Effects of Intervening Sinus Beats on Ectopic Cycle Length in a Patient with Ventricular Parasystole

Akira Furuse, M.D., Hiroshi Matsuo, M.D.,* and Masahiro Saigusa, M.D.

Summary

The intrinsic cycle length of the ventricular parasystolic pacemaker may be modulated by electrotonic influence of intervening ventricular depolarizations. In order to quantify the magnitude of modulation in relation to the timing of the intervening depolarization, a curve of influence was constructed in a patient with ventricular parasystole. The current communication deals with the detailed description of a step-by-step method for the construction of the curve of influence.

Additional Indexing Words:
Electrotonic modulation Curve of influence

In a previous communication, we documented the apparent suppression of parasystolic activities by cardiac pacing when the pacing rate was near multiples of the parasystolic rate. In order to explain the apparent suppression, we assumed that the ectopic cycle length should shorten or lengthen so that the sinus beat could entrain every ectopic activity.

Recent experimental studies of Jalife and Moe have shown that the ectopic cycle length may be prolonged or abbreviated depending on the timing of the electrotonic influence imposed upon the parasystolic focus. Early electrical discharges around the focus delayed, and late discharges accelerated the ectopic pacemaker. A biphasic curve of electrotonic influence was presented relating the timing of electrical discharges during the intrinsic ectopic interval to the magnitude of changes in the ectopic interval. A rather critically timed 'break' in the curve relating the 2 events was present at about the midpoint of the ectopic cycle.

Clinical application to patients with ventricular or junctional parasystole was tried by Moe and his associates. They were able to construct the 'curve of influence' by trial-and-error guesses.

In this communication we will present a clinical case with ventricular parasystole...
parasystole in which the curve of influence was constructed by several steps of mathematical calculation with minimal presumption.

CASE PRESENTATION

The patient was a 39-year-old male with aortic stenosis and regurgitation who underwent aortic valve replacement on February 28, 1979. Postoperatively no arrhythmia was detected until September 29, when the electrocardiogram revealed frequent ventricular ectopic contractions. They had typical features of ventricular parasystole characterized by varying coupling intervals, ventricular fusion beats, and approximate mathematical relationships of interectopic intervals (Fig. 1).

Construction of the curve of influence was done using a continuous electrocardiographic strip of lead III for about 3 min which was taken on November 24, 1979.

Definitions
Interectopic interval: the interval between two consecutive manifest ectopic beats separated by one or more intervening sinus beats.
Intrinsic ectopic cycle length (ICL): the unmodulated, free-running ectopic cycle length.
Actual ectopic cycle length: the modulated, intervened ectopic cycle length.
Percentage modulation of ectopic cycle length (PME): the actual ectopic cycle length normalized as percentage of the intrinsic cycle length.
Percentage postectopic interval (PPI): the interval between the ectopic beat and the first postectopic sinus beat, normalized as percentage of the intrinsic ectopic cycle length.

Construction of curve of influence
It required 5 steps for us to construct the curve of influence as described below.

Step 1
The initial step was to establish the intrinsic ectopic cycle length. This was
Fig. 2. Interectopic intervals containing no or one intervening beat.
A. Intrinsic ectopic cycle length = 1,220 msec.
B. Delay of ectopic beat by early intervening sinus beat.
Postectopic interval = 528 msec, actual ectopic cycle length = 1,340 msec.
C. Acceleration of ectopic beat by late intervening sinus beat.
Postectopic interval = 768 msec, actual ectopic cycle length = 1,180 msec.

directly measured, in this strip, from three intervals between two ectopic beats in succession (Fig. 2A). Thus the intrinsic ectopic cycle length in this patient was 1,220 msec.

**Step 2**
The second step was to look at the interectopic intervals separated by only one intervening sinus beat. There were four such interectopic intervals in the strip (Fig. 2B, 2C). When the sinus beat fell at 43.2, 43.6, 60.7, or 63.0% of the intrinsic ectopic cycle length, the actual ectopic cycle length became 109.8, 110.8, 103.3, or 96.7% of the intrinsic ectopic cycle length respectively, as plotted in Fig. 3. Then we knew that the break point was located at 61.9% of the intrinsic ectopic cycle length, namely 754 msec following the initial ectopic beat.

