Parasystole with Fixed Coupling

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SUMMARY

Four examples of spontaneous and six of artificial ventricular parasystole exhibiting fixed coupling of the premature beats were presented. In five cases, one of the pacemakers was located in the S-A node, in four in the A-V junction, and in one at first in the A-V junction and later in the ventricles. The material was divided according to the presence either of mutual protection of the two pacemakers (group I, four cases) or of unilateral protection of the parasystolic pacemaker (group II, five cases).

When the rates of both pacemakers are regular and the conditions for impulse propagation remain stable, fixed (forced) coupling of parasystolic beats develops under the following conditions: in group I (with mutual protection) (a) if there is—by chance or by experimental adjustment—a simple numerical relation between the two pacemaker rates, or (b) if subthreshold stimuli become effective only during a short supernormal period of excitability in the early portion of the ventricular cycle; and in group II (with unilateral protection) as a result of a fixed relationship that develops secondarily to the discharge of the dominant pacemaker by the parasystolic pacemaker, a mechanism referred to as "reversed coupling." This mechanism may be combined with that of group Ib.

Additional Indexing Words:
Premature beats
Sinus and ectopic rhythms
Allorhythmia
Sinus tachycardia

One of the hallmarks of parasystole is a changing interval between premature ectopic beats and those of the dominant rhythm. However, a constant coupling interval does not rule out a parasystolic rhythm. Fifty years ago Kaufmann and Rotherberger found in dog experiments—much to their surprise—that simultaneous action of two regular pacemakers of different rate invariably resulted in the development of repetitive group beating, an allorhythmia, with recurrence of one or more identical coupling intervals. This was true regardless of the presence or absence of a simple numerical relation between the two rhythms and regardless of whether or not they remained entirely independent. With total independence, a group was concluded and started to repeat itself as soon as the least common multiple of the two rates was reached. Obviously this multiple had to be a whole number since a group can consist only of a whole number of beats. In its simplest form the allorhythmia resulted in bigeminy with fixed coupling.

Allorhythmias with repetitive coupling intervals can be observed in clinical electrocardiography with spontaneous as well as artificial parasystoles, usually as a temporary event since the dominant pacemaker, usually located in the sinus node, is more or less irregular. If this irregularity is eliminated during simultaneous action of two artificial pacemakers, a lasting parasystolic allorhythmia becomes established and the different factors determining the pattern of group beating and the development of fixed coupling can be readily studied in the human heart.
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Figure 1

Fixed coupling due to a simple numerical relation between the parasystolic and basic rate. (A) In spontaneous parasystole after acceleration of sinus rate; (B) in artificial parasystole after adjustment of pacing rate. \( Si = \) sinus rate/min; \( P = \) parasystolic rate/min. Numbers between horizontal arrows are multiples of cycle length in sec/100, of parasystole in A and of parasystole and sinus in B. Numbers within records are coupling intervals in sec/100. The first three pacemaker signals in B are retouched.

This report deals with 11 cases in which this line of study was pursued. In accordance with the experiments of Kaufmann and Rothberger, the material was divided into group I, in which there was mutual protection of dominant and parasystolic impulse formation and group II, in which this protection was unilateral in that the regular parasystolic impulses could reach and discharge prematurely the predominant rhythm and thus reset it. Two different causes of constant coupling could be established in both groups. Furthermore, the factors that prevent the development of a permanent allorhythmia under such circumstances became apparent, permitting the recognition of a parasystolic rhythm.

The Material and Its Interpretation

Group I. Parasystole with Mutual Pacemaker Protection

Fixed Coupling due to a Simple Numerical Relation Between Basic and Parasystolic Rhythms

In the upper record of figure 1A four ventricular premature beats occur during sinus tachycardia (rate 115) at different coupling intervals, as indicated. The longest coupling (measuring 0.50 sec) permits the fusion of a sinus and an ectopic impulse. Measurements of the inter-ectopic intervals reveal a common denominator of 0.75 sec and, thus, the presence of a spontaneous ventricular parasystole discharging at a regular rate of 80. None of the parasystolic impulses is transmitted back to the atria (mutual protection of both sinus and ectopic rhythms). To explain the long inter-ectopic interval of 3.75 (5 \( \times \) 0.75) sec at the beginning of the record, exit block of one parasystolic impulse has to be postulated. Elsewhere, failure of every other ectopic impulse to propagate is accounted for by ventricular refractoriness after a sinus beat.

