

TOPIC REVIEW

Electrocardiographic Analysis for His Bundle Pacing at Implantation and Follow-Up



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ABSTRACT

His bundle pacing (HBP) is steadily gaining interest for providing physiological cardiac stimulation. Careful analysis of the electrocardiogram (ECG) is crucial to confirm capture of conduction tissue, which is a prerequisite for successful HBP at implantation and follow-up. However, interpretation of the ECG with HBP can be challenging. This review provides the reader with practical guidance on how to best use and troubleshoot the 12-lead ECG for HBP in daily clinical practice. (J Am Coll Cardiol EP 2020;6:883-900) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

His bundle pacing (HBP) has the advantage of avoiding the electrical (and thereby mechanical) dyssynchrony induced by myocardial pacing by recruiting the intrinsic conduction system to activate the ventricles. It may therefore be used in lieu of right ventricular (RV) pacing. Furthermore, HBP can correct bundle branch block (BBB) in a subset of patients with a proximal site of conduction disturbance (1). Therefore, HBP may be used in lieu of biventricular (BiV) pacing for cardiac resynchronization therapy (CRT) (e.g., as rescue therapy in patients with failed coronary sinus lead implantation). It may also be used in combination with RV, left ventricular (LV), or BiV pacing as His-optimized CRT (HOT-CRT) (2). A recent consensus document outlined the various types of His capture and stressed the importance of 12-lead electrocardiography (ECG) analysis

(3). There is a wealth of information that can be gleaned from careful analysis of the ECG, but interpretation may sometimes be challenging and fraught with pitfalls. The purpose of this review is to help the reader overcome these challenges by providing a practical guide on ECG interpretation in the setting of HBP.

TYPES OF HIS CAPTURE

It is mandatory to record a 12-lead ECG at HBP implantation and at follow-up because changes in QRS morphology during threshold tests may be subtle and missed if only limb leads are used. ECG belts (available from different manufacturers) are useful to speed up recordings in the busy device follow-up clinic.

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ABBREVIATIONS AND ACRONYMS

BBB	= bundle branch block
Biv	= biventricular
CRT	= cardiac resynchronization therapy
ECG	= electrocardiography
ERP	= effective refractory period
HB	= His bundle
HBP	= His bundle pacing
HOT-CRT	= His-optimized cardiac resynchronization therapy
HV	= His-ventricular
LBBB	= left bundle branch block
LV	= left ventricle
NIVCD	= nonspecific intraventricular conduction delay
NS	= nonselective His bundle pacing
RBBB	= right bundle branch block
RV	= right ventricle
RWPT	= R-wave peak time
S-HBP	= selective His bundle pacing

In addition to capture of the HB, the His lead can capture ventricular and/or atrial tissue that can affect ECG findings. Type of capture depends on His lead position (Figure 1), the anatomical variant of the HB (4), capture thresholds (Figure 2, Central Illustration), pacing output, and pacing configuration (e.g., anodal capture in case of extended bipolar pacing). Furthermore, in case of underlying BBB, HBP may correct the conduction disease either partially or totally, leading to additional variations in QRS morphology.

It is important to correctly identify the following entities.

SELECTIVE HIS BUNDLE PACING. With selective His bundle pacing (S-HBP), an isoelectric interval is visible in all 12 leads, that corresponds to the His-ventricular (HV) interval and separates the pacing spike from QRS onset (Figures 1 and 3). Analysis of the device electrogram also shows a delay between delivery of pacing and the local ventricular electrogram (5). The QRS morphology is most often identical to that in intrinsic rhythm. However, slight differences in the paced QRS morphology may be observed (Figures 4A and 4B) in case the His lead is positioned distally, such as at the right bundle

branch and/or HB bifurcation. In these instances, activation through the conduction system will follow a different course compared with intrinsic conduction. The His lead may be positioned on either the atrial aspect or the ventricular aspect of the tricuspid valve (6). Leads implanted at sites where the HB is sub-endocardial and distant from the ventricular myocardium will display S-HBP as the only form of capture.

NONSELECTIVE HIS BUNDLE PACING. With nonselective His bundle pacing (NS-HBP), the lead is usually positioned in the ventricle at a site where the HB is surrounded by or at proximity to myocardial tissue. However, supra-ventricular lead position with either higher output and/or ventricular myocardium adjacent to the atrioventricular septum (4) near the HB can also result in NS-HBP. The ECG shows a pseudo-delta wave corresponding to local myocardial capture, resembling pre-excitation of a para-Hisian or fasciculo-ventricular accessory pathway (Figures 1 and 3B). The amplitude and duration of the pseudo-delta wave will depend on the surrounding myocardial fiber density and mass of the captured myocardium and the HV interval (Figures 3B, 3C, and 4C). The pseudo-delta wave may also be slightly shorter than the HV interval in case the His lead is

HIGHLIGHTS

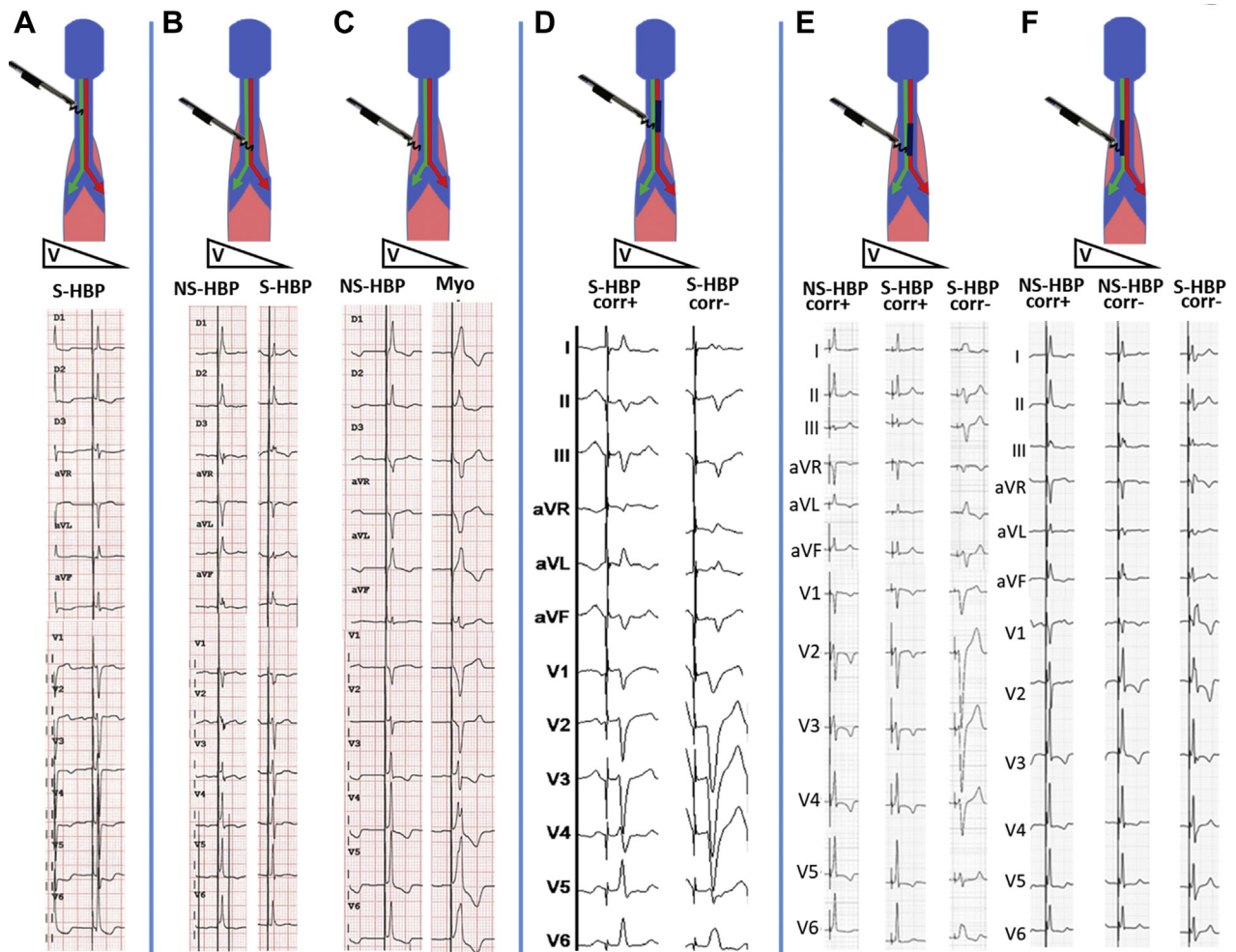
- At HBP implantation and follow-up, the ECG is central for threshold testing and optimizing programming.
- Selective and nonselective His capture, with and without correction of bundle branch block, should be recognized.
- Physicians should be aware of ECG features and pacing maneuvers that help identify type of His capture.

