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. 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. 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, 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

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was hyperresonant to percussion. Fine rales were present in both lung bases. A systolic gallop rhythm was present. The liver was two fingerbreadths 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 parasystolic focus in lead aVF that was presumed to be 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 digitalis 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 99F. 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 bilaterally. At this time the electrocardiogram was interpreted as showing an incomplete atrioventricular block of the Wenckeback type with premature ventricular systoles. The patient's arrhythmia was thought possibly to be due to coronary artery disease, since he had received no digitalis 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.

| Table 2 |
| Parasystolic Intervals in Seconds |
| Case 1 (fig. 1) | Case 2 (fig. 2) |
| 0.76 ± 0.04 | 3.24 | (1.08 × 3) |
| 0.88 — 0.90 | 1.04 |
| 0.88 — 0.90 | 2.12 | (1.06 × 2) |
| 0.88 — 0.90 | 1.04 |

| Table 1 |
| Coupling Intervals in Seconds of Parasystolic P to Preceding P * |
| Case 1 (fig. 1) | Case 2 (fig. 2) |
| 0.36 | 0.36 | 0.40 |
| 0.24 | 0.24 | 0.40 |
| 0.32 | 0.26 | 0.46 |
| 0.36 | 0.36 | 0.56 |
| 0.32 | 0.40 | 0.62 |
| 0.32 | 0.28 | 0.30 |
| 0.28 | 0.30 |
| 0.32 | 0.28 |
| 0.46 | 0.32 |
| 0.30 | 0.42 |
| 0.32 |

* 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.
cultured from the urine, and except for a hematocrit level of 33 per cent 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

**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>
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<tr>
<td>0.20</td>
<td>0.28*</td>
<td>0.24*</td>
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<tr>
<td>0.20</td>
<td>0.19</td>
<td>0.16</td>
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<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>
</tbody>
</table>

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

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 aVF. 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.5

![Figure 2](image)

The three strips illustrated above were obtained simultaneously. The upper strip is from a unipolar esophageal lead with recording electrode 42 cm. from the nares. The second strip is from a bipolar esophageal lead with the midpoint between poles lying 41 cm. from the nares. The poles were 2 cm. apart. The lower strip is conventional lead I. Complexes labeled P are from the sinoatrial node. P1 represents the parasystolic focus. P2 represents a possible fusion between the parasystolic and sinus focuses. Pn is an atrial complex of uncertain origin. It is possible that it represents a return atrial systole conducted from the ventricular premature systole just preceding it; however, its positive direction rather than a negative one from a recording site at this level in the esophagus makes this interpretation subject to question. The basic rhythm of this recording is an incomplete atrioventricular block with Wenckebach phenomenon best demonstrated in the early portion of VE42 and BE41. The sinus interval is approximately 0.68 second. The basic parasystolic interval is about 1.06 with a variation of 0.02 second. The longer parasystolic intervals are approximate multiples of the basic interval (table 2). Coupling intervals of the P1 with the preceding P vary from 0.30 to 0.62 second (table 1).
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') 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' 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 interpolated 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 non-dominant 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

![Figure 3](image)

Diagram illustrating the pathway of parasystolic P waves that are conducted to the ventricles. From figure I this includes the first and last P' of the upper strip; the second, third, and fifth P' of the middle strip; and the third, seventh, and eighth P' of the lower strip. These waves appeared to occur late enough in the cycle, i.e., the R-P' is long enough (table 3) to allow the parasystolic impulse to find the atrioventricular node and the ventricle nonrefractory enough to allow conduction. In figure 2 the possibility was considered that the first and second parasystolic impulses (P₁) are conducted to the ventricle in an aberrant fashion demonstrated by the bizarre QRS that follows. In favor of these bizarre QRS complexes being conducted rather than being ventricular prematurity systoles are the facts that the R-R intervals immediately preceding them are long and their own coupling interval to the immediately preceding R is relatively short. However, the fact that the last bizarre QRS in the tracing is of similar configuration in the esophageal leads and of identical configuration in the extremity lead to those in question, and is obviously nonconducted (a definite ventricular prematurity systole) would lead one away from aberrant conduction as an explanation for the occurrence of the QRS's that follow the P₁ complexes. They are likely ventricular prematurity systoles.

![Figure 4](image)

Schematic diagram representing circumstance in which the parasystolic impulses are able to depolarize the atrium, which apparently has recovered sufficiently from its previous depolarization to respond to the ectopic impulse, but finds the atrioventricular node and ventricle refractory from the immediately preceding sinus discharge. Shaded areas represent refractoriness; light areas represent recovery. (Diagram is not necessarily meant to imply partial penetration of the atrioventricular node.)

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

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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.
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