Effect of Extrasystoles on Idioventricular Rhythm

Clinical and Electrophysiologic Correlation

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SUMMARY

When premature ventricular contractions (PVCs) occur in the idioventricular rhythm of patients with complete heart block (CHB), one of several rhythm alterations may occur: the postextrasystolic or returning cycle (RC) may be prolonged, unaltered, or even shortened without any apparent reason. To elucidate the mechanisms by which these alterations occur, the effect of spontaneous and of electrically induced PVCs was studied in five patients with CHB. Over 500 observations were obtained. PVCs occurring late in the basic cycle, i.e., after the T wave of the last spontaneous idioventricular beat, almost uniformly resulted in lengthening of the first RC. In sharp contrast, PVCs elicited early in the cycle, i.e., during the T wave, were followed by a shortened RC in 95% of instances. The underlying electrophysiologic mechanisms responsible for the relationship of RC length to degree of prematurity of the PVC are discussed.

Additional Indexing Words:
Complete heart block  Pacemakers  Transmembrane action potentials

The effect of extrasystoles on normal sinus rhythm has been the object of intense investigation. In contrast, the effect of premature ventricular contractions (PVCs) on idioventricular rhythm during complete heart block (CHB) has received relatively scant attention, perhaps unfortunately so, since such a study might have given valuable information on the electrophysiologic behavior of pacemaker cells. Only one clinical study of PVCs in idioventricular rhythm has been published.1 Although there are isolated references in the early electrocardiographic literature, most of the reports antedate the electrocardiographic era.2–17 The conclusion reached by previous authors has been that PVCs ordinarily reset the idioventricular rhythm in such a manner as to produce a lengthening of the postextrasystolic, or returning, cycle (RC). However, many unexplained instances of shortening of the RC also were noted,1–4, 6, 9, 11, 16, 18–21 and occasionally there was no alteration of the RC.1, 6, 14, 22 Various mechanisms were postulated to explain these findings, but never documented by experimentation.23

Recently, the effect of premature stimulation on idioventricular rhythm was systematically studied in an in vitro model consisting of spontaneously beating canine and ungulate Purkinje fibers attached to segments of ventricular muscle.24 It became clear that the timing of the extrasystole within the dominant cycle (DC) was of crucial importance in determining the duration of the RC of the Purkinje fiber: early extrasystoles usually resulted in shortening and late extrasystoles in lengthening of the RC. It also became clear that although the ventricular RC duration often reflected accurately the Purkinje RC duration, this was not a universal finding. Discrepancy between Purkinje and ventricular RC was sometimes observed.

This report presents observations in patients which are consistent with the experimental results obtained from the cardiac tissue in vitro and which indicate that extrasystoles influence ventricular pacemaker cells in idioventricular rhythm in the human heart in a fashion similar to that in the experimental model.
Methods

Observations were obtained in five patients with idiopathic CHB who required a temporary pacemaker prior to the insertion of a permanent pacemaker. These patients were studied after a temporary bipolar electrode catheter had been introduced transvenously into the right ventricle and connected to an external, battery-powered pacemaker. Myocardial infarction had previously been ruled out by the negative history and by negative enzyme studies.

With the consent of the patients, the pacemaker rate and amperage were gradually decreased to allow the reestablishment of a stable idioventricular rhythm. Once this rhythm had been attained, single PVCs were induced in the dominant cycle by briefly turning the pacemaker on, in the "demand" mode at a previously selected rate. This resulted in effect in the introduction of PVCs at previously selected intervals after the last spontaneous idioventricular beat sensed by the pacemaker. At least two, and more usually three, single PVCs were elicited with similar coupling intervals at different times during the testing period. The entire duration of the dominant cycle was "scanned" in such a fashion in each patient. More than 15 consecutive spontaneous cycles were allowed to pass between each stimulus. A total of over 50 separate observations were recorded in each patient on each of two separate days. A second set of observations was obtained in idioventricular rhythm interrupted by spontaneously occurring PVCs. Tracings were recorded on electrocardiographic paper at a speed of 25 or 50 mm/sec and analyzed for cycle length and degree of prematurity of the stimulus and of the PVC.

Results

Figure 1 illustrates in a graphic form the spectrum of responses obtained in a patient representative of the group. PVCs elicited after the T wave were followed by a lengthened RC. The degree of lengthening tended to be more accentuated with longer coupling intervals. Fairly consistent returning cycles were found at various coupling intervals, but exceptions were frequent. In sharp contrast, PVCs elicited during the T wave of the last spontaneous beat were almost invariably followed by a short RC. The degree of shortening of the RC tended to be greater with shorter coupling intervals, but exceptions were again found.