positioned distally. QRS may show an initial isoelectric segment in some leads (especially lead III) if the initial vector of depolarization is perpendicular to those leads. In addition to the pseudo-delta wave, fusion with myocardial activation results in an increase in R-wave amplitude in leads I, II, and V₆ (Figures 1B, 3, 4A, and 4B) due to summation of the vectors of myocardial capture and activation via the His-Purkinje system. A reduction in R-wave amplitude in these leads helps identify the transition between NS-HBP and S-HBP in cases where the pseudo-delta wave may not be apparent. NS-HBP has the advantage of providing backup myocardial capture in case of loss of His capture or exit block. It is also associated with higher R-wave sensing amplitudes compared with S-HBP. No significant differences in cardiac mechanical synchrony (7) or clinical outcome have been found between S-HBP and NS-HBP, although there was a trend in lower mortality with S-HBP (however, this group had fewer comorbidities) (8).

CORRECTION OF BBB. It has been reported that approximately 60% to 90% of patients with BBB have QRS narrowing with HBP (9-15). The finding that the level of conduction block may be located proximally within the HB with longitudinal dissociation of the conduction fibers was originally described in the 1970s both for left bundle branch block (LBBB) (1) and for right bundle branch block (RBBB) (16). BBB may be totally or partially corrected by HBP. Conduction blocks may exist at different levels in a given patient, and HBP may correct BBB at a proximal level, which may then unmask distal conduction disease, which results in a different QRS morphology.

MYOCARDIAL CAPTURE ONLY. The QRS complex may sometimes be relatively narrow (and narrower than in intrinsic rhythm with BBB). It may be difficult in some instances to distinguish myocardial capture from NS-HBP. This is covered separately in a following section.

FIGURE 1 His Lead Position and Examples of Different Types of His Capture and Transitions in QRS Morphology With Decrementing Voltage Output



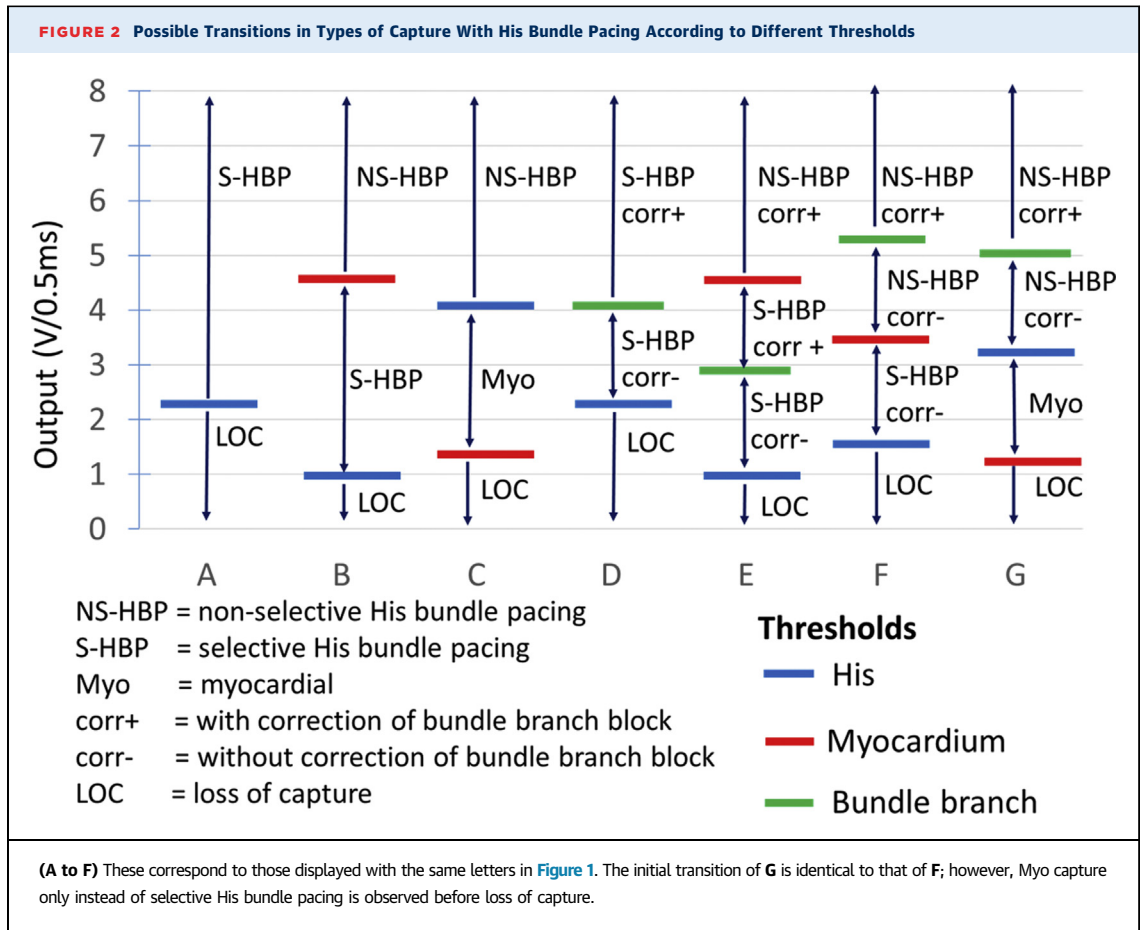
(A) Proximal position over a subendocardial His bundle with obligatory selective His capture (selective His bundle pacing [S-HBP]). Note the isoelectric interval between the pacing spike and QRS onset, visible in all 12 leads. **(B)** More distal position with a His bundle surrounded by a myocardial sleeve. There is close contact of the pacing lead and conduction tissue, resulting in nonselective His bundle pacing (NS-HBP) with a pseudo-delta wave, which transitions to S-HBP with a reduction in output. **(C)** The lead is in less close contact with the conduction tissue, resulting in a transition between NS-HBP and myocardial (Myo) capture only. **(D)** Proximal position of the His lead with S-HBP in the setting of left bundle branch block (LBBB). At higher output, there is recruitment of conduction tissue resulting in correction (corr+) of the conduction delay and loss of correction (corr-) at reduced output. **(E)** More distal position of the lead with initial NS-HBP and correction of LBBB. **(F)** Right bundle branch block with initial correction and NS-HBP, with loss of correction (note the presence of an s wave in lead I and a more prominent R-wave in lead V₂) followed by uncorrected S-HBP.

