How to perform permanent His bundle pacing in routine clinical practice

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Over the years, various sites of ventricular pacing have been evaluated in clinical trials. Earlier trials established the detrimental effects of right ventricular (RV) apical pacing, including increased risk of atrial fibrillation, heart failure (HF), and mortality. Alternate RV pacing sites have yielded mixed results.1 Biventricular (BiV) pacing in advanced HF and electrical dyssynchrony reduced HF hospitalizations and mortality. Recently, 2 trials evaluated the clinical utility of BiV pacing in the setting of heart block and demonstrated equivocal results.2,3 The biventricular pacing for atrioventricular block and systolic dysfunction (BLOCK-HF) trial showed benefits of BiV over RV pacing, mostly driven by change in left ventricular (LV) systolic volumes. Preliminary results of the biventricular pacing for atrioventricular block to prevent cardiac desynchronization (BioPace) trial showed no significant clinical benefit of BiV over RV pacing. There still remains an unmet need in populations with first-degree heart block and HF, populations with right bundle branch (RBB) block (Online Supplemental Figure 1) and HF, and cardiac resynchronization therapy nonresponders (Online Supplemental Figures 2 and 3), where A-V and V-V synchrony may improve outcomes.

Clinical permanent His bundle pacing (PHBP) was first described in 2000.4 Since then, several investigators5–8 across the world have published the safety and feasibility of PHBP. We attempt PHBP in all patients with indication for permanent pacemaker implantation and have performed more than 500 implants in past 5 years (success rate of 85% in recruiting the HB). We have performed PHBP in more than 100 patients with heart block (atrioventricular nodal block and intra-His heart block) (Online Supplemental Figure 4).7 Benefits include ability to achieve physiological pacing by recruiting the native His Purkinje system and avoiding electrical dyssynchrony, potentially translating into reduced HF incidence.9 The anatomy of the HB has been well characterized.9 Also, limited clinical observations have demonstrated that the lead does not cross the tricuspid valve and this may result in lower incidence of valvular regurgitation.10 We describe our method of performing PHBP along with potential device/lead issues that one may encounter at the time of implantation and during follow-up. Most of these issues can be successfully addressed with appropriate programming.

Equipment requirements

Following are the equipment requirements:

1. Medtronic (Minneapolis, MN) 3830 69-cm His lead with a 1.8-mm exposed helix, lead outer diameter (OD) 4.2F (Figure 1A). It is a solid core pacing lead with no central lumen to deliver a stylet, requiring a long sheath for delivery.

2. Medtronic C315 His nondeflectable sheath (43 cm), inner diameter (ID) 5.5F, OD 7.0F (see asterisk in Figure 1B), primary curve to reach the superior aspect of the tricuspid annulus, secondary curve to reach the septum. A Medtronic deflectable sheath (C304-69; ID 5.7F and OD 8.4F) is also available with unidirectional deflection (Figure 1C); it can be helpful in challenging anatomical situations (dilated right atrium [RA], an inferiorly displaced HB, etc.).

3. Short 7F peel-away sheath to place the C315 His sheath through it. It allows continued vascular access after the His sheath is split (guidewire can be retained as well).

4. Pace-sense analyzer (PSA) to record intracardiac electrograms (EGMs). Because of the inherent sensing algorithms built into the Medtronic PSA, we usually connect the pacing lead to the atrial channel (higher gain setting of 0.05 mV/mm). For PSAs of other manufacturers, the lead can be connected to the ventricular channel.

5. Unipolar connection to map the HB EGMs with the pacing lead. We use the pacing lead to map the HB with PSA EGMs, without the need for a mapping catheter. Also, an electrophysiology (EP) recording system can be used (PSA EGMs are adequate).

6. Twelve-lead electrocardiogram for the procedure (critical to analyze pacing morphologies to confirm recruitment of the HB).
Procedure description

Once vascular access is obtained (cephalic, axillary, or subclavian vein), a guidewire is advanced into the RA or RV (see Online Supplemental Movie). A short 7F sheath can be advanced over the wire to retain access. Otherwise, the C315 His sheath is advanced into the RA or RV and the guidewire removed. The pacing lead is advanced to the tip of the sheath, with the distal tip of the lead exposed minimally. Unipolar connections are made with the tip of the lead and cardiac tissue.

If the sheath and the lead tip are in the RV, the apparatus is gently pulled back to the atrioventricular groove with minimal counterclockwise rotation to ensure that the lead tip is abutting the septum. If the sheath and the lead are in the RA, gentle forward clockwise rotation tends to move the apparatus to the summit of the tricuspid annulus.

While this is being performed, it is important for both the operator and the person operating the PSA to pay careful attention to intracardiac EGMs. Small movements are encouraged as HB deflections can be easily missed. We usually set the sweep speed to 50 or 100 mm/s to allow better separation of atrial, HB, and local ventricular EGMs.