Figure 2 illustrates typical responses to electrically induced PVCs in a patient with CHB. The PVCs elicited after the end of the T wave are followed by an RC which is either unchanged or longer than the dominant cycles (fig. 2A, B). In contrast, the PVCs elicited before the end of the T wave are followed by an RC which is shorter than the dominant cycles (fig. 2C, D). Figure 3 illustrates similar responses to spontaneous PVCs in the same patient on a different day. The pacemaker was stopped shortly before this record was obtained and the basic rate of the idioventricular rhythm gradually increased in the expected fashion from A to D. In A, B, and C, a late PVC is followed by a lengthened RC. In contrast, the RC following the early PVC in figure 3D is shorter than the dominant cycle. In one patient, early PVCs which were induced electrically failed to shorten the RC. However, the RC became less prolonged with successively shorter coupling intervals, and the shortest RC was equal to the dominant cycle after a PVC induced 400 msec after the last spontaneous beat (fig. 4C). In contrast, spontaneous premature beats produced shortening of the RC when they occurred early (fig. 4D).

Discussion

Although variable responses of an idioventricular rhythm to PVCs have been reported by various authors, the simple relationship of returning cycle length to the degree of prematurity of the PVC has not previously been recognized. As illustrated in figures 1–3, a sharp difference in responses is seen.
between PVCs occurring during the T wave and those occurring later. Recent studies using microelectrode technics demonstrated that this difference is due to a difference in the response of the transmembrane potential of Purkinje cells after early and late extrasystoles. This is illustrated in figure 5 in which the electrophysiologic events underlying the electrocardiographic observations of figures 1–3 are depicted. The RC is lengthened after a late extrasystole (fig. 5A) because of the greater time duration required by the transmembrane potential to return to threshold potential. This may be due to depression of the slope of spontaneous diastolic depolarization or to greater negativity of the maximum diastolic potential, or to both. Since these two factors vary from extrasystole to extrasystole, variable degrees of lengthening of the RC with PVCs of a similar degree of prematurity are not unexpected, as indeed is the case in figure 1. The action potential of a late extrasystole is usually somewhat shortened, and this shortening is occasionally substantial enough to counterbalance the effect of the depression of spontaneous depolarization, so that the ultimate RC length may be unaltered (fig. 5B). It is, however, usually not substantial enough to shorten the RC.

In contrast, the action potential of an extremely early extrasystole undergoes repolarization very quickly and is therefore extremely short, as in figure 5C. This abbreviation of the extrasystolic action potential is so marked that it counterbalances any subsequent depression of the slope of spontaneous diastolic depolarization and is responsible for shortening of the RC. Shortening of the extrasystolic action potential also occurs in ventricular myocardial cells and is often reflected on the electrocardiogram by a shortened Q-T interval of early PVCs when compared to that of the beats of.
Figure 3

Effect of increasing prematurity of spontaneous PVCs on RC duration. The pacemaker had been shut off, and the rate of the idioventricular pacemaker is unstable and becomes gradually faster from A to D. Later PVCs in A, B, and C are followed by lengthened RC and an early PVC in D by a shortened RC. Q-T is shortened by 50 msec in D (see discussion). II = lead II. Paper speed = 25 mm/sec.

the spontaneous idioventricular rhythm (figs. 2, 3).

However, the shortening of the His-Purkinje RC after early PVCs may not always be demonstrable electrocardiographically. The ECG fails to give any direct indication of the actual duration of the Purkinje pacemaker cell RC or about other factors which affect the ultimate ventricular RC, such as (1) the relationship of origin and direction of propagation of the PVC with respect to the spontaneous pacemaker site in the His-Purkinje system, and (2) the degree of conduction delay of the PVC or of the postextrasystolic beat. Given the proper set of conditions, a ventricular RC may be equal to the dominant cycles, or it may even be lengthened, while the His-Purkinje RC is shortened. This would occur, as illustrated in figure 6C, D, in the case where the PVC originates in the periphery.
Figure 4

Effect of different extrasystolic focus on ventricular RC. In A, B, and C, PVCs are induced through a pacemaker with the electrode tip located in the right ventricular apex. The ventricular RC is lengthened in A and B and unchanged in C, and presumably the Purkinje RC is shortened (see discussion). In D, a spontaneous premature beat originating in a different focus is followed by a shortened RC. II = lead II.

