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**Modulation of Parasystolic Activity by Nonparasystolic Beats**

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**SUMMARY** We studied 12 patients with ventricular parasystole in whom pacemaker activity could be modulated by nonparasystolic beats (NPBs). In six patients (group 1) in whom the intrinsic parasystolic cycle length (XX interval) was obtained without interposed NPBs, we found that NPBs falling during the first half of the cycle prolonged the XRX interval (containing one NPB) and that NPBs falling during the second half of the cycle abbreviated the XRX interval; both effects were maximal when NPBs fell close to the middle of the cycle and were separated by a reversal point. However, because of mutual interference between parasystolic beats and NPBs, only 13.2–43.4% of the parasystolic cycle could be effectively scanned. We also found that the XRX and RX intervals were linearly related. This relationship served to establish that in six patients in whom the XX interval was not obtained (group 2), modulation showed a similar behavior, although neither the reversal point nor the sense of the modulation could be determined. In this report, we suggest diagnostic criteria of parasystolic modulation.

IN CLASSIC parasystole, an automatic focus is assumed to be totally independent of the electric activity elsewhere in the heart. Entrance block (a form of unidirectional block) is the barrier that protects the parasystolic center from exogenous influences. However, a mechanism by which nonparasystolic beats (NPBs) can modify the activity of a protected automatic center has been conceived by Jalife and Moe. They reasoned that "if an impulse cannot invade the pacemaker, but the pacemaker impulses can escape, then clearly there must be a viable ionic pathway across the blocked region." Using an in vitro preparation — the sucrose gap model — they established the existence of an electrotonic action exerted, by evoked action potentials or current pulses, on the activity of a protected pacemaker. Electrotonic modulation of a protected automatic center was demonstrated, and it was assumed that similar events had to occur in clinical cases of ventricular parasystole (VP). Although isolated reports indicate that this may be the case, to the extent to which and the mechanism by which NPBs modulate parasystolic activity in man are still unknown.

In this report, we present 12 patients with VP in whom the activity of the parasystolic center was predictably enhanced or depressed by the effect of NPBs, depending on their timing within the ectopic cycle. These cases are a clinical counterpart of the experimental findings and serve to delineate the diagnostic criteria of parasystolic modulation, underline the differences from the experimental results, and show the limitations of extrapolating the latter to the clinical situation.

**Material and Methods**

The ECGs of 50 patients with VP were carefully examined for the presence of ectopic cycle lengths that varied because of the presence of interposed NPBs, as described by Jalife and Moe. Fifteen such patients (30%) were found, but two were excluded because the
interictopic interval lengthened substantially during carotid sinus massage and one was excluded because the analyzed strip did not contain enough beats. The remaining 12 patients form the material of the present study. Long rhythm strips were obtained in each patient, and carotid sinus massage was repeatedly performed to change the relationship between the dominant (supraventricular) rhythm and the parasystolic rhythm and to obtain, whenever possible, the intrinsic parasystolic cycle length (without interposed NPBs). In five patients, the study was repeated one or more times during a period of 4 days to 24 months. The results were similar in every repeat study.

The following intervals were measured (fig. 1): XX — duration of the intrinsic parasystolic cycle length; XR — duration of the parasystolic cycle containing a single interposed NPB; RX — coupling of the interposed NPB to the preceding parasystolic beat; XR — coupling of the parasystolic beat to the preceding NPB. Prolonged interictopic intervals (multiples of the parasystolic cycle) and XR intervals containing more than one NPB were not considered in the present study.

