Double Irregular Ventricular Parasystole: Rate-Dependent Entrance Block and “Supernormal” Exit Conduction

JÓZSEF TENCZER, M.D., AND LÁSSLÓ LITTMANN, M.D.

SUMMARY An analysis of electrocardiograms from a patient with spontaneous double irregular ventricular parasystole presented. Irregularity in one of the two parasystoles was produced by intermittence based on rate-dependent (phase 3) entrance block, and in the other parasystole it was attributed to “supernormal” exit conduction. Critical analysis of electrocardiograms revealed that first degree block, rate-dependent block, and “supernormal” conduction in the exit pathway may account for the alterations in the arrangement and manifestation of the parasystolic beats. An electrocardiographic approach to these properties of the parasystolic structure and demonstration of double ventricular parasystole with irregularity in both parasystoles has not previously been found in the literature.

AMONG THE FEW REPORTS on spontaneous double and multifocal ventricular parasystole, only three cases with intermittence in one of the parasystoles were demonstrated. Temporary loss of protection of the parasystolic focus was postulated for the interpretation of intermittence in these cases. Resetting of the parasystolic pacemaker was ascribed to supernormal conduction and to rate-dependent conduction disturbance in the surrounding tissues.

The purpose of our report is: 1) to demonstrate a patient with double irregular ventricular parasystole, 2) to approach some electrophysiological properties of the parasystolic structure, and 3) to review the diagnostic criteria of intermittence based on phase 3 conduction defect.

Demonstration and Interpretation of Electrocardiograms

The ECG tracings analyzed were selected from several long rhythm strips (standard lead II) obtained over a seven-day period from a 64-year-old male patient with coronary heart disease. ECG on admission (fig. 1A, continuous tracings) shows QRS complexes of sinus origin (labelled R) and two types of ectopic QRS complexes (labelled A, ventricular ectopy and labelled B). The wide variations in coupling intervals and spacing of both A and B ectopic beats fulfill the criteria for regular double ventricular parasystole with short runs of the double parasystolic rhythm. Beats labelled F are fusions of B and R in the first strip, and of A and R in the second one. Occasionally, the ventricles and the atria were controlled by pure bifocal parasystolic rhythm over longer periods, as seen in figure 1B. Manifest and calculated cycle lengths of parasystole A and of parasystole B are indicated above and below the strips, respectively. All time intervals in this and in subsequent figures and in the text are expressed in hundredths of a second.

The consecutive records in figure 2A were obtained the next day. Parasystole B appears for only one instance in the form of a fusion beat (the second beat in the third strip). The smallest manifest interectopic A-A intervals range from 184-188. The longer interectopic intervals are not simple multiples of the smallest ones. The differences between calculated and manifest cycle lengths exceed that accepted for simple parasystole, and the characteristic structure of Wenckebach type exit block cannot be recognized.

Analysis of the ECG reveals that if the coupling time of a sinus beat to the previous parasystolic beat is below a critical time, the corresponding A-A interval remains about 188, but if it is beyond this critical value, the A-A interectopic interval is prolonged. The time intervals between the sinus beats exceeding the critical coupling time (indicated by circles in the figure) and the next parasystolic beats are only a little longer than the manifest parasystolic cycle length. These intervals in figure 2A are: 192, 192, 196, 196, 196, 192, 196, 196, 188, 192, 192, 196, 196, 196 and 196. The demonstrated phenomena can all be well explained by rate-dependent (phase 3) entrance block of parasystole A. This means that if an impulse reaches the parasystolic focus or the adjacent tissues after their refractory periods, the parasystolic center will be discharged and its timing reset by the entering impulse, and a new cycle will start. Disregarding the possible depressant effect of the invading impulse, the parasystolic beat after the resetting QRS is likely postponed with the sum of entrance and exit conduction times. With coupling times of sinus beats to parasystole A being 102 or shorter, the parasystolic focus is not reset, whereas with those being 104 or longer, the parasystolic center is reset in the tracings of figure 2A. The coupling times of sinus beats to the resetting QRS complexes in this figure are always below the critical interval (104) so that they do not permit successive passive discharge of the parasystolic center.

