Facilitation of macroreentry within the His-Purkinje system with abrupt changes in cycle length

Stephen Denker, M.D., Michael H. Lehmann, M.D., Rehan Mahmud, M.D., Carol Gilbert, R.N., and Masood Akhtar, M.D.

ABSTRACT We have recently described the ability of abrupt short-to-long changes in atrial cycle length (CL) to prolong refractoriness of the His-Purkinje system (HPS) and increase the likelihood of aberrant ventricular conduction. We have also shown similar functional behavior in retrograde refractoriness of the HPS during changes in ventricular CL. To further assess these characteristics we evaluated the effect of abrupt short-to-long change in ventricular CL on the phenomenon of macroreentry within the HPS (Re-HPS) in 20 patients in whom Re-HPS occurred during application of a ventricular extrastimulus (V,) at a constant ventricular CL (method I) and/or with abrupt short-to-long change in CL (method II). For both methods V, was coupled to a CL of identical duration, designated the reference CL (CLr). In method I the CLs preceding (CLp) the CLR equaled CLr, whereas in method II CLp was less than CLr. The results showed a dramatic increase in occurrence of Re-HPS with abrupt short-to-long change in CL with Re-HPS occurring in 19 patients with this method compared with in 11 patients during constant CL. In 10 patients manifesting Re-HPS with both methods the associated retrograde conduction (VH1) delays were equal or less during abrupt short-to-long changes in CL and, remarkably, there were concomitantly shorter antegrade conduction (H2V,1) delays compared with at a constant CL. Moreover, despite the resulting shorter VH1, additional Re-HPS beats were also more likely to occur with abrupt short-to-long change in CL compared with at a constant CL. These findings suggest that (1) there is an earlier onset or more distal site of retrograde block in the right bundle–Purkinje system or Purkinje-myocardial junction with abrupt short-to-long change in CL, which is consistent with previous findings of increased refractoriness of HPS with this method, and (2) that such changes in CL can facilitate reentry involving the HPS.


RECENT STUDIES have demonstrated that abrupt short-to-long changes in cycle length (CL) affect the His-Purkinje system (HPS) in an unexpected manner. The classic concept that duration of HPS refractoriness varies directly with CL could not account for the following observations: (1) Abrupt short-to-long changes in CL during studies of the antegrade refractory period significantly affected the phenomenon of aberrant ventricular conduction (VAb), such that VAb occurred exclusively or occurred at longer atrial coupling intervals (A,A,) and H1H2 intervals than it did at constant CLs. (2) Similarly, abrupt short-to-long changes in CL during studies of the retrograde refractory period produced retrograde conduction delay in the HPS at longer ventricular coupling intervals (V,V,) than those observed at constant CLs. (3) Abrupt changes in CL produced a divergence in the refractory characteristics of the HPS and ventricular muscle (VM). These observations suggested that the type of HPS behavior associated with abrupt changes in CL was fundamentally different from the so-called cumulative response to preceding CLs that occurs in VM. To further assess the fundamental nature of this seemingly paradoxical behavior, we systematically examined the effect of abrupt short-to-long changes in CL on another common electrophysiologic phenomenon in man, macroreentry within the HPS (Re-HPS), which occurs during ventricular premature stimulation. Re-HPS is a well-elucidated model for reentry in the intact human heart that could permit analysis of the effects of short-to-long changes in CL on reentry. The possible mechanisms of the effects of abrupt short-to-long changes in CL on this model and the clinical significance of the findings are discussed.
Methods

Electrophysiologic studies were performed in all patients after obtaining signed consent to the explained procedure. Patients were studied in the unsedated, postabsorptive state. With the use of local anesthesia and fluoroscopic guidance, multipolar electrode catheters were percutaneously introduced and positioned in the right high atrium, the atrioventricular junction, and the right ventricle to record local electrical activity or for local pacing. All intracardiac electrograms (filter frequency 30 to 500 Hz), three surface electrocardiographic (ECG) leads (I, II, VI), and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on magnetic tape for later reproduction. Programmed electrical stimulation was accomplished with a digital stimulator delivering rectangular impulses of adjustable amplitude and duration. In none of the patients was the magnitude or duration of the electrical impulse altered during the study. All equipment was grounded and an isolation unit was used for electrical stimulation.

