Mechanisms of Intermittent Ventricular Bigeminy

II. Parasystole, and Parasystole or Re-entry with Conduction Disturbance

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In a previous report dealing with intermittent ventricular bigeminy due to ventricular premature systoles with fixed coupling in the presence of a grossly irregular dominant pacemaker, it was shown that the appearance and disappearance of the ectopic beats were dependent upon the length of the ventricular cycle ("rule of bigeminy"). In the present report, dealing with ventricular premature systoles with varying coupling in the presence of a regular dominant rhythm, the mechanism of ectopic impulse formation is shown to be either parasystole or re-entry, and the intermittence of bigeminy is due to simple interference or to a disturbance in conduction of a parasystolic or of a re-entrant impulse. Intermittent parasystole is illustrated and presented as a possible link between the two fundamental mechanisms of parasystole and re-entry.

In a preceding report,¹ a group (A) of cases of intermittent ventricular bigeminy was analyzed in which the latter was caused by the occurrence of ventricular premature systoles with fixed coupling during a grossly irregular dominant rhythm. Under such circumstances, intermittence and recurrence of bigeminy were shown to depend largely on the duration of the ventricular cycle. In the course of the study, another group (B) of intermittent bigeminy was identified in cases with a regular dominant rhythm and marked variability of the coupling of the ectopic beats. This group, too, was submitted to extensive analysis and, as in group A, certain conclusions were reached regarding the mechanism involved in the intermittence and reappearance of ventricular premature systoles. The results of this study, and their implications pertaining to the genesis of ventricular premature systoles, are presented in this report.

Material and Methods

The material comprises 14 records from 12 patients with a regular dominant rhythm and intermittent ventricular bigeminy selected on the basis of sufficient material for a detailed study. Since in all cases the coupling of the ectopic beats varied over a wide range, the methods of analysis were those applied customarily in the recognition or exclusion of a parasystolic mechanism.² Careful measurements were made of the intervals separating ectopic beats, or ectopic beats and fusion beats, and these were examined as to the presence of a least common divisor, or the presence of a structure characteristic for a conduction disturbance of the Wenckebach type.* Furthermore, the duration of the coupling of the first premature systole upon resumption of bigeminy, whether fixed or variable, was noted; and finally, the cause of intermittence of bigeminy was examined, whether attributable to physiologic refractoriness of the ventricles subsequent to a preceding response, to a conduction disturbance, to intermittence of ectopic (parasystolic) impulse formation or a combination of these mechanisms:

Results

Five representative cases are illustrated in figures 1, 2, 3, 4 and 5, and the analysis of the

* The characteristic arrangement of the irregularity of rhythm caused by the Wenckebach phenomenon of impaired conduction is as follows: (1) Groups of short cycles are separated by a long cycle. (2) The long cycle separating the short cycles measures less than double that of any of the short cycles. (3) Within the groups of short cycles there is progressive shortening of cycle length. (4) The first cycle after the intermission (long cycle) is longer than the last cycle preceding the intermission.
MECHANISMS OF INTERMITTENT VENTRICULAR BIGEMINY. II

Fig. 1. Intermittent ventricular bigeminy, due to continuous parasystole, showing progressive shortening and temporary fixed coupling of the ectopic beats. (Lead II). A continuous long strip obtained on another occasion is reproduced in Katz, Electrocardiographyb). The sinus rate is uneven, varying between 60 and 71. In the first portion of the strip each sinus beat is succeeded by a premature ventricular beat, the coupling of which shortens progressively from 0.92 to 0.52 second. After two successive sinus beats in the middle of the record bigeminy occurs again; here, however, the coupling is fixed (0.52 second) and equal to that of the last premature beat of the first group. Another bigeminus, with the shortest coupling (0.46 second), is seen at the end of the strip. Thus, while the coupling of the premature systole varies within a wide range, the intervals separating them are as follows: 1.76, 1.70, 3.40, 1.70, 1.70 and 3.38 seconds. This establishes the presence of a continuous ventricular parasystolic pacemaker operating at a rate of 34 to 35—a little faster than half the sinus rate. The two latent discharges of the parasystolic focus (causing the long interectopic intervals) occur 0.04 and 0.06 second respectively following completion of the T wave of a sinus beat and thus within the refractory phase of the ventricles. Thus, in this instance, intermittent ventricular bigeminy is caused by ventricular interference of sinus impulses with those of a ventricular parasystolic focus.