TRANSITIONS IN QRS MORPHOLOGY WITH DECREMENTING OUTPUT

A number of different transitions in QRS morphology before loss of capture may be observed with decrementing pacing output (and starting at maximum output) (Figures 1 and 2) and is summarized in Table 1.

In patients with RBBB, it may be difficult to distinguish NS-HBP with and without correction of

the conduction disorder (Figure 1F). This is because myocardial capture in these instances serves to narrow the QRS (Figure 3D and section on HOT-CRT), much as LV fusion pacing with coronary sinus leads serves to narrow the QRS complex in patients with underlying LBBB (17). Therefore, measuring the duration from the stimulus to the end of the QRS is not useful in this instance to determine whether RBBB has been corrected. The presence of an S- and/or s-wave in leads I and/or aVL and a qR- and/or



Qr-wave in leads V_1 and/or V_2 is suggestive of NS-HBP with uncorrected RBBB.

Additional transitions to those previously mentioned may be due to inhomogeneous capture of conduction tissue at different outputs (probably due to longitudinal dissociation of the HB). Inhomogeneous capture may result in slight variations in QRS morphology, both in patients with a narrow intrinsic QRS (Figure 4) and in those with underlying BBB. Inhomogeneous capture may also be due to slight variations in lead orientation with the cardiac or respiratory cycle and may result in alternating BBB morphology (Figure 5). Furthermore, transitions in morphology due to anodal capture or capture of the right atrium may be sometimes observed and should not be confounded with transitions due to loss of capture of conduction tissue (see section on Pitfalls in ECG Analysis of HBP).

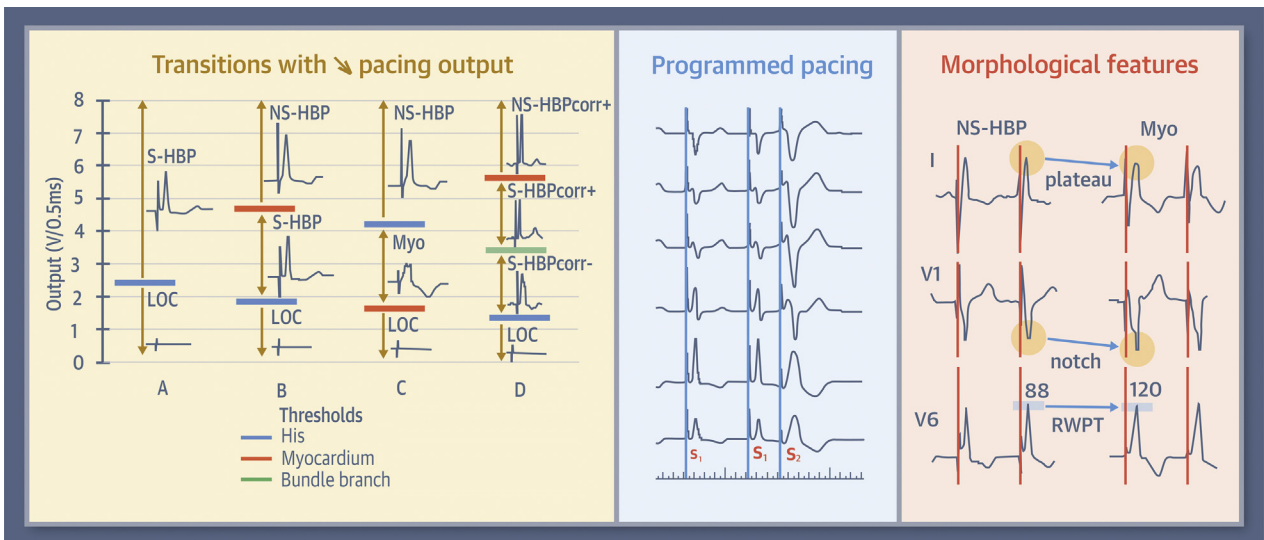
Absence of transitions in QRS morphology before total loss of capture with decrementing output indicates either: 1) S-HBP; 2) ventricular myocardial capture only (i.e., para-Hisian pacing, which

cannot be considered as being true HBP); and 3) similar thresholds of different tissues that are simultaneously captured. To better distinguish the different capture thresholds, pulse width may be narrowed because this serves to widen the difference in capture thresholds between the HB and the adjacent myocardium (Figure 6A) (18). Other maneuvers, based on differences in tissue refractory periods, to distinguish between NS-HBP and myocardial capture only, are discussed in the following section.

Impossible transitions with decrementing output are: 1) S-HBP → NS-HBP; 2) S-HBP → myocardial capture only; and 3) without correction of BBB → with correction of BBB.

Thresholds of different tissues may be almost identical, and different types of capture may be observed during consecutive beats at a given output, probably due to slight variations in lead orientation and tissue contact. This is usually observed during pacing with output close to the capture threshold(s).

CENTRAL ILLUSTRATION Fundamentals of Electrocardiographic Analysis With His Bundle Pacing



Burri, H. et al. *J Am Coll Cardiol EP.* 2020;6(7):883-900.

(Left panel) The 12-lead electrocardiogram (ECG) is central in confirming His capture. A number of different transitions in QRS morphology may be observed with decrementing pacing output, reflecting loss of His capture, myocardial capture, or correction of bundle branch block. The sequence of transition will depend upon the respective thresholds. **(Middle panel)** When transitions in QRS morphology are not obvious, programmed pacing maneuvers can be useful to distinguish nonselective His bundle pacing (NS-HBP) from myocardial-only (Myo) capture. **(Right panel)** Morphological features combining absence of plateaus, notching and/or slurring in leads I, V₁, and V₄ to V₆ and R-wave peak time (RWPT) <100 ms indicates NS-HBP and allows to distinguish this entity from Myo capture. corr+ = with correction of bundle branch block; corr- = without correction of bundle branch block; LOC = loss of capture; S-HBP = selective His bundle pacing.

DISTINGUISHING NS-HBP FROM MYOCARDIAL CAPTURE ONLY

As previously mentioned, it may be sometimes difficult to distinguish NSH-HBP from myocardial capture only. The latter instance is important to identify because it cannot be considered as being HBP.

