Wenckebach (Type 1) Behavior of Ventricular Reentry*

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Electrocardiographic excerpts from three patients demonstrate bigeminy, concealed bigeminy, trigeminy, concealed trigeminy, and Wenckebach's pattern of variation in the coupling interval from sinus beat to ventricular ectopic beat. These diverse manifestations of ventricular ectopy support the hypothesis that reentry in man reflects conduction of an initial impulse, with delay, to cause the coupled reentrant beat.

Reentry is considered to be a generic mechanism by which a wide diversity of abnormal rhythms occur. Among these are the tachyarrhythmias of the syndromes of preexcitation1 and other paroxysmal atrial, junctional, or ventricular tachyarrhythmias.2 Many hypotheses have been put forth to explain the process of reentry. These include those which presume disorders of impulse propagation,3 those which reflect pacemaker-like activity of cardiac cells,4 and those which invoke excitation of or by cells adjacent to injured myocardium.5 The conductive mechanism of reentry has been modeled on theoretic grounds,6 produced in preparations of isolated tissue,7 and deduced to be present in man.8 Ventricular premature beats may occur as a sporadic unexplained event, as a consequence of parasystolic activity, or with an apparent cause-effect relationship to the preceding beat. The latter phenomenon, a fixed coupling interval, has been taken as a hallmark of reentry caused by abnormal conduction; however, a fixed coupling interval has also been cited as evidence of reexcitation of or by damaged neighboring fibers9 and as evidence of extrasystolic enhancement.8 Schamroth10 has particularly championed the notion that most, if not all, ventricular ectopic rhythms reflect ectopic enhancement of latent pacemaking cells, rather than a disorder of impulse propagation.

Schamroth and Marriott10 originally described an interesting pattern of intermittent ventricular premature beats. They spoke of the phenomenon as concealed extrasystoles. These investigators postulated intermittent facilitation of an extrasystolic pacemaking focus as the mechanism. Subsequently, Levy and co-workers11,12 have described similar in-

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Manuscript received September 28; accepted October 12.
and no other abnormalities. Routine laboratory studies confirmed the presence of an infection of the urinary tract with *Escherichia coli*.

An ECG demonstrated normal sinus rhythm, normal QRS-T complexes, and frequent ventricular premature ectopic beats. During ten minutes of electrocardiographic recording, two temporal patterns of ventricular premature beats occurred. The QRS morphology of all ventricular premature beats was similar, suggesting a single focus of origin within the ventricles. The first temporal pattern persisted for more than one minute on two occasions. This consisted of sinus rhythm with the P-P interval varying between 760 and 810 msec and a constant bigeminal pattern. The coupling interval between the sinus beat and the ventricular ectopic beat was 440 msec in one series and 480 msec in the second series. The remainder of the ten minutes of sinus rhythm was interrupted by 24 episodes analogous to those illustrated in Figures 3 and 4.

**CASE 3**

A 46-year-old man entered the hospital for evaluation of angina pectoris produced by moderate effort and relieved by rest. The angina occurred several times each week, despite therapy with isosorbide dinitrate and propranolol hydro-
chloride.

Electrocardiographic studies demonstrated a normal tracing at rest, 2 mm of flat ST-segment depression of the inferior wall in leads 2, aVF, and 3 during stress testing, and intermittent periods of bigeminal rhythm related to unifocal ventricular premature ectopic beats. A representative strip taken from lead 2 is displayed in Figure 5.

**Analysis of ECGs and Discussion**

Each figure includes a ladder diagram indicating coupling intervals, interectopic intervals, and postulated participation of a reentrant pathway. Figures 1 and 2 show leads 1, 2, and 3 recorded simultaneously; Figures 3 to 5 show continuous strips of lead 2.

Strips recorded from case 1 demonstrate normal sinus rhythm and ventricular premature beats. The ventricular premature beats appear to be dissociated from and usually obscuring the sinus-initiated P waves. The sinus rate is approximately 72 beats per minute, with the minor variation of sinus arrhythmia. Ventricular premature beats occur as the second, fourth, sixth, eighth, and 13th complexes in Figure 1. The interectopic intervals of 1,730, 1,730, 1,760, and 4,040 msec do not lend themselves to a parasystolic model; the 4,040-msec interval is neither a multiple of 1,730 msec, of 1,760 msec, nor of these intervals divided by an integer. Moreover, the interval of 1,760 msec before the 13th complex does
not lend itself to a hypothesis that a parasystolic focus was "reset" either by the tenth, 11th, or 12th complex.