**Step 3**
In Steps 3 to 5, we dealt with the interectopic intervals separated by two intervening sinus beats. These intervals ranged 2,280 to 2,500 msec, indicating that they were approximately twice the intrinsic ectopic cycle length. Most probably they contained two ectopic cycles, the one ectopic discharge firing during the re-
Then we could depict a schema of the time sequence of the interectopic interval as illustrated in Fig. 4. $S_1$ or $S_2$ is the first or second intervening sinus beat respectively. $P_0$ or $P_2'$ is the initial or terminal ectopic beat respectively, while $P_1'$ is the intermediate occult ectopic beat. $P_3$ is the expected timing for $P_1'$ when it is not influenced by $S_1$ ($P_0P_1'=ICL$). $P_4$ is the expected timing for $P_2'$ when $P_1$ is modulated by $S_1$ but $P_2$ is not influenced by $S_2$ ($P_1'P_2'=ICL$). The percentage modulation of ectopic cycle length was calculated by $PME(1)=P_0P_1'/ICL\times100$ for the first ectopic cycle and by $PME(2)=P_1'P_2'/ICL\times100$ for the second ectopic cycle. The percentage postectopic interval was obtained by $PPI(1)=P_0S_1/ICL\times100$ for the first sinus beat and by $PPI(2)=P_1'S_2/ICL\times100$ for the second sinus beat.

We had 39 such interectopic intervals in the strip, the percentage postectopic interval for the first ectopic cycle ranging 66.6 to 99.0%. Since the critical percentage postectopic interval at the break point was 61.9%, every $S_1$ abbreviated the first ectopic cycle length. Therefore $P_1'$ occurred prior to $P_1$. On the other hand, $P_2'$ might come either before or after $P_2$ depending on $P_1'S_2$ interval.

When $P_1'S_2$ interval is shorter than 754 msec, $P_1'P_2'$ will be longer than the refractory period of the first sinus beat.

Fig. 3. Construction of the curve of influence (Steps 2 to 4). See text for explanation.

Fig. 4. Time sequence of the interectopic interval separated by two intervening sinus beats. Shaded portions represent refractory period.

Abbreviations: $V=ventricle; E=ectopic focus; S=sinus beat; P=para-systolic beat.$

Full description in text.
intrinsic ectopic cycle length. Since $P_1'S_2 = P_0S_2 - P_0P_1'$ and $P_1'P_2' = P_0P_2' - P_0P_1'$, these 2 conditions can be written as:

$$P_0S_2 - P_0P_1' < 754,$$

and

$$P_0P_2' - P_0P_1' > 1220$$

Hence:

$$P_0S_2 - 754 < P_0P_1' < P_0P_2' - 1220$$

When $P_1'S_2$ interval is longer than 754 msec, $P_1'P_2'$ will be shorter than the intrinsic ectopic cycle length. In this situation the following can be derived in a similar fashion:

$$P_0S_2 - 754 > P_0P_1' > P_0P_2' - 1220$$

Suppose $(P_0S_2 - 754)$ and $(P_0P_2' - 1220)$ take an identical value, $P_2'$ has fixed coupling to $S_2$, because:

$$S_2P_2' = P_0S_2 - P_0P_2' = 1220 - 754 = 466$$

$(P_0P_2' - 1220)$ was plotted against $(P_0S_2 - 754)$ in Fig. 5. It was noted that in a number of interectopic intervals both took values very close to each other. In nine interectopic intervals the differences between $(P_0S_2 - 754)$ and $(P_0P_2' - 1220)$ were less than 12 msec, within 1% error in the scale of the intrinsic ectopic cycle length. In these nine interectopic intervals, $P_0P_1'$ was estimated as an average between $(P_0S_2 - 754)$ and $(P_0P_2' - 1220)$. It was then possible to calculate the magnitude of influence of the first sinus beat upon the first ectopic cycle length in these nine interectopic intervals, as plotted in Fig. 3.