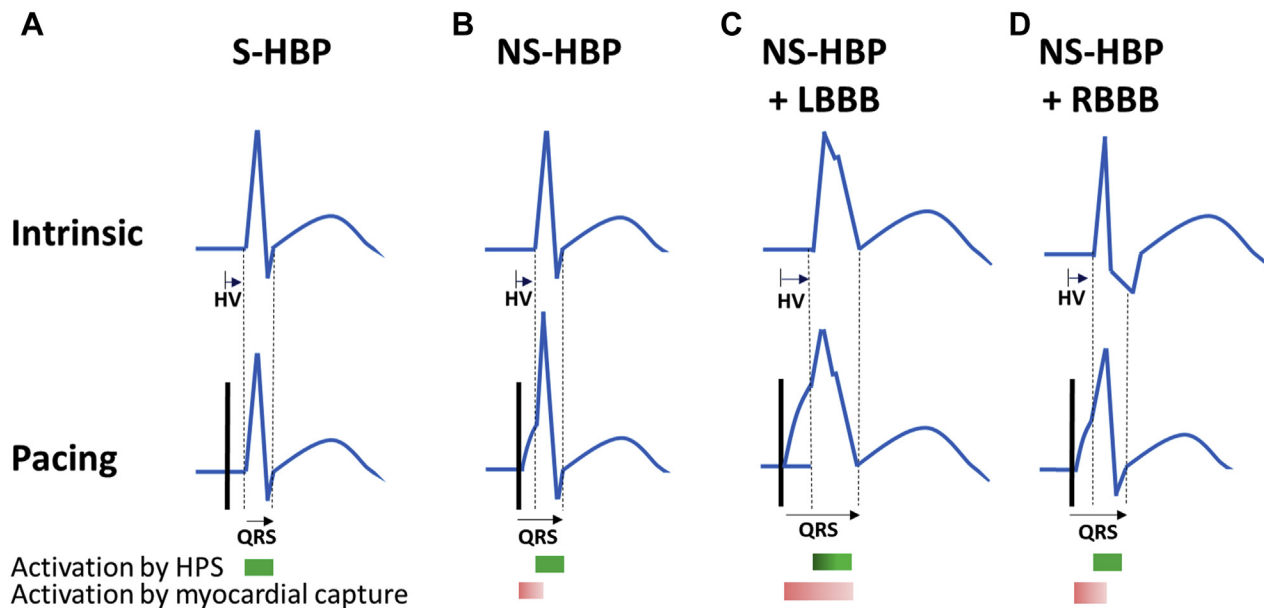
DECREMENTING PACING OUTPUT. Decrementing the pacing output and observing a transition in QRS morphology between NS-HBP and myocardial capture only is the routine method because it is fast and straightforward in most cases. However, in some instances, the transition in QRS morphology may be subtle and missed. In patients in sinus rhythm and preserved ventriculo-atrial conduction, sudden lengthening of the stimulus to a retrograde P-wave (or the atrial electrogram [5]) can be a useful sign that there has been a transition from NS-HBP to myocardial capture only. This maneuver is similar to parasympathetic pacing used to evaluate the presence of an

accessory pathway (19). However, a potential trap is direct atrial capture by the His lead (see section on *Pitfalls in ECG Analysis of HBP*).

In our experience, an estimated 5% of patients have equal thresholds of HB and local ventricular myocardium (despite narrowing of the pulse width) (Figure 6A), and there is no transition in QRS morphology before complete loss of capture. It is important to distinguish this situation from myocardial capture only (i.e., lack of HB capture), in which no transitions in QRS morphology are observed either.

ANALYSIS OF PACED QRS DURATION AND MORPHOLOGY. ECG criteria have been used to identify loss of LV or RV capture with BiV pacing (20,21). Analysis of the ECG can also identify loss of His capture in HBP and distinguish NS-HBP from myocardial capture only.

QRS duration. In the setting of NS-HBP or myocardial capture only, QRS duration is measured from the

FIGURE 3 Schematic Representation of Lead I and Ventricular Activation With Different Types of His Bundle Capture

(A) S-HBP with stimulus to QRS-onset interval corresponding to the His-ventricular (HV) interval. (B) NS-HBP in the setting of a normal HV interval, with a small pseudo-delta wave and an increase in QRS amplitude. (C) NS-HBP in the setting of uncorrected LBBB and prolonged HV interval with widening of the paced QRS due to a large pseudo-delta wave and predominant activation of the left ventricle via cell-to-cell depolarization due delayed activation of the His-Purkinje system (D) NS-HBP in the setting of uncorrected right branch bundle block (RBBB) with narrowing of the QRS complex by virtue of pre-excitation of the right ventricle (activated late in intrinsic rhythm) and fusion with ventricular activation via the His-Purkinje system. In case of intrinsic bundle branch block which is corrected by His bundle pacing, the resulting paced QRS will resemble that of (A and B) in case of S-HBP or NS-HBP, respectively. HPS = His Purkinje system; other abbreviations as in Figure 1.

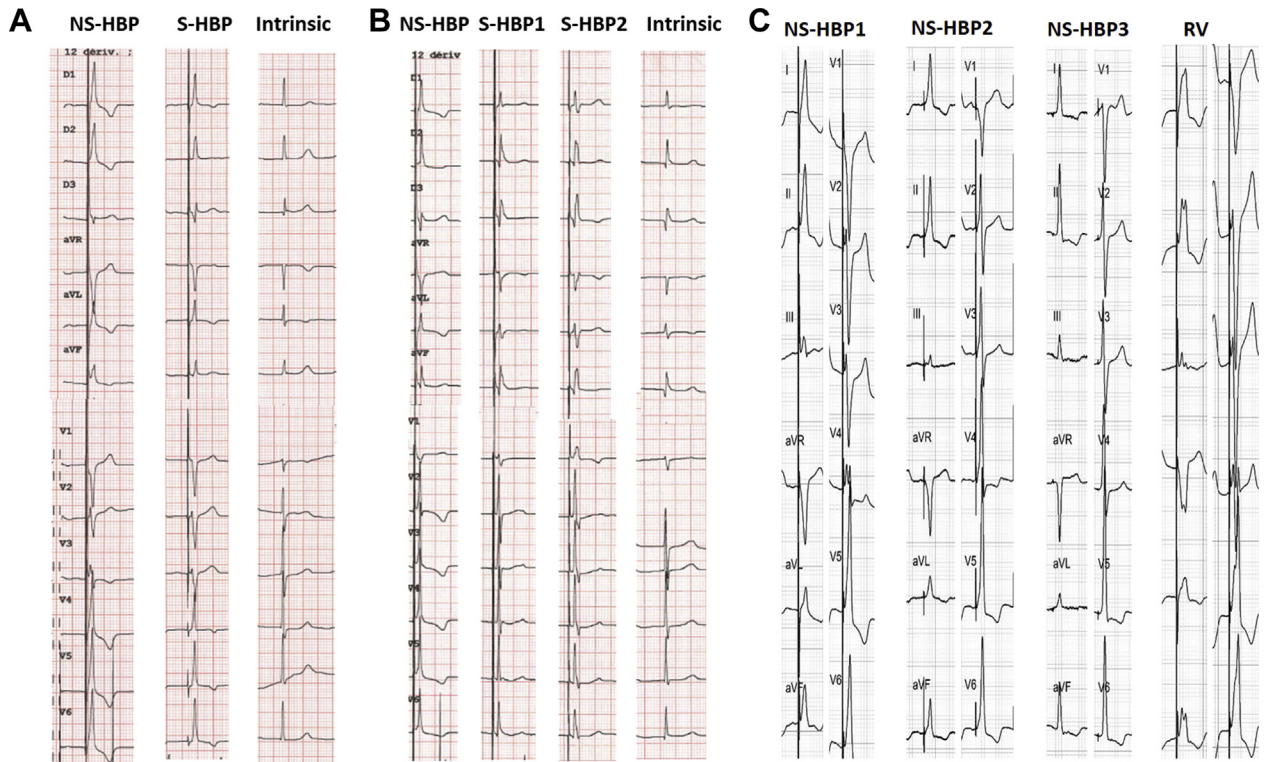
pacing stimulus to the latest QRS component in any of the 12 simultaneously recorded leads (whereas with S-HBP, it is measured from QRS onset). In patients with a narrow QRS, NS-HBP results in QRS duration that is the sum of the HV interval and the intrinsic QRS duration (Figure 3B). Because the upper normal values of the HV interval and QRS duration are 55 and 110 ms (22), respectively, even in the absence of any conduction disturbances, NS-HBP can result in a QRS complex as wide as 165 ms (due to the “pseudo-delta” wave) (Figure 3B). Although on average, reported QRS duration with NS-HBP is approximately 140 ms (8,23) (HV of 45 ms + QRS of 95 ms), some HV interval prolongation or intraventricular conduction delay can result in a NS-HBP with a QRS of 180 ms (23). This results in a significant overlap of QRS duration between NS-HBP and myocardial capture only (Figure 6C). However, a QRS duration <130 ms is almost never observed with myocardial capture only (23).