**TABLE 1. Twelve Cases of Modulated Ventricular Parasystole**

<table>
<thead>
<tr>
<th>Pt</th>
<th>XX interval (msec)</th>
<th>XRX range (msec)</th>
<th>XRX variation (% change of XX)</th>
<th>Reversal point (msec)</th>
<th>XRX range (msec)</th>
<th>RX range (msec)</th>
<th>RX interval (msec)</th>
<th>Alpha angle</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1400–1500 (1440)*</td>
<td>1080–2020</td>
<td>25</td>
<td>46.8</td>
<td>500–780</td>
<td>19.4</td>
<td>400–1320</td>
<td>42°</td>
</tr>
<tr>
<td>2</td>
<td>1100</td>
<td>990–1180</td>
<td>10</td>
<td>45.4</td>
<td>440–620</td>
<td>16.4</td>
<td>450–700</td>
<td>34°</td>
</tr>
<tr>
<td>3</td>
<td>1100–1200 (1180)*</td>
<td>1020–1600</td>
<td>13.5</td>
<td>53</td>
<td>560–740</td>
<td>15.3</td>
<td>320–1020</td>
<td>39°</td>
</tr>
<tr>
<td>4</td>
<td>1270</td>
<td>1120–1480</td>
<td>11.8</td>
<td>59</td>
<td>660–860</td>
<td>15.7</td>
<td>340–700</td>
<td>30°</td>
</tr>
<tr>
<td>5</td>
<td>2080–2160 (2120)*</td>
<td>1940–2330</td>
<td>8.5</td>
<td>69.3</td>
<td>940–1860</td>
<td>43.4</td>
<td>360–1200</td>
<td>27°</td>
</tr>
<tr>
<td>6</td>
<td>1520</td>
<td>1340–1551</td>
<td>11.8</td>
<td>2</td>
<td>750–950</td>
<td>13.2</td>
<td>390–760</td>
<td>28°</td>
</tr>
<tr>
<td>7</td>
<td>920–1240</td>
<td></td>
<td></td>
<td></td>
<td>560–740</td>
<td>13.2</td>
<td>300–540</td>
<td>55°</td>
</tr>
<tr>
<td>8</td>
<td>1500–2100</td>
<td></td>
<td></td>
<td></td>
<td>770–1270</td>
<td>23°</td>
<td>500–1120</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>2120–2720</td>
<td></td>
<td></td>
<td></td>
<td>1280–1460</td>
<td>37°</td>
<td>680–1440</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1530–1930</td>
<td></td>
<td></td>
<td></td>
<td>800–1210</td>
<td>30°</td>
<td>440–1040</td>
<td></td>
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<tr>
<td>11</td>
<td>1960–2360</td>
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<td></td>
<td></td>
<td>1160–1520</td>
<td>28°</td>
<td>500–1170</td>
<td></td>
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<tr>
<td>12</td>
<td>1680–2000</td>
<td></td>
<td></td>
<td></td>
<td>780–1200</td>
<td>23°</td>
<td>480–1220</td>
<td></td>
</tr>
</tbody>
</table>

See text for definition of XX, XRX, XR and RX intervals, reversal point and alpha angle.

*The average of 30, seven and 14 XX intervals from patients 1, 3 and 5 respectively. This average was used when the data were expressed as a percentage of the XX interval.

**Results**

Since the parasystolic cycle length varies because of the modulating action of NPBs, the need to know the intrinsic or unmodified ectopic cycle length becomes of paramount importance. Therefore, a special effort was made to secure recordings in which this information was spontaneously available or was obtained through vagal stimulation. The intrinsic parasystolic cycle length could be obtained in six patients (nos. 1–6, group 1). In the other six patients (nos. 7–12, group 2) the intrinsic ectopic cycle length could not be determined (table 1). The two groups are analyzed separately.

**Group 1**

*The Intrinsic Parasystolic Cycle*

The XX intervals were obtained during vagal stimulation in all six patients in group 1 and occurred spontaneously in three. The XX intervals (table 1) were fixed in three patients and varied by 80–100 msec in the other three. This variation was erratic and apparently independent of vagal stimulation, because both slight acceleration and slight slowing occurred in each patient during the same maneuvers. In these six patients, the intrinsic rate of the parasystolic focus was 27–54 impulses/min.

*The Modulated Parasystolic Cycle*

The XX intervals were either constant or slightly variable, but the XRX intervals varied widely (table 1). NPBs falling during the first half of the parasystolic cycle prolonged the XRX interval by postponing the next automatic discharge, whereas NPBs falling dur-
ing the second half of the cycle abbreviated the XRX interval by accelerating the next discharge (figs. 2–5). The XRX interval at the beginning of the upper strip in figure 2 measures 1700 msec and contains an NPB with a coupling interval of 600 msec. The XRX interval at the end of the strip measures 1800 msec and contains an NPB with a coupling of 640 msec. The 100-msec difference may not appear to be significant. Moreover, because the interval between the second and third automatic beats is a perfect multiple of the first interectopic interval (6800 = 1700 × 4), one might believe that the closest approximation to the intrinsic interectopic interval is 1700 msec. However, the bottom strip, recorded during carotid sinus massage, reveals that the nonmodulated parasystolic cycle measures 1440 msec and is practically constant when "undisturbed" by NPBs. Moreover, NPBs with a coupling interval of 640 msec or shorter prolong the corresponding XRX interval, whereas the single NPB with a coupling interval of 720 msec caused instead a great shortening (to 1160 msec).