Example 1 (Fig. 6A): From the direct measurements we obtained $P_0P_2' = 2380$ msec, $P_0S_1 = 1152$ msec, and $P_0S_2 = 1916$ msec. Therefore, $P_0S_2 - 754 = 1162$, and $P_0P_2' - 1220 = 1160$. Hence the estimated value for $P_0P_1'$ was 1161 msec. Then final solutions were:

$$PPI(1) = 94.4\% \text{ and } PME(1) = 95.2\%$$

Step 4

The fourth step was to broaden the curve of influence by applying the obtained data to the interectopic intervals separated by two intervening sinus beats, which
had not been utilized in Step 3. For this particular step we selected 14 interectopic intervals in which the percentage postectopic interval of the first sinus beat ranged between 83.6 to 93.0%, since the magnitude of modulation in this range had been known by Step 3. All data obtained from Step 4 analysis were also plotted in Fig. 3.

Example 2 (Fig. 6B): From the direct measurements we obtained $P_0P_2' = 2320$ msec, $P_0S_1 = 1100$ msec, and $P_0S_2 = 1880$. Since $PPI(1) = 90.2\%$, $PMI(1) = 91.6\%$ could be read from Fig. 3. Then $P_0P_1'$ was calculated as 1117 msec. The solutions were:

$PPI(2) = 62.5\%$ and $PME(2) = 98.6\%$

*Step 5*

The final step was to fill the gap in the curve of influence by trial-and-error guesses, dealing with the interectopic intervals separated by two intervening sinus beats, which had not been used in Step 3 or 4. In the following example, the
second intervening event, $S_2$, is an ectopic response from another ventricular focus instead of a second beat of sinus origin, but the effect on the parasystolic pacemaker should be the same.

Example 3 (Fig. 6C, 7): From the direct measurements we obtained $P_0P_2' = 2480$ msec, $P_0S_1 = 920$ msec, and $P_0S_2 = 1656$ msec. Since $PPI(1) = 66.6\%$, we estimated that $PME(1)$ lay between 90 to 95\%. When a value of $PME(1)$ was selected in this range, $PPI(2)$ and $PME(2)$ were determined, as shown in Table I. Possible combinations of $PME(1)/PPI(1)$ and $PME(2)/PPI(2)$ were plotted in Fig. 7. It was noted that one of the points D lay on the curve of influence so far obtained. Therefore, in this example, the combination of the points D—D offered the most probable fit to the curve. Thus our estimations were:

$PPI(1) = 66.6\%$, $PME(1) = 93.0\%$ and $PPI(2) = 42.7\%$, $PME(2) = 110.3\%$

Example 4 (Fig. 6D, 7): From the direct measurements we obtained $P_0P_2' = 2460$ msec, $P_0S_1 = 940$ msec, and $P_0S_2 = 1704$ msec. Since $PPI(1) = 77.0\%$, we estimated that $PME(1)$ lay between 85 to 90\%. A similar guessing table was

Table I. Estimation of Percentage Modulation of the First and Second Ectopic Cycle Lengths

<table>
<thead>
<tr>
<th>Estimation</th>
<th>$PME(1)$</th>
<th>$PPI(2)$</th>
<th>$PME(2)$</th>
<th>Estimation</th>
<th>$PME(1)$</th>
<th>$PPI(2)$</th>
<th>$PME(2)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>90.0</td>
<td>45.7</td>
<td>113.3</td>
<td>G</td>
<td>85.0</td>
<td>54.7</td>
<td>116.6</td>
</tr>
<tr>
<td>B</td>
<td>91.0</td>
<td>44.7</td>
<td>112.3</td>
<td>H</td>
<td>86.0</td>
<td>53.7</td>
<td>115.6</td>
</tr>
<tr>
<td>C</td>
<td>92.0</td>
<td>43.7</td>
<td>111.3</td>
<td>I</td>
<td>87.0</td>
<td>52.7</td>
<td>114.6</td>
</tr>
<tr>
<td>D</td>
<td>93.0</td>
<td>42.7</td>
<td>110.3</td>
<td>J</td>
<td>88.0</td>
<td>51.7</td>
<td>113.6</td>
</tr>
<tr>
<td>E</td>
<td>94.0</td>
<td>41.7</td>
<td>109.3</td>
<td>K</td>
<td>89.0</td>
<td>50.7</td>
<td>112.6</td>
</tr>
<tr>
<td>F</td>
<td>95.0</td>
<td>40.7</td>
<td>108.3</td>
<td>L</td>
<td>90.0</td>
<td>49.7</td>
<td>111.6</td>
</tr>
</tbody>
</table>
Verification of the curve of influence.