Alternatively, it is attractive to postulate that the initial coupling interval of 580 msec represents successful propagation of the first sinus complex through an intraventricular zone of slow conduction. This postulated pathway of impulse propagation is represented as a reentrant loop. The fourth complex also represents an impulse initiated by the antecedent sinus complex; however, the coupling interval has increased to 610 msec. The sixth complex couples with an interval of 630 msec and the eighth with an interval of 750 msec. The eighth coupling interval is sufficiently long to permit the sinus impulse to traverse the atrioventricular junctional system and to produce a fusion beat. The ladder diagram indicates speculation that the ninth complex initiates an impulse into the reentrant circuit, but that sufficient delay is encountered in the circuit to prevent evident ectopic activity. This presumed block is analogous to the "concealed" extrasystoles reported by others.8,11,12 Whether an impulse enters the postulated reentrant loop during the tenth or 11th complex is pure speculation and is not indicated on the ladder diagram. The 12th complex is followed by an ectopic beat with a coupling interval similar to that which precedes the sixth complex.

Figure 2, taken about one minute after Figure 1, shows a slower sinus rate, approximately 67 beats per minute. The interectopic intervals have also increased over those in Figure 1; between the second and fourth complexes, it is 1,930 msec, and between the fourth and sixth complexes, it is 1,910 msec. The interval between the sixth complex and the 11th complex is 4,840 msec, an interval which is not a multiple of the other interectopic intervals. The interectopic intervals in Figures 1 and 2 bear a close relationship to the prevailing sinus rhythm. Despite the patterned variation in the coupling between sinus beat and ectopic beat, the rhythms in case 1 do not fit a parasystolic model.

The coupling interval between the preceding sinus beat and the ventricular ectopic beat in the first three couplets of Figure 2 progressively increases from 520 to 640 msec. The ladder diagram indicates the postulate that impulse penetration through a reentrant circuit after the seventh complex is blocked. The presumed reentrant circuit again becomes visibly operative after the tenth complex. With slight variations in the basic sinus rate, it becomes difficult to apply a rigid Wenckebach model12 to this tracing; however, the cou-
pling interval preceding the second ventricular ectopic beat increases by 90 msec to 610 msec, compared with that before the first ventricular ectopic beat. The third coupling interval increases by an additional 30 msec to 640 msec. The coupling interval after the tenth complex shortens again to restart the sequence. These observations support the hypothesis of Wenckebach's pattern of impulse propagation within a reentrant ventricular pathway of conduction. Successful traversal of the pathway produces ventricular premature ectopic beats; successive traversals encounter progressive delay in conduction until a failure in conduction occurs.

Figures 3 and 4 show moderate sinus arrhythmia. Each has ventricular ectopic premature beats which appear to dissociate from the sinus P waves, as indicated on the ladder diagram. The sinus rate varies between approximately 68 and 88 beats per minute in Figure 3 and between approximately 75 and 88 beats per minute in Figure 4. The interectopic intervals follow no predictable pattern, and neither suggest sustained parasystole nor a parasystolic focus "reset" by an intervening sinus beat.

In Figure 3 the coupling interval after the third, the fifth, and the seventh QRS complexes increases progressively with increments of 40 and 20 msec; an ectopic premature beat does not occur after the ninth complex. A single premature beat occurs as the 13th complex. The bottom strip shows two ectopic beats coupled at 490 and 560 msec, respectively. The fifth complex is not followed by an ectopic beat. This sequence is diagrammed to suggest a 3:2 ratio of impulses entering and impulses leaving the reentrant loop. The latter portion of the bottom strip demonstrates trigeminal ectopic beats with a constant coupling interval of 550 msec. This patient, then, demonstrates both bigeminal and trigeminal ventricular ectopic activity. There are one, two, three, or four normal beats intervening between ectopic beats in Figure 3. Thus, this tracing conforms neither to the pattern proposed for concealed bigeminy nor to that proposed for concealed trigeminy.9 This variability between concealed bigeminy and concealed trigeminy further suggests that variable conduction in the reentrant pathway was the mechanism of arrhythmia in this patient. A similar alternation between concealed bigeminy and trigeminy has been described by Levy and co-workers.11 Indeed, scrutiny of their Figure 6 discloses progressive increases in the coupling interval from sinus beat to ectopic beat similar to that observed in the patients reported in the present study.

