Paroxysmal Ventricular Tachycardia with Rate-Dependent Coupling Intervals

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SUMMARY

A case of repetitive ventricular tachycardia is reported in which the duration of the coupling interval of the first ectopic beat of each paroxysm is related to the duration of the preceding R-R interval. Long coupling intervals are preceded by relatively long R-R intervals, and vice versa. The postulate is presented that this phenomenon may be due to varying conduction times of the extrasystolic impulse through the ectopic-ventricular junction.

Additional Indexing Words:
Extrasystoles Ectopic beats Arrhythmia Rule of bigeminy

Ventricular Extrasystoles are usually associated with fixed or constant coupling intervals,1 that is, the interval between the extrasystole and the preceding sinus beat is the same for all extrasystoles arising from the same focus. This indicates that the extrasystole is in some way dependent upon, precipitated by, or forced by, the preceding sinus beat. In contrast to this, parasystole is associated with varying coupling intervals, for this condition reflects the coexistence of two independent pacemakers beating asynchronously; the ectopic beats therefore bear no relationship to the sinus beats.

On rare occasions, ectopic beats which are unequivocally extrasystoles may also be associated with variation of the coupling intervals. The cause of this has not as yet been established. We report a case of repetitive paroxysmal ventricular tachycardia in which an unequivocal correlation is demonstrated between the coupling intervals and the length of the preceding R-R intervals.

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*All time intervals are represented in hundredths of a second, that is, 33 = 0.33 sec.

Report of Case

The electrocardiogram (fig. 1) was recorded from a 54-year-old man with coronary insufficiency. Physical examination revealed an irregular cardiac rhythm and diminished arterial pulsation in the left lower limb. The blood pressure was 150/90 mm Hg. Roentgenograms of the chest were normal. The fasting blood sugar level, the blood urea, and the erythrocyte sedimentation rate were all normal. Postural and respiratory maneuvers, physical effort, as well as administration of digitalis, quinidine, procainamide, and reserpine were without effect on the arrhythmia. A follow-up period of 16 months revealed no change.

The tracing (fig. 1) shows repetitive paroxysms of ventricular tachycardia. A relatively long pause follows the termination of each paroxysm. The coupling intervals of the first ectopic beat of each paroxysm and its preceding sinus beat vary. These coupling intervals may be classified into two distinct groups (fig. 2; table 1): Group 1 shows relatively short coupling intervals which range from 32 to 39 (mean, 34.6).* Group 2 shows relatively long coupling intervals which range from 46 to 52 (mean, 50.4). The short coupling intervals are all preceded by relatively short R-R intervals which range from 39 to 63 (mean, 50.7). The long coupling intervals are all preceded by relatively long R-R intervals which range from 72 to 90...
Figure 1

Electrocardiogram (continuous strip of lead V1) showing paroxysms of ventricular tachycardia. The coupling intervals of the first ectopic beats of each paroxysm are related to the length of the preceding R-R interval. Long coupling intervals are preceded by long R-R intervals and vice versa.

(mean, 81.4). The ectopic beats of each paroxysm are arrhythmically distributed in two well-defined patterns.

**Pattern I.** The ectopic beats have a 2-1-2 distribution, that is, the first two beats follow each other in close succession and are succeeded after a pause by a single ectopic beat which, after another pause, is followed by a further pair of beats in close succession. At times, only the first two beats of this pattern are manifest. This pattern always follows a relatively short R-R interval, and hence, always begins with a short coupling interval.

**Pattern II.** The ectopic beats have a 1-2 distribution, that is, the sequence begins with a single beat which is followed by a pause and then two beats in close succession. This pattern is similar to the second part of Pattern I. As this pattern is always preceded by a relatively long R-R interval, it always begins with a long coupling interval.

**Discussion**

Paroxysmal ventricular tachycardia may be viewed as a series of three or more consecutive ventricular extrasystoles. The coupling interval of the first extrasystole of each paroxysm has the same significance whether it is followed by further extrasystoles or not. The coupling intervals of the first ectopic beats of each paroxysm in this case showed a clear-cut correlation with the length of the preceding R-R intervals. Relatively long coupling intervals were associated with long preceding R-R intervals and vice versa.

It is of interest to note that Lepeschkin and Rosenbaum in 1957, in an analysis of large numbers of cases of ventricular extrasystoles, observed that slower heart rates are usually associated with longer coupling intervals than faster heart rates. Surawicz and MacDonald in 1964, however, in an analysis of ventricular ectopic beats with varying coupling intervals (including parasystole and intermittent parasystole) found that a significant positive or negative correlation between the coupling interval and the preceding R-R interval was rarely present.