R-wave peak time in lead V₆. It has recently been reported that R-wave peak time (RWPT) in lead V₆ is significantly shorter during NS-HBP than during

myocardial capture only, which represents more rapid electrical activation of the LV by the His-Purkinje system, although there is some overlap in values. An RWPT <90 ms is almost never observed with myocardial capture only (Figure 6C) (23). The lead V₆ RWPT has better discriminating value than paced QRS duration, is easier to measure (because the end of the QRS may be difficult to determine, whereas R peak is relatively easy), and is not affected by delay in RV activation (i.e., in case of noncorrected RBBB and NS-HBP).

QRS slurring, notching, and/or plateau. During RV pacing from the His region, a LBBB pattern is observed. It is therefore not surprising that presence of notches, slurs, and/or plateaus in the LV leads are useful for distinguishing myocardial capture only from NS-HBP (Figure 7A). Mid-QRS notches, slurs, and/or plateaus in leads I, V₁, and V₄ to V₆ are specific for loss of HB capture during NS-HBP in patients with a narrow intrinsic QRS, but may also be observed in case of NS-HBP without correction of LBBB or nonspecific intraventricular conduction delay (NIVCD) (Figures 3C and 7B) (23).

FIGURE 4 Examples of Minor Differences QRS Morphology With His Bundle Pacing in Three Patients With Normal Baseline QRS Complexes



Initial NS-HBP is shown in all cases for comparison. **(A)** Slight difference in QRS morphology between S-HBP compared with intrinsic rhythm. The His lead was implanted in a distal position, probably at the level of the right bundle branch. Note that with S-HBP, the QRS width is slightly wider than during intrinsic rhythm, with differences in QRS morphology (e.g., leads III and V₂). **(B)** S-HBP1 shows that QRS morphology was initially identical compared with intrinsic rhythm. S-HBP2 shows that with a decrease in pacing output, QRS morphology resembles incomplete RBBB (widening of QRS with an S-wave in lead I and a positive QRS in lead V₁), indicating loss of capture of conduction fibers to the right ventricle. **(C)** Three different NS-HBP morphologies in the same patient during His lead implantation. This was most probably related to acute trauma of the His bundle with varying degrees of HV prolongation that recovered during the course of the procedure (NS-HBP was performed at the same pacing output). It is also possible that the variations may reflect changes in local myocardial capture because acute injury subsides directly after lead fixation. Right ventricular (RV) myocardial capture at a lower output is shown for comparison. Abbreviations as in [Figures 1 and 3](#).

On the basis of the previous observations, it has been shown (23) that NS-HBP can be confirmed and distinguished from myocardial capture only with a sensitivity of 64% and a specificity of 100% using the following criteria—lack of any mid-QRS notch, slur, and/or plateau in leads I, V₁, and V₄ to V₆, and lead V₆ RWPT <100 ms.

Exploiting differences in refractoriness between HB and myocardium. Programmed pacing with a single extra-stimulus delivered at decreasing coupling intervals during an HBP drive train or during intrinsic rhythm (preferably both) can reveal the individual components of the fused QRS of NS-HBP (i.e., myocardial QRS and S-HBP QRS). With a drive train of 600 ms, the effective refractory period (ERP) of the HB is 353 ± 30 ms, whereas that of the ventricular myocardium is 272 ± 38 ms [with only 2 of 32 patients having a

shorter ERP for the HB than for the myocardium (24)]. Therefore, with a drive train of 600 ms and an extra-stimulus at a coupling interval of 300 ms, sudden QRS prolongation of the extra-stimulus occurs in most patients with NS-HBP, which corresponds to the refractoriness of the HB and depolarization of the myocardium only (Figure 8). In case of myocardial capture only during the drive train, no sudden change in QRS morphology will occur, nor will it occur with further reduction in coupling intervals until loss of capture. However, slight changes in QRS morphology of the extra-stimulus may be observed due to intra-myocardial conduction delay (especially when one encroaches upon the relative refractory period) and should not be mistaken as confirmation of NS-HBP during the drive cycles. Another caveat is BBB with extra-stimuli that should not be mistaken for

TABLE 1 Summary of Types of Transitions in QRS Morphology With Decrementing Pacing Output**Transitions in QRS Morphology Observed With Decrementing Pacing Output**

Patients with an intrinsic narrow QRS

1. (NS-HBP) → S-HBP → LOC.
2. NS-HBP → myocardial capture only → LOC.

Patients with underlying BBB

1. (NS-HBP with correction of BBB →) (NS-HBP without correction of BBB →) S-HBP without correction of BBB → LOC.
2. (NS-HBP with correction of BBB →) S-HBP with correction of BBB → S-HBP without correction of BBB → LOC.
3. (NS-HBP with correction of BBB →) NS-HBP without correction of BBB → myocardial capture only → LOC.

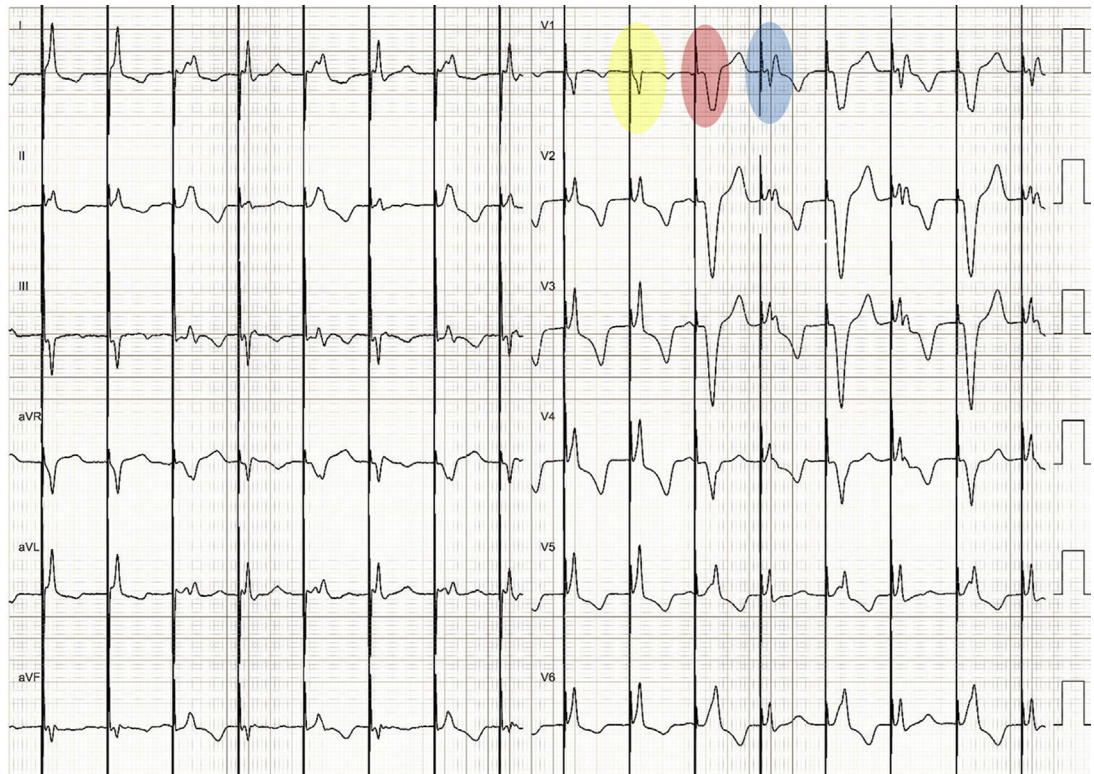
Rare transitions when the His lead is placed beyond the site of BBB

4. NS-HBP with correction of BBB → myocardial capture only → LOC.
5. (NS-HBP with correction of BBB →) S-HBP with correction of BBB → LOC.