The sinus arrhythmia and the development of a fixed coupling appear to be interrelated in the following way: sinus cycles not including a ventricular beat, or starting at the time of an ectopic discharge, are longer than those which include a ventricular beat at a normal P-R distance, or one paired with an ectopic beat (ventriculophasic sinus arrhythmia14, 15). Whereas the ectopic rate remains constant, the sinus rate changes depending on the occurrence of an effective ectopic impulse within its cycle. Temporarily the sinus rate becomes exactly twice the rate of the parasystole and bigeminy with fixed coupling ensues.

Fig. 2. Intermittent ventricular bigeminy due to intermittent parasystole, with progressive shortening of the coupling of the ectopic beats (in a case with healing posterior wall infarction). The regular succession of sinus beats (rate 71) is periodically disturbed by ventricular premature beats. They are similar in contour, but some alterations in the contour of QRS and/or T can be noted in the first premature beat of a series. The coupling varies considerably. There is progressive shortening from the first premature beat (0.56 to 0.54 second) to the last (0.44 second) of a sequence. The intervals separating the premature beats are fairly regular, corresponding to a rate of 36 to 37. This suggests the presence of a parasystolic ventricular focus with a rate a little faster than half the sinus rate. On this assumption, an ectopic beat would fail to occur after a series of four as a result of the progressive shortening of the coupling, causing a fifth ectopic discharge to coincide with the absolute refractory period following a sinus beat. However, this is not true for the calculated ectopic discharge preceding a series. Note that the first coupling of each series of ectopic beats remains the same.

The factors which determine this unusual appearance of parasystolic rhythm in the form of intermittent ventricular bigeminy are: (a) the rate of the ectopic discharge, which is a little faster than half the sinus rate, (b) the normal refractory period of the ventricle, (c) a temporary breakthrough of a sinus impulse past the barrier of protection of the ectopic focus, or a spontaneous intermittence of the parasystolic activity of this focus. The possibility of temporary exit block of a continuously acting parasystolic focus appeared to be unlikely in view of the fixed coupling of the first ectopic beat of each run of bigeminy and could, in this instance, definitely be ruled out after studying the entire material.
Fig. 3. Intermittent ventricular bigeminy due to intermittent parasystole showing (a) progressive shortening of the coupling of the ectopic beats and (b) a conduction disturbance of the parasystolic impulses. The two lower strips are consecutive (but not continuous) portions of a long strip of lead III obtained several hours after the upper tracing (lead II). The sinus rate is 94 in lead II and 107 in lead III. There is an intermittent ventricular bigeminy, in that after one undisturbed sinus cycle two or three sinus beats are paired with ventricular premature systoles. The latter vary in coupling and contour. The coupling of a first of a series is fixed in each lead (0.56 second in II and 0.54 second in III) and shortens progressively to 0.38 and 0.44 second respectively. Because in lead III the duration of the first coupling approaches the cycle length of the sinus (0.56 second), the sinus and ectopic impulses occasionally come into competition for ventricular activation with a ventricular fusion beat (VF) resulting. During bigeminy the intervals between the ectopic beats shorten progressively.

The repetitive occurrence of the premature systoles, their constant coupling, and the occurrence of ventricular fusion beats suggest a parasystolic mechanism. A simple parasystole cannot be present since the short intervals are of varying duration and the long intervals are not simple multiples of any short one; nevertheless, the spacing of the ectopic and fusion beats show an arrangement suggestive of a Wenckebach type of block affecting the impulses of a continuous parasystolic pacemaker; however, the fact that the coupling of the ectopic beats initiating each run of bigeminy is fixed, regardless of whether the run of bigeminy consists of two or three couples, rules out a continuous parasystole with conduction disturbance (Wenckebach phenomenon) and suggests an intermittent parasystole with conduction disturbance.

On this assumption the intermittence of the resulting bigeminy can be explained in this case in a two-fold way: (a) by temporary intermittence of the parasystolic impulse formation, similar to that seen in figure 2 or (b) by an intermittent “exit block” of the parasystolic impulse. The latter may result from coincidence of the ectopic impulse with normal ventricular refractoriness or may be the result of an abnormal progressive delay of its propagation. This interpretation is supported by the constant coupling of the first of a series of premature systoles, a finding characteristic for intermittent parasystole.

cases is given in the respective legends. The characteristic findings of the spacing of the ectopic beats, the interpretation as to the underlying mechanism, and the cause of intermittence of the ventricular bigeminy are summarized in the accompanying table.

