Mechanisms of Intermittent Ventricular Bigeminy

I. Appearance of Ectopic Beats Dependent Upon Length of the Ventricular Cycle, the “Rule of Bigeminy”

By R. Langendorf, M.D., A. Pick, M.D., and M. Winternitz, M.D.†

In order to analyze the cause for the appearance and disappearance of ventricular premature systoles giving rise to temporary bigeminal rhythm, the electrocardiographic material was divided into two major groups: group A, consisting of ectopic beats with fixed coupling occurring during grossly irregular dominant rhythms, and group B, consisting of ectopic beats with varying coupling occurring during regular dominant rhythms. Analysis of group A, the subject of the present report, shows a definite relationship between the duration of the ventricular cycle and the occurrence of ventricular premature systoles, in that long cycles favor their appearance whereas short cycles tend to preclude it. This “rule of bigeminy” is best explained on the basis of a re-entry mechanism of the ventricular premature systoles. Criteria are given for the differential diagnosis between ventricular premature systoles and aberrant ventricular conduction of supraventricular impulses which is prone to occur under similar circumstances. The analysis of group B is presented in a subsequent report.

BIGEMINY, as a descriptive term of cardiac irregularity refers to a continuous alternation of short and long cardiac cycles, corresponding to the phenomenon of pulsus bigeminus diagnosed at the bedside on palpation of the radial pulse. Such a grouping of ventricular beats may be the result of a number of different mechanisms involving a disturbance of impulse formation or impulse conduction, or a combination of both; an unusual example of the latter as a result of reciprocal beating was presented in a previous report from this department.3

The present report is confined to an investigation of the various mechanisms operating in the most common variety, intermittent bigeminy due to ventricular premature systoles. For many years we have been impressed by the observation that in auricular fibrillation the occurrence of ectopic ventricular beats causing bigeminy seems to bear a certain relation to the duration of the ventricular cycle, a fact which has received only scarce attention in the literature.3, 4 A different group of cases in which intermittence of ventricular bigeminy could be ascribed to a re-entry mechanism was described from this laboratory.5 Since our attention has been focused on the problem, a third group of cases caused by a parasystolic mechanism was recognized. It seemed to us that the various circumstances under which ventricular bigeminy starts and ends might have wider implications and that the understanding of the factors responsible for initiation and termination of ventricular bigeminy may contribute to the clarification of ectopic premature systoles in general. The first part of our study deals with the factor of ventricular cycle length influencing the appearance and disappearance of ventricular premature systoles. Other causes of intermittence of ventricular bigeminy as revealed in the course of the analysis of the material form the basis of a subsequent paper.

Material and Methods

Thirty-one examples of intermittent ventricular bigeminy caused by ventricular premature systoles were selected from the electrocardiographic files of the Heart Station, the only criterion for selection being a sufficiently long recording to permit a de-
Fig. 1. *Intermittent ventricular bigeminy caused by ventricular premature systoles in auricular fibrillation.* The three strips are portions of a long record in lead II. The conventions in the diagram below the record are as follows: The scale in the center gives the range (in hundredths of a second) of all R-R intervals terminated by conducted (supraventricular beats) in the entire tracing. Those R-R intervals succeeded by a ventricular premature systole are plotted as dots to the right of the scale; those R-R intervals not followed by a premature systole appear as circles on the left of the scale.

Auricular fibrillation is diagnosed in the usual way. In all three strips the sequence of supraventricular beats (average rate 85) is temporarily replaced by bigeminy due to ventricular premature systoles with fixed coupling and uniform contour (except for one beat labeled Vb). Bigeminy starts when the R-R between two conducted beats lengthens; it continues as long as the pause after the premature systole is long; and it ends when this pause shortens. Thus, in A, bigeminy consists of a short run, in B it continues to the end of the strip and in C it terminates with the first premature beat. The beat labeled V4 is a variety of the same phenomenon. Subsequent to a long R-R, an ectopic beat appears at the same coupling as the others, but is intermediate in contour between the dominant and the bizarre ectopic beats. This is a ventricular fusion beat caused by ventricular interference of the ectopic ventricular impulse (occurring because of the long pause) and a conducted auricular impulse which crossed the A-V junction at the same time that the ectopic impulse was released.