The patterns shown in parentheses may or may not be present initially.

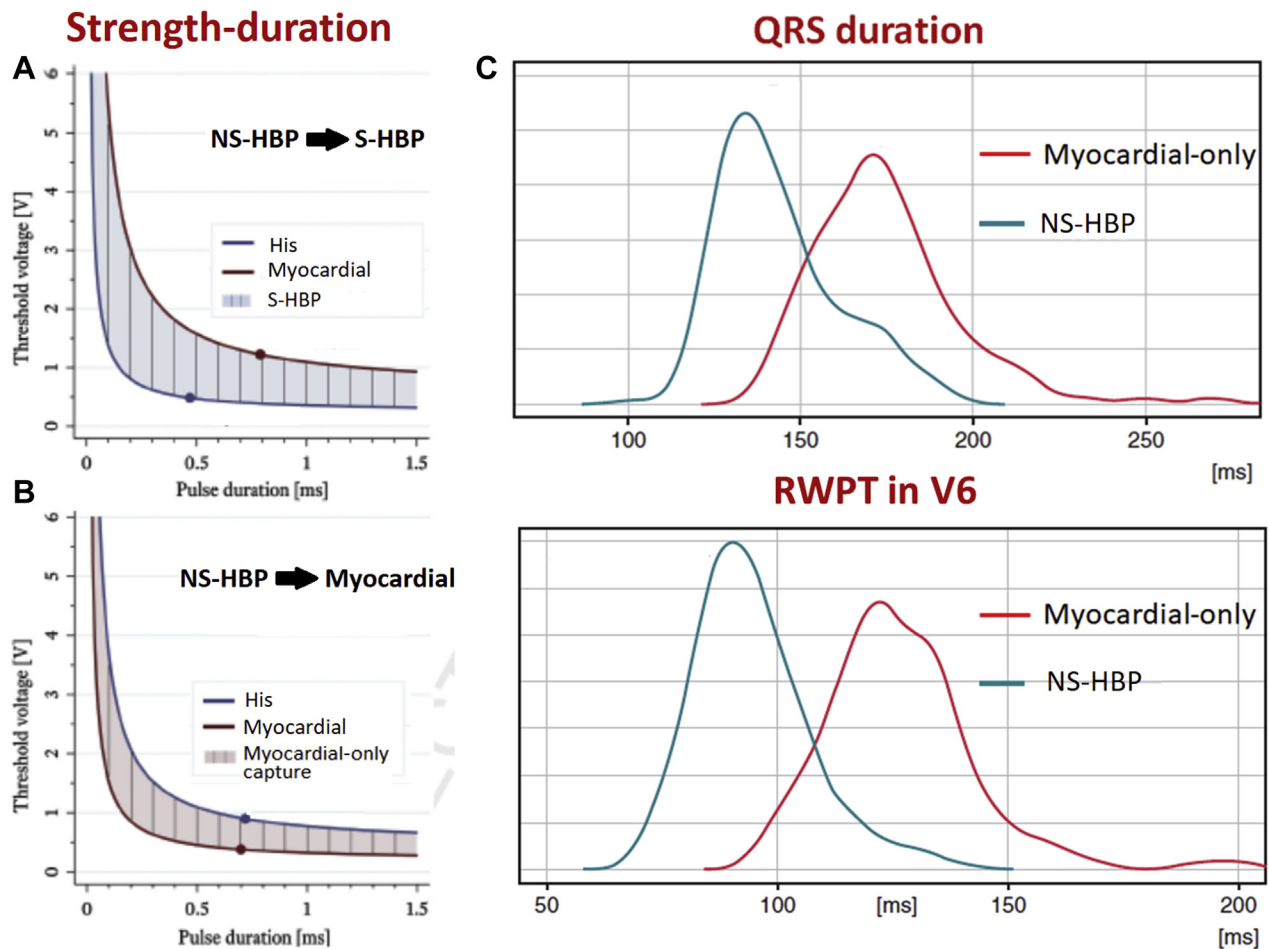
BBB = bundle branch block; LOC = loss of capture; NS-HBP = non-selective His bundle pacing; S-HBP = selective His bundle pacing.

myocardial capture only. Delivering extra-stimuli during native conducted rhythm rather than after a drive train has the advantage of visualizing S-HBP despite HB ERP being greater than myocardial ERP. This is possible because during intrinsic-conducted rhythm, the HB is activated 70 to 100 ms earlier than the adjacent myocardium, and in 70% of patients, this is enough to offset the difference in ERP (24). Slow asynchronous pacing (e.g., VOO of 40 beats/min) may be more practical to perform than programmed stimulation because it results in variable coupling intervals and usually allows prompt visualization of variable-paced QRS complexes (Figure 9). Another option is to perform incremental pacing, during which progressive shortening of ERP of both HB and myocardium occurs. However, a sudden change of paced QRS morphology due to loss of HB capture or myocardial capture can usually be observed, often when the pacing cycle length approaches 320 to 250 ms, but may also be due to BBB (Figure 10).

FIGURE 5 Example Showing Different Types of Capture in a Patient With LBBB in Intrinsic Rhythm

The first 2 cycles show NS-HBP with correction of bundle branch block (yellow). The following cycles are all S-HBP with alternating left (red) and right (blue) bundle branch patterns, indicating selective capture of the right and left bundle branches, respectively. Abbreviations as in Figures 1 and 3.

FIGURE 6 Strength Duration Curves for His Bundle and Myocardial Capture



The curves represent different patients and illustrate the effect of pulse width on differences in capture thresholds between the His bundle and ventricular myocardium. The chronaxie of each curve is shown as a point. **(A)** Curves for transition from NS-HBP to S-HBP. **(B)** Transition from NS-HBP to myocardial only capture. In both cases, the difference in capture thresholds between the His bundle and the myocardium is greater at lower pulse widths. This difference is usually more marked in patients with S-HBP. **(C)** Overlap between myocardial only capture and NS-HBP for QRS duration and R-wave peak time (RWPT) in lead V₆. Note that a QRS duration of <130 ms and an RWPT of <90 ms are almost 100% specific for NS-HBP (the latter criterion is 100% specific when combined with lack of mid-QRS notch, slur, and/or plateau in leads I, V₁, and V₄ to V₆). Adapted with permission from Jastrzebski et al. (18) and Jastrzebski et al. (23). Abbreviations as in Figure 1.