Coupling times of the resetting and non-resetting beats to the previous parasystole are represented in figure 2B. Small circles in the clear area indicate
coupling times of resetting beats and large circles in the stippled area represent coupling times of non-resetting beats of the tracings in figure 2A.

Figure 3A is a schematic representation, according to Cohen and coworkers, of the last strip of figure 2A. Only the ventricular level is shown. Full circles represent spontaneous firing of the parasystolic pacemaker A, horizontal shaded bars indicate protective block of the parasystolic focus and the interruptions of the bars represent temporary loss of protection. Vertical arrows circumscribing the area of the parasystolic focus represent beats that do not disturb the parasystolic rhythm. Vertical arrows transsecting the interruptions indicate beats that enter, depolarize, and reset the parasystolic center.

Regular arrangement of parasystole B and remarkable irregularity of parasystole A are apparent in figure 3B. Parasystole A appears only after relatively short QRS-QRS intervals. The spacing of beats A can be explained by successive resetting of parasystole A. Extraneous impulses, whether they are of sinus or of parasystole B origin, with appropriate coupling times of 100 or longer, may reset the parasystolic focus. If the intervals between successive extraneous impulses all exceed the critical value, temporary dominance over the parasystolic center will occur. A diagrammatic representation of this proposed mechanism is given below the tracing.

Parasystole B is regular in the continuous records in figure 4, with the exception of an interectopic interval of 140. This shortest interectopic interval (indicated by two arrows) differs more from the longer manifest or calculated cycle lengths than is acceptable for simple parasystole. Shortening in the manifest interectopic interval from 180–140 nearly equals the

lengthening in the next interectopic interval (548) compared to the other three long interectopic intervals (524, 524, 520).

The continuous tracings in figure 5A are parts of the same ECG as in figure 4. Once again, a regular parasystole B is interrupted by an interectopic interval of 140. Apparent shortening of the cycle length and the lengthening of the succeeding interectopic interval is nearly the same as in figure 4. This characteristic arrangement of beats B shown in figures 4 and 5A was observed many times.

It is well-known that cycle lengths calculated from long interectopic intervals are often shorter than manifest cycle lengths. This phenomenon was also observed in our case, except for the interectopic intervals following the ones of 140. Interectopic intervals composed of three cycles varied within narrow limits (520–526) with the exception of those following the shortest ones, which ranged from 548–560. The average difference between the two types of long interectopic intervals measured 32. It was also noted that beats terminating the shortest interectopic intervals of parasystole B were always preceded by QRS complexes A with an interval of 52.

Diagrammatic representation of the proposed mechanism for irregularity of parasystole B is given below the tracings in figure 5A. Intermittent shortening of the interectopic interval of parasystole B with the comparable consecutive lengthening of the following interectopic interval can be readily explained by intermittent improvement in exit conduction of the parasystole. This means that unaltered regularity of impulse formation may be postulated, despite the irregularity of the parasystole.

Analysis of coupling times of the postulated dis-
FIGURE 2. Intermittence — coupling time-dependent resetting — of parasystole A.
Panel A: Numbers above the continuous curves are A-A interectopic intervals. QRS complexes marked by circles represent beats that reset the parasystole.
Panel B: Large circles in the stippled area represent A-R intervals of non-resetting and small circles in the clear area indicate those of the resetting beats. Circle crossed by X represents the coupling time of a ventricular extrasystole (ES) to the preceding beat A.

FIGURE 3. Panel A: Diagrammatic representation of the suggested mechanism to the last strip of figure 2.
Panel B: Successive resettings of parasystole A. Horizontal shaded bars represent protection of parasystole A, with intermittent interruptions, and the stippled bar represents protection of parasystole B. Full circles indicate spontaneous firing of the parasystoles. Numbers above the curves show interectopic intervals of parasystole A.
FIGURE 4. Irregular double ventricular parasystole. Interectopic intervals of parasystole A are given above, those of parasystole B, below the continuous curves. Two arrows indicate the shortest interectopic interval of parasystole B.

