Effects of Abrupt Changes in Cycle Length on Refractoriness of the His-Purkinje System in Man

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SUMMARY Abrupt changes in cycle length (CL) occur frequently in the clinical setting of atrial fibrillation. However, the effects of such changes on the His-Purkinje system (HPS) have not previously been considered during aberrant ventricular conduction (VAb). In 12 patients who manifested VAb with atrial premature stimulation (A2) during sinus rhythm, the relative refractory period (RRP) of the HPS was evaluated during a constant atrial CL (method I) and during abrupt changes in the CL (method II), wherein the A2 was coupled to an atrial CL (last A1A1) comparable to method I. This last A1A1 during method II was preceded by a series of constant atrial CLs 100–200 msec longer (method IIA) in 11 of 12 patients, or 100–300 msec shorter (method IIB) in all 12 patients. Although abrupt changes in the atrial CL using method IIA resulted in a longer HH interval (by 0–30 msec; mean 13.2 ± 9.2 msec) preceding the A2, the RRP-HPS was 5–20 msec shorter (mean 9.3 ± 5.3 msec) compared with method I in eight patients. The effect of abrupt changes was further evaluated in nine patients using method III, with a constant atrial CL, with a duration equal to the last H2H2 interval of method IIA. The VAb that occurred with method III was not manifested with method IIA in seven of nine patients, and in two patients the RRP-HPS was the same or less. Conversely, method IIB resulted in a shorter HH interval (by 0–110 msec; mean 28.9 ± 21.1 msec) preceding A2, but in 10 patients, RRP-HPS was 5–40 msec longer (mean 20.7 ± 10.5 msec) than that of method I and in two, VAb was only manifested using method IIB. Further scanning with method III, derived from the HH interval immediately preceding A2 of method IIB, was performed in seven patients and compared with method IIB. Prolongation in the RRP-HPS was shown using the latter method. The results indicate that abrupt changes in CL influence the functional behavior of the HPS in a manner not anticipated. Such changes may have important implications in determining the occurrence of VAb during atrial fibrillation.

THE PHENOMENON of aberrant ventricular conduction (VAb) during antegrade propagation of premature supraventricular impulses has been recognized for many decades.1-7 The classic paper by Gouaux and Ashman described the occurrence of VAb during atrial fibrillation in association with the sequence of a long cycle length (CL) followed by short CL.1 Intracardiac electrophysiologic techniques and His bundle electrogram recordings have demonstrated that such VAb is a result of functional conduction delay in the His-Purkinje system (HPS) and have confirmed the physiologic relationship between CL and VAb: a long CL followed by an antegrade propagated premature supraventricular impulse favors the occurrence of VAb, while a short CL decreases the likelihood of VAb.8-13 that is, the relative refractory period (RRP) of the HPS varies directly with CL.

Such refractory period determinations are usually performed during a constant CL or sinus rhythm and do not consider the varying CL pattern characteristic of atrial fibrillation.5-13 Although varying CL provides the setting for long CL—short CL sequences and, therefore, engenders the occurrence of VAb, the effect, if any, on VAb of variation in the cycle length preceding (CLp) the long CL has not been determined. We studied the effects on the functional properties of the HPS of varying CLp. In this report, we discuss several new findings and their clinical implications.

Materials and Methods

The 12 patients (six males and six females, ages 22–77 years) included in this series were studied for a variety of reasons, including palpitations, syncope and previously suspected or documented tachyarrhythmias. The underlying structural heart disease was arteriosclerotic in four, primary myocardial in two and valvular in one. Five patients had no detectable structural heart disease. Electrophysiologic studies were performed in the postabsorptive, unsedated state.4 The nature of the procedure was explained and each patient gave signed consent. Under local anesthesia, multipolar electrode catheters were introduced percutaneously and, with fluoroscopic guidance, were positioned in the high right atrium, atrioventricular junction, and right ventricle to record local electrical activity or for local pacing. All intracardiac electrograms (filter frequency 30–500 Hz), three surface ECG leads (I, II and V1) and time lines were simultaneously displayed on a multichannel oscilloscope and recorded on magnetic tape. Programmed electrical stimulation was accomplished with a digital stimulator delivering rectangular impulses with adjustable amplitude and duration. All equipment was grounded. Patients were electrically isolated during the studies.

