Overcoming left bundle branch block by permanent His bundle pacing: Evidence of longitudinal dissociation in the His via recordings from a permanent pacing lead

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Introduction
Narrowing of the QRS and overcoming left bundle branch block (LBBB) with His bundle pacing (HBP) has been previously described.1–3 We present the first 2 reported cases of left bundle branch (LBB) delay with evidence of a split His electrogram during unipolar mapping from the tip of the His bundle (HB) lead during pacemaker implantation. These findings suggest that the site of LBB delay is intra-Hisian, thus further validating the theory of “longitudinal dissociation in the HB.”1

Case report
Case 1
The patient is a 94-year-old woman with ischemic cardiomyopathy and declining left ventricular ejection fraction (LVEF) despite optimal medical management. Over the past year her LVEF decreased from 35% to 20%, correlating with progression to NYHA class III heart failure. Her electrocardiogram was notable for a wide LBBB of 164 ms and she was referred for cardiac resynchronization therapy (CRT).

She was felt to be a reasonable candidate for CRT; however, given her advanced age and comorbidities a decision was made to consider a CRT pacemaker and not a CRT defibrillator. Owing to the patient’s frail stature without appreciable pectoral muscle, a decision was made to attempt resynchronization by placing the ventricular lead at the HB. We hoped to recruit the LBB and implant a smaller dual-chamber pacemaker generator.

The HB was mapped using unipolar recordings from a Medtronic 3830 lead (Medtronic Inc, Minneapolis, MN) through a His C315 sheath. HB recordings and pace mapping at different HB sites are shown in Figure 1. The final location of the HB lead resulted in nonselective HBP with recruitment of the LBBB and narrowing of the QRS to 90 ms. Total procedure time was 113 minutes and fluoroscopy time was 8.4 minutes.

Case 2
A 79-year-old man with a history of hypertension and type II diabetes was admitted with dizziness and fatigue. These episodes correlated with significant sinus bradycardia and junctional rhythm in the 30s. He was also noted to have a rate-related delay in the LBB at 60 beats per minute (bpm). A decision was made to implant a dual-chamber pacemaker for highly symptomatic sinus node dysfunction. Given the LBBB, we also planned for an HBP lead implant with an attempt to recruit the LBB delay.

We started with implantation of the atrial lead so as to unmask the rate-related delay in LBB. AAI pacing at different rates showed progressive delay in the LBB (Supplemental Figure 1). The HB was mapped using unipolar recordings from a Medtronic 3830 lead (Medtronic Inc, Minneapolis, MN) through a His C315 sheath. Figure 2 demonstrates HB recordings and pace mapping (5 V at 1 ms) at different HB sites. The final location of the HB lead resulted in selective HBP with recruitment of the LBBB and narrowing of the QRS to 90 ms. Total procedure time was 105 minutes and fluoroscopy time was 7 minutes.

Discussion
We present 2 patients with a wide LBBB in whom unipolar mapping from the HB pacing lead identified a split His potential. Pacing at a site distal to the split His recruited the LB. This suggests that the level of block/delay in the left
bundle was likely within the HB in fibers that were predes-
tined to the LB.

In case 1, Figure 1 demonstrates proximal, mid, and
distal HB recordings with corresponding paced morphol-
gies (5 V at 1 ms). The mid and distal recordings revealed
a split His potential, suggesting intra-Hisian disease as the
possible site of the LBBB or left bundle delay. Pacing from
the distal His site resulted in both capture of the HB and
local right ventricular capture (nonselective HB capture)
and successful recruitment of intra-Hisian LBBB
(Figure 1). Distal HBP provided successful CRT with a nar-
rowing of the QRS from 164 ms to 90 ms. HBP resulted in
normalization of LVEF to 55% and clinical improvement to
NYHA class I.

Case 2 demonstrates a patient with a rate-related LBB
delay who was noted to have a split HB electrogram with
a wide separation (H1-H2 interval of 150 ms at 60 bpm,
Figure 2) during HB mapping from the tip of the pace/
sense lead. As seen in Figure 3, pacing the atrium at faster
rates (from AAI 70 bpm to 100 bpm) resulted not only in
A-H1 prolongation (representing normal atrioventricular
nodal physiology), but also in a prolongation of H1-H2
interval from 150 ms to 180 ms. This increase in
H1-H2 intervals corresponded precisely with a progres-
sive delay in the LBBB and QRS prolongation from
140 ms to 170 ms, suggesting that the delay was within
the His bundle. Pacing at the distal HB (H2) resulted in
complete recruitment of LBB delay (Figure 2) and
normalization of the QRS duration, confirming the
intra-His site of delay.

The normalization of bundle branch block by distal HBP
was first reported in the 1970s by Narula.1 HB pacing has
also been shown to provide pacing support for heart failure
patients with and without LBBB heart failure.2,4 The ability
to recruit the left bundle with pacing at the distal HB
supports the concept of functional longitudinal dissociation
in the HB. Previous studies on permanent HB pacing
validate this concept.3,5,6 The postulated mechanisms for
this recruitment of branches are as follows: (1) longitudinal
dissociation in the HB with pacing distal to the site of
delay/block, and/or (2) differential source-sink relationships
during pacing vs intrinsic impulse propagation, and/or (3)
virtual electrode polarization effect.6

These cases support the theory of longitudinal dissociation
in the HB and provide the first documentation of the abil-
ity to record a split His potential using an HB permanent
pacing lead.

**Figure 1** Unipolar recordings from the lead tip at different sites the His bundle region. Top panel with unipolar recordings (filtered and unfiltered electrograms)
from the His bundle pacing lead using alligator cables. As noted, mid and distal His electrograms reveal a split His electrogram. The bottom panel represents paced
QRS morphologies at the respective mapped sites.
Figure 2  Unipolar recordings and pacing from the lead tip at different sites in the His bundle (HB) region. Proximal HB electrograms demonstrate a split HB (H1-H2 of 150 ms). Right anterior oblique fluoroscopic view demonstrates location of the tip electrode. Pacing at this site demonstrates a similar QRS with left bundle branch (LBB) delay. Moving from proximal to distal HB position results in a distal site that in 0.5 ms more distal fluoroscopically, with a larger distal HB electrogram. Pacing at this site results in narrowing of the QRS and loss of LBB delay. There is no local ventricular capture, suggesting selective HB capture and LBB recruitment.

Figure 3  AAI pacing at incremental rates with progressive intra-His and left bundle branch (LBB) delay. Similar to Figure 2, there is progressive prolongation in LBB (140 ms to 170 ms). Note the prolongation in H1-H2 intervals (from 150 ms to 180 ms) suggestive of intra-Hisian LBB delay.
Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2017.02.003.

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