Once HB EGMs are obtained, unipolar pacing is performed since the proximal pole of the lead is within the sheath. We start pacing at 5 V @ 1 ms and assess 12-lead QRS morphologies. The following patterns can be observed:

1. Pure HB pacing where stimulus to ventricular activation is equal to the intrinsic HV interval and paced QRS morphology is identical to the intrinsic QRS complex. We term this as selective HB pacing (Figures 2 and 3).
2. HB capture with local ventricular fusion: stimulus-ventricular capture is shorter than the HV interval. The pacing output is decremented to assess the changing QRS morphologies (akin to para-His pacing performed to assess septal accessory pathways). We term this as nonselective HB pacing (Figures 2 and 3). Various responses can be observed:
   (a) At high output, the HB is preferentially recruited with progressive widening as output is reduced, resulting in more local ventricular capture.
   (b) At high output, more fusion is encountered because of local ventricular capture, and at lower output, HB is preferentially activated, resulting in less fusion (Figures 2 and 3).
   (c) Sometimes just before loss of capture, pure HB pacing can be seen. Also, selective RBB or left bundle branch (LBB) capture can be demonstrated (Figure 3).
   (d) Sometimes because of the proximity of RBB and more ventricular placement of the lead, RBB capture can occur. This results in an LBB block pattern-wide QRS complex. It may be difficult to determine whether local myocardial capture occurs along with RBB capture. If intrinsic conduction is present, measuring the HV interval can help determine RBB capture. Typically, a local HV interval would be short (<30 ms), with no far-field atrial EGM seen on the PSA.

![Figure 1](image-url)

**Figure 1**  
A: Select Secure 3830 lead. B: Several nondeflectable sheaths used for the 3830 lead. The first sheath marked by the asterisk is a C315 His nondeflectable sheath used for His bundle mapping and lead placement. All other sheaths are used for either right atrial or right ventricular lead placement. C: Deflectable sheath used occasionally to place the His bundle lead (Medtronic, Inc.).
Figure 2  At pacing outputs above 2 V @ 0.5 ms, there is fusion with His bundle (HB) and local ventricular capture (nonselective HB capture). Below 2 V @ 0.5 ms, there is pure HB capture with paced QRS morphology identical to the native QRS complex (selective HB pacing). Note that once pure His capture is obtained as the pacing threshold, intermittent fusion is still high enough to intermittently recruit the local ventricular myocardium. ECG = electrocardiogram.

Figure 3  At higher output (up to 1.5 V @ 1 ms), there is fusion between His bundle and local myocardial capture (nonselective response). At lower outputs (up to 0.5 V @ 1 ms), there is pure His bundle capture with stimulus to QRS onset (S-QRS) equal to the native HV interval (selective response, *). Below this output, there is selective right bundle branch (RBB) capture.
We believe that these responses are due to anatomical and functional aspects of the HB system. Anatomy of the HB has been well characterized. Preferential pacing of RBB and LBB can be explained in part by the phenomenon of longitudinal dissociation of the His-Purkinje conduction system. Other properties such as favorable source-sink ratios and virtual electrode polarization may play a part as well.

Once HB capture is ascertained, the lead is turned 4–5 times in a clockwise direction. Usually, torque is built up and transmitted to the end of the lead. Once it is released, the lead may unwind 1–2 rotations. It is important to use the sheath to support the lead and make good contact with the septum. The sheath is pulled back, and the lead is advanced to allow for slack. Current of injury, either of the HB or local ventricular tissue, is usually seen (Figure 4). However, there are cases where the lead is well affixed with good pacing thresholds and minimal local tissue injury. Testing is performed in both unipolar and bipolar configurations. Starting at 5 V @ 1 ms, output is decremented to assess response to pacing. Either selective or nonselective HB pacing is accepted, and pacing thresholds less than 2 V @ 1 ms are deemed acceptable. If the patient is pacemaker dependent, lower pacing thresholds are sought after before accepting the final lead position. If the pacing threshold margins are not deemed acceptable by the operator after multiple attempts, the lead is placed slightly more anteriorly, with larger ventricular sensing and pacing morphology consistent with septal pacing. The patient is monitored overnight on telemetry, and the device system is interrogated before discharge the following day.

**Intraprocedural findings and troubleshooting**

**Issues with mapping the HB**

The C315 His sheath is well shaped to reach the septal isthmus and record HB EGMs in 80% of cases within the first 5 minutes. In difficult cases, the following strategies can be used:

1. Pace map to assess HB capture based on 12-lead morphology.
2. Use a deflectable C304 sheath with challenging anatomy such as a very dilated right atrium.
3. Use a mapping catheter from the above (using the access site for the atrial lead) or placing it from the femoral vein (we rarely use it).