Patients were studied with the conventional technique of atrial premature stimulation (A2), designated as method I (fig. 1), wherein the A2 was introduced after a series of six atrial beats of predetermined CLs (A1A1). To evaluate the effect of abrupt variation in CL on aberrant conduction, all patients were also studied
METHOD I: \( \text{CL}_p = \text{CL}_r \)
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_r \\
\end{array}
\]
METHOD II A: \( \text{CL}_p > \text{CL}_r \)
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_r \\
\end{array}
\]
METHOD II B: \( \text{CL}_p < \text{CL}_r \)
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_p & \text{A}_1 & \text{CL}_r \\
\end{array}
\]

Figure 1. Stimulation protocols. The main difference between methods I and II is that in method I the \( \text{A}_1 \) is preceded by a series of constant atrial CLs, whereas in method II the CL just before \( \text{A}_1 \) is abruptly altered (\( \text{A}_1, \text{A}_1' \)) compared with the preceding CLs (\( \text{A}_1, \text{A}_1 \)). In all methods, the reference CL (\( \text{CL}_r \)) is the atrial CL to which \( \text{A}_1 \) is coupled, whereas the CLs preceding the \( \text{CL}_p \) are designated as \( \text{CL}_p \). In method IIA, the \( \text{CL}_p > \text{CL}_r \), whereas in method IIB, the \( \text{CL}_p < \text{CL}_r \). In method I, \( \text{CL}_p = \text{CL}_r \).

with method II (fig. 1), in which, after a series of constant atrial CLs, the last \( \text{A}_1, \text{A}_1' \) (i.e., immediately preceding the \( \text{A}_1 \)) was altered. The data presented here include analysis of the effect on aberrant conduction of both the last and next-to-last \( \text{A}_1, \text{A}_1' \) cycles; therefore, the two are designated differently to avoid confusion. The last \( \text{A}_1, \text{A}_1' \) cycle during both methods I and II is designated as the reference CL (\( \text{CL}_r \)); all CLs preceding the \( \text{CL}_p \) are called \( \text{CL}_p \). The stimulation technique of method II is further divided into A and B (fig. 1), depending on whether the \( \text{CL}_p \) was larger or smaller. All \( \text{A}_1, \text{A}_1' \) cycles preceding the \( \text{CL}_r \) are of the same duration and equal \( \text{CL}_p \) (fig. 1). In the schema and tracings, the atrial and His deflections of the altered CL (\( \text{CL}_p \)) of method II are depicted as \( \text{A}_1, \text{A}_1' \) and \( \text{H}_1, \text{H}_1' \), for a clearer separation from all other constant CLs.

With method II, by pacing design, only the atrial (rather than HH) CL could be altered from \( \text{CL}_p \) to \( \text{CL}_r \). However, the occurrence of aberrant conduction relates to the HH rather than the AA CL unless the two are identical. The \( \text{A}_1, \text{A}_1' \) equals \( \text{H}_1, \text{H}_1' \) with method I and also during the \( \text{CL}_p \) of method II, but not during the \( \text{CL}_p \) of method II. How the \( \text{A}_1, \text{A}_1' \) or \( \text{H}_1, \text{H}_1' \) could differ from the corresponding \( \text{H}_1, \text{H}_1' \) CL is shown in figure 2. The terms \( \text{CL}_p \) and \( \text{CL}_r \) refer to only the atrial CL, whereas the corresponding HH CLs are depicted as \( \text{CL}_p \)-HPS and \( \text{CL}_r \)-HPS. For a given \( \text{CL}_p \), a longer \( \text{CL}_p \) lengthens \( \text{CL}_r \)-HPS and a shorter \( \text{CL}_p \) shortens \( \text{CL}_r \)-HPS. The amount of change is equal to the difference in AH intervals between \( \text{CL}_p \) and \( \text{CL}_r \). Therefore, although the atrial CL (\( \text{CL}_r \) of method I) measuring exactly equal to the atrial \( \text{CL}_p \) of methods IIA and IIB was available in all patients, due to small but definite differences between atrial \( \text{CL}_p \) and \( \text{CL}_r \) CLs during method II, additional atrial CLs were scanned; these are referred to as method III (fig. 3). This was done in order to obtain a constant \( \text{H}_1, \text{H}_1' \) CL equal to the \( \text{H}_1, \text{H}_1' \) interval of methods IIA and IIB. During method III, the \( \text{A}_1, \text{A}_1' \) and \( \text{H}_1, \text{H}_1' \) CLs are constant, as in method I. However, the exact CL of method III could be derived only after obtaining the \( \text{H}_1, \text{H}_1' \) values of methods IIA and IIB by direct measurement; hence the designation of method III.