The six strips in figure 3 were aligned to show in greater detail the relation between the XR and XRX intervals. Strips A–C show that as the XR interval increases progressively from 580 to 680 msec, prolongation of the XRX cycle also increases. In strip D, a slight increment of the XR interval (from 680 to 720 msec) causes a sudden inversion of the effect, giving rise to a very short XRX cycle. Further increases of the XR interval to 820 and 1020 msec resulted in a maximal shortening of the XRX cycle (strip E), followed by a lesser degree of shortening (strip F), respectively. In strips E and F, the accelerated automatic impulse fell during the refractory period of the modulating NPB, giving rise to a concealed discharge (arrow), whose timing could only be estimated from the succeeding unmodulated parasystolic cycle. Thus, by attracting and "swallowing" the next automatic discharge, a late NPB can greatly change the sequence of events of VP.

The magnitude of the modulating effect varied from patient to patient (table 1, columns 4 and 5). Although in some cases the prolongation and shortening were of similar degree, substantial differences were also observed. In patient 2 (fig 4), for example, the magnitude of the modulation was much less than that in patient 1. NPBs provoked variations of the parasystolic cycle no greater than 10%. Nevertheless, because the XX interval was constant, such variations could be accurately determined and followed essentially the same pattern as in case 1. In patient 3, modulation apparently caused much more prolongation than shortening of the parasystolic cycle (fig. 5).

**Reversal Point of the Modulating Effect**

Progressive increments in the coupling interval of the NPB result in increasingly greater prolongations of the parasystolic cycle, until the effect changes direction rather abruptly from maximal prolongation to maximal shortening. The critical XR interval at which
the reversal occurs was expressed as a percentage of the intrinsic parasystolic cycle length (table 1, column 6). Although the reversal point was generally close to the middle of the XX cycle, in patients 4 and 5 it was moderately shifted to the right.

**Mutual Interference of Parasystolic and Nonparasystolic Beats**

In clinical parasystole, there are obvious reasons why parts of the parasystolic (or sinus) cycle cannot be occupied by other beats. Therefore, we tried to estimate how much of the ectopic cycle was effectively scanned by NPB (giving rise to a modulated manifest response). For XR intervals, the shortest coupling was 440–940 msec, while the longest was 620–1860 msec (table 1, column 7). When each range was expressed as a percentage of the corresponding automatic cycle length (column 8), it became apparent that the part of the cycle that was effectively scanned was surprisingly small, 13.2–43.4%, and usually less than 20%. However, in every patient, the scanned section belonged to the central part of the cycle and contained the reversal point. This limitation resulted from several factors. Refractoriness induced by each manifest parasystolic beat precluded conduction to or impulse formation in the ventricles of NPBs with XR intervals shorter than 440 msec. Moreover, the common occurrence of post-extrasystolic pauses (particularly if fully compensatory) prevented scanning of nearly the first half of the parasystolic cycle in several cases (compare XX intervals in column 2 with shortest XR intervals in column 7). Refractoriness after each NPB precluded the occurrence of manifest parasystolic beats with RX intervals shorter than 320–450 msec (column 9, table 1). Furthermore, by accelerating and concealing the succeeding parasystolic discharge (fig. 3), relatively late NPBs thwarted effective scanning of the second half of the parasystolic cycle (compare XX intervals in column 2 with longest XR intervals in column 7) in most of the cases. If the timing of the accelerated concealed discharges cannot be precisely established (as happened only in patient 1), this part of the accelerating effect cannot be quantitated. The fact that parasystolic cycles containing more than one NPB were excluded was a further limitation related to the methods used in the present study.