DISCUSSION

All cases included in this group, in contrast to group A, showed marked variations in the coupling of the ectopic beats. Since the cycle length of the dominant pacemaker was constant, it could play no role in the initiation or intermittence of the bigeminy as was the case in group A. The alteration in coupling of the premature systoles occurred either in the form of a progressive shortening (figs. 1 to 3) or progressive prolongation (figs. 4 and 5) before intermittence of bigeminy took place. Only in one case (fig. 1) was the coupling of the ectopic beats temporarily fixed.

Evidently, if progressive shortening of the coupling is found during ventricular bigeminy, a re-entry mechanism can be excluded and parasystole has to be suspected. Measuring of the interectopic intervals then permits differentiation between two mechanisms. (1) The long interectopic intervals may be simple multiples of the short ones; this can be considered to indicate continuous parasystole with a rate a little faster than half the rate of the dominant pacemaker. Under such cir-
MECHANISMS OF INTERMITTENT VENTRICULAR BIGEMINY. II

Fig. 4. Intermittent ventricular bigeminy probably due to intermittent parasystole, with progressive prolongation of the coupling of the ectopic beats. A regular sinus rhythm (rate 88) is disturbed on three occasions (in the middle of the strip) by premature auricular impulses, the transmission of which to the ventricles is interfered with by the occurrence, just after the premature auricular discharge, of ventricular premature systoles. The ventricular premature beats are part of a sequence of four which starts after the second sinus beat, is interrupted after the sixth sinus beat, and resumed after the seventh. The coupling of the ectopic beats shows progressive prolongation (0.40 to 0.56 second); however, the first coupling of the two runs of bigeminy is fixed. While the short interectopic interval preceding, and that following, the intermittence of bigeminy are almost identical (the difference being 0.04 second), the long interectopic interval that bridges the intermittence is considerably shorter than two short interectopic intervals. This rules against a continuous parasystole with or without conduction disturbance and suggests an intermittent type of parasystole with a rate (42) less than half of the sinus node. A re-entry mechanism with progressive delay in the re-entry path (see fig. 5) cannot be ruled out entirely, but the even spacing of the ectopic beats during bigeminy is definitely in favor of a parasystolic mechanism.

Under these circumstances interruption of bigeminy will take place when parasystolic impulses occurring progressively earlier in the wake of a dominant impulse eventually coincide with the normal refractory phase of the ventricle (fig. 1). When the dominant sinus rhythm is uneven, its rate may temporarily become exactly half the parasystolic rate. As a consequence, the coupling of the ectopic beats may become transiently fixed within the cycle of the sinus beats, an unusual feature in parasystole. In a case like this the diagnosis of parasystole and the exclusion of a re-entry mechanism can be made only if long records are available, as was the case in figure 1. (2) The spacing of the ectopic beats may appear to be quite irregular; however, close examination of the arrangement of the ectopic beats may reveal the characteristic structure of Wenckebach periods. On this basis, too, operation of a regular parasystolic pacemaker can be implied with progressive impairment of the spread of successive parasystolic impulses. Here the intermittence of bigeminy can be ascribed to interference or to “dropped beats,” and conditions then become comparable to the familiar mechanisms operating in second degree A-V (and S-A) block. If, at the same time, the coupling of the first ectopic beat of each run of bigeminy is fixed, an intermittent parasystole with conduction disturbance can be diagnosed (fig. 3).

If progressive lengthening of coupling is found, there are several possibilities to be considered. The spacing of the interectopic intervals may clearly indicate simple parasystole, but this time at a discharge rate just a little slower than half the rate of the dominant pacemaker. Unless the activity of the parasystolic pacemaker itself shows intermittence (as in fig. 4), the bigeminy would be expected to continue for some time, depending on the spacing of the primary pacemaker, before intermittence of bigeminy would be caused by interference of the two competing impulses. If, on the other hand, progressive lengthening of the coupling is associated with a Wenckebach structure of the interectopic intervals, parasystole or re-entry may be operating. Under either circumstance a progressive conduction delay is conceivable in parasystole involving pathways from the ectopic pacemaker to the rest of the myocardium, in re-entry affecting the re-entry path itself or pathways leading to the area of the sweep. In both cases, dropping out of an ectopic impulse causes intermittence of bigeminy. In the case of re-entry the intermittence is expected to follow the longest coupling, and in each run of bigeminy the first ectopic beat would tend to have a coupling
of equal length and shortest duration; the same would be true for an intermittent parasystole (see below) but would not apply to a continuous parasystole.