The diagram reveals that premature systoles occurred whenever an R-R interval became longer than 0.70 second, and failed to appear when the R-R was shorter than 0.60 second. Within a small range of R-R between 0.60 and 0.70 second, the presence or absence of premature systoles had about the same frequency. Thus, the dependence of bigeminy upon the duration of the cardiac cycle is clearly revealed.

tailed study. No attempt was made at an exhaustive survey of the many records exhibiting this type of arrhythmia. It soon became obvious that on the basis of the spacing of the dominant and ectopic beats the material could be divided into two main groups, namely, (A) a group of 17 records from 14 patients in which the coupling of the premature systoles was practically fixed and the dominant rhythm was grossly irregular, the cases considered in this paper, and, (B) a group of 14 records from 12 patients in which the coupling of the premature systoles varied but the dominant rhythm was practically regular, the subject of a second communication.

In group A, the irregular ventricular beating had been recorded in long strips. The duration of each ventricular cycle was carefully measured. The various R-R intervals encountered in each case, except the fixed coupling of the ectopic beats, were charted in relation to the occurrence or the absence of a subsequent ectopic beat. Representative examples of such electrocardiograms and graphs are reproduced in figures 1, 2, 3 and 4, and details of the analysis are presented in the respective legends.

**Results**

In 16 of the records, the ventricular irregularity was marked, in 15 caused by auricular
The presence of auricular fibrillation is suggested by the irregular undulations of the baseline and was clearly seen in numerous preceding and subsequent records. In the record shown, there is complete A-V dissociation, the ventricles being under the command of an accelerated nodal pacemaker; temporarily its action is disturbed by ectopic ventricular premature systoles (the bizarre beats) appearing after every nodal beat (ventricular bigeminy). The sequence of nodal beats is otherwise regular (R-R equals 0.74 second, corresponding to a rate of 81), except for the beat ahead of the first ventricular premature systole in the bottom strip; here, the nodal interval lengthens to 0.80 second. The premature ventricular beats show some variation in contour and have with one exception (see below) a constant coupling (0.54 second) to the nodal beats. Their distance to the subsequent nodal beats is 0.94 second, 0.20 second longer than the interval between two consecutive nodal beats. This is due to (concealed) retrograde discharge of the A-V nodal pacemaker by the ectopic ventricular impulse, and possibly to some depression of the nodal pacemaker consequent to this premature extraneous discharge. Thus, at the time of ventricular bigeminy, A-V dissociation persists as in the other parts of the tracing. The exception to be noted concerns the first premature beat in the bottom strip. Its coupling is shorter (0.40 second) and the subsequent nodal interval is longer (1.04 second) than elsewhere. The shortening of the coupling can be ascribed to recovery of impulse conduction in a re-entry path; the prolongation of the nodal interval can be ascribed to a delay of retrograde transmission of the ectopic impulse to the nodal pacemaker, since it occurred earlier in the cycle.

An analysis of the entire tracing, summarized in the diagram, shows that the A-V dissociation was not complete, occasional conducted auricular impulses being represented by the short R-R intervals. The diagram also shows slight irregularity of the nodal pacemaker with variations in its cycle by ±0.05 second. Finally, the diagram shows clearly the relationship of appearance of ectopic beats to the length of the cycle. With a few exceptions, long ventricular intervals are followed by an ectopic premature beat, whereas short ones appear to preclude its appearance. As seen in the bottom strip of the tracing, even slight spontaneous prolongation of the nodal interval is sufficient to initiate a bigeminus, with subsequent self-perpetuation of bigeminy on account of the long ventricular intervals following each premature beat. This continues until a premature beat fails to appear (upper strip) and the original short nodal interval is restored, which in turn prevents the reappearance of the ectopic beats (middle strip).
fibrillation (figs. 1 and 3), in one by a sinus arrhythmia (fig. 4). One case of auricular fibrillation (fig. 2) showed less pronounced variations in ventricular cycle length due to the presence of incomplete A-V dissociation with a slightly irregular nodal pacemaker. In no case did the coupling of the ectopic beats vary by more than 0.08 second. The intervals separating the ectopic beats were neither equal nor reducible to a common denominator. The irregularity of the interectopic intervals followed the irregularity of the fundamental rhythm and revealed in no instance an arrangement characteristic of Wenckebach periods.6,7 Thus, the possibility of a parasystolic mechanism, without or with exit block, could be ruled out in each case.