**The Biphasic Phase-Response Curve and the Alpha Angle**

Two types of curves were constructed for each of the 6 patients (fig. 6). The curves in figure 6A relate the XRX intervals to the XR intervals, both normalized as percentage of the corresponding XX cycle, an approach similar to that one used by Jalife and Moe in their experimental model. In figure 6B, the data are plotted using an alternate format in which absolute values of the XRX interval are related to the RX interval. In every case, the curves in figure 6A were biphasic and show the percent prolongation of the XRX cy-

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**Figure 4.** Case 2. Small modulating effect caused by supraventricular beats and ventricular extrasystoles in the presence of atrial fibrillation. The intrinsic parasystolic cycle is fixed and measures 1100 msec. XR intervals are modestly prolonged by the ventricular extrasystoles falling during the first half of the cycle (XR intervals of 440–460 msec) and moderately shortened by supraventricular beats falling during the second half of the cycle (XR intervals of 600–590 msec). E = ventricular extrasystole. Format as in figure 2.

**Figure 5.** Case 3. Parasystolic modulation during spontaneous rhythm (A) and during carotid sinus massage (CSM) (B and C). (A) Each parasystolic beat is followed by a compensatory pause that prevented the occurrence of nonparasystolic beats (NPBs) within the first half of the parasystolic cycle. Under this condition, the parasystolic cycles measure 1020 and 1160 msec when the couplings of NPBs are 680 and 660 msec, respectively. However, the nonmodulated parasystolic cycle is actually longer and measures constantly 1200 msec in B and C. An early NPB in C (XR = 560 msec) produces a substantial prolongation of the parasystolic cycle (XRX = 1560 msec). This prolonging effect of modulation prevails over the accelerating effect. See also figure 6.
Graphic representation of the modulating effect for the six patients in group 1. (A) Curves relating the XRX intervals (ordinate) to the XR intervals (abcissa), both normalized as a percentage of the intrinsic parasystolic cycle length. (B) Curves relating the XRX interval (ordinate) to the RX interval (abcissa), both expressed in msec. Open circles in both curves from case 1 indicate concealed discharges of the parasystolic center. Broken lines indicate the range of variation of the pure parasystolic cycle length in cases 1, 3 and 5, whereas solid lines indicate their average value or the fixed value in the other three cases.

Circles modulated by NPBs falling during the first half of the cycle and the percent shortening caused by NPBs falling during the second half of the cycle. Both changes were statistically significant \( p < 0.01 \), except the prolongation in patient 6. The phases were separated by the horizontal line representing the duration of the unmodulated XX cycle. The intercept of the biphasic curve with the horizontal line indicates the reversal point (not always well defined). However, when NPBs fell close to the reversal point, identical XR intervals correlated with very different XRX cycles and had even an opposite modulating effect. This overlap, the fact that a complete curve could never be obtained, and, above all, the need to find a common graphic representation that would also describe the cases of group 2, in whom the intrinsic interectopic interval is unknown, indicated the need to construct the curves in figure 6B, which contain similar information but are displayed in a way that better suits the clinical observations. These curves show a linear relationship between the XRX and RX intervals \( r = 0.75-0.99, p < 0.005 \); the slope of this relation becomes steeper when the modulating effect is greater. As in the curves in figure 6A, the XX line separates the lengthening from the shortening effect, and the intercept defines the reversal point. The magnitude of the modulating effect is indicated by the length of the straight line obtained from the display of the data points, and probably by the value of the alpha angle that such a line forms with any horizontal line.

Patient 1, who had the widest alpha angle \( (42°) \), exhibited additional interesting features. The abrupt transition from the lengthening to the shortening effect was a consequence of the high degree of modulation. In other words, all interectopic intervals were substantially longer or shorter than the intrinsic parasystolic cycle length, as shown by the absence of points around the XX line. Were it not for the vagal maneuvers (see figs. 2 and 3), a nonmodulated parasystolic cycle could never have been recorded, and the timing of the nonmanifest discharges (included in the curves as open circles) would not have been possible. Patient 3, with
an alpha angle of 39°, showed similar behavior, except that the shortening effect was apparently much smaller than the lengthening effect. In the four other patients, who had narrower alpha angles, the transition from the lengthening to the shortening phase was more gradual, and the reversal point became a band of overlapping points. In patient 5, a small modulating effect (alpha angle of 27°) coexisted with a long intrinsic parasystolic cycle, bringing out the fact that the earliest NPB (shortest XR in figure 6A, longest RX in figure 6B) had little or no effect.