In the lower section of figure 1A, the same ventricular parasystole, with corresponding inter-ectopic intervals of 1.50 sec persists in the form of a simple allorhythmia. A premature beat occurs after every second sinus beat at a constant coupling of 0.36 sec. This change from the previously variable to fixed coupling is the consequence of a slight acceleration of the sinus rate to 120 so that

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now two parasystolic cycles \((2 \times 0.75\text{ sec})\) precisely equal three sinus cycles \((3 \times 0.50\text{ sec})\). As before, alternate parasystolic impulses fall into the ventricular refractory phase and none is conducted back to the sinus node (mutual protection).

In figure 1B, a similar event is illustrated by an artificial parasystole which developed when A-V conduction was restored during transvenous pacing in a patient with advanced A-V block. Responses to the artificial stimulation are represented by premature beats occurring at a constant coupling of 0.60 sec after every third sinus beat. Here the arrhythmia is the result of appropriate adjustment of the pacing rate so that: (a) the sum of three parasystolic cycles precisely equals the sum of four regular sinus cycles \((3 \times 1.147 = 3.44\text{ sec} = 4 \times 0.86\text{ sec})\); (b) only one of three successive pacemaker impulses elicits a ventricular response whereas the others fall into the ventricular refractory period after a sinus beat; and (c) none of the artificial impulses is conducted back to the atria to disturb the sinus activity (mutual protection).

More complex allorhythmias may result from a simple temporal relation of two mutually protected regular pacemakers as exemplified by figure 2, another instance of A-V block with resumption of A-V conduction after several days of artificial pacing. Here, groups of four sinus beats alternate regularly with four artificial beats. The transition from artificial to natural beating always takes place via a ventricular fusion beat whereas the switch to the parasystolic rhythm occurs at a constant short coupling interval of 0.40 sec. Again, the explanation of this unusual behavior of a parasystole is found in the comparative measurements indicated in the record. The sum of eight sinus cycles \((8 \times 0.70\text{ sec})\) equals the sum of seven artificial ones \((7 \times 0.80\text{ sec})\). This, of necessity, must result in strictly periodic appearance and disappearance of the two independent rhythms with constant coupling of the ectopic (parasystolic) one. Fusion beats occur once the least common multiple of the two cycles is reached, representing a new "starting point" for the repetitive interplay of the two rhythms.

**Fixed Coupling due to a Supernormal Phase of Ventricular Excitability**

Figure 3 illustrates a different cause of fixed coupling in the presence of two mutually independent pacemakers. A, B, and C are successive records in a case of 2:1 A-V block (combined with an intraventricular block) paced via a transvenous catheter by subthreshold stimuli. In all, the spontaneous (sinus) rhythm is regular (rate 83). In A, none of the artificial stimuli is effective. In B, every third stimulus elicits a ventricular response in the form of a premature

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**Figure 2**

Repetitive group beating (allorhythmia) with fixed coupling as a result of a simple temporal relation between a regular sinus rate and the rate of pacing. P indicates consecutive sinus impulses. Other symbols as in figure 1B.
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Figure 3

Fixed coupling as consequence of a supernormal phase of excitability in a case of 2:1 A-V block paced by subthreshold stimuli. Heavy upright arrows indicate ventricular responses. Other symbols as in figures 1 and 2. C is a continuous record with the last three QRS complexes of the upper strip reproduced as the first three in the lower strip. The greater number of conducted sinus impulses between supernormal responses to pacing compared with B depends on the different relation existing between the natural and artificial rhythm.

Comparison of records A and B (fig. 3) reveals that here fixed coupling is caused by the fact that responses to artificial stimulation are limited to a short supernormal phase rather than by a certain numerical relation of two regular rhythms. However, the latter relationship determines the pattern of the resulting arrhythmia. Thus in B, when four sinus cycles (with two ventricular responses) equal three artificial cycles, premature beats with fixed coupling recur more frequently than in C where 14 sinus cycles (with seven ventricular responses) equal 13 artificial ones.