Definition of Terms

\( \text{A}_1, \text{H}_1, \text{V}_1 \) — the atrial, His bundle, and ventricular depolarizations during all constant atrial CLs.

\( \text{A}_1', \text{H}_1', \text{V}_1' \) — the atrial, His bundle, and ventricular depolarizations of the altered CL just before \( \text{A}_1 \) during method II.

\( \text{A}_2, \text{H}_2, \text{V}_2 \) — the atrial, His bundle, and ventricular depolarizations resulting from coupled premature atrial stimulation.

Functional refractory period (FRP) of the atrioventricular (AV) node — the shortest \( \text{H}_1, \text{H}_1' \) interval in response to any \( \text{A}_1, \text{A}_1' \).

RRP of the HPS — the longest \( \text{H}_1, \text{H}_1' \) interval at which \( \text{H}_1 \) conducts to the ventricles with a longer HV interval than the basic drive beat or with a QRS showing a definite bundle branch block (BBB) pattern.

Results

None of the patients had evidence of preexisting BBB, although in three cases a nonspecific intraventricular conduction defect was noted on the resting ECG. All patients manifested VAb with either premature atrial stimulation (\( \text{A}_1 \)) at atrial paced CLs slightly shorter than sinus CL or with \( \text{A}_1 \) coupled to sinus beats. Patients were in sinus rhythm and had normal AH and HV intervals and were not taking any cardioactive medication at the time of study. The electrophysiologic data are listed in table 1. During VAb, a right BBB (RBBB) morphology was noted in eight patients, a left BBB (LBBB) configuration in one (patient 7), and both RBBB and LBBB morphologies in three (patients

METHOD I
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{H}_1 & \text{CL}_p \text{HPS} & \text{A}_1 & \text{CL}_r \text{HPS} & \text{A}_1 \\
\end{array}
\]
METHOD II A
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{H}_1 & \text{CL}_p \text{HPS} & \text{A}_1' & \text{CL}_r \text{HPS} & \text{A}_1' \\
\end{array}
\]
METHOD II B
\[
\begin{array}{c|c|c|c|c|c}
\text{A}_1 & \text{H}_1 & \text{CL}_p \text{HPS} & \text{A}_1' & \text{CL}_r \text{HPS} & \text{A}_1' \\
\end{array}
\]
2, 5 and 9). The pertinent data for each CL are provided in table 1.

The range in which CLₚ was varied was limited at longer CLs by the ability to maintain constant atrial capture without spontaneous sinus beats and at shorter CLs by the occurrence of pacing-induced AV nodal Wenckebach phenomenon. Within these limits, CLₚ was 100–200 msec longer than CLᵣ or 100–300 msec shorter than CLᵣ.

A change of atrial CL from CLₚ to CLᵣ (method II) generally produced a change in CLᵣ-HPS that did not exactly equal the atrial CLₚ due to a concomitant change in AV nodal (AH) conduction (fig. 2). In fact, the change in the atrial CLᵣ resulted in reciprocal change in the corresponding HH' CL. During this study, when atrial CLₚ was longer than CLᵣ (method IIA), the CLᵣ-HPS was 0–30 msec longer (mean 13.2 ± 9.2 msec) than the corresponding atrial CLᵣ (or CLᵣ-HPS of method I). Likewise, when the atrial CLₚ was shorter than CLᵣ (method IIB), the CLᵣ-HPS was 0–110 msec shorter (mean 28.9 ± 21.1 msec) than the corresponding atrial CLᵣ (or CLᵣ-HPS of method I). The greater change in CLᵣ-HPS at shorter CLₚ was attributed to a greater effect on AV nodal conduction at a shorter paced atrial CL.

