<thead>
<tr>
<th>Case 1 (fig. 1)</th>
<th>Case 2 (fig. 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.76 ± 0.04</td>
<td>3.24</td>
</tr>
<tr>
<td>0.88 - 0.90</td>
<td>1.04</td>
</tr>
<tr>
<td>0.88 - 0.90</td>
<td>2.12</td>
</tr>
<tr>
<td>basic P1-P1 interval</td>
<td>1.06 ± 0.02</td>
</tr>
</tbody>
</table>

In the upper strip of case 1 (fig. 1) the parasytolic interval was found to be 0.76 second with a variation of plus or minus 0.04 second. In the lower two strips of figure 1 the parasytolic rate had slowed somewhat so that the interval became 0.88 with a variation of only ± 0.02 second. In case 2 the basic parasytolic interval was measured in the case of the third and fifth P1-P1 intervals to be 1.04 second while in the case of the second P1-P1 interval the measurement was 1.08 second. The first P1-P1 interval represents a multiple of three times 1.08 second while the interval in the case of the fourth P1-P1 interval is a multiple of two times 1.06 second.
Table 3

R-P Intervals in Seconds, Case 1 (Fig. 1)

<table>
<thead>
<tr>
<th>Upper strip</th>
<th>Middle strip</th>
<th>Lower strip</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.25*</td>
<td>0.18</td>
<td>0.16</td>
</tr>
<tr>
<td>0.15</td>
<td>0.20*</td>
<td>0.22</td>
</tr>
<tr>
<td>0.20</td>
<td>0.28*</td>
<td>0.24*</td>
</tr>
<tr>
<td>0.20</td>
<td>0.19</td>
<td>0.16</td>
</tr>
<tr>
<td>0.24</td>
<td>0.20*</td>
<td>0.20</td>
</tr>
<tr>
<td>0.27*</td>
<td>0.17</td>
<td>0.23*</td>
</tr>
<tr>
<td></td>
<td>0.17</td>
<td>0.24*</td>
</tr>
</tbody>
</table>

* R-P interval of P' waves which are conducted to the ventricle.

Figure 1

All strips are from lead aV_p on the same date (June 8, 1962). The lower two strips are from the same electrocardiogram but are not continuous. The sinus impulse is labeled P, the parasystolic impulse P'. P_1 and P_2 refer to ectopic atrial systoles from still other sites. The first F? in the middle strip refers to a possible fusion between P' and one of the other ectopic P waves. The second F? suggests a fusion between P and P_1. Because a P' is invariably interposed within each P-P interval, the P-P interval of about 0.80 in the upper strip and about 0.88 in the lower strip may not be without influence from the parasystolic focus. The P-P interval from other parts of the upper tracings in the absence of parasystole was about 0.72 second. Coupling varies between 0.24 and 0.46 second (table 1). In the upper strip the first and last P' waves occur slightly later in the cycle than the others and are conducted to the ventricles. This is also true of the second, third, and fifth P' in the middle strip and the third, seventh, and eighth P' in the lower strip (table 3).

cultured from the urine, and except for a hematocrit level of 33 percent the other laboratory studies were noncontributory. A chest film showed the lungs to be clear and the heart was thought to be of normal size. Esophageal electrocardiography was obtained and is discussed below. The incomplete atrioventricular block remained in spite of isoproterenol (Isuprel) therapy and small amounts of atropine. The patient underwent a transurethral resection, had an uncomplicated postoperative course, was placed on maintenance antibiotics, and was discharged in good condition.