Panel A: Schematic representation of the proposed mechanism for irregularity of parasystole B ("supernormal" exit conduction). Numbers above the dots represent cycle lengths of the parasystolic firing. Obliquity of arrows emerging from the focus indicate proportionate values of exit times. Numbers between the arrows show manifest interectopic intervals of parasystole B.

Panel B: Coupling times of focus activities in parasystole B (B₁) to beats A and to sinus beats (R) from a continuous recording including strips in figures 4 and 5A. Small circles represent coupling times associated with prolonged exit conduction time and large circles in the shaded area indicate those with enhanced exit conduction.
charges of parasystole B \( (B_r) \) to the previous beats A and sinus beats \( (R) \) revealed the following: (fig. 5B) Whenever the firing of parasystole B was preceded by a QRS A with 48, shortening of the corresponding B-B interval occurred. Such a reduction was never noted when parasystole B was preceded by a sinus beat. These observations raise the possibility of summation underlying enhanced exit conduction from parasystole B. For spacial summation to occur, there should be a particular time relationship between two impulses from particular sites of origin in a depressed area. In our case, these criteria were fulfilled by impulses A and B, causing intermittent relief of exit conduction delay in fibers surrounding parasystole B.

To further support “supernormal” exit conduction, it is essential to rule out reentry as a mechanism responsible for the occasional premature appearance of beats B. It was noted that coupling times of beats A not causing resetting of parasystole B exceeded those coupling times of beats A to beats B preceding early complexes B (figures 4 and 5A). This observation is against premature beats B being of reentrant origin, as reentrant extrasystoles during parasystole from distant sites are usually elicited by beats falling at the boundaries of refractori-ness of the parasystolic structure. Moreover, a reentrant extrasystole which resets the parasystole would be expected to cause shortening of the subsequent interectopic interval, as such beats manifest after significant delay in the reentry loop. In figures 4 and 5A the interectopic interval following the shortest ones exceeded the other interectopic intervals also comprising three cycles, and were not shorter than three manifest cycle lengths.

The irregularity of parasystole B can be best explained by “pseudointermittence” of a continuous parasystole caused by “supernormal” exit conduction. The marked occasional acceleration of the emerging impulse involves significant basic conduction delay (first grade exit block) as indicated by the more oblique lines in the diagram (fig. 5A).

In figures 4 and 5A, intermittent parasystole A with a critical resetting interval of about 90 and several successive resettings are present. Only infrequent active parasystolic firings are seen.

The continuous ECG tracings reproduced in figure 6 were selected from the ECG obtained on admission. Both types A and B parasystole are very regular. Parasystolic cycle lengths of parasystole A are: 156, 156, 156, 5 × 154, 160, 4 × 154, 160 and 7 × 156; those of parasystole B are: 3 × 154, 160, 6 × 155, 158, 5 × 156, 160 and 3 × 158. B, opening the first, and both A and B, closing the last interectopic intervals, are not shown for technical reasons.

Intensive horizontal lines above and below the ECG tracings represent coupling times of manifest parasystole A and B, respectively, whereas thin horizontal
FIGURE 7. Intermittent parasystole A (with interectopic intervals above the continuous curves) and regular parasystole B (with cycle lengths and coupling times below the recordings). All symbols above the continuous curves are for parasystole A, below the tracings for parasystole B. Note that lengthening of the parasystolic cycle B did not alter the critical coupling time of manifestation.

lines above and below the tracings represent coupling times of calculated parasystolic activities of A and B, respectively. Scanning of the long interectopic intervals reveals that the expected discharges of parasystole A and parasystole B are never inscribed if they fall within 56 and 52 after the preceding QRS complexes, respectively. With coupling times of 60 and 64, the respective parasystolic beats can already appear. Manifestation of parasystole B is also coupling time-dependent at a cycle length of about 188 (fig. 7). Decrease in parasystolic rate had no influence on the critical time of emergence from the parasystolic focus into the ventricles. In figure 7, this critical time is between 68–56.