Group II. Parasystole with Unilateral Pacemaker Protection (Fixed Coupling due to “Reversed Coupling”)

Figure 4 shows an artificial parasystole in a case of complete A-V dissociation due to advanced A-V block in combination with a right bundle-branch block. A spontaneous pacemaker originating in the A-V junction (R) at a rate of 59 to 60 competes for ventricular activation with a transvenous artificial one with a rate of 53. Every fourth pacer stimulus is effective and elicits a premature beat (X); the others fall into the refractory phase after spontaneous beats. The pause following these premature beats is not compensatory and the returning cycle (X-R) is slightly longer than the spontaneous junctional cycle (R-R), an indication that effective artificial impulses reach the junctional pacemaker and reset its cycle (concealed retrograde conduction). Since both
acting pacemakers remain otherwise regular and since the propagation time of their impulses is constant (a) premature retrograde discharge of the spontaneous by the artificial pacemaker must recur after the same number of spontaneous beats, in this instance after every fourth one and (b) the coupling of the premature artificial beats becomes constant. Fixed coupling of the premature beats, is, therefore, a secondary effect of the “reversed coupling” of spontaneous (A-V junctional) beats to the parasystolic ones.

A supernormal phase of excitability as a cause of the fixed coupling of the premature beats, as illustrated by figure 3, was ruled out in this instance by responses to artificial stimuli of the same strength occurring in later portions of diastole when the rate of pacing was changed (not illustrated).

That “reversed coupling” is the cause of fixed coupling of parasystolic beats is again demonstrated in figure 5 from a case of ventricular bigeminy in atrial fibrillation with complete A-V dissociation. The spacing of the supraventricular beats (an A-V junctional rhythm) is regular (1.28 sec) and the coupling of the premature ventricular beats is constant (0.50 sec) except in the middle of the record. Here one junctional cycle (X₃-R) is shortened to 1.04 sec, and the coupling of the subsequent premature beat (R-X₄) is lengthened by the same amount to 0.74 sec. However, the distance between the premature beats remains constant (1.78 sec) revealing their parasystolic nature. Interpretation of this disturbance in the otherwise orderly arrangement of the bigeminy is indicated in the diagram in figure 5.

With the exception of X₃, all premature impulses are conducted back to the site of the junctional pacemaker, discharge it, and thus postpone its spontaneous firing; the
A-V dissociation and retrograde conduction junction. Lower beats alters their otherwise retrograde impulse because it encounters partial refractoriness in the lower A-V junction, and the next junctional impulse is blocked (postponed compensatory pause). Thus, an occasional change in the extent of concealed retrograde conduction of ventricular premature beats alters their otherwise fixed coupling and unmasks the parasystolic mechanism. Without this transient change of unilateral to mutual protection of the two pacemakers, A-V dissociation and bigeminy with fixed coupling (in a digitalized patient) could hardly be considered a result of parasystole.

An analogous persistent allorhythmia due to unilateral “protection” may develop in incomplete A-V dissociation where the premature beats represent ventricular captures (fig. 6). Here it is a conducted (usually sinus) impulse that intermittently disturbs the regularity of an accelerated subsidiary (usually A-V junctional) pacemaker, whereas impulses of the latter do not reach the higher (slower) pacemaker. If both remain regular, fixed “coupling” at regular intervals of single captures or of the first of a series of captures is enforced. Thus in figure 6, a capture occurs after every second automatic beat at a stable R-R interval of 0.44 sec.

Given two regular or nearly regular pacemakers with unilateral protection, a variety of allorhythmias with repetitive coupling intervals may develop, determined by pacemaker rates on the one hand and refractory periods of the A-V junction and ventricles on the other. One such variety is illustrated in figure 7, an instance of spontaneous ventricular parasystole (rate 41 to 42) during sinus rhythm (rate 65 to 70) in which coupling intervals are limited to two ranges (0.80 to 0.92 and 0.44 to 0.48 sec). Premature beats with the shorter coupling, like X₁, are followed by retrograde P waves (−P). The retrograde impulse reaches the sinus node and postpones the next spontaneous sinus impulse. Thereby, the timing of the two pacemakers attains such a relation that

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**Figure 6**

Allorhythmia with fixed coupling of ventricular captures in incomplete A-V dissociation. Lack of protection causes “reversed coupling” of A-V junctional beats (R) to sinus beats (R). P = sinus P wave.