**Acute ventricular sensing issues**

Because of the location of the lead tip, we see a wide range of ventricular sensing values (1.2–10 mV). We compare ventricular EGMs with atrial EGMs to assess sensing margins. Typically, the lead tip records small atrial EGMs and atrial oversensing is not a major issue. Unipolar and bipolar sensing is assessed, and if no far-field sensing is seen, unipolar sensing can be used if it offers better sensing safety margins. However, it is rare that we program unipolar ventricular sensing. In patients who are pacemaker dependent, we do not use unipolar sensing and ventricular sensitivity is usually set to >1.2 mV on the basis of measured values and care is taken to assess far-field atrial sensing before accepting the final location of the ventricular lead. It is important to choose the appropriate devices with higher programmable sensitivity (eg: the Medtronic Sensia system has a ceiling of 1.2 mV for sensitivity, while the Advisa device can be programmed to a more sensitive value of 0.3 mV).

**Acute pacing issues**

The HB is encased in a fibrous sheath with various anatomical variations. Pacing thresholds are expected to be inherently higher because of the lead-tissue interface. Therefore, if pacing thresholds are lower than 2 V @ 1 ms at implantation with good HB recruitment, we accept the lead position. Typically, as the pacing output decreases, there is more fusion with ventricular capture, thereby providing good safety margins (nonselective HB pacing cases only). The pacing output is set to 5 V @ 1 ms at implantation and
decreased at 2-month check. In dependent patients, we look for at least 2–3 times safety margin. We aim for nonselective HB pacing in such cases and determine safety margins from the lowest output capture of either the HB or local ventricular tissue. If selective HB pacing is desired in the setting of higher pacing thresholds, we place a backup lead in the RV. This necessitates placement of a BiV pacemaker with the HB lead plugged into the LV port. We program an LV to RV offset of 80 ms. If HB capture occurs, the RV stimulus falls in the refractory period, resulting in functional noncapture. If HB capture does not occur, the RV stimulus acts as safety pacing.

Because of the annular nature of the lead tip position, it is important to exclude simultaneous atrial capture during high-output HB pacing. With DDD pacemakers, the atrial lead can be placed simultaneously to assess atrial capture during ventricular pacing. This is a nonissue in patients with permanent atrial fibrillation.

Postprocedural follow-up and troubleshooting
We set up follow-up at 2 weeks and 2 months postimplantation. Thereafter, we use remote monitoring with yearly follow-up in a device clinic. Our device technicians are trained to assess pacing thresholds where HB capture is lost (QRS complex widening occurs on the PSA) and when ventricular capture is lost. These are documented separately. Potential oversensing and undersensing issues are carefully assessed. If any significant issues are encountered, the patient is seen by one of the electrophysiologists. Twelve-lead electrocardiograms are performed to assess the loss of HB capture in cases where it cannot be reliably demonstrated using the programmer.

Long-term sensing issues
Long-term issues with oversensing and undersensing are relatively rare. Less than 1% of patients have required lead revision due to sensing issues. With modern devices and a myriad of programmable parameters, devices can be programmed to avoid such issues. However, it is important to obtain good sensing safety margins at the time of implantation.

Long-term pacing issues
The biggest concern is that pacing thresholds may increase over time. In less than 10% of patients, thresholds may increase in the first 3 months. This may be due to microdislodgement vs local fibrosis. Acute thresholds may increase, but chronic thresholds remain constant. We have close to 7 years of follow-up and more than 100 dependent patients with complete heart block. We have not had any cases where patients presented with syncope or had a fatal outcome due to loss of capture. Lead revisions may be necessary in up to 5% of patients because of intermittent loss of capture or significant increase in thresholds.

Because of higher pacing thresholds than those of conventional RV pacing, there is increased battery drain. We use the largest pacemaker battery available from each manufacturer. We consider HB thresholds to be similar to LV pacing thresholds rather than to RV pacing thresholds because of inherent differences in the tissue that is being excited. We program a pulse width of 1.0 ms and decrease the amplitude to reduce battery drain.

Conclusion
In our experience, PHBP is safe and feasible in daily clinical practice and there is a definite learning curve. We started out by performing the procedure in patients with sinus node dysfunction who were not ventricular pace dependent. Once we gained experience (~20 cases per operator), we expanded to include all patients. PHBP is physiological in nature, and with more physicians adopting it, device and lead manufacturers will be prompted to develop better leads, devices, and delivery technologies to improve ease of PHBP in clinical practice, thereby putting “physiology” at the forefront of bradycardia pacing. In the long run, large randomized multicenter clinical trials will be welcomed to establish safety, feasibility, and outcomes.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrt hm.2016.03.040.

References