Duration of refractoriness of the ventricles is thought to be delineated by determining the longest latent and/or the shortest manifest coupling of regular parasystolic beats. In our case, however, ventricular extrasystoles were observed with coupling times ranging from 44–52, as shown at the end of the tracings in figure 6 and in figure 8. These extrasystoles are not likely to contain parasystolic beats arising in focus A, as they do not correspond to calculated parasystolic activities. Loosely speaking, this means that para-

FIGURE 8. Coupling times of ventricular extrasystoles from selected tracings. Panels A and B are not continuous with each other.
systolic impulses must have encountered refractoriness at least 120 msec greater compared to the extrasystolic ones. This observation may be explained by depressed parasystolic surroundings compared to the ventricles and/or by divergence in stimulus strength of the parasystole and extrasystole.

To be sure that coupling time-dependent disappearance of the parasystole is caused by the presence of tissues refractory to the parasystolic impulses, it is essential to prove rate-independent entrance block of the parasystolic focus. Figure 9 demonstrates stability of protection (non-resetting) of parasystole B. Panels A and B are selected parts of one long rhythm strip. Strips comprising panel B are continuous. Manifest and calculated cycle lengths of a regular parasystole B are shown in the first row below the ECG tracings. Whenever coupling time of parasystole B is within a critical value, parasystole B does not appear. Presuming that QRS complexes (marked by an arrow), appearing at long distances after parasystolic activity, reset the parasystolic pacemaker, the estimated sum of exit and entrance conduction times would vary between 26–60, as can be calculated from the intervals between the supposedly resetting QRS complexes (marked by an arrow) and the next beats B. Such a variability in the sum of entrance and exit times of the parasystole does not seem probable. Moreover, the coupling time of the presumably resetting QRS complex plus the estimated sum of entrance and exit conduction times are always the same as the given calculated parasystolic cycle length. Such a congruence may exist with fixed coupling times, but is practically impossible if the coupling times of the resetting QRS complexes are variable. Accordingly, parasystole B does not seem to reset.

The coupling times of QRS complexes not followed by beats A did not show such a variability at times when parasystole A was regular (figs. 1 and 6). Therefore, non-resetting of parasystole A cannot be proven, despite its regularity.

Discussion

Entrance Block

The theory of entrance block for the interpretation of parasystole has been confirmed by microelectrode technics. This protective block inhibits extraneous impulses in disturbing the regular rhythm of the parasystolic pacemaker. The possibility of temporary loss of protection was postulated by Kaufmann and Rohtberger as early as 1920, and later by Katz and Pick, Watanabe, and Steffens. Cohen and coworkers proved that impulses at given phases of the parasystolic cycle may discharge and reset the parasystolic focus. According to their opinion, this time interval is determined by and falls between refractoriness and phase 4 depolarization of the parasystolic center and/or of the surrounding tissues. Wenckebach type and 2:1 entrance block was demonstrated by Kinoshita in intermittent ventricular parasystole. Stimuli arriving at the supernormal phase of the parasystolic structures are also thought to be able to penetrate into and reset the parasystolic focus, as was demonstrated by Scherf and Boyd and by Hiejima and Poh.

In our case of double irregular ventricular para-
systole, entrance block was complete only in one of the two parasystoles (B), and was rate-dependent in the other one (A). A characteristic feature of the latter was the appearance of successive passive depolarizations of the parasystolic center as shown in the diagram of figure 3B. When transiently active and passive parasystolic discharges alternated, a seeming temporary regularity of this intermittent parasystole occurred (fig. 2A).