Abbreviations: S = sinus beat; P = parasystolic beat; V = ventricle; E = ectopic focus; PPI = percentage postectopic interval; PME = percentage modulation of ectopic cycle length. Actual ectopic cycle lengths are shown in msec. S₁ is a fusion beat. P₀P₁' equals the intrinsic ectopic cycle length. S₂, S₅, and S₈ with longer PPI than the break point accelerate, but S₃ and S₆ with shorter PPI delay the ectopic pacemaker. Following P₄', S₆ delays the expected timing of ectopic discharge to P₅'. Since S₇ occurs earlier than P₅', S₇ brings the timing back to P₅''.

made (Table I). In this example no combinations of PME(1)/PPI(1) and PME(2)/PPI(2) were on the curve of influence so far obtained. Then we had to guess the best fit to the curve. In this particular example the combination of I—I was chosen as the best approximation. Therefore our estimations were:

PPI(1) = 77.0%, PME(1) = 87.0% and PPI(2) = 52.7%, PME(2) = 114.7%

Verification of curve of influence

The completed curve of influence was applied to the record in which eight sinus beats were interposed between manifest ectopic beats, as reproduced in Fig. 8. The intervals between the ectopic discharge and the following sinus beats were sequentially measured. Then the degree of modulation of the next ectopic discharge was obtained from Fig. 7. Thus actual timings of all occult ectopic discharges were determined.

It was found by this analysis that the predicted interectopic interval (P₀P₆') was 7289 msec while the measured interectopic interval was 7272 msec. Therefore the error in prediction was only 18 msec for this interval.

COMMENTS

In this communication we presented a clinical electrocardiographic record of ventricular parasystole which showed significant variations of the apparent ectopic cycle length. By means of the step-by-step analysis, we could
construct a curve of influence on this ectopic pacemaker. The curve thus obtained had a biphasic pattern quite similar to that constructed in the experimental study of Jalife and Moe.\textsuperscript{2)} This indicates that there exists electrotonic modulation in the ectopic pacemaker of patients with ventricular parasystole.

As far as the construction of the curve of influence is concerned, the current case is a lucky one. Firstly, in this case, the intrinsic ectopic cycle length was directly measurable as the interval of two ectopic beats in succession. When such uninterrupted ectopic cycles are not available, one has to take another laborious step to estimate the intrinsic ectopic cycle length as has been done by Moe and his associates.\textsuperscript{4)} Secondly, the approximate timing of the break point was apparent from the interectopic intervals separated by one sinus beat. This facilitated the analysis of the interectopic intervals separated by two sinus beats. Thirdly, we had a series of trigeminy with fixed coupling. These trigeminal sequences were used to establish the accelerating slope of the curve of influence.

Generally speaking, however, the analysis of electrotonic modulation in the parasystolic pacemaker would be more complex. Nevertheless, the curve of influence may be constructed in many cases with ventricular parasystole, if one follows the steps described here with relatively long rhythm strips. The major difficulty one may encounter in the course of the analysis is related to the fact that the postectopic intervening sinus beats are confined to a limited range in a certain rhythm strip. This can be overcome to some extent by procedures that alter the sinus rate. If one proceeds with atrial pacing, one may certainly solve this problem.

At any rate, once the curve of influence is established in patients with ventricular parasystole, apparently complicated arrhythmias may be analyzed with ease.

**Acknowledgment**

The authors wish to acknowledge the valuable review of this manuscript by Dr. Gordon K. Moe, Director of Research, Masonic Medical Research Laboratory.

**References**