Paper speed = 25 mm/sec.
IDIOVENTRICULAR RHYTHM

Figure 5
Effect of increasing prematurity of extrasystoles on trans-
membrane action potentials (AP) of a spontaneously beating 
Purkinje fiber preparation. (A) A relatively late extrasystole 
is followed by lengthening of the RC. The extrasystolic AP 
is not shortened enough to counterbalance the increased 
duration of spontaneous phase 4 depolarization. (B) An 
earlier extrasystole is induced. The moderate shortening of 
the extrasystolic AP counterbalances the increased duration 
of phase 4, and the entire RC is unchanged. (C) A very early 
extrasystole is followed by a markedly shortened RC because 
of the extreme abbreviation of the extrasystolic AP. The 
altered duration of the conjoined cycle in C (2730 msec) 
when compared to the dominant cycles indicates resetting 
of the pacemaker rhythm by the extrasystole. Time lines = 
1000 and 100 msec. Arrows indicate the point of maximum 
diastolic potential.

of the ventricular conduction system and has to 
propagate retrogradely before reaching the sponta-
neous pacemaker site.

This is probably the mechanism responsible for 
the unaltered RC length following pacemaker beats 
in patients with partial pacemaker failure reported 
by Walker,22 Pick and Langendorf,26 and Sham-
roth's case 153,27 and in our patient illustrated in 
figure 4C. In contrast, an early extrasystole may 
originate somewhere above the idioventricular 
pacemaker site and, propagating in the same 
general direction as the idioventricular beats, it will 
first depolarize the idioventricular pacemaker site 
and then, and only after a variable degree of 
conduction delay, reach the myocardium, as illus-
trated in figure 6A. The shortening of the 
ventricular RC then reflects the shortening of the 
His-Purkinje RC. Such a response has recently been 
published by Shamroth27 (case 152), and figure 4D 
may represent another example of it.

Exceptionally, shortening of the ventricular RC 
may occur on the electrocardiogram after late PVCs 
too. True interpolation of a PVC may occasionally 
occur.3 Three other mechanisms may be responsible 
for ventricular RC shortening. The first is illustrated 
by figure 6B: a late PVC originating above the site 
of origin of the idioventricular pacemaker site may 
produce a Purkinje RC of unaltered length such as 
that of figure 4B and a shortened ventricular RC if 

Figure 6
Effect of relative location of intrinsic pacemaker site and 
eccentric focus on the direction of propagation of the extra-
systole and on ventricular RC duration. (A) The intrinsic 
pacemaker site (black circle) is located distal to the eccentric 
focus (white circle). When the latter fires, the extrasystole 
first depolarizes the intrinsic pacemaker site on its way down 
through the ventricular conduction system and is then re-
corded by the surface electrocardiogram, as shown in B. 
The ventricular RC is shortened as much as the pacemaker 
fiber RC, or may even be more so, provided there is suffi-
cient conduction delay of the extrasystole (dashed line). (C) 
The intrinsic pacemaker site is located proximally in the 
conduction system. The eccentric focus is distal. When it fires, 
the extrasystole is insulated almost immediately on the ECG, 
but activity must spread retrogradely before depolarizing 
the pacemaker site. If conduction delay is marked, the ven-
tricular RC may not reflect the shortening of the pacemaker 
fiber RC (D). P = Purkinje fiber events; V = ventricular 

it reaches myocardium only after a marked 
conduction delay, reach the myocardium, as illus-
observed in hypoxia or exposure to excessive drug 
concentrations. In such conditions, Purkinje cells 
may respond to extrasystoles in an abnormal 
fashion. The point of maximal diastolic potential of 
such sick cells may be quite delayed in diastole. An 
extrasystole is capable of shortening the RC simply 
by displacing the point of maximum diastolic 
potential to a point earlier in diastole and thus 
resulting in the earlier onset of spontaneous 
depolarization.24 The third mechanism is the 
availability of latent pacemaker cells to transiently 
take over the control of the ventricles.1, 28, 29

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Notwithstanding these exceptions, and in spite of the limitations of the electrocardiogram and more primitive recording methods, a review of the literature makes it clear that lengthening of the ventricular RC has usually been observed after late PVCs and that almost all recorded instances of shortened or unchanged ventricular RCs occur after early PVCs, near the end of or shortly after the T wave of the last spontaneous beat. The T wave represents ventricular repolarization, which terminates before repolarization in the His-Purkinje system. It is therefore evident that the PVCs which shorten the ventricular RC on clinical electrocardiograms occur early in the dominant Purkinje RC. The basis for this shortening of the RC in man is probably similar to that in the experimental animal, i.e., shortening of the extrasystolic action potential.

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Correction

Klein HO: Circulation 47: 758, 1973. On page 759, in the legend to figure 1, line 9, sentence should read: "Extrasystoles induced thereafter lead to lengthened post extrasystolic cycles." On page 763, the second line of column two should read, "conduction delay. The second mechanism may be..."