Some features of intermittent parasystole based on rate-dependent entrance block were critically analyzed by Kinoshita. He gave a possible explanation for the shortening of cycle lengths calculated from long interectopic intervals, and for entrance block of relatively late stimuli. He established that resetting of the parasystole is not necessarily accompanied by irregularity. Whenever the coupling time of the resetting QRS plus the entrance and exit conduction times of the parasystole precisely or nearly equal the parasystolic cycle length, the parasystole will be regular but not simple (continuous). On the other hand, we believe that if the differences between manifest and calculated cycle lengths do not exceed that accepted for simple parasystole, and if the coupling times of QRS complexes not followed by parasystolic QRS are within a narrow range, the disappearance of parasystolic beats may be explained either by resetting of the parasystolic focus or by refractoriness of the parasystolic surroundings or of the ventricles (see parasystole A in figs. 1 and 6). Accordingly, in most of the published cases of intermittent (phase 3) parasystole, the exact differentiation between these two alternatives is not possible, and the suggested resetting of the parasystole does appear to be proven. The spontaneous or induced changes in coupling times of the apparently resetting beats may aid in the differentiation. If the alterations in coupling times of the presumably resetting QRS complexes are accompanied by similar changes in the corresponding interectopic intervals, intermittent parasystole is most likely present, even when only small deviations from regularity are seen. If the changes of coupling times of the presumably resetting QRS complexes have no influence on the corresponding interectopic intervals, as shown for parasystole B in our case (fig. 9), the possibility of resetting can practically be excluded.

Exit Block

Exit block in parasystole is no longer a hypothetical postulate, and has gained a firm experimental basis. If non-propagation of a parasystolic impulse is not due to ventricular refractoriness, exit block is present. Among the possible conduction patterns of the emerging impulse, Mobitz I and Mobitz II-type exit block and Mobitz II-type exit block and manifestation of a subthreshold impulse due to supernormal excitability of the ventricles have been described in clinical electrocardiography. In this case, rate-dependent block in the exit pathway (figs. 6–8), first degree exit block (fig. 5A), and “supernormal” exit conduction (figs. 4 and 5A) in one of the parasystoles (B), and a possible phase 3 exit block (fig. 6) in the other one (A) could be postulated.

The occasional premature appearance of parasystole B in figures 4 and 5A is not a result of supernormal excitability (unexpected propagation of a subthreshold stimulus), but may be related to supernormal conduction velocity of the emerging impulse. Since the apparent shortening in cycle length is closely related to a critical A and focus B relationship, the acceleration of the emerging impulse may probably be due to a summation of the two stimuli, resulting in enhanced conductivity in the surroundings of the parasystolic center. The role of summation in development of a parasystolic rhythm has been disclosed by Cranefield and associates. The mechanism of summation in our case did not play a role in the development, but it could be related to the peculiar arrangement of parasystole B.

It has been held that first grade exit block from a parasystolic focus was unrecognizable in clinical electrocardiography. But regular discharge of the parasystolic pacemaker with the demonstrated temporary acceleration of the emerging impulse of parasystole B could only occur with a prolonged basic exit conduction time (first degree exit block). Thus, it seems that there may be specific electrophysiological constellations which provide an opportunity to deduce a simple delay in exit conduction from the parasystole. In rate-dependent intermittent parasystole, the sum of entrance and exit conduction times may also be approximated by the parasystolic escape interval lengthening after a resetting beat, similar to the method used for the estimation of sinoatrial conduction times. The minimal basic exit time of parasystole B in our case exceeds this calculated joint value of parasystole A.

The observation that stimuli from extrasystolic foci capture the ventricles at shorter coupling times than those from the parasystolic centers can be explained by alternative mechanisms. More depressed parasystolic than extrasystolic surroundings — the latter may be the myocardium itself — may be responsible for the observed difference in ventricular excitability depending on the site of stimuli. It follows that disappearance of a parasystolic QRS may be due to rate-dependent (phase 3) exit block and may refer to electrophysiological heterogeneity in the parasystolic structures. An impulse not discharging the parasystolic focus, but influencing the emerging impulse, suggests at least two levels of the parasystolic structure. If the alternative mechanism works, a weaker parasystolic than extrasystolic impulse may also cause manifest ventricular depolarization, but only in later phases of ventricular repolarization.

References

16. Fleming GB: Triple rhythm of the heart due to ventricular extrasystoles. Q J Med 5: 318, 1912
Double irregular ventricular parasystole: rate-dependent entrance block and "supernormal" exit conduction.
J Tenczer and L Littmann

Circulation. 1978;58:723-731
doi: 10.1161/01.CIR.58.4.723

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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/58/4/723

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/