In all records a striking consistency was found in that ectopic beats occurred only after long ventricular cycles, and failed to do so when the cycle was short. In fact, a dividing line could be drawn in the ventricular cycle lengths in relation to whether or not an ectopic beat appeared (see charts in figs. 1 to 4). However, exceptions occurred mainly in the range of the longer cycles. Actually, in some cases a considerable overlap was found in that many long R-R intervals failed to be followed by an ectopic impulse. The significance of these findings will be discussed below.

**Comment**

The separation of a certain number of cases with intermittent ventricular bigeminy into a single group was based upon two common findings, the gross irregularity of the dominant rhythm and the fixed coupling of the ectopic beats. Such a combination presents strong support for the concept that premature systoles with precisely uniform coupling are in some way related to, and initiated by, the beat to which they are coupled. While this viewpoint has found wide acceptance in contrast to other past and present viewpoints,8,9 no agreement has been reached as to the actual mechanism in operation. Based on experimental evidence, supported by data of neurophysiologic investigation, the theory has been advanced that cardiac tissue may under certain circumstances yield a repetitive response to a single stimulus.10 In contrast to this assumption stands the theory of a re-entry mechanism11,12 presupposing an area of unidirectional block in the periphery of the Purkinje tissue. When such a local impedance to impulse transmission develops, the cardiac impulse, unable to pass through from one direction may reach, enter and traverse it in a reversed direction and restimulate the chamber involved; the result being a premature systole.

The fact, as shown in this report, that the occurrence of a premature systole may be a function of cycle length is hard to reconcile with any other explanation but the re-entry concept. Any alteration in cardiac rhythm which can be shown to depend on the duration and variation of the cardiac cycle, can be viewed in the light of refractoriness and recovery of cardiac tissue. The re-entry concept,
because it implies a local disturbance of conductivity, can readily be applied to the explanation of the phenomenon under discussion. It would appear that either the pathway leading to the region of unidirectional block (causing the re-entry) is open only after a certain period of rest, or that the region of block itself becomes passable in the reverse direction only after a certain period of rest. The latter assumption is the more likely one since the Purkinje network undoubtedly provides a number of avenues leading to the region of block. Instead of postulating depressed conductivity in all of them, it is simpler to assume that there is but one region of block which is bidirectional at a fast rate, and becomes unidirectional at a slower rate of stimulation. In this way the occurrence of ventricular premature systoles which are dependent upon the length of the cycle can be explained on the assumption of a single region of unidirectional block in the periphery of the conduction system permitting retrograde propagation of the impulse, but only after a long ventricular pause.

Once ventricular bigeminy is initiated in this matter, it tends to persist because of the long pause which follows each premature systole. This pause in auricular fibrillation may be accounted for on the same basis as the ordinary compensatory pause after premature systoles during sinus rhythm, that is, by A-V interference engendered by retrograde transmission of the ectopic ventricular impulse, which
renders the A-V junction temporarily refractory to auricular impulses. Bigeminy usually is stopped when a fibrillation impulse succeeds in traversing the A-V junction at an earlier time and abbreviates this pause. Thus, perpetuation of bigeminy follows the same rule as its initiation.