An instance illustrating a re-entry mechanism associated with progressive lengthening of the coupling is illustrated in figure 5. In this case re-entry was implied because no consistency in the interectopic spacing was found in the presence of progressive lengthening of the coupling of the premature systoles. Throughout the record, with one exception, the shortest coupling occurs after the intermittenct of bigeminy, and the longest just preceding it. The exception is the last premature systole in lead I which has a coupling...
Table 1.—Summary of the Analysis and Interpretation of Cases 1 to 5

<table>
<thead>
<tr>
<th>Coupling</th>
<th>First after intermittence</th>
<th>Mode of alteration</th>
<th>Interectopic intervals</th>
<th>Mechanism</th>
<th>Cause of intermittence of bigeminy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>variable</td>
<td>Progressive shortening</td>
<td>Short: regular Long: multiples</td>
<td>Continuous Parsonsystole (R-R) &lt; 2(P-P)*</td>
<td>Interference</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(temporarily fixed?)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>fixed</td>
<td>Progressive shortening</td>
<td>Short: regular Long: not multiples</td>
<td>Intermittent Parsonsystole (R-R) &lt; 2(P-P)</td>
<td>(a) Interference and (b) Intermittence of activity</td>
</tr>
<tr>
<td>Case 3</td>
<td>fixed</td>
<td>Progressive shortening</td>
<td>Irregular with Wenckebach structure</td>
<td>Intermittence of activity or interference, or exit block due to conduction disturbance</td>
<td></td>
</tr>
<tr>
<td>Case 4</td>
<td>fixed</td>
<td>Progressive prolongation</td>
<td>Short: regular Long: not multiples</td>
<td>Intermittent Parsonsystole (R-R) &gt; 2(P-P)</td>
<td>Intermittence of activity</td>
</tr>
<tr>
<td>Case 5</td>
<td>fixed (except one)†</td>
<td>Progressive prolongation</td>
<td>Irregular, No Wenckebach structure</td>
<td>Re-entry with conduction disturbance</td>
<td>Block in re-entry path</td>
</tr>
</tbody>
</table>

* R-R = Parasytolic interval  † discussed in text  P-P = Sinus interval

longer than anticipated, and corresponding to the second coupling of the other groups. This apparent inconsistency can be explained by the assumption that the sinus beat ahead of this bigeminus initiated a re-entry which was not completed because the impulse penetrated only partially into the re-entry path. This would represent an unusual variant of "concealed conduction," occurring in a re-entry path and manifested by its effect on the duration of the subsequent re-entry sweep (coupling), analogous to a phenomenon ordinarily encountered in pathways of A-V conduction. A similar mechanism was proposed by Schott, affecting the conduction of parasytolic impulses.

The duration of the coupling of the first premature beat initiating a run of bigeminy was fixed in four of the five illustrated cases. Only in one case (fig. 1) was there a variation exceeding 0.08 second. This and the type of the interectopic spacing left no doubt as to the operation of a continuous parasytole in that instance. Ordinarily, fixed coupling of premature systoles is considered to indicate dependence of the ectopic beat upon the beat to which it is coupled, and a re-entry mechanism can be assumed to be present. However, a repetitive occurrence of a constant coupling is also seen in intermittent parasytole, a condition in which the regular activity of the ectopic focus is periodically disturbed, either by transient disappearance of its "protection," or by spontaneous intermittence of the parasytolic impulse formation. Such fixed coupling is, of course, limited to the first of a sequence of parasytolic beats. Simple cases of intermittent parasytole causing intermittent bigeminy are illustrated in figures 2 and 4; here, the interectopic intervals within the runs of bigeminy are constant. In figure 2, on the other hand, the arrangement of the ectopic beats would indicate intermittent parasytole with conduction disturbance of the Wenkebach type. It would appear that the same mechanism which is responsible for sporadic or repetitive appearance of isolated premature systoles, may, on occasion, initiate continuous
ectopic impulse formation. A series of such ectopic beats, when rapid, constitutes a paroxysmal tachycardia, when occurring at a slow rate, parasystole of an intermittent type. Thus, intermittent parasystole may represent a link between the two fundamental mechanisms of re-entry and parasystole.