Interpretation of Electrocardiograms

Based on the criteria listed, both of these cases present examples of supraventricular parasystole (figs. 1 and 2). In table 1 the varying coupling intervals of the parasystolic P waves with the preceding P waves are recorded. In table 2 the rather constant parasystolic intervals may be seen. In case 1 these P waves occur at essentially the same interval. In case 2 the basic parasystolic interval is about 1.06 ± 0.02 second with
this first interval representing a multiple of three times the basic interval and in the case of the fourth interval the multiple is about two times the basic interval. In case 1 (fig. 1) the three strips are not continuous. The first strip was obtained the same day as the latter two but some hours earlier. The parasystolic focus is somewhat faster in the upper strip of figure 1 than in the lower two strips, but nevertheless the regularity is apparent. In case 1 the question certainly arose as to whether we were not dealing with return atrial systoles rather than with parasystole. We think that this is more likely parasystole because the ectopic P waves seem quite regularly related to each other while their relationship to either the preceding P wave or the preceding R wave is much less constant. The possibility that this inverted ectopic P wave might represent a focus in the atrioventricular node rather than in the atrium must be considered. Unfortunately, because the parasystolic focus was inconstant in some of the leads and esophageal electrocardiography, though attempted, was unsuccessful due to the irrational state of the patient, we are unable to evaluate P-wave configuration in leads other than aV2. If this evaluation were possible, we would probably be in a better position to postulate the exact site of origin.

Figure 2 represents simultaneous electrocardiographic recordings from the esophagus and standard lead I. Probably the basic rhythm here is an incomplete atrioventricular block of the Wenckebach type. A parasystolic focus is in evidence and is labeled P1. Ectopic atrial systoles from still another site are labeled P2. Occasionally bizarre ventricular complexes, which are likely ectopic ventricular systoles rather than aberrantly conducted QRS complexes from the parasystolic focus, should be noted.

![Figure 2](image-url)
Discussion

Langendorf et al.\textsuperscript{4} reported a case of atrial parasystole with interpolated atrial premature systoles. These investigators noted marked variation in the duration of the returning cycle, that is, the parasystolic to succeeding P interval. When a parasystolic P-sinus P interval longer than the basic sinus or P-P interval occurred, discharge of the sinus pacemaker by the ectopic impulse was considered the likely explanation. This was seen primarily when parasystolic P waves occurred in the middle part of the cycle. In figure 2 this possibly would be the case with the fourth and fifth parasystolic P waves (P\textsubscript{1}). It could be the case with the first P\textsubscript{1} in figure 2, but the intervening ectopic ventricular systole makes this a less analogous case. Langendorf et al.\textsuperscript{4} thought that parasystolic P-sinus P intervals about equal to the basic sinus (P-P) interval could be accounted for on the basis of interference with the next sinus impulse by the parasystolic impulse, thus demonstrating a fully compensatory pause. We do not have an example of this in either of our cases. Parasystolic P-sinus P intervals, which were found to be shorter than the P-P cycle, were considered to be due to interpolation of the parasystolic cycle. It was thought that in this case the parasystolic impulse did not reach or discharge the sinoatrial node, but did render the atrial muscle partially refractory for the sinus impulse that was to follow, thus accounting for the difference in the P-P interval and the somewhat longer P-P-P interval. This may have represented concealed atrio-sinus conduction, comparable to its counterpart, concealed ventriculo-atrial conduction with interpolated ventricular premature systoles that show postectopic prolongation of atrioventricular conduction.

We were unable definitely to place the parasystolic impulses in case 1 in any of these three categories for the following reasons. First, in every instance in which a sinus P followed a parasystolic P(P\textsuperscript{}) the P-P interval was shorter than the postulated P-P interval, thus making the first two explanations untenable, and strongly suggesting interpolation with concealed atrio-sinus conduction. This would be difficult to substantiate definitely, however, since our P-P\textsuperscript{P} and P-P intervals are so nearly the same so far as we are able to determine, and we have no instance of a P-P interval available for measurement without an interposed P\textsuperscript{P}. So in actuality in case 1 we are unable to state with assurance what the basic P-P interval is, for the P-P cycle never occurs free from the influence of the parasystolic focus.