**Figure 7**

Two magnitudes of coupling in spontaneous parasystole due to resetting of the sinus cycle by retrograde conduction to the atria of alternate parasystolic impulses (unilateral protection causing “reversed coupling”). Symbols explained in text.

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(a) the next parasystolic impulse ($X_2$) always falls into the ventricular refractory period after a sinus impulse, and (b) the third, $X_3$, collides in the A-V junction with a sinus impulse preventing retrograde premature activation of the atria. Allorhythmia develops as a result of this repetitive 3:2 ventricular and 3:1 atrial response to the parasystolic impulses, in which failure of propagation in both directions coincides ($X_2$). Alternation of coupling intervals of two magnitudes in parasystole is unusual and attributable to the link between the two rhythms intermittently established via capture of the atria (and of the sinus node) by the ectopic impulse (“reversed coupling”).

The factors that prevent every parasystole with unilateral protection from causing persistent fixed coupling or uniform allorhythmia, or both, are illustrated by two additional cases. Records A and B of figure 8 were obtained in the same patient in short succession. Throughout B, short ventricular cycles (0.50 sec) alternate with cycles which are twice as long (bigeminy). Beats terminating the longer cycles have a widened QRS but resemble previously recorded sinus beats (not illustrated). QRS is more prolonged in the premature, larger complexes. Both types are followed by a retrograde P wave, the earlier one at a longer R-P interval. This record could be interpreted as a regular A-V junctional tachycardia (rate 120) with a 3:2 antegrade and retrograde block, the former with aberrant ventricular conduction, the latter with Wenckebach’s phenomenon. However, the true mechanism of the bigeminy is disclosed by record A. Here the ventricular bigeminy

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**Figure 8**

Spontaneous ventricular parasystole during A-V junctional rhythm causing, in B, persistent bigeminy with fixed coupling due to “reversed coupling.” In A, this allorhythmia is disturbed, and the coupling is variable, as the result of an intermittent exit block of the parasystole. The two portions of record A are continuous with the last QRS of the upper strip reproduced as the first in the lower strip.
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is intermittent with marked variations of coupling intervals (0.50 to 0.86 sec). Furthermore, the long intervals between premature beats in A are multiples of the shorter ones recorded in A as well as in B. This reveals that in both tracings the premature beats are caused by the same independent parasystolic mechanism. In record A, some of the parasystolic impulses are ineffective, apparently due to a transient exit block. This permits several successive spontaneous discharges of the junctional center, thereby changing the time relation to the regular parasystole and resulting in marked differences of coupling intervals when parasystolic beats reappear. Contrariwise, in record B, every parasystolic impulse is effective. Hence, retrograde conduction occurs regularly and each such impulse, on its way to the atria, discharges the junctional pacemaker prematurely and resets its cycle (0.90 sec). In this way, a link is maintained between the junctional and the ventricular (parasystolic) rhythms ("reversed coupling") and, in turn, parasystolic beats are forced to maintain a fixed coupling to the junctional ones.

In figure 9 is shown artificial parasystole in atrial fibrillation with incomplete A-V dissociation that has features in common with figures 3 and 4. As in figure 3, responses to subthreshold pacing of the right ventricle (heavy arrows) are limited to a supernormal phase of excitability causing premature beats with fixed coupling and, as in figure 4, these effective parasystolic impulses depolarize not only the ventricles but are conducted part way back to the A-V junction to the site of a junctional pacemaker (R') to prolong its returning cycle from 1.22 to 1.32 sec (concealed retrograde conduction causing "reversed coupling"). But, in addition to these retrograde discharges and in contrast to figures 3 and 4, occasional ventricular captures (C) by fibrillation impulses disturb the regular sequence of spontaneous junctional beats and this prevents the development of arrhythmia, that is, recurrence of premature beats with fixed coupling at predictable intervals.

Finally, figure 10 illustrates fixed coupling associated with different types of arrhythmia which develop in the same case when unilateral protection of two simultaneously acting regular pacemakers changes to mutual protection. Records A and B were obtained in a patient with complete A-V dissociation due to advanced A-V block, who was admitted to the hospital because of malfunctioning

Figure 9

Artificial ventricular parasystole with fixed coupling (due to a supernormal phase of excitability) and "reversed coupling" in atrial fibrillation with incomplete A-V dissociation. Development of arrhythmia is prevented by irregular occurrence of ventricular captures. Symbols explained in text.