The dependence of the coupling interval on the length of the preceding R-R interval in our case suggests a possible effect on the
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Table 1

representation of Coupling Intervals and Their Respective Preceding R-R Intervals

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<th>Group 1</th>
<th>Coupling interval*</th>
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*All time intervals are in hundredths of a second, that is, 34 = 0.34 sec and so on.

conduction of the ectopic impulse through the ectopic-ventricular (E-V) junction, namely, the junction between the ectopic focus and the surrounding ventricular myocardium. In other words, an effect on the exit of the extrasystolic impulse from its ectopic focus. This postulate is illustrated in figure 3. The refractory period of a conducting tissue is directly proportional to the length of the preceding R-R interval.\(^4\) The longer the R-R interval, the longer the ensuing refractory period and vice versa, that is, the refractory period shortens with acceleration. A long R-R interval would conceivably have the same effect on the E-V junction. In the presence of a long R-R interval (fig. 3, I), the following refractory period of the ectopic-ventricular junction is relatively long; the ensuing extrasystolic impulse, therefore, is conducted with a relatively long conduction time through the ectopic-ventricular junction, and this results in a long coupling interval (labeled C1). In the presence of a short R-R interval (fig. 3, II), the subsequent refractory period of the ectopic-ventricular junction is relatively short; the ensuing extrasystolic impulse, therefore, is conducted through the junction with a shorter conduction time, and this results in a short coupling interval (labeled C2).

Another mechanism, based on the same principles, may be deduced from the allo-
Figure 2

Graphic representation of the relationship between the coupling intervals and the preceding R-R intervals. Time intervals in hundredths of a second.

Figure 3

Diagram illustrating the effect of long and short preceding R-R intervals on the conduction of extrasystolic impulses through the ectopic-ventricular junction. (I) A long preceding R-R interval results in a long ensuing coupling interval (C 1). (II) A short preceding R-R interval results in a short ensuing coupling interval (C 2). See text. A = atrial level; E = ectopic ventricular level.

The grouping of two beats in close succession followed by a pause suggests a 3:2 exit block at the E-V junction. And as the pause is less than twice the interval between the first two ectopic beats, conduction through the junction is probably of the Wenckebach type. This sequence is followed by a 2:1 exit block, which in turn is succeeded by another 3:2 Wenckebach sequence.

\[A-V = A-V \text{ nodal level; } V = \text{ventricular level; } E-V = \text{ectopic-ventricular junction; } E = \text{ectopic pacemaker.}\]
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Pattern II is illustrated in diagram II of figure 4. The paroxysm is preceded by a relatively long R-R interval (R1-R2). The ensuing refractory period of the E-V junction is thus relatively long. The first ectopic impulse (E1), therefore, encounters refractory tissue and is blocked. This blocked impulse is followed by a 2:1 exit block of impulses E2 and E3, which is succeeded by a 3:2 Wenckebach exit of impulses E4 to E5. It will be noted that pattern II is similar to the second part of pattern I, that is, conduction of impulses E1 to E6 of pattern II is equivalent to conduction of impulses E3 and E8 of pattern I.

The aforementioned postulates indicate that the long coupling interval is due to an exit block or an exit delay of the first ectopic beat of the paroxysm as a result of the increased refractoriness following a long interval.

Langendorf and associates in 1955 showed that the precipitation of an extrasystole was favored by a long preceding R-R interval. The compensatory pause of the precipitated extrasystole in turn constitutes another long R-R interval, and this favors the precipitation of a further extrasystole. The bigeminal rhythm thus tends to be perpetuated—the “rule of bigeminy.” It must be stressed, however, that only the precipitation of the extrasystole is favored by the preceding long R-R interval; the rule of bigeminy does not apply to any subsequent variation of the coupling intervals. These authors postulated that the rule of bigeminy was explicable on the basis of varying intraventricular conduction, but the precise mechanism was not elaborated.

On the basis of the observations in this case, a long R-R interval causes a delay or block, or both, of the first ensuing extrasystole. Under such circumstances, it would appear that the long R-R interval should militate against the appearance of an extrasystole. The rule of bigeminy is thus not necessarily dependent upon a conduction disturbance. This is in keeping with recent observations and postulates. It has been shown that extrasystoles are not all dependent upon the rule

Figure 4

Diagrams illustrating conduction of the ectopic impulses through the ectopic-ventricular junction. See text. A-V = A-V nodal level; V = ventricular level; E-V = ectopic-ventricular junction; and E = ectopic focus.
of bigeminy, and theories other than conduction disturbances have been presented to explain this phenomenon.

Acknowledgment
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References

100 Years Ago—Correspondence—Lancet—Heart Disease and Tobacco
Few of us can doubt that the habit of indiscriminate smoking has led to much mischief, especially when indulged in by immature and etiolated youths, who nauseate themselves with cheap cigars and English tobacco. An Irish registrar, who is therefore we presume a medical man, calls attention to the number of sudden deaths from heart disease, which are thought to be much more common now than they were thirty or forty years ago, and he raises the question whether the now almost universal use of tobacco has anything to do with this. He thinks it has; but it strikes us as being an exceedingly difficult theory to establish satisfactorily. The mere fact of the increased consumption of tobacco would hardly be a sufficient ground to go upon, unless it could be proved by experiment upon a given number of persons with heart disease that the non-smokers of this class were (coeteris paribus) less liable to sudden death than the smokers.—Anonymous: Heart Disease and Tobacco. Lancet 2: 119, 1867.
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