All patients were studied with the conventional method (method I) of ventricular pacing at a constant CL (S1S2 or V1V3) and scanning was done with progressively decreasing coupling (S1S2 or V1V3) intervals until the effective refractory period (ERP) of the VM was attained. The effect of an abrupt short-to-long change in CL (method II) was evaluated by shortening the constant CL preceding (CLp) the last beat of the ventricular drive in method I. By design, the CL of the 1 beat just before V2 was of identical duration in both methods and, therefore, was designated as the reference CL (CLR). In method I, CLR was equal to CLR, whereas in method II, CLR was less than CLR (figure 1). Simultaneous atrial and ventricular pacing during the basic ventricular drive was performed in those patients without intact ventriculoatrial conduction in whom sinus beats interrupted the ventricular drive.

Statistical analysis of data was done by Student’s t test, except when additional methods were indicated.

Definition of terms. A complete list of definitions of terms that are used in this type of clinical investigation have been published previously.5,6 Only the intervals pertinent to this report are defined below.

Antegrade HPS conduction. The HV interval was measured from the onset of the His bundle (HB) deflection to the earliest detectable ventricular activation on the surface ECG or local electrogram recordings.

Retrograde HPS conduction. The retrograde HB potential for the basic drive beats (H1) could not be identified. When the HB deflection emerged from the local V1 electrogram the S1H2 (V1H2) interval was measured from the stimulus artifact (or local ventricular electrogram) to the onset of the HB potential.

Retrograde ERP of the HPS. The longest S1S2 (V1V2) interval at which S2 (V2) retrogradely blocked below the HB.

ERP of ventricular muscle. The longest S1S2 interval at which S2 did not produce a ventricular response.

V3 phenomenon. A spontaneous ventricular depolarization due to Re-HPS following S2 (V2).

Zone of reentry within the HPS. The range of S1S2 (V1V2) intervals at which S2 (V2) produced the V3 phenomenon. The outer and inner limits of Re-HPS zone were the longest and shortest S1S2 (V1V2) intervals, respectively, which resulted in V3 due to Re-HPS.

Critical retrograde (V1H2 or S1H2) and antegrade (H2V3) conduction delays. The resulting V1H2 (S1H2) and H2V3 at the longest comparable V1V3 (S1S2) at which Re-HPS occurred with both methods of stimulation; when Re-HPS occurred exclusively with either method the critical values are indicated for the longest V1V3 (S1S2) at which Re-HPS occurred.

Results

Essential electrophysiologic data are listed in table 1. Only patients in sinus rhythm with normal intraventricular conduction manifesting the V3 phenomenon (Re-HPS) during V2 with either constant CL and/or abrupt increase in CL were included in this series. The 20 consecutive patients (14 men, six women) had a mean age of 52 ± 15 years and were undergoing electrophysiologic studies for symptomatic or suspected cardiac arrhythmias. Cardioactive drugs were discontinued for 48 to 72 hr before the study. The underlying structural heart disease was arteriosclerotic in 10 patients, mitral valve prolapse in three, valvular heart disease in one, and cardiomyopathy in one, and there was no clinically detectable heart disease in five.

The features of Re-HPS have been previously described and are not presented in detail here.5,7,8 In all cases the occurrence of Re-HPS was preceded by the emergence of retrograde HB deflection from the local ventricular electrogram, indicating retrograde conduction delay in the HPS. With progressive shortening of V1V2 the continued occurrence of V3 at longer V1H2 delays was typically associated with shorter H2V3 delays and longer V1V3 intervals. In all patients V3 occurred with a left bundle branch (LBB) block configuration, indicating retrograde block in the right BB-Purkinje system (RBB-PS) and retrograde conduction delay in the LBB.5

Effect of constant CL vs abrupt increase in CL on occurrence of Re-HPS. When method I was used and V2 was preceded by a constant CL (CLp = CLR) Re-HPS was manifest in 11 of 20 patients, compared with 19 of 20 patients with method II, in which V2 was preceded by an abrupt increase in CL (CLp < CLR). The increased likelihood of Re-HPS occurring with method II compared with method I was statistically significant (p <
### TABLE 1

**Electrophysiologic data**

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<th>Patient No.</th>
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All measurements are in milliseconds.

*V1,H2 and H2,V1 intervals at the longest V1,V2(V1,V2) at which Re-HPS occurred with both methods; when Re-HPS occurred exclusively with either method the values are indicated for the longest V1,V2 (V1,V2) at which Re-HPS occurred.

VCL = ventricular cycle length.