Scherf, Yildiz, and DeArmas\textsuperscript{5} made the point that in contrast to ventricular parasystole, in atrial parasystole there is less variation in coupling, and the presence of fusion P waves is rare. Our cases would in general support this. Those authors further commented on the relationship of parasystole to healthy versus diseased hearts.\textsuperscript{5} Heart disease was present in both our cases. Digitalis was involved in only one of our cases.

Relationship of Atrial Parasystole to Atrial Dissociation

Igarashi et al.\textsuperscript{6} commented that atrial dissociation and atrial parasystole have a common feature in the sense that both have constantly active ectopic pacemakers in the atrium that can produce atrial contraction independent of the sinus rhythm. They further stated, however, that parasystole may be differentiated from atrial dissociation in that parasystolic impulses may be conducted to the ventricles and in atrial dissociation the impulse is never conducted to the ventricles. We have been unable to find substantiating experimental evidence for this and rather doubt the justification of this distinction. If true dissociation of the two atria does occur outside the experimental situation, it would seem more logical to consider the nondominant atrium as simply a large parasystolic focus. We suspect then that the determining factor of whether atrioventricular conduction occurs from such a focus is more likely the time at which the ectopic impulse occurs in relation to the cardiac cycle. If the parasystolic impulse falls at a time when the intervening atrial muscle, the atrioventricular conduction system, and the ventricle are recovered from the previous sinus-originated wave of activa-
tion, conduction to the ventricle should occur (figs. 3 and 4).

The bulk of evidence seems to be that the atria are not physiologically separate entities. It is readily admitted that much experimental evidence is yet necessary fully to elucidate the mechanism of parasystole, and that the question of whether atrial dissociation is simply a fortuitous expression of atrial parasystole or is indeed a distinct entity remains unanswered.

Summary and Conclusions

Two cases of supraventricular parasystole have been reported. The criteria of (1) variation in coupling between the ectopic P waves and the preceding P wave and (2) an interparasystolic interval which was constant or a multiple of the basic ectopic interval were met. The possibility that case 1 might well represent atrioventricular nodal parasystole was discussed.

In case 1 the inverted parasystolic P waves were considered not to be return atrial systoles because of their consistent relationship to each other and varied relationship to the preceding R wave.

The relationship of atrial parasystole to atrial dissociation was discussed, and the possibility that it represents a single mechanism was suggested.

Acknowledgment

We would like to express appreciation to Dr. Albert D. Kistin, Chief of Medicine, Beckley Memorial Hospital, Beckley, West Virginia, for his assistance in compiling the information in the first case.

References

William Harvey The Manuscript and Its Publication

Harvey wanted to publish his book on the Continent, in Frankfurt, in order to penetrate the old curricula more effectively and to be sure leading European scientists in the Italian universities, in Switzerland, and last but not least in Paris should be able to acquire it . . .

The road for the manuscript was not the smoothest possible to a place as distant as Frankfurt-am-Main. Having survived the fogs and storms of the Channel, it had to find its way by lumbering cart through the Dutch theatre of war, and the printer had to puzzle out the handwritten text, unable to discuss incomprehensible passages with the author. Reading through the book, he picked out about a page and a half of errors. Even so he was not sure of himself and felt it necessary to append at the end: "Benevolent Reader, the directors of the printer's shop ask your indulgence for the many errors in a book of such small dimensions (the whole book was only 72 folio pages) in view of the author's absence a distance of a long journey by land and water at a period so unfavorable to postal communication, and also due to the novelty of the subject to our proof readers, and the strangeness of the handwriting . . ."

The editor was very polite. Harvey's handwriting is hard to read even when we know the words in question.—Tibor Doby, M.D. Discoverers of Blood Circulation. From Aristotle to the Times of Da Vinci and Harvey. New York, Abelard-Schuman, 1963, p. 206.
Two Cases of Supraventricular Parasystole
NANCY C. FLOWERS, G. DANIEL COPELAND and DANIEL A. BRODY

Circulation. 1964;29:440-446
doi: 10.1161/01.CIR.29.3.440

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1964 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/29/3/440.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/