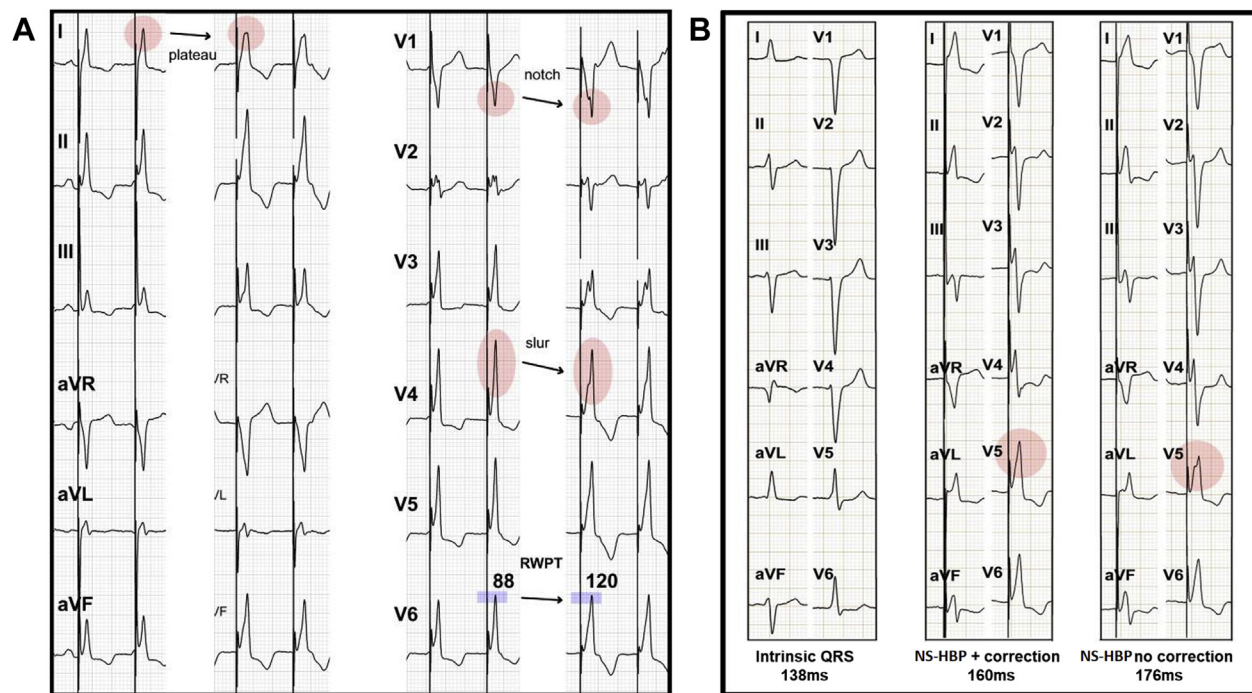
HOT-CRT

In patients with BBB due to conduction disease in the proximal HB, HBP often results in complete correction of the underlying conduction patterns. However, in patients with advanced cardiomyopathy, proximal BBB and peripheral conduction delay may coexist, and resynchronization by HBP is often incomplete in these patients.

Optimization of AV and VV intervals to achieve the narrowest QRS has been used in BiV CRT and has been shown in a randomized trial to result in greater

LV remodeling compared with nominal settings (25). HOT-CRT allows maximization of electrical resynchronization by further narrowing the QRS complex compared with BiV CRT (2,26).

HOT-CRT IN LBBB. In patients with coexisting proximal LBBB and peripheral conduction delay, HBP may result in correction of LBBB but with a residual wide QRS due to the uncorrected peripheral conduction delay. In these patients, HBP may be coupled with sequential LV pacing from a coronary sinus lead, which results in significant QRS

FIGURE 7 Morphological Criteria for Distinguishing NS-HBP From Myocardial Capture Only

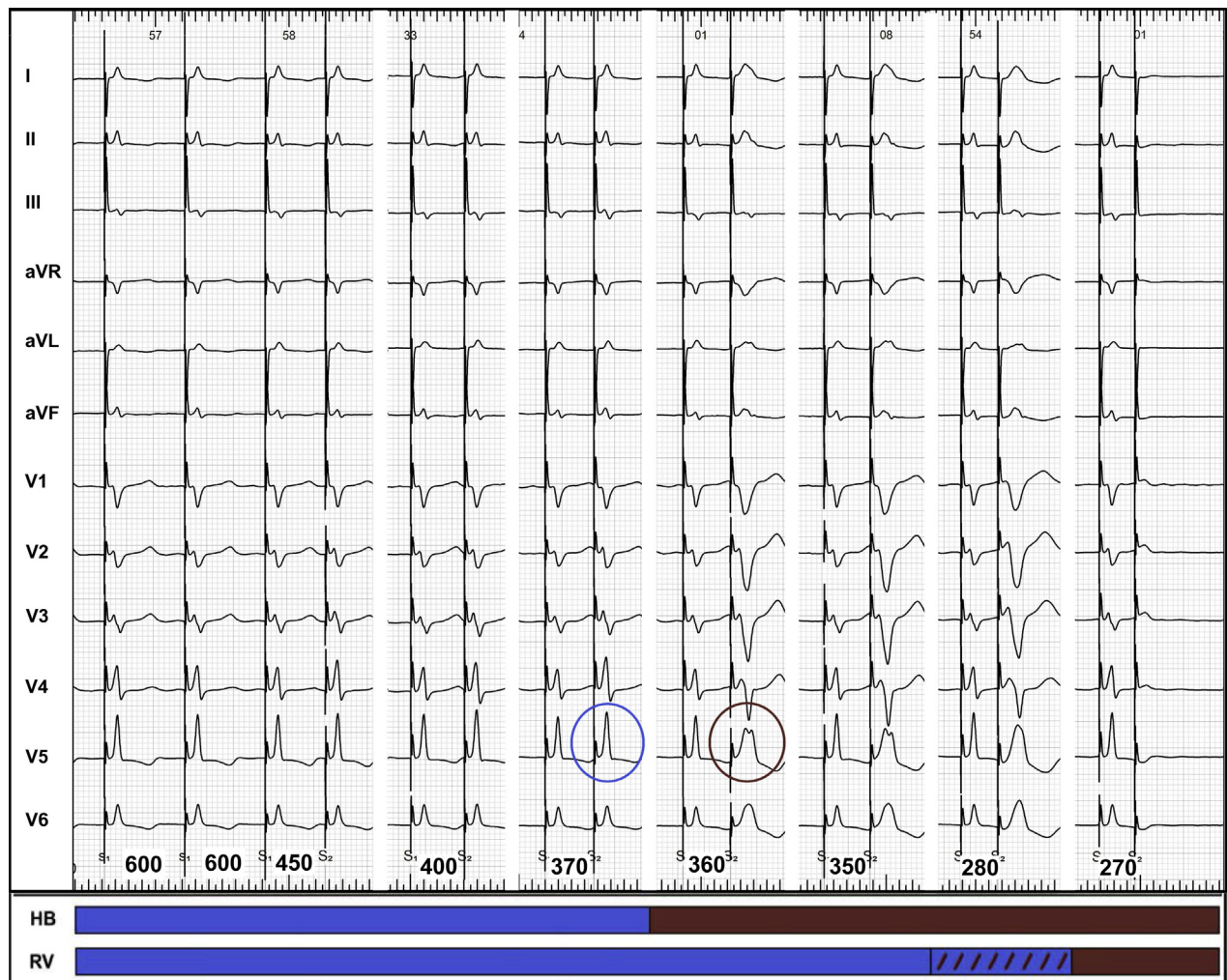
(A) Typical changes in QRS morphology during loss of His capture and transition from NS-HBP to myocardial capture only. In lead I, the pointy peak changes into a slur and/or plateau, in leads V₁ and V₃, a notch appears, and in leads V₄ and V₅ a slur develops. Global QRS duration changes from 128 to 162 ms and RWPT in lead V₆ prolongs from 88 to 120 ms. Note that there is a notch in the transitional QRS (lead V₂) already during NS-HBP; this is why notching in transitional QRS complexes is not diagnostic. **(B)** In patients with nonspecific intraventricular conduction disturbances (**left panel**), NS-HBP (**right panel**) often resembles QRS typically observed during right ventricular myocardial capture only. With high-output pacing (**middle panel**), there is some correction of conduction disturbances as evidenced by QRS shortening by 20 ms and disappearance of the notch and/or slur in lead V₅. Reproduced with permission from Jastrzebski et al. (22). Abbreviations as in [Figures 1 and 6](#).

narrowing. This approach has been reported to result in superior electrical resynchronization compared with HBP or BiV pacing alone. In a series of 25 patients with successful HOT-CRT (2), QRS duration decreased from 183 ± 27 ms at baseline to 162 ± 17 ms with BiV pacing ($p = 0.003$), to 151 ± 24 ms during HBP ($p < 0.0001$), and further to 120 ± 16 ms during HOT-CRT ($p < 0.0001$). It is essential that optimization of the VV delay be performed with careful analysis of 12-lead ECG to properly evaluate electrical resynchronization. The HBP lead and the LV lead may be connected in different ports (atrial, RV, or LV port) of the device. Pacing output required to (partially) correct LBBB needs to be determined, and voltage has to be programmed above this threshold, whereas the HBP to LV pacing interval needs to be determined based on the baseline HV interval or the stimulus to ventricular onset (depending on NS-HBP or S-HBP). This approach can result in maximization of conduction through the

His-Purkinje system and fusion with LV pacing ([Figure 11](#)). This approach is also applicable in patients who are in atrial fibrillation in whom HBP is unable to correct LBBB (and allows constant timing for LV fusion pacing).