Exceptions to this "rule of bigeminy" occurred and consisted mainly in the absence of ventricular premature beats after long ventricular cycles. However, this failure of premature systoles to occur when expected may be only an apparent exception. It is possible that on occasion, subsequent to a longer ventricular pause, the impulse may penetrate, as do the others, into the re-entry pathway, but be stopped within the path, or fail to fan out to the rest of the chamber after completion of the re-entry sweep. In either event the ordinary electrical manifestation and consequence of re-entry, a premature systole, will be absent. In support of the assumption of such a "concealed", completed or attempted re-entry are observations of a similar mechanism in reciprocal beating and the unexpected prolongation of the first coupling in instances in which intermittence of ventricular bigeminy can be ascribed to a progressive block within the re-entry pathway, as will be shown in our subsequent paper.\(^7\)

In one instance in which the operation of the "rule of bigeminy" was observed during sinus arrhythmia (fig. 4), the fixed coupling was long and on occasion approached the duration of the variable sinus cycle. This led to competition of the sinus and ectopic impulse for the control of the ventricles resulting in ventricular fusion beats. The fact of a long coupling cannot be used as evidence against a re-entry mechanism, since two or more re-entry sweeps may occur before the impulse spreads out to activate the ventricles.\(^13\) Thus, the occurrence of fusion beats does not always rule against re-entry and in favor of parasystole, just as their absence does not exclude parasystole.\(^14\)

When early beats with bizarre contour and

![Fig. 5. A case of auricular fibrillation in which aberrant ventricular conduction of supraventricular impulses imitates the occurrence of ventricular premature systoles related to cycle length. The ventricular rate is rapid and irregular (130 in the average). Once in lead II, and twice in lead CF₂, ventricular complexes have a prolonged QRS and a contour of a right-sided intraventricular block. In all three instances these beats terminate a short cycle which succeeds an exceptionally long one. The former are of unequal length and not the shortest in the record. This exemplifies that aberrant ventricular conduction, common in cases with rapid heart action is not merely a function of rate, but largely depends on its sudden variations as the refractory phase of the ventricular conduction system lengthens with lengthening of the cycle. Hence, when a short cycle follows a long one, the beat terminating the short cycle tends to show aberrant ventricular conduction.](image)
prolonged QRS occur in auricular fibrillation (or auricular flutter with irregular ventricular response) it is of considerable practical importance to distinguish between ectopic ventricular premature systoles and aberrant ventricular conduction of early supraventricular impulses. Such a distinction may sometimes be difficult since under both circumstances the resulting bizarre beat tends to be “coupled” to a beat terminating a long cycle (fig. 5). Where the occurrence of an ectopic premature systole in the wake of a long ventricular pause can be ascribed, as outlined above, to partial recovery of conductivity in a re-entry path, aberrant ventricular conduction occurring under such circumstances is explained by impairment of conductivity in ordinary ventricular conduction pathways caused by prolongation of the normal ventricular refractory phase concomitant with the lengthening of the cardiac cycle. Usually it is the right sided bundle-branch system which is affected by this mechanism.  

The following three criteria in most instances should permit the correct interpretation of early bizarre beats during irregular ventricular beating. (a) Beats of ectopic origin tend to have a fixed coupling while the short R-R interval of aberrant ventricular conduction tends to vary in a wider range. (b) Aberrant ventricular complexes almost invariably show a pattern of right bundle-branch system block, with QRS prolonged in its terminal portion in contrast to ectopic beats which show a variety of bizarre-ness, with QRS widened throughout. (c) Unlike premature beats of ectopic origin, aberrant ventricular beats do not give rise to a “compensatory” pause, and it is the absence of such a pause which prevents continuation of aberrant conduction in the form of bigeminy. Aberrant ventricular conduction may, however, continue in the form of consecutive rapid beats with a similar bizarre contour and prolonged QRS duration and thus imitate ventricular paroxysmal tachycardia.  