.01) by Fisher's exact test. Moreover, Re-HPS occurred exclusively in only one patient (No. 10) during constant CL, but occurred exclusively in nine patients (Nos. 1 through 9) during abrupt increase in CL despite attainment of comparable retrograde (V1,H2) conduction delays in most instances (table 1, figure 2). In one patient in whom Re-HPS was manifest only during a constant CL a sudden increase in V1,H2 delay was observed at the V1,V2 interval at which Re-HPS occurred. This degree of delay was not present at shorter V1,V2 intervals and also was not obtained with method II. Similar unexpected changes in V1,H2 delays have been previously reported.9

**Effect of abrupt increase in cycle length on V3 zone (table 1).** In 10 patients (Nos. 11 through 20) Re-HPS occurred at both a constant CL and after an abrupt increase in CL, which permitted comparison of the V1,V2 zones during which V3 was manifest (table 1). The outer and inner limits of the V3 zone were 282 ± 15 and 239 ± 43 msec, respectively, with method I and 295 ± 32 and 231 ± 20 msec, respectively, with method II. These differences between methods I and II were not statistically significant. The range of the V3 zone with method I was 43 ± 27 msec compared with 64 ± 34 msec with method II. This difference was significant (p < .01) based on a two-sided paired t test. In three patients (Nos. 18 through 20) Re-HPS first became manifest at shorter coupling intervals after bilateral retrograde BB block below the HB (retrograde gap phenomenon).9 In these three patients bilateral retrograde BB block below the HB occurred at a longer V1,V2 after an abrupt increase in CL than at a constant CL.

**Effect of the abrupt increase in CL on critical conduction delays associated with Re-HPS (table 1).** Evaluation of the retrograde (V1,H2) and antegrade (H2,V1) conduction delays associated with the V3 phenomenon was performed at the longest comparable V1,V2 coupling interval at which V3 occurred both at a constant CL and after an abrupt increase in CL in 10 patients (Nos. 11 through 20). The outer limit of the V3 zone was chosen for comparison because at the inner limit (shortest V1,V2) appreciable antegrade conduction delay may not occur and latency between the premature stimulus (S3) and local ventricular electrogram was frequently encountered.
In seven patients (Nos. 11 through 17) there was gradual prolongation of the V2H2 interval as V1V2 shortened at the outer limit of the V3 zone. In the remaining three patients (Nos. 18 through 20) V3 was manifest only with sudden prolongation of V1H2 after retrograde gap in the HPS. At the outer limit of the Re-HPS zone the S,H2 was 193 ± 29 msec for method I and 192 ± 37 msec for method II (p = NS). Despite the comparable S2H2 delays the antegrade conduction delay (H2V3) was significantly shorter after an abrupt increase in CL (65 ± 21 msec) as compared with at a constant CL (77 ± 26 msec; p < .005) (table 1, figure 3). The resulting V1V3 interval for these patients was shorter after an abrupt increase in CL compared with at a constant CL (256 ± 35 and 269 ± 39 msec, respectively; p < .05).

FIGURE 2. Macroreentry zone during abrupt increase in CL. The data plotted from patient No. 4 show that the Re-HPS occurs over a wide range of V1V2 coupling intervals following abrupt short-to-long changes in CL. The typical inverse relationships between V2H2 and H2V3, and between V1V2 and V3 are seen. Note that when a constant CL is followed by an identical CL of 600 msec during similar scanning with V2 and comparable retrograde conduction delay (V2H2) Re-HPS is not observed.

FIGURE 3. Effect of abrupt increase in CL on critical conduction delays associated with Re-HPS. The tracings taken from patient No. 17 show the occurrence of Re-HPS with both methods I and II at the same coupling interval of 280 msec. Note that with abrupt short-to-long change in CL (bottom) both the retrograde (V2H2) conduction delay and the antegrade (H2V3) conduction delay are shorter compared with constant CL (top). 1. V1 = surface ECG leads; HB = HB electrogram; T = time line at 1000, 100, and 10 msec intervals.
**Occurrence of repetitive ventricular beats due to Re-HPS.** At a constant CL, Re-HPS either did not occur or was limited to V3 in all but one patient (No. 13) in whom V4 due to Re-HPS occurred. In comparison, repetitive ventricular beats due to Re-HPS up to V6 occurred in seven patients (Nos. 1, 6, 7, 9, 13, 14, and 16) after an abrupt increase in CL (figure 4). In four of these patients the V3 phenomenon was never observed during a constant CL (table 1).