**Effect of a CLₚ Longer Than CLᵣ on VAb (table 1)**

The effect of method IIA, with which the CLₚ was longer than the CLᵣ, could be compared in 11 of 12 patients (nos. 1–11) with method I (table 1). At the same atrial CLₚ, the RRP-HPS was 5–20 msec (mean 9.3 ± 5.3 msec) shorter with method IIA than with method I in eight patients (nos. 1–4, 7–9 and 11) (figs. 4A–C) at one or more CLs. Patients 5 and 6 showed a 10–20-msec increase in the RRP-HPS with method IIA compared with method I. In patient 10, the FRP of the AV node limited determination of the RRP-HPS during method IIA. In nine cases (patients 1–4 and 6–10), the CLᵣ-HPS during method IIA exceeded the atrial CLᵣ of method I by 5–30 msec despite the same atrial CLᵣ. Method III was therefore used to approximate the H₁H₁' CL of method IIA to detect small changes in the RRP-HPS that might have been caused by subtle variation in the HH CL between the CLᵣ of methods I and IIA. In patients 5 and 11, the CLᵣ-HPS with method IIA was the same as during method I and, therefore, derivation of method III was unnecessary.

Analysis of RRP-HPS showed that this variable either decreased or did not change whenever the CLᵣ-HPS of method IIA was compared with CLᵣ-HPS of the same but constant duration, i.e., method I or III (table 1). The shortening of RRP-HPS with method IIA was particularly noticeable in patients 1, 2, 4, 7, 8, 9 and 10 at CLₚ-CLᵣ sequences of 700/500, 800/600, 800/600, 800/600, 600/500, 800/600 and 650/500, where the RRP-HPS shortened to less than the FRP of
1. However, tated shorter manifestation the CLp H,HI' CLR produced proportionately longer patients, for the AV III, that the AVB could not be achieved and were limited by the AV nodal FRP (figs. 4E and F).

Effect of a CLp Shorter Than CLR on VAb (table 1)
The influence of method IIb, with which the CLp was shorter than the CLR, was compared with method I in all patients at one or more basic CLs (table 1). At the same CLR as method I, the RRP-HPS with method IIb was 5–40 msec longer (mean 20.7 ± 10.5 msec) in 10 of 12 cases (fig. 5A; compare with fig. 4A). This occurred although the CLp-HPS during method IIb was invariably shorter than with method I. In the other two patients (nos. 8 and 12), VAb was noted only during method IIb at comparable H1H2, which did not produce VAb with method I (fig. 6), although the CLp-HPS during the latter was longer. In seven patients (nos. 1–5, 9 and 10) an H1H2' CL equal to the CLp-HPS of method IIb was scanned using the method III.

### Table 1. Electrophysiologic Data

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<td>400/600 430</td>
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<td>400/600 650 445</td>
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Values listed as < under RRP-HPS represent the functional refractory period of the atrioventricular node. All electrophysiologic data are in msec.

Abbreviations: Clp = atrial preceding cycle (A1A2); CLR = atrial reference cycle length (A1A2 or A1A2'); CLp-HPS = reference cycle length His-Purkinje system (H1H2 or H1H2'); RRP-HPS = relative refractory period of the HPS.
(table 1). As expected, compared with method III, the RRP-HPS with method IIB showed even a greater degree of prolongation at most CLs tested (figs. 5A–F).

When the atrial CL was altered before A₂, such that the CLₚ was shorter than CLₕ (figs. 5 and 6), three events were consistently noted: (1) The H₃H₄′ CL preceding A₂ was shorter than if the A₁ was coupled to a series of constant CLs, i.e., CLₚ = CLₕ. (2) The magnitude of change from CLₚ-HPS to CLₕ-HPS was proportional to the degree of change from CLₕ to CLₚ. (3) Despite the shorter CLₕ-HPS with method IIB, the RRP-HPS was invariably longer than the RRP-HPS during method I, and the appearance of VAb therefore generally occurred at longer A₁A₂ and H₁H₂ intervals than during method I or III. The VAb at times could not be demonstrated at constant atrial CLs (methods I and III) with a short CLₚ, but was achieved at a comparable CLₚ or shorter CLₚ-HPS using method IIB.