A search of the literature reveals a number of cases with intermittent ventricular bigeminy in which appearance, perpetuation and disappearance of ectopic premature beats follow the stated “rule of bigeminy.” The role of the ventricular cycle length is seen in a unique case of Scherf and Schott showing, in auricular fibrillation, parasystole and coupled ventricular premature systoles, the latter occurring exclusively after the long cycles of the parasystolic beats; the only instance of failure of a premature ventricular systole to appear was in a parasystolic beat with a short R-R interval. Likewise, in a case of Holzmann showing a ventricular parasystole and, in addition, ectopic beats with fixed coupling giving rise to temporary bigeminy, the latter was interrupted when the cycle length shortened. A similar mechanism prevails in a case reported by Rachmilewitz and Scherf illustrating in figure 374 an unusual case of sinus bradycardia and arrhythmia with bigeminy caused by interpolated ventricular premature systoles; intermission of the bigeminy coincides with the shortest ventricular cycle. Figures 111 and 441 are more common examples of the “rule of bigeminy” in auricular fibrillation.

**Summary and Conclusions**

1. An analysis is presented of selected electrocardiograms with intermittent bigeminy caused by ectopic impulse formation in the ventricles. The material could be divided into two groups. (A) Cases with fixed coupling of the ectopic beats in the presence of grossly irregular ventricular beating. (B) Cases with variable coupling of the ectopic beats in the presence of otherwise regular ventricular beating. Group A forms the basis of this report.

2. In the presence of irregular ventricular beating the appearance of ventricular premature systoles with fixed coupling, their continuation in the form of bigeminy, and the termination of the latter, all tend to depend on the duration of the cycle of the beat to which the ectopic beat is coupled. Lengthening of the ventricular cycle favors the appearance of ventricular premature systoles. The term “rule of bigeminy” is proposed as a short designation of this phenomenon.

3. An adequate explanation of this “rule of bigeminy” can be based on the concept of a re-entry mechanism. Conversely, the existence
of such a rule lends strong support to the view that re-entry is the mechanism responsible for premature systoles with fixed coupling in general.

4. Whenever there is a rapid and irregular response to supraventricular impulses—in auricular fibrillation in particular—a beat terminating a short cycle subsequent to a long one tends to exhibit a bizarre contour and QRS prolongation, caused by aberrant ventricular conduction. Criteria are presented for the differential diagnosis between such aberrant supraventricular beats and ectopic beats of ventricular origin.

5. Cases of intermittent bigeminia of other groups listed under (B) will be the subject of a subsequent report.

**Summary e Conclusiones in Interlingua**

1. Es presentate un analyse de seligite electrocardiogrammas obtenite ab casos de intermitente bigeminia causate per un formation ectopic de impulsos in le ventriculos. Le material es classificabile in duo gruppas. Le prime gruppus include le casos de fixe accopulamento del pulso ectopic in le presentia de grossier irregularitades del pulsation ventricular. Le secunde gruppus consiste de casos de variabile accopulamento del pulso ectopic in le presentia de un pulsation ventricular sin alte irregularitades. Le prime de iste gruppas es tractate in le presente reporto.

2. In le presentia de irregularitades del pulsation ventricular, le apparition del prematur systoles ventricular a fixe accopulamento, le continuation de iste systoles in le forma de bigeminia, e le terminasion del bigeminia, onne iste eventos inclina a depender del duration del cyclo del pulso con que le pulso ectopic es accopulate. Le allongamento del cyclo ventricular tende a coincider con le apparition de prematur systoles ventricular. Nos propone le termino “regula del bigeminia” como designation abbreviate de iste phenomenon.

3. Un adequate explication del “regula del bigeminia” pote esser basate super un mecanismo de re-entrata. Reciprocamente, le existentia de un tal regula supporta le concep-

4. Quandoque il ha irregular e rapide responsas a impulsos supraventricular—specialmente in fibrillation auricular—un pulso al fin de un breve cyclo que seque un longe cyclo tende a exhibir un bizarre contorno e un prolongation de QRS in consequentia de un aberrante conduction ventricular. Nos presenta criterios pro le distinction diagnostico de tal aberrante pulso supraventricular e pulsos ectopic de origine ventricular.

5. Casos de intermittente bigeminia con un rhythm dominante regular e un variabile accopulamento del pulsos ectopic va esser tractate in un reporto separate.

**References**

MECHANISMS OF INTERMITTENT VENTRICULAR BIGEMINY