The recognition of an intermittent type of parasystole introduces another factor which may be responsible for intermittence of bigeminy, in addition to physiologic ventricular interference and conduction disturbance of the parasystolic impulses. In fact, in the case of figure 3, it cannot be decided which of these three mechanisms causes the intermittence of bigeminy. Moreover, as pointed out before, when the rate of the parasystolic pacemaker is slower than half the rate of the dominant rhythm, so that there is progressive lengthening of the coupling of successive ectopic beats, it may become very difficult or impossible to determine whether a conduction disturbance involves an intermittent parasystole or a re-entry mechanism (fig. 4). The distinction can be made when a Wenckebach structure of the interectopic intervals is associated with progressive shortening of the coupling, since this obviously cannot occur with re-entry (fig. 3).

No case was encountered in our material in which intermittent bigeminy was caused by a continuous parasystole with regular spacing at a rate approaching half the sinus rate, and in which intermittence could not be attributed to the normal refractory state of the ventricles. Such cases are on record (see below), and an "exit block" without the Wenckebach phenomenon has to be postulated to account for the intermittence.

A number of cases can be found in the literature complying with the classification of parasystolic bigeminy outlined above. Ventricular bigeminy due to continuous parasystole and intermittence of the bigeminy as a result of the normal refractory period (with progressive shortening of the coupling of the ectopic beats) is seen in a case of Faltitschek and Scherf,9, case 5 and (with progressive lengthening of the coupling) in two cases of Scherf and Schott,10, figs. 102 and 103 a case of Vedoya,11, fig. 5 and a case of Gentile,12, fig. 1

Intermittent bigeminy due to continuous ventricular parasystole with exit block (with progressive lengthening of the coupling) can be recognized in a case of Scherf and Schott,10, fig. 10 and with both progressive lengthening and progressive shortening in the same record as a result of marked sinus arrhythmia, in a case of Goldenberg and Scherf,12, figs. 1 and 2

Unusual instances of intermittent parasystole (without conduction disturbance of the ectopic impulse during the runs of bigeminy) were reported by Scherf and Boyd.8 Their case 2 shows progressive shortening of the coupling, case 3 progressive lengthening in a case of a parasystolic pacemaker almost identical in rate with that of the sinus node, and bigeminy due to interpolation of the ectopic beats. The difficulty in distinguishing between bigeminy due to intermittent parasystole and re-entry with progressive conduction delay in the re-entry path is well illustrated by the cases of Zander14 and Scherf and Schott,10, fig. 130

A review of the entire material, both of the first part of our study1 and of the present report, permits the following regrouping and classification of intermittent ventricular bigeminy due to ventricular premature systoles, based on the two mechanisms of ectopic impulse formation.

A. Bigeminy due to re-entry (1) With fixed coupling and intermittence of bigeminy due to critical shortening of the ventricular cycle of the dominant rhythm,1 (2) With varying coupling and intermittence of bigeminy due to a conduction disturbance in the re-entry path.

B. Bigeminy due to parasystole with varying coupling and intermittence due to (1) The refractory phase of the ventricles following a response to the dominant pacemaker (interference), (2) Exit block of the parasystolic impulse with or without the Wenckebach phenomenon, (3) Intermittence of ectopic impulse formation, spontaneous, or due to temporary disappearance of protection (intermittent parasystole).

Our study and classification do not include cases in which intermittent bigeminy with
fixed coupling of the premature systoles occurred in the presence of a regular dominant rhythm. Although such cases are numerous and actually represent the most common variety of intermittent ventricular bigeminy in clinical electrocardiography, their study seemed redundant, since no insight can be gained into the mechanism of ectopic impulse formation in the face of constant spacing of both the dominant and ectopic beats. For that reason their inclusion in the above genetic classification did not appear warranted. Their mode of initiation and termination of bigeminy remains undetermined. Either mechanism, re-entry or intermittent parasystole with a rate of the parasystolic pacemaker half that of the dominant rhythm, could account for such ectopic beats.

**Summary and Conclusions**

1. As a continuation of a preceding report on the mechanisms of intermittent bigeminy caused by ventricular premature systoles, an analysis is presented of five selected cases exemplifying a group characterized by a regular dominant rhythm and variable coupling of the ectopic beats.