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of a previously implanted left ventricular pacemaker. Record A was taken after disabling the "runaway" epicardial pacemaker and insertion of a temporary endocardial one into the right ventricle. When isoproterenol, given by intravenous drip, had accelerated a spontaneous A-V junctional pacemaker to a rate of 52, the strength of artificial right ventricular pacing was reduced to subthreshold values (upright arrows). It is seen to be ineffective until the fifth stimulus (first heavy arrow) falls into the supernormal phase of excitability induced by a spontaneous beat. The resulting premature ventricular response resets the timing of the spontaneous junctional pacemaker which, thereby, gets into a fixed time relation to the artificial impulses. From then on, alternate artificial impulses fall into the supernormal phase which occupies a time span of about 0.08 sec within the T wave of spontaneous beats. This, in combination with "reversed coupling," results in bigeminy with fixed coupling illustrated in the second half of record A.

Record B (fig. 10) was obtained after repair of the permanent left ventricular pacemaker while the transvenous right ventricular one was still in operation. Each left-sided impulse (↓) occurring at a rate of 72 (cycle length, 0.83 sec) elicits a response, but the subthreshold right-sided pacer impulses (↑) occurring at a rate of 50 (cycle length, 1.20 sec) are effective as in record A only during the supernormal phase of excitability following a left-sided beat (heavy upright arrows). They appear as interpolated premature complexes with two magnitudes of coupling intervals (0.42 and 0.34 sec). Unlike record A, the two rhythms, being artificially maintained, are entirely independent (a model for mutual protection). Nevertheless, a repetitive arrhythmia is bound to develop as long as the two pacemakers remain regular. Given the particular
rate relationship, responses to the right-sided pacemaker, limited to a supernormal phase, occur alternately after 3 and 13 left-sided beats, with coincidence at X of every ninth left-sided and every thirteenth right-sided artificial stimulus (9 × 120 = 1,080 and 13 × 83 = 1,079). Interval X-X represents the least common multiple of the two regular rates and point X becomes the moment at which one group of beats ends and the next starts.

Comment

The analysis of our material permits certain generalizations summarized in table 1:

When two pacemakers are protected from each other and, at the same time, compete for activation of the same cardiac chamber, fixed coupling will develop provided that: (a) by chance or by design (during artificial pacing), the rates of the two pacemakers maintain a simple numerical relationship or (b) the parasystolic impulses elicit a response only during a supernormal phase of excitability engendered by a beat of the other pacemaker.

If the dominant rhythm fails to disturb the parasystolic one but not vice versa (unilateral protection), fixed coupling of parasystolic beats becomes the consequence of the recurrent link of dominant to parasystolic beats, a phenomenon for which we propose the term "reversed coupling."

Under both circumstances, various arrhythmias may develop and will be maintained as long as the rates of spontaneous discharges of the two pacemakers remain regular and the speed of propagation of the impulses remains constant.

| Table 1 |
|---|---|
| **Parasystole with Fixed Coupling** | |
| Protection of pacemakers | Mechanism of fixed coupling |
| **Mutual** | a. Simple numerical relation of pacemaker rates |
| | b. Stimuli effective only during supernormal phase of excitability |
| **Unilateral** | "Reversed coupling" |

The results of this study are at variance with the notion that, on the basis of persistent constant coupling, a line can be drawn between premature beats of parasystolic nature and the more common type induced by, and dependent on, impulses of the dominant rhythm (extrasystoles). However, this does not imply that the distinction between the two types of ectopic beats has become futile. On the contrary, our presentation has shown that in cases of fixed coupling a longer period of observation may disclose the parasystolic origin of the ectopic beats during a spontaneous change in rate of the dominant pacemaker (fig. 1A) or, if there is evidence of unilateral protection of the ectopic pacemaker, following the development of an exit block of the latter (fig. 8). The fixed coupling may then change to a varying one while the inter-ectopic intervals remain constant, or the longer ones can be shown to be multiples of the shortest one or a simple fraction thereof.

