LETTERS TO THE EDITOR

Letters to the Editor will be published when suitable and as space permits. They should not exceed 1,000 words (typed double spaced) in length, and may be subject to editing or abridgment.

Mechanisms of Parasystole

To the Editor:

Kinoshita et al.1 reported two cases of supraventricular ectopic activity in which the classic criteria for the diagnosis of parasystole were not applicable. To explain the mechanism of the arrhythmic patterns in these and other cases,2,3 the authors suggested the presence of Mobitz type I second-degree block in the entrance pathway, and contended that most cases of parasystole are explained by intermittent or continuous parasystolic focus discharge coexisting (or alternating) with reentrant activity.

In the discussion of their results, these investigators compared their hypothesis with what they interpreted to be the "modulated parasystole" hypothesis4-6 and concluded that biphasic modulation of an ectopic pacemaker could not explain the arrhythmic patterns in their cases of ventricular2,3 or supraventricular4 parasystole.

The explanation of Kinoshita and co-workers2 involving a relatively long "absolute refractory period" and a later period in which the sinus impulse "reaches and discharges the focus after marked delay" is, of course, valid, and cannot be refuted at this time. However, their interpretation of the "modulated parasystole" hypothesis is in error, at least as it applies to the cases presented in their recent articles.1,6 The arrhythmic patterns in most of those cases can be explained by the electrotonic interactions between an ectopic pacemaker "protected" by entrance block and the activity of the surrounding tissue.

Figure 1 shows the results of two types of analysis in the interpretation of case 1 of Kinoshita et al.1 In figure 1A, the data are presented in terms of Kinoshita's interpretation. In figure 1B, the phase-response curve (PRC) represents the "inverse solution" of the same trace when the analysis was performed in accordance to the rules dictated by the biologic and mathematical models of parasystole.5,6 Even though both theories are based on completely different assumptions, either hypothesis can explain the mechanism of the arrhythmic pattern in this case.

Kinoshita and co-workers1 illustrate their hypothesis (fig. 1A) by plotting the interectopic intervals (E1,E2) containing one sinus beat as a function of the ectopic-to-sinus (E,S) interval. The intrinsic ectopic pacemaker period ("pure parasystolic cycle") was not re-

![Figure 1](http://circ.ahajournals.org/attachment/34226/1036-figure1.jpg)

**Figure 1.** (A) Relationship between interectopic (E1,E2) and ectopic-to-sinus intervals (E,S) as interpreted by Kinoshita et al.1 from the record in their figure 1. The dotted parallel lines indicate the "ranges" of long (1820-1840 msec) and short (1600-1630 msec) E1,E2 intervals that should have occurred to fit their theory. Open circles indicate manifest E1,E2 intervals; closed circles indicate concealed E1,E2 intervals. (B) PRC derived from the same record as predicted by the "modulated parasystole" theory.5,6 The intrinsic ectopic pacemaker cycle (1725 msec) was estimated by comparing the ectopic patterns in the clinical trace with those obtained by the mathematical model at an intrinsic ectopic-to-sinus-cycle-length ratio of 1.8 (mean sinus cycle length ~ 960 msec).
corded in this case. Kinoshita et al. assumed that this intrinsic period was "always" equal to the long interectopic intervals (open circles, fig. 1A) containing one sinus beat and that the concealed (closed circles) long or short interectopic intervals followed the same rules as their manifest counterparts.

According to the "modulated parasystole" theory (fig. 1B), electrotonic depolarizations across an area of entrance block can entrain an ectopic pacemaker and force it to beat at cycle lengths that may be shorter or longer than its own intrinsic period, depending on phase relationships (i.e., E,S). As a result of this biphasic influence, a number of patterns may emerge that depend on the magnitude of the electrotonic influence and on the relationship between the intrinsic ectopic pacemaker period and the basic heart rate.

Contrary to the interpretation of Kinoshita et al., the long manifest interectopic intervals in this case need not represent the intrinsic period of the ectopic pacemaker. Indeed, the inverse solution of figure 1B indicates that the ectopic patterns in the case presented by Kinoshita et al. as figure 1 could have been generated by a supraventricular parasystolic pacemaker that had an intrinsic period of 1725 msec, but was forced to beat at periods that ranged between 1840 and 1600 msec depending on E,S.

This suggestion is only a guess; I have no way of proving it. However, Kinoshita et al. are also guessing, and, unless my assumptions remain postulated by the demonstration that the "pure parasystolic cycles" are equal to the longer interectopic intervals and by the documentation of the existence of an "absolute refractory period" that extends beyond 56% of the pure parasystolic cycle, my guess is just as good as theirs.

JOSE JALIFE, M.D.
Masonic Medical Research Lab
Utica, New York
Department of Pharmacology
S.U.N.Y. Upstate Medical Center
Syracuse, New York

References

The authors reply:
To the Editor:

We thank Dr. Jalife for his interest in our paper. We reported two cases of supraventricular parasystole in which "pure" parasystolic cycles containing no intervening sinus beat were not seen. As mentioned in that paper and as shown in Dr. Jalife's letter, these cases can be explained both by the mechanism postulated by Jalife et al. and by our theory. Jalife et al. postulated that when a sinus impulse falls early in the parasystolic cycle, it delays the next discharge of the pacemaker. In our previous cases of parasystole\(^4\) in which pure parasystolic cycles were seen, however, it was not shown that the interectopic interval containing such an early sinus impulse was longer than the pure parasystolic cycle.

One of our cases of parasystole with type I second-degree entrance block is shown in figure 1. Figure 1 shows parts of a long continuous recording. In this case, when a sinus beat conducted to the ventricles occurred within 1.16 seconds after the preceding ectopic beat, the interectopic interval containing this sinus beat (for example, the beats S\(_1\) and S\(_2\) in the top strip of figure 1) was always almost equal in length to the pure parasystolic cycle (E\(_1\)E\(_2\) in the middle strip), irrespective of marked changes in the interval between the preceding ectopic beat and the intervening sinus beat (ES interval). Such ES intervals in this case ranged from 0.71 to 1.15 seconds. Thus, we do not believe that the mechanism postulated by Jalife et al. can explain all cases of parasystole.

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Ventricular parasystole with type I second-degree entrance block. This ECG was taken from a previously reported patient,\(^6\) but more than 5 years after the previous recording. The middle and bottom strips are continuous, but the top and middle strips are not continuous. Time intervals are expressed in hundredths of a second. Shaded bars in the diagrams below the strips indicate the absolute refractory period of the reentrant pathway. S and (S) = sinus beats conducted and not conducted to the ventricles, respectively; E and (E) = manifest and concealed ectopic beats, respectively; E + S = fusion beat; V = ventricles; RP = reentrant pathway containing the parasystolic focus.
Mechanisms of parasystole.
J Jalife

Circulation. 1982;65:1036-1038
doi: 10.1161/01.CIR.65.5.1036

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/65/5/1036.citation