HOT-CRT IN NIVCD. HBP usually does not correct the underlying conduction disturbance in patients with NIVCD. Traditional BiV pacing has not been shown to be beneficial in these patients. It is unclear if LV fusion pacing alone improves clinical outcomes in these patients by avoiding ventricular dyssynchrony induced by RV pacing. In patients with NIVCD and long PR intervals or chronic atrial fibrillation, fusion LV pacing is not feasible. In these patients, HOT-CRT provides the ability to achieve fusion LV pacing by sequential HBP followed by LV stimulation. In patients with chronic atrial fibrillation, the HBP lead is generally connected to the atrial port, and the AV (His-LV) delay is programmed at the HV or stimulus to

FIGURE 8 Programmed HBP Technique for Confirmation of HB Capture During Presumed Nonselective Pacing

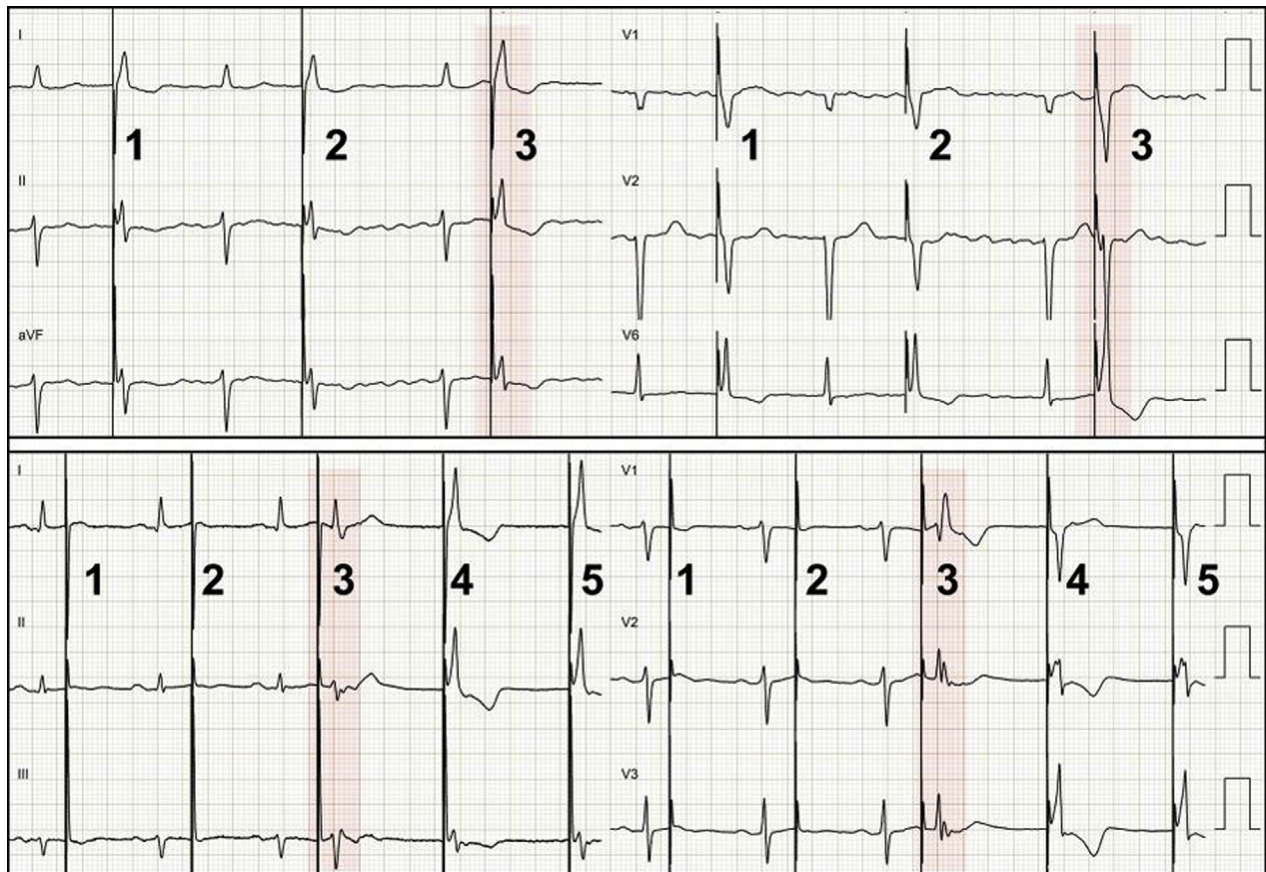


A single premature extra-stimulus is delivered at a drive cycle of 600 ms with a sudden dramatic change in the paced QRS at a coupling interval of 360 ms. This change is due to a transition from NS-HBP to myocardial capture only, due to the HB being refractory at this coupling interval. Note the slight change in QRS morphology of the extra-systolic beat at 280 ms, due to prolongation of intraventricular conduction. The **blue bar** corresponds to HB/RV capture, the **dashed bar** corresponds to capture with decremental conduction (relative refractory period), and the **brown bar** to loss of capture (effective refractory period). Reproduced with permission from Jastrzebski et al. (24). Abbreviations as in Figures 1 and 4.

ventricular interval to achieve maximal electrical synchronization. In patients with sinus rhythm, the VV (His-LV) delay is programmed appropriately to provide LV fusion pacing (Figure 12). Although this approach has not been systematically studied in a large population, 4 of 5 patients with NIVCD demonstrated both echocardiographic and clinical response in the previously mentioned study (2).

HOT-CRT IN RBBB. In a recent study of HBP in patients with RBBB who require CRT, HBP was effective

in correcting underlying RBBB in 29 of 39 (74%) patients (27). In some patients with HBP and uncorrected RBBB, nonselective capture of the surrounding RV septal myocardium may present the appearance of complete or partial correction of the RBBB pattern. As previously discussed, careful analysis of ECG is essential to differentiate correction of RBBB from NS-HBP. When NSHBC is associated with RBBB correction, 3 distinct morphological transitions should be observed (i.e., corrected NS-HBP, NS-HBP without correction of RBBB, and S-HBP). In patients with

FIGURE 9 Asynchronous Pacing in VOO Mode Results in Scanning of Diastole With Pacing Stimuli

(Top panel) The first 2 stimuli result in NS-HBP, whereas the third (highlighted) occurs when the HB is refractory and depolarizes only the RV myocardium, thus providing a diagnostic response (note QRS broadening and axis change). **(Bottom panel)** The first 2 stimuli fall on the effective refractory period of the HB and RV myocardium and result in noncapture. The third stimulus (highlighted) occurs when the ventricular myocardium is refractory but the HB is excitable again and propagates with selective HB pacing and RBBB (note isoelectric interval before QRS). The slight resulting delay allowed the ventricular myocardium