If a change in rate does not occur spontaneously, it can be provoked by exercise, carotid sinus pressure, or administration of a chronotropic drug, and this will help to identify a parasystolic mechanism.

A supernormal phase of excitability, responsible for the appearance of premature systoles with fixed coupling, was first suggested by Lewis and Master in 1924. Actually, in their case it was not a question of a supernormal phase of excitability but of conductivity in a high degree A-V block. Soloff and Fewell, Linenthal and Zoll, Burchell, and Walker and co-workers demonstrated a supernormal phase of excitability during artificial pacing of the human heart. Goldenberg and Rothberger explained the activation of subthreshold stimuli by the preceding beat as a special form of the Wedensky phenomenon and concluded that this was the mechanism of parasystole with fixed coupling. Recently, Castellanos and associates using two artificial pacemakers simultaneously, demonstrated the Wedensky effect in the human heart and attributed to it the effectiveness of subthreshold impulses late in
the cycle, when a supernormal phase of excitable could no longer be invoked.

Actually, implication of Wedensky facilitation or of a supernormal phase as a cause of long or short fixed coupling presupposes an autonomous center capable of initiating impulses and shielded in some manner from discharge by other impulses—the basis and very nature of a parasystolic rhythm. Our figures 3C and 9 suggest that a parasystolic mechanism should be considered even in cases with very sporadic premature beats with fixed coupling. In those cases in which the inter-ectopic intervals do not permit the assumption of a parasystolic pacemaker—best exemplified by atrial fibrillation with an irregular ventricular response and ventricular bigeminy with fixed coupling—the concept of a re-entry mechanism, strongly supported by recent experiments,14 seems more acceptable to us than that of a repetitive response to a single stimulus be it due to a Wedensky mechanism or a supernormal phase of excitability.

That parasystolic rhythms with unidirectional protection may force the development of fixed coupling was clearly established by Kaufmann and Rothberger4 during experimental studies on atrial premature systoles and can be demonstrated in the human heart by artificial pacing of the atria during a regular sinus rhythm.15 However, only Schaefer,16 a strong proponent of a parasystolic genesis of all extrasystoles, refers to such premature beats as “rückgekoppelte Extrasystolen” and speaks of fixed coupling by “Rück-Koppelung” in analogy to the term “reversed coupling” used by us in the same context.

Runs of ventricular premature beats with fixed coupling of the first one may, on occasion, be due to continuous activity of a parasystolic center (fig. 2). This, however, will have to be distinguished from intermittent parasystole likewise characterized by fixed coupling of the first ectopic beats.17 In such cases, long inter-ectopic intervals are not multiples of a short one nor do they have a common denominator. Also certain observations on transition of typical parasystolic rhythms into extrasystoles with fixed coupling cannot be explained by any of the mechanisms described in this report.

The unsolved riddle of the mechanism of premature beats, the most common disturbance of cardiac rhythm, has hounded physiologists and clinicians alike for many years. New avenues of investigation have been opened by modern refined experimental techniques applicable in the animal laboratory and by simple bedside techniques after acceptance of artificial pacing as a routine therapeutic procedure. There is now reason to believe that several of the various theories concerning the genesis of ectopic beats may have validity, and it would appear that proponents of any single one have made a mistake in excluding all others. This study demonstrates that strict categorization of premature beats into dependent and independent21 (parasystolic) ones on the basis of coupling intervals is no longer feasible.

Conclusions

1. Although variable coupling is the most conspicuous feature of ectopic beats due to parasystole, fixed coupling does not rule out a parasystolic mechanism.

2. Identification of a parasystolic mechanism despite fixed coupling is possible in the presence of mutual protection of both pacemakers during a period of temporary irregularity of one of them, and in the case of unilateral protection of the parasystolic pacemaker during intermittent cessation of discharge of the dominant by the parasystolic pacemaker (caused by intermittent exit block or physiological interference).

3. The ease with which parasystole with fixed coupling can be produced by subthreshold artificial pacing lends support to the assumption of a similar genesis of some forms of spontaneous ectopic beats with fixed coupling.

4. The old abandoned theory of Kaufmann and Rothberger of a parasystolic origin of some premature beats with persistent, fixed coupling appears corroborated in the human
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heart by recent observations during artificial pacing.

References


