

Gradual prolongation of PR interval followed by a pause. What's the mechanism?

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Title page

Title: Gradual prolongation of PR interval followed by a pause. What's the mechanism?

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CASE

A 68-year-old lady with a history of diabetes mellitus and hypertension was diagnosed with nonischemic dilated cardiomyopathy (left ventricular ejection fraction of 30%). She presented with one episode of syncope. The 12-lead electrocardiogram (ECG) revealed what seems to be sinus rhythm with 4:3 AV Wenckebach pattern, atypical right bundle branch block (RBBB) with left anterior hemiblock, QRS duration of 150 ms and a frontal QRS axis of -30 degrees. Figure 1 shows 12-lead ECG along with the distal and proximal bipolar His catheter signals obtained during the electrophysiology study. What is the rhythm that the intracardiac signals reveal?

DISCUSSION

A closer look at the 12-lead ECG (Figure 1) reveals findings suggestive but not typical of Wenckebach pattern because the 1stPR interval after the pause is longer than that of the first beat of the tracing and hence raises a question about the nature of the rhythm.

The P waves are of sinus morphology and appear to occur at constant intervals. Further analysis of the intracardiac electrograms (EGMs) points to three distinct signals. Apart from the obvious ventricular (V) signals on the distal His electrode, the signals with largest maintain a constant relationship with the P waves and likely represent atrial activity. The lowest amplitude signals which precede the apparent atrial signals (best seen in the first beat of the trace), occur at a regular timing and are likely to be the His potentials (as annotated in Figure 2). They are related with Ventricular signals in a 4:3 Wenckebach pattern. Of note, the varying HA intervals suggest dissociation. As inferred from the explanation in figure 2, it is clear that A-A and H-H intervals are slightly dissimilar, the HA intervals are not fixed and hence, are dissociated isorhythmically suggesting Hisian automaticity competing with sinus rhythm. The H-V intervals, however, are progressively increasing until the fourth H blocks and fails to activate the ventricle, thus suggesting a 4:3 infra-Hisian Wenckebach. The characteristic V-V interval shortening before the pause during a typical Wenckebach phenomenon is also seen (depicted with a ladder diagram in figure 2). To note, in this tracing, there is no opportunity for the Hisian impulse to capture the atrium retrogradely.

One other possibility could be that each atrial impulse conducts antegradely to the subsequent His with a very long AH interval and then to the ventricle in a Wenckebach pattern. However, the constant HH intervals with varying AA intervals is against this possibility.

At times (Figure 3A), when the Hisian automaticity usurped the sinus rate (represented by asterisk), the atrium was captured retrogradely as can be inferred by the inverted P waves in inferior leads (Figure 3B). Thus, there was intermittently an H-A association with regular HA intervals. However, the infra-Hisian Wenckebach pattern persisted in varying ratio, throughout the study. Here again, the possibility of an ectopic atrial rhythm conducting down to the next H cannot be ruled out despite the constant HH intervals.

During the EP study, it was noted that whenever the ventricle was captured by RV apical pacing, the infra-Hisian conduction system remained perpetually refractory and there was no antegrade ventricular capture by Hisian automaticity even at a ventricular pacing rate of 50 bpm.

No ventricular tachycardia was inducible during the EP study.

The patient eventually had a dual-chamber pacemaker without any drugs to suppress the automatic focus. She had nonischemic cardiomyopathy, and cardiac MRI had ruled out inflammatory or infiltrative pathologies.

Hence the rhythm in figure 1 can be described as ‘Hisian automaticity in isorhythmic dissociation with the atrium and infra-Hisian typical Wenckebach conduction’.

This is in accordance with Occam’s Razor where a single explanation (of Hisian automaticity) gains precedence over two separate anomalies (abnormally long AV nodal conduction and an ectopic atrial rhythm) to explain the same phenomenon.

Gradual prolongation of PR interval followed by a pause usually represents Atrioventricular (AV) Wenckebach and has classically been considered an AV nodal phenomenon. However, an open mind should be kept

regarding less common mechanisms as presented in this unusual case. Infra-Hisian Wenckebach has been scarcely reported in the literature. (1) The distinction, sometimes, becomes critical as infra-Hisian Wenckebach is a harbinger of complete AV block and demands permanent pacing contrary to AV nodal Wenckebach. (2)

In summary, our case documents a rare and hitherto unreported phenomenon of persistent Hisian automaticity in isorhythmic dissociation with the atrium and varying patterns of infra-Hisian Wenckebach conduction.

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Figure legends:

Figure 1 shows 12 lead ECG lined up with intracardiac His electrogram (His-d represents the distal bipole and His-p represents the proximal bipole) traces

Figure 2 Explanation for Figure 1

Upper panel corresponds to the 12 lead ECG and intracardiac EGMs as in figure 1 (with annotated EGMs). It shows that the atrial and His potentials are occurring near simultaneously and hence could be isorhythmically dissociated or each His potential could be the result of the previous atrial potential. There is progressive HV prolongation and RR shortening followed by a block; Annotations H- His, A- atrium, V- ventricle. The origin of atrial depolarisation is depicted by the dotted lines

Lower panel shows *ladder diagrams* corresponding to the intracardiac EGMs depicts 4:3 infra-Hisian Wenckebach sequence and presumed isorhythmic dissociation of atrium and His signals.

Figure 3

At times (Figure 3A), when the Hisian automaticity usurped the sinus rate, the atrium was captured retrogradely as can be inferred by the inverted P waves in inferior leads. Thus, there was intermittently, a H-A association with regular HA intervals (Figure 3B).

Panel A shows 12 lead ECG, intracardiac EGMs and corresponding ladder diagrams, which depicts atrioventricular dissociation with the Junctional/ Hisian escape rate exceeding the sinus rate leading to 1:1 retrograde conduction and activation of the atrium (note the transition in P wave morphology in the fourth beat- fusion in the atrium between sinus and His discharge depicted in the ladder diagram attached)

Panel B corresponds to 12 lead ECG, the intracardiac EGMs and corresponding ladder diagrams, which depicts atrioventricular dissociation with the Hisian escape rhythm conducting antegradely with 3:2 infra-Hisian Wenckebach sequence (note the progressive HV prolongation and then drop) and conducting retrogradely with a 1:1 HA (HA interval constant) association. The retrograde activation of atrium is also evident from the surface P wave morphology (onset marked by dotted lines); Annotations H- His, A- atrium, V- ventricle. Ladder diagrams {interatrial intervals (763 ms), inter-Hisian intervals (763 ms) and V-V intervals (763ms) are depicted in milliseconds}; corresponds to the intracardiac EGMs in panel A. HV progressively prolongs followed by the block to the ventricle

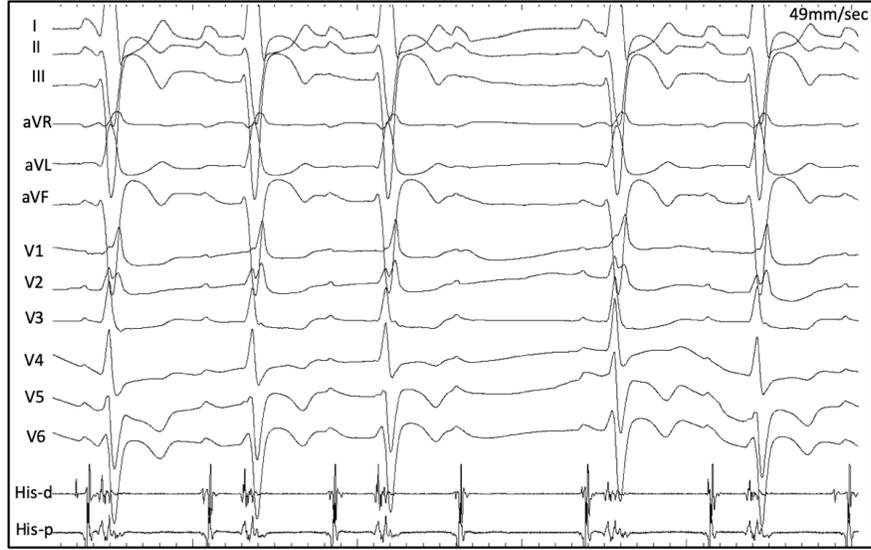


Figure 1

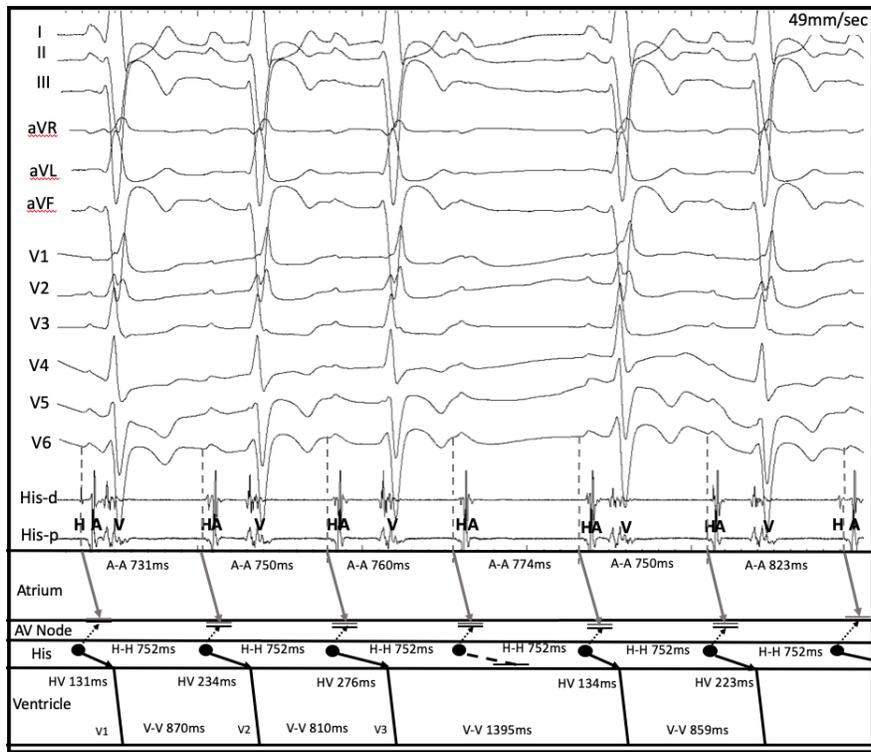


Figure -2

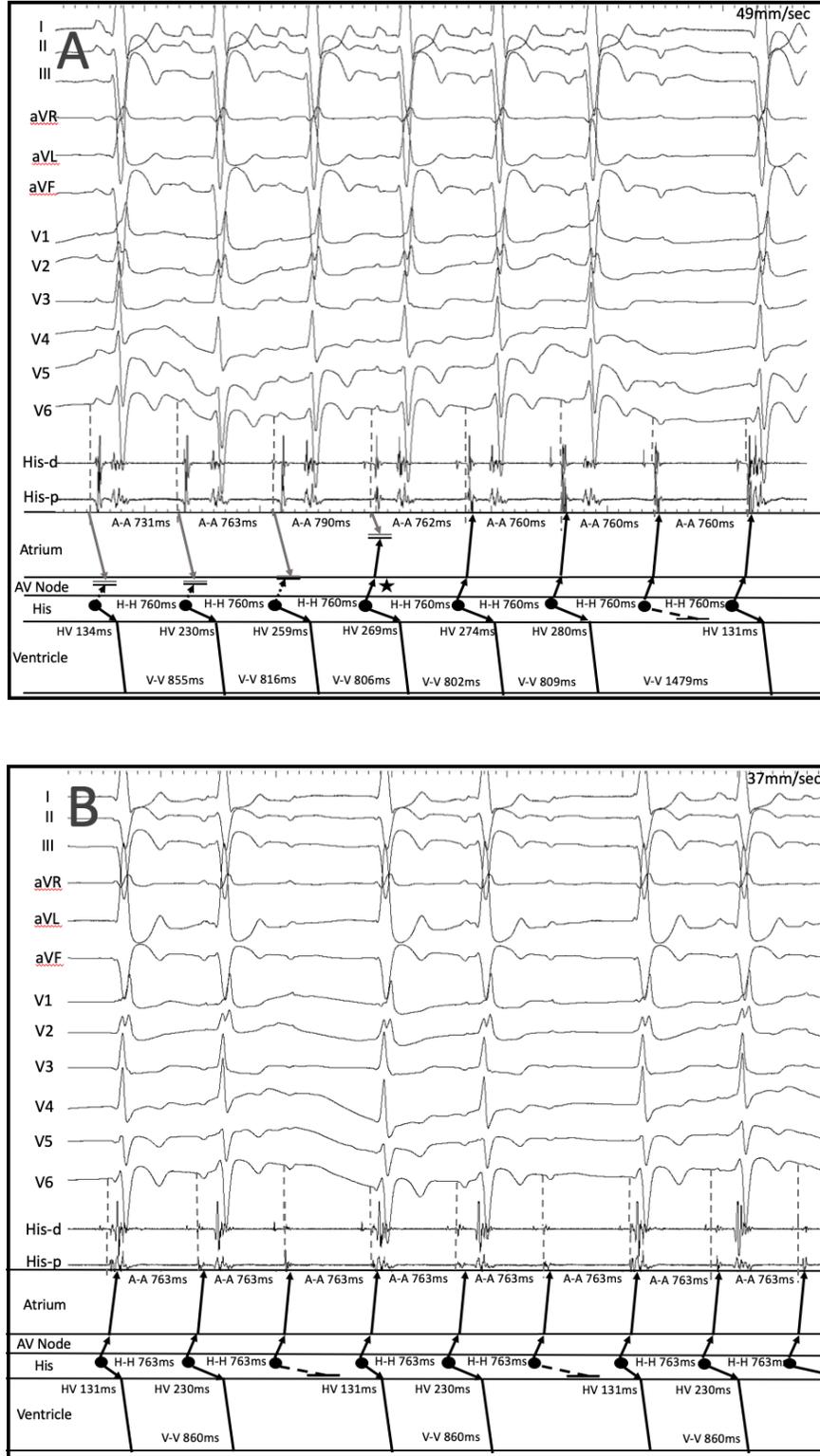


Figure 3