The rhythm strip demonstrated in Figure 4 also reveals intermittent bigeminy, with a single instance of a trigeminal rhythm. The first two ventricular ectopic beats in the top strip couple at 490 and 540 msec after their corresponding sinus beats; the sixth QRS complex is not followed by a ventricular ectopic beat. The ladder diagram indicates the supposition that this reflects a 3:2 ratio for entry/emergence of the impulse in a reentrant circuit. The fourth ventricular ectopic beat in the top strip initiates a run of bigeminal rhythm with an initially stable coupling interval of approximately 520 msec. In the bottom strip, this coupling interval sequentially increases from 520 to 550, 570, and 630 msec. The ventricular ectopic beat which occurs after the coupling interval of 630 msec is a fusion beat. Egress of the impulse from the reentrant circuit after the tenth QRS complex of the bottom strip is presumed to be blocked.

The sinus rate varies slightly in Figure 4 without disrupting the basic cause-effect relationship between an antecedent sinus beat and the subsequent ventricular ectopic beat. There is an accompanying variation in the interectopic intervals, which include 1,530, 1,560, 1,660, 2,200, and 2,850 msec. These facts do not lend themselves to a parasystolic model of arrhythmia.

The electrocardiographic strip shown in Figure 5 is representative of many similar sequences demonstrated in case 3. The interectopic intervals are more closely related to the slightly varying sinus intervals than to any presumed parasystolic interval. The top strip demonstrates bigeminy, with a progressive increase in the coupling interval between the sinus beats and the ectopic beats from 400 to 450 msec. The bottom strip is continuous with the top and demonstrates failure of coupling after the first sinus beat of the bottom strip. This is presumed to represent failure of the impulse to successfully traverse the reentrant pathway.

A second pattern commonly displayed by case 3 is illustrated in the bottom strip. This is an apparent quadrigeminal rhythm. At other times, instances of five and seven sinus beats intervening between ventricular premature beats occurred; no instances of even numbers of sinus beats between ectopic beats were seen. This observation is analogous to the phenomenon of concealed bigeminy, as reported by Kerin and co-workers.12

The electrocardiographic excerpts from the three cases share many common features. In each, ventricular ectopic activity is intermittent. In none does the intermittence of the ventricular activity fit well with a parasystolic model, even if one invokes only intermittent expression of the parasystole or intermittent protection of the parasystolic focus, or both.

Each tracing demonstrates some of those features.
which have led to a formulation of concealed bigeminy or concealed trigeminy.\textsuperscript{9,11,12} The formulation of concealed ventricular ectopic activity, as originally proposed by Schamroth and Marriott,\textsuperscript{4} has been considered to represent intermittent and patterned failure of ectopic enhancement\textsuperscript{4,8,10} or of impulse propagation through a reentrant circuit.\textsuperscript{11,12}

In 1950, Mack and Langendorf\textsuperscript{13} presented ECGs from two patients similar to those in this report. They pointed out that the Wenckebach structure of the arrhythmia constituted evidence of an abnormal path of conduction of the impulse as the mechanism of the rhythm. One of the two patients whose ECG was presented in 1950 was also discussed by Langendorf and Pick\textsuperscript{14} in 1955. Two very early reports presented similar ECGs,\textsuperscript{15,16} although the mechanism postulated is similar only in one.

I have been unable to locate descriptions of similar arrhythmias in the literature after 1955, although Figure 6 in the report by Levy and co-workers\textsuperscript{11} appears to show the same phenomenon.

The phenomena demonstrated by the cases presented in this report include ventricular ectopic bigeminal rhythm, trigeminal rhythm, presumed concealed bigeminy, presumed concealed trigeminy, and Wenckebach-like behavior with increasing coupling intervals prior to interruption of bigeminal sequences. The phenomena provide evidence that a derangement of intraventricular impulse propagation is the mechanism which produced the arrhythmias. By inference, the observations reported herein constitute evidence that disordered impulse propagation underlies most clinical instances of ventricular reentrant arrhythmias.

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