2. Under such circumstances, ventricular bigeminy may be due either to re-entry or parasystole. Criteria for the differentiation between the two mechanisms are based upon (a) the presence or absence of fixed coupling and (b) the numerical relationship of the interectopic intervals which may point to the presence of a simple parasystole or one complicated by a conduction disturbance of the Wenckebach type.

3. When spacing of the ectopic beats characteristic of a parasystole was found only in the individual runs of bigeminy, but not throughout the record, an intermittent form of parasystole was diagnosed. Under such circumstances the coupling of the first premature beats after the interectopic intervals of bigeminy was fixed. The latter phenomenon supported the diagnosis of intermittent parasystole.

4. Intermittent parasystole appears to represent a link between the two fundamental mechanisms of re-entry and parasystole.

5. Intermittent bigeminy in parasystole occurs when the rate of the parasystolic pacemaker approaches half the rate of the dominant pacemaker. If it is slightly faster the coupling of successive ectopic beats exhibits progressive shortening; if it is slightly slower, the coupling shows progressive lengthening. Intermittence of bigeminy occurs as a result of (a) the normal ventricular refractory period, (b) a conduction disturbance of the parasystolic impulses, (c) intermittence of ectopic impulse formation, and (d) a combination of these mechanisms.

6. Differentiation between a re-entry mechanism and intermittent parasystole is not always possible. This is true for both short runs of bigeminy with fixed coupling and certain cases with progressive shortening of the coupling.

7. The literature is reviewed for cases with intermittent ventricular bigeminy, and some unusual instances are pointed out, revealing mechanisms similar to those presented above.

8. A classification is proposed of intermittent bigeminy due to ventricular premature systoles based on genetic principles.

**Summario e Conclusiones in Interlingua**

1. In continuation de un previe reporto super le mechanismos de bigeminia intermittente causate per prematur systoles ventricular, nos presenta un analyse de cinque seligite casos que exemplifica un typo characterisate per un rhythmo dominante regular e un variabile accopulamento del pulsos ectopic.

2. Sub tal conditiones, bigeminia ventricular pote esser causate per re-entraga o per parasystole. Criterios pro differenciar le duo possibile mechanismos es basate (a) super le presentia o absentia de un accopulamento fixe e (b) super le relation numeric del intervallos interectopic que indica le presentia de o un simple parasystole o un parasystole complicate per un disturbance del conduction del typo Wenckebach.

3. Quando le spatiamento del pulsos ectopic eseva del typo caracteristic de parasystole sed occurreva solo in le cursos individual de bigeminia e non in le integre registration, nostre diagnose eseva "un forma intermittente de parasystole." Sub iste conditiones le accopu-
lamento del prime pulsos prematur post le intermittentias del bigeminia esseva fixe. Iste ultime phenomeno supportava le diagnose de intermittente parasystole.

4. Intermittente parasystole pare reprezentar un nexo inter le duo mechanismos fundamental de re-entra e parasystole.

5. Bigemina intermittente in parasystole occurre quando le tempo del pacemaker parasytolic approcha medie le tempo del pacemaker dominante. Quando le tempo del pacemaker parasytolic excede illo del pacemaker dominante levemente, le accopulamento de successive pulsos ectopic exhibi un accurement progressive; in le caso contrario, le accopulamento exhibi un allongamento progressive. Intermittentia de bigemina occurre in consequentia (a) del normal periodo refractori ventricular, (b) de un disturbation conductional del impulsos parasytolic, (c) de intermittentia del formation de impulsos ectopic, e (d) de un combination de iste mechanismos.

6. Le differentiation inter un mechanismo de re-entra e intermittente parasystole non es semper possibile. Isto es ver tanto pro breve cursos de bigemina con accopulamento fixe como etiam pro certe casos con allongamento progressive del accopulamento.

7. Es presentate un revista del litteratura in re cases de intermittente bigemina ventricular. In isto nostro presta atttention specialmente a alcun cases inusual que exhibi mechanismos simile a illos reportate in le presente studio.

8. Super le base de principios genetic nos propone un classification del casos de intermittent bigemina causate per premmatur systoles ventricular.

REFERENCES

5 Mack, I., and Langendorf, R.: Factors influencing the time of appearance of premature systoles (including a demonstration of cases with ventricular premature systoles due to reentry but exhibiting variable coupling). Circulation 1: 910, 1930.
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