**Discussion**

Within the range of CLs used the results of the present study demonstrate an increased likelihood of macroreentry within the HPS after abrupt short-to-long changes in CL compared with at a constant CL. We have also previously reported a paradoxical increased likelihood of VAb during antegrade studies, and with similar changes in CL have demonstrated an unanticipated increase in retrograde refractoriness of the HPS with ventricular premature stimulation after abrupt short-to-long changes in ventricular CL.1-4

It has been demonstrated in the macroreentry model that the V3 phenomenon with LBB block configuration during right ventricular stimulation is dependent on retrograde block in the RBB-PS and delayed conduction in the LBB.5-10-12 Depending on the level of retrograde penetration in the RBB-PS and the degree of delay of retrograde conduction in the LBB, recovery of excitability permitting antegrade conduction along the RBB-PS axis may occur, resulting in V3. Remarkably, the present study demonstrates a higher incidence of Re-HPS with abrupt short-to-long changes in CL despite comparable retrograde conduction delay at a constant CL. Moreover, the shorter H3V3 intervals in association with less retrograde conduction delay at the outer limit of the V3 zone with abrupt short-to-long changes in CL are indeed unique.

**Facilitation of Re-HPS with abrupt short-to-long CL change.** For more frequent occurrence of macroreentry with abrupt short-to-long changes in CL despite comparable or longer retrograde delay during constant CL an earlier recovery of the antegrade limb, or RBB-PS, has to be postulated. This could occur as a result of an earlier onset or more distal site of retrograde block in the RBB-PS or at the Purkinje element of the Purkinje-muscle junction (PMJ). The most probable mechanism

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**FIGURE 4.** Effect of abrupt increase in CL on occurrence on repetitive ventricular beats due to Re-HPS. The tracings taken from patient No. 14 show that at constant CL of 600 msec a coupling interval of 290 msec results in an S1H2 of 190 msec and that Re-HPS is not manifest (A). During abrupt short-to-long change in CL with an identical coupling interval and retrograde delay Re-HPS occurs with an associated H3V3 interval of 55 msec followed by repetitive Re-HPS beats to V3 (B). C. During constant CL a shortening of S1S2 to 280 msec again results in S1H2 of 190 msec, but Re-HPS occurs with an associated H2V3 interval of 65 msec. No additional Re-HPS beats occur. D. In contrast, during abrupt short-to-long change in CL with comparable coupling interval and S2H3 delay as in C. repetitive Re-HPS is again manifest. Note the shorter antegrade conduction time (H2V3) associated with Re-HPS at comparable coupling intervals after abrupt short-to-long change in CL compared with at a constant CL. 1, 2. V1 = surface ECG leads; HB = HB electrogram; T = time line.
to explain this phenomenon is that an abrupt increase in CL results in increased refractoriness in the RBB-PS or Purkinje element of the PMJ. This is consistent with our previous findings of increased refractoriness in the HPS with abrupt short-to-long changes in CL,\(^1\)\(^2\) as well as with the higher incidence of bilateral retrograde BB block below the HB observed in the present study. Also, the intrinsically longer refractoriness of the RBB compared with that of the LBB\(^3\) may be further exaggerated after an abrupt increase in CL. A second factor possibly contributing to these findings is a more rapid input of the propagated S\(_2\) impulse to the distal RBB-PS or PMJ after abrupt short-to-long changes in CL by facilitation of conduction in the myocardial element of the PMJ. That this may occur is suggested by the shorter V\(_2\)H\(_2\) at a comparable V\(_1\)V\(_2\) with method II in some patients. Nevertheless, it would seem that a shortening in the refractoriness of VM or the myocardial element of PMJ per se could not account for the increased incidence of Re-HPS after abrupt short-to-long changes in CL. This is suggested by the observation that in almost 50% of patients studied, further shortening of V\(_1\)V\(_2\) intervals did not result in Re-HPS during constant CLs despite the occurrence of the V\(_1\) phenomenon at longer V\(_1\)V\(_2\) intervals with abrupt short-to-long changes in CL. It appears that increased refractoriness of the RBB-PS or Purkinje element of the PMJ and/or earlier effective input of S\(_2\) (due to a possible decrease in refractoriness of the myocardial element of the PMJ) causes an earlier onset or a more distal site of retrograde block along this axis and therefore earlier recovery for antegrade propagation.