**Effect of Varying CLₚ at the Same CLₕ**

At the same CLₕ, two or more values for CLₚ shorter than CLₕ were available in seven patients (nos. 1–3, 5, 7, 9 and 11, table 1). The difference between the CLₚ and CLₕ in these cases was 100–300 msec. When the RRP-HPS with method IIB exceeded the RRP-HPS of method I at a minimum CLₚ → CLₕ difference, an increment in the CLₚ → CLₕ difference generally resulted in a further increase of 5–35 msec in the RRP-HPS. This occurred despite a shorter CLₕ-HPS with a maximal CLₚ → CLₕ difference. However, for a given

**FIGURE 4. Effect of method IIA on aberrant ventricular conduction.** These tracings, taken from patient 1, show the relative refractory period of the His-Purkinje system (RRP-HPS) during methods I, II A and III. (A) With method I, complete right bundle branch block (RBBB) is noted at an H₂H₃ interval of 390 msec. (B and C) A change of CLₚ → CLₕ from 700–600 (method II) shortens the RRP-HPS such that only an incomplete RBBB occurs at an H₂H₃ of 370 msec. The H₂H₄′ CLs in panels B and C were 610 msec. The latter CL is also scanned with method III (panel D) and shows that the RRP-HPS is even longer (400 msec) compared with method IIA (panel C). (E) A change of CLₚ → CLₕ from 700 to 500 msec. The RRP-HPS results in a decrease of this variable. The H₃H₄′ CL is 510 msec. (F) This CL is scanned with method III and again shows that RRP-HPS is longer than with method IIA (E). I and V₁ = surface ECG leads; HRA = high right atrial electrogram; HBE = His bundle electrogram; T = time lines.
CL<sub>R</sub>, we could not predict the relative increase in RRP-HPS for any CL<sub>p</sub> → CL<sub>R</sub> difference.

At the same CL<sub>R</sub>, two CL<sub>p</sub> values longer than the CL<sub>R</sub> were available in only three patients (nos. 3, 5 and 8). Insufficient data were therefore available to draw conclusions about the effect of varying CL<sub>p</sub> at the same CL<sub>R</sub> on VAb when the CL<sub>p</sub> is longer than the CL<sub>R</sub>.

**Discussion**

The VAb in relation to CL was originally described by Lewis<sup>1</sup> and was attributed by Scherf<sup>2</sup> to the effect of CL on refractory period. More recently, electrophysiologic studies by Moe et al.<sup>5</sup> in the canine heart and by Wit et al.<sup>7</sup> in the human heart have shown that VAb in relation to early premature supraventricular beats is due to conduction delay in the HPS and is a functional property of the normal conduction system.<sup>5,7</sup> It is now universally accepted that the onset of VAb or RRP-HPS is CL-dependent, varying directly with CL.<sup>9-13</sup>

These studies are consistent with the observation of VAb during atrial fibrillation, where the sequence of long followed by short CL can occur frequently. Since the report by Gouaux and Ashman,<sup>3</sup> it has become accepted clinical practice to distinguish VAb from ventricular beats by such CL changes.<sup>4</sup> Therefore, while general clinical observation during atrial fibrillation and electrophysiologic studies have described the phenomenon of VAb as it relates to long CLs followed by short coupling intervals, the effect on VAb of variations in the interval preceding the long CL has not been described.

In this report, the effect on VAb of the CL<sub>p</sub> of the long-short CL sequence was systematically studied. The results indicate that the RRP-HPS is appreciably altered by CL<sub>p</sub> such that the RRP-HPS may shorten or the functional BBB may be abolished when the long CL is preceded by an even longer CL. Conversely, a functional BBB may first become manifest and/or
RRP-HPS lengthened if the long CL is preceded by relatively shorter CL. Moreover, the findings show that the dictum that RRP-HPS varies directly with CL must be reevaluated. While this characteristic of the HPS is true when CLₚ is constant, it is not what occurs in most patients when CLₚ is changing. In such instances, the RRP-HPS generally varies inversely with CLₚ, i.e., the RRP-HPS of a given CL will be longer if preceded by a shorter CL and shorter if preceded by a longer CL.

A possible explanation for these findings may again relate to the effect of varying CLₚ on CLₚ-HPS, as shown in figure 7. In all panels, the CLₚ is 700 msec. In panel A, where CLₚ is also 700 msec, the CLₚ-HPS is 700 msec. However, when CLₚ is varied such that it is 900 msec (B), the CLₚ-HPS is longer (720 msec) and when CLₚ is 500 msec (C), the CLₚ-HPS is shorter (650 msec) than the corresponding atrial CLₚ. Therefore, compared with panel A, one would expect the RRP-HPS in panel B to be longer and RRP-HPS in panel C to be shorter. However, the increase in CLₚ-HPS in panel B is the result of a shortening in HPS CL from 900 to 720 msec and the decrease in CLₚ-HPS in panel C is the result of a lengthening in HPS CL from 500 to 650 msec. Thus direction of change in the HPS CL significantly alters the findings expected from considering absolute HPS CL alone.

In addition, the shorter CLₚ lengthened the RRP-HPS more than longer CLₚ shortened RRP-HPS, although the shorter CLₚ lengthened the CLₚ-HPS more than longer CLₚ lengthened CLₚ-HPS. This may indicate that the HPS is also sensitive to rate of change in CL. Again, using the schema in figure 7, where CLₚ is 900 msec and CLₚ-HPS is 720 msec, the percent shortening for the HPS CL is 180/900, or -20%, and where CLₚ is 500 msec and CLₚ-HPS is 650 msec, the percent lengthening for HPS CL is 150/500, or 30%. For the same variance in CLₚ, relative to CLₚ, the percent lengthening will be greater than the percent shortening, and therefore, if the HPS is sensitive to
rate of change in CL, changes of short to long CL will have a greater effect on RRP-HPS than equal changes of long to short CL.

This explanation only provides some insight into the directional changes regarding the onset of VAb with a given alteration in CL\(_p\) relative to CL\(_r\); the present results do not explain the mechanism of the observed phenomena. The adjustment in refractory period of the HPS to a change in CL, although it starts immediately, takes several CLs to stabilize. The number of beats required to achieve this new steady state seems to vary considerably in different species.\(^{14, 15}\) It is tempting to postulate, therefore, that the refractoriness of the HPS in man may also be a dynamic phenomenon that is continuously influenced by absolute duration, rate and direction of change of several preceding CLs. A sudden change in the CL may produce an overadjustment in the duration of RRP-HPS in the direction dictated by the preceding sequence of events. This could explain why the RRP-HPS values with CL\(_p\)-HPS shorter than CL\(_r\)-HPS sequence may exceed those expected from a CL\(_r\)-HPS of constant duration. Similarly, a CL\(_p\)-HPS longer than the CL\(_r\)-HPS sequence may produce RRP-HPS values shorter than the CL\(_r\)-HPS of a constant CL.

A fatigue effect after short CLs (i.e., a CL\(_p\) shorter than a CL\(_r\)) and faster recovery after CL\(_p\) longer than CL\(_r\) may have produced time-dependent changes in the RRP-HPS. This mechanism would not explain the present results because in several of these cases, relatively short CLs (400–500 msec) were also scanned with method I (table 1), and yet, the RRP-HPS was always shorter compared with that during longer CLs. In fact, in patients 1 and 10 (table 1), the RRP-HPS was not encountered despite achievement of H\(_1\)H\(_2\) values measuring significantly less than longer CL where VAb was clearly noted. Whether similar or other mechanisms are operative cannot be determined from this study.

Although an exact model of atrial fibrillation requires an extremely complex stimulation protocol, this study has shown the pronounced effect on the functional behavior of the HPS of the CL before the long-short sequence usually described. Such effects include significant changes in RRP-HPS such that VAb may occur at longer or shorter coupling intervals than expected when CL\(_p\) is not considered. In a setting of atrial fibrillation where the long CL could be preceded by considerably longer or shorter CL, this could lead to the misdiagnosis of VAb as beats of ventricular origin. The coupling intervals (RR) of short CL at the onset of VAb could vary markedly during atrial fibrillation due
to the wide range of CLp values that may be recorded in these settings. During this study, the range of long and short CLs was limited by spontaneous sinus CLs and pacing-induced onset of AV nodal Wenckebach phenomenon, respectively.

This simplified model demonstrates that the absolute CL preceding premature supraventricular beats is not always the only determinant of VAb, but that the direction and rate of change in CLs preceding the long-short CL sequence both determine VAb in the human heart.

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