**Group 2**

When the duration of the intrinsic parasystolic cycle is not known, as in group 2, the modulating effect cannot be directly quantitated and the point of reversal cannot be determined. However, the overall magnitude of the modulation was no less in group 2 than in group 1, as shown by the wide range of variation of the XRX intervals (third column, table 1), which was similarly determined by the variable timing of NPB within the encompassing parasystolic cycle (columns 7 and 9, table 1). Also, small changes of the XR interval provoked great variations of the XRX cycle (fig. 7).

As in group 1, there was a linear relationship between the RX and XRX intervals in group 2 patients (fig. 8). According to the alpha angle (table 1), these six patients can be analyzed as follows. Patients 8, 10, 11 and 12 showed a moderate effect (alpha angle of 23–30°) and a large variation of the XRX intervals, probably related to a long intrinsic cycle length. Patient 9 showed an important effect (alpha angle of 37°) that caused the greatest variation of the XRX cycles in this group, favored by an extremely long parasystolic cycle. Patient 7 was the only one who had an alpha angle larger than 45°, which suggests a large modulating effect. However, variations of the XRX interval were moderate, which we attributed to a relatively shorter intrinsic cycle length, and most of the NPBs fell close to the reversal point. This case stresses the problems of interpretation that can arise when the intrinsic cycle length of the parasystolic pacemaker is unknown.

**Discussion**

The six patients in group 1 represent the clinical counterpart of the experimental results reported by Ja-
life and Moe. We thus confirmed that (1) NPBs falling during the first half of the parasystolic cycle prolong the XRX interval; (2) NPBs falling during the second half of the cycle abbreviate the XRX interval; (3) both effects are maximal when NPBs fall close to the middle of the cycle and are separated by a critical or reversal point, under the form of a biphasic phase-response curve; (4) the modulating effect of NPBs varies from case to case, and when it is only slight or moderate, the reversal point becomes a band of overlapping points. Similar differences occurred when current pulses of different intensity were used to provoke the electrotonic modulation.

A major problem in extrapolating from the sucrose-gap preparation to man is that in the intact heart, various hemodynamic, endocrine and neurogenic factors come into play. For example, it is conceivable that an earlier NPB with a small end-diastolic volume, combined with carotid massage, would affect the parasystolic impulse differently than a late NPB with a larger ventricular volume. Although this or other related mechanisms cannot be totally excluded, the clear tendency for a diphasic response to occur suggests that electrotonic modulation played a significant role in our patients. The separation of patients into groups 1 and 2 was not the result of a different behavior, but was because in the latter patients, no intrinsic parasystolic cycle against which to measure the modulating effect of NPBs was available. However, group 2 better represents the clinical situation in that a nonmodulated parasystolic cycle can be obtained rarely or only if vagal maneuvers are effective. Accordingly, we developed diagnostic criteria that might help to recognize the modulating effect even under the latter conditions. Thus, parasystolic modulation can be suspected when there is a wide range of variations in the XRX intervals (column 3, table 1), confirmed by demonstration of a practically linear relationship between the RX and XRX intervals (the straight line in the curves of figure 8), and even quantitated by the length of such straight line and possibly the amplitude of the alpha angle. When all these features were considered, it was apparent that the six patients in group 2 were essentially similar to the six patients in group 1, although in none of the former could the reversal point or the sign of the modulation be determined.

The present study lends strong support to the contention that the protection block in cases of VP need not provide complete insulation of the automatic center, and that NPBs commonly exert an electrotonic influence across the region of unidirectional block. But does this indicate that modulation must be present in every case of VP? Although a definitive answer to this question cannot be provided, such modulation was demonstrated in only 15 of 50 cases (30%). If modulation is no longer than the spontaneous variability of the intrinsic parasystolic cycle length occurring in some cases (column 2, table 1), it may certainly escape notice. If the automatic focus is not very close to the region of block, the electrotonic influence may be extremely small or even absent.

**Differences Between the Clinical and Experimental Models: Limitations**

Although it was remarkable to find so many similarities between the experimental and clinical findings, some important differences need further comment. In the experimental model of VP, in addition to having complete control of both the automatic center and the modulating impulses, it was possible to scan the entire automatic cycle because such study was done when block across the sucrose gap was bidirectional, obviating the physiologic interference provoked by refractoriness at both sides of the gap. Although that was indeed convenient for constructing the entire biphasic phase-response curve, the part of the parasystolic cycle that could be effectively scanned by NPBs in our patients was substantially curtailed (column eight, table 1) and was the main reason why the curves in figure 6A differed from their experimental counterparts. In fact, the curves of the clinical cases (figs. 6 and 8) corresponded only to the central part of the experimental curves.

This portion of the curve included the maximal modulating effect, as well as the reversal point. The linear relation of our curves in figure 6B was partially a consequence of the above limitation. Earlier and later NPBs should have yielded XRX intervals closer to the control XX cycle, as in the experimental studies. This tendency was noted in patient 1, the only one in whom nonmanifest parasystolic discharges could be used to construct the curves, and in patient 5, in whom NPB with the longest XR intervals yielded XRX cycles similar to the XX interval. Such a tendency was favored by the long intrinsic parasystolic cycle length. Although the alpha angle was apparently useful in analyzing our patients, its precise significance is still unclear.

**Differential Diagnosis of Parasystolic Modulation**

Electrotonic modulation by NPBs is not the only mechanism that can change the manifest rate of discharge of a parasystolic focus. Vagal stimulation substantially slowed the rate of discharge in seven of our 50 cases of VP. This was not the case in the six patients in group 1, in whom the intrinsic parasystolic cycle length was fixed or scarcely variable. Even in the six patients in group 2, in whom the intrinsic interectopic interval was not available, the electrotonic modulation could be readily recognized in the presence or absence of vagal stimulation A first-degree exit block may change the duration of a parasystolic cycle. It should be suspected when the XRX cycle lengthens in response to a short RX interval; but this response is totally opposite to that occurring in electrotonic modulation. Kinoshita described a shortening of the XRX interval when NPBs occurred late during a parasystolic cycle, and attributed it to reentry within or close to the

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parasystolic focus. This could simply reflect the accelerating phase of parasystolic modulation.

**Intermittent Parasytology: Active vs Electrotonic Capture of the Pacemaker**

In one of the best-known varieties of intermittent VP, long interectopic intervals are not multiples of the shortest one, due to the discharge and resetting of the pacemaker by relatively late NPBs. According to Moe et al., this ‘‘is not fundamentally different in concept from the acceleration phase of the electrotonic influence.’’ Furthermore, they suggest that the sharp temporal boundary between protection and capture of the pacemaker described by Cohen et al. represents the reversal point in the curve of electrotonic influence. However, our studies do not support such contentions. We believe that relatively late NPBs can capture the pacemaker either by electrotonic facilitation or by full active propagation, and that both can be distinguished electrocardiographically. Thus, in 12 of our cases of intermittent VP (unpublished data), as well as in nine similar cases reported by Castellanos et al., the active capture of the pacemaker (and its resetting) always occurred when the QRS of the capturing NPB was being inscribed.

This finding is consistent with the idea that in intermittent VP the protection block consists simply of a prolonged refractory period, such that every NPB falling after refractoriness is over (after a critical XR interval) will rapidly discharge the pacemaker because the barrier determining the entrance block has been temporarily lifted. However, in the present study, when NPBs ‘‘captured’’ the pacemaker, the discharge and resetting occurred not during the QRS, but much later, and with a highly variable latency (strips E and F, fig. 3). This ‘‘deferred’’ capture, due to the passive electrotonic action of the NPB, appears to differ from the active and rapid capture occurring in intermittent VP, in which no variable latency is observed. In fact, in the experimental studies, a considerable delay was a constant feature, except when the electrotonic influence was very large. However, in our 12 patients with intermittent VP, as well as in others reported in the literature, the XRX cycles did not differ significantly from the XX cycles, which indicates that parasystolic modulation was small or absent. Why electrotonic modulation is paradoxically less marked or absent in cases in which entrance block is only partial is still unclear. Jalife and Moe stated that the region of impaired conductivity, like the sucrose gap, does not need to conduct an impulse; but it may, as seems to be the case in intermittent parasytology.

**References**

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