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**FIGURE 5.** Comparison of rate of HPS CL change following V\(_2\) between methods I and II. A illustrates the rate of change in CL for method I vs method II. At a constant CL of 600 msec (method I), percent change in CL equals zero. After abrupt short-to-long change of 400 → 600 msec (method II) CL increased 50%. An increasing rate of CL change has been shown to result in an increase in HPS refractoriness.\(^1\)\(^2\) Therefore, a constant CL would have to be >600 msec to produce equivalent refractoriness of the HPS. In **B** a constant CL of ">600 msec" is substituted for the 400 → 600 msec change in CL of method II. For the same degree of CL shortening, i.e., V\(_1\)V\(_2\) of 300 msec, there is a greater rate of CL abbreviation with ">600 msec" (substituted for 400 → 600 msec of method II) than with a constant CL of 600 msec (method I). It is this greater rate of CL shortening during V\(_2\) that may account for the greater decrease in refractoriness of the HPS (LB) encountered by V\(_3\) due to Re-HPS with method II compared with method I.\(^1\)\(^2\)

**Repetitive Re-HPS.** The increased incidence of repetitive macroreentry beats may be similarly explained. Again, according to conventional concepts derived from studies performed during constant CL, the increased likelihood of repetitive macroreentry beats with abrupt short-to-long changes in CL appears paradoxical. It has been previously noted that spontaneous termination of Re-HPS in most instances occurs in the retrograde limb of the reentrant circuit between the VM and HB, i.e., along the LBB-Purkinje muscle axis.\(^3\) Generally, sufficient recovery of the retrograde limb to permit propagation of the V\(_i\) impulse would be less likely with a shorter V\(_2\)V\(_3\). Nonetheless, after abrupt short-to-long changes in CL V\(_2\)V\(_3\) was shorter (due to shorter V\(_2\)H\(_2\) and H\(_2\)V\(_3\) or shorter H\(_1\)V\(_3\), alone) than it was at a constant CL, but remarkably there was a higher incidence of repetitive macroreentry beats. For this to occur it must be postulated that V\(_i\) encountered less refractoriness as it transversed the retrograde limb after abrupt short-to-long changes in CL than it did at a constant CL. When both V\(_2\)H\(_2\) and H\(_2\)V\(_3\) are shorter, this may be due to a shortening in refractoriness of the entire reentrant circuit. Another possibility and explanation when only H\(_2\)V\(_3\) is shorter may relate to the effect of abrupt short-to-long changes in CL on the duration of refractoriness at two possible sites of block in the retrograde limb,\(^14\)\(^15\) i.e., in the LBB-PS or in the PMJ. Decreased refractoriness in the LBB-PS may occur due to shortening of LBB-PS CL by the propagated V\(_2\) impulse. A greater decrease in refractoriness may occur when V\(_2\) is preceded by an abrupt short-to-long change in CL than when CL is constant.
because of the so-called dynamic response of the HPS to abrupt CL changes.\textsuperscript{1,2} Previously, we have suggested that one of the determinants of HPS refractoriness is the rate of change of preceding CLs.\textsuperscript{1,2} Conceivably, greater rates of change might occur if CLs were alternately short-long-short, which would accentuate this dynamic response (figure 5). A similar response may occur in the Purkinje element of the PMJ after abrupt changes in CL. Refractoriness in the myocardial element of the PMJ is likely to be shortened during abrupt short-to-long changes in CL due to the possible cumulative effect of the shorter preceding CLs.

Electrophysiologic and clinical implications. The findings of this study are consistent with our previous observations that abrupt short-to-long CL changes can markedly affect HPS refractoriness in a fashion heretofore not described. In the present study, the increase in HPS refractoriness with abrupt short-to-long changes in CL was probably reflected by the earlier onset or more distal site of retrograde block in the distal RBB-Purkinje-muscle system. Moreover, the capability of this method of stimulation to alter the incidence of macroreentry is significant. While Re-HPS is considered a functional phenomenon of the human conduction system the present study suggests that clinical reentrant arrhythmias incorporating the HPS may also be more likely to be initiated by abrupt short-to-long changes in CL than by a constant CL. These considerations require further study.

We thank Ms. Ann Edwards, Mr. Brian Miller, and Mr. Ray Grenier for their assistance in the preparation of this manuscript.

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Circulation. 1984;69:26-32
doi: 10.1161/01.CIR.69.1.26
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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