Paradoxical increase of stimulus to atrium interval despite His-bundle capture during para-Hisian pacing

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Para-Hisian pacing at high output simultaneously captures the His bundle or proximal right bundle, as well as the adjacent ventricular myocardium. At lower output, direct His-bundle, or right-bundle, capture is lost which causes an increase in the stimulus to atrium interval. We describe a case with an increment of 68 ms with capture of the His bundle. This seems to be a paradoxical response, which however can be explained by the presence of retrograde dual AV-node physiology. Continuation of this phenomenon could be related to concealed anterograde invasion of the fast pathway thereby maintaining the retrograde activation during para-Hisian pacing on the slow pathway despite paced cycle lengths (His to His intervals) where retrograde fast pathway conduction proved to be possible.

Introduction
The para-Hisian pacing site is unique because it is anatomically close but electrically distant from the His bundle. Para-Hisian pacing at high output simultaneously captures the His bundle or proximal right bundle, as well as the adjacent ventricular myocardium.

At lower output, direct His-bundle, or right-bundle, capture is lost and retrograde activation of the His bundle is delayed because the His bundle and right bundle are insulated from the adjacent myocardium and the peripheral input to the Purkinje system is located far from the para-Hisian pacing site.

When the right ventricle (RV) and His bundle are captured simultaneously, the wavefront travels down the His-Purkinje system which results in a relatively narrow, almost normally shaped QRS. The wavefront can also travel retrogradely over the AV node to activate the atrium with a stimulus to atrium (SA) interval that represents conduction time over the proximal part of the His bundle and AV node.

When the ventricle is captured, but not the atrium or His bundle, the wavefront travels through the ventricle by muscle to muscle conduction resulting in a wide QRS with left bundle branch morphology. Once the wavefront reaches the distal RV, it conducts retrogradely up the right bundle and then over the His bundle and AV node to activate the atria. In this case, the SA interval represents the conduction time from the RV base to the His bundle plus the conduction time over the His bundle and AV node. Thus, normally, para-Hisian pacing results in a shorter SA interval when the His bundle is captured than when only the RV is captured. A potential pitfall is widening of the paced QRS complex during para-Hisian pacing despite His-bundle capture due to loss of left bundle branch capture.

In the presence of a septal AV-bypass tract, the SA interval usually remains fixed regardless of whether or not the His bundle is being captured, because in both cases the impulse travels retrogradely over AV-bypass tract, with constant conduction time to the atrium as long as local ventricular myocardium is being captured.

We describe a case with an ‘increment’ of the SA interval despite direct His-bundle capture.

Case report
In a 41-year-old woman with symptomatic palpitations, an electrophysiological study was performed. Endocardial catheters were placed via the femoral veins in the His-bundle region, coronary sinus (CS) and right ventricular apex.

Para-Hisian pacing was performed to exclude a septal bypass tract.

With stimulus to stimulus intervals of 600 ms and direct capture of the His bundle, a decrement in the SA interval of 80 ms when compared with non-capture of the His bundle (Figure 1) was observed. This is a normal response when no fast retrogradely conducting septal AV-bypass tract is present.

However, with a stimulus-to-stimulus interval of 530 ms (and shorter) reproducibly, an increment of 68 ms with capture of the His bundle (narrow QRS) was noticed (Figure 2).

This seems to be a paradoxical response, which however can be explained by the presence of retrograde dual AV-node physiology.

When looking carefully at Figure 2, the QRS complex with direct His-bundle capture (third, narrow QRS complex) leads to first retrograde atrial activation in CS 9–10. This suggests conduction over the CS os or base of the triangle of Koch instead over the anteroseptal region (i.e. HBE).
Figure 1  Para-Hisian pacing at a cycle length of 600 ms: the first two (wide) QRS complexes do not capture the His bundle directly, leading to an SA interval of 140 ms. The following (narrow) QRS complexes capture the His bundle directly resulting in a shorter SA interval of 60 ms. Both show first retrograde activation in the HB signals (fast pathway region).

Figure 2  Para-Hisian pacing at a cycle length of 530 ms: the first two (wide) QRS complexes do not capture the His bundle directly leading to an SA interval of 140 ms (identical to Figure 1) with first retrograde activation on the HB signals (fast pathway region). Although the third (narrow) QRS complex captures the His bundle directly, this results in an increase of the SA interval to 208 ms. This paradoxical behaviour can be explained by retrograde block in the fast pathway leading to conduction over the slow AV-nodal pathway (first retrograde atrial activation in CS 9–10 instead of HB) and a paradoxical increase of the SA interval despite His capture (see text for details). The following SA intervals remain prolonged because retrograde activation remains on the slow pathway area as a result of concealed anterograde invasion of the fast pathway.
Thus, the activation pattern indicates retrograde conduction over the slow AV-nodal pathway area instead of conduction over the fast AV-nodal pathway. Presence of dual retrograde AV-nodal physiology was also confirmed with decremental ventricular pacing and single retrograde AV-nodal echo beats.

An explanation for the observed behaviour could be close proximity of the stimulus site to the posterior AV-nodal extension that might represent the slow pathway. This would also account for the earliest atrial activation in the proximal CS.

However, it does not clarify why the paradoxical para-Hisian response was related to the decrement in the paced cycle length from 600 to 530 ms.

Importantly, the His-to-His interval from the paced wide QRS complex to narrow QRS complex is even shorter than 530 ms as the stimulus with non-His-bundle capture (first and second wide QRS complexes in Figure 2) activates the His only after the wavefront travels through the ventricle by muscle-to-muscle conduction, reaches the RV apex, conducts retrogradely up the right bundle, and then reaches the His bundle (Figure 3). Conversely, the following stimulus (third complex in Figure 2) captures the His bundle directly (narrow QRS), resulting in a factual His-to-His time shorter than 530 ms and retrograde conduction block in the fast AV-nodal pathway.

As the impulse now has to travel through the slow AV-nodal pathway (first retrograde atrial activation in CS 9–10), the SA time paradoxically increases despite direct His-bundle capture (Figure 3). Although the following QRS complex is also narrow (direct His-bundle capture, Figure 2), and the His-to-His time is now the same as the paced para-Hisian interval (530 ms), the retrograde conduction remains on the slow pathway. This is remarkable as retrograde fast pathway conduction proved to be possible at His-to-His intervals of 530 ms (first two complexes in Figure 2). Also with RV-apex pacing a retrograde jump with conduction over the slow pathway occurred only at cycle lengths lower than 420 ms.

The second phenomenon could be related to concealed anterograde invasion of the fast pathway thereby maintaining the retrograde activation during para-Hisian pacing on the slow pathway despite paced cycle lengths (His-to-His intervals) where retrograde fast pathway conduction proved to be possible previously (Figure 3).

No ablation of the slow pathway area was performed, which would have further supported our findings, because no AV-nodal re-entrant tachycardia could be evoked and no ECG registrations were made during the complaints of palpitations.

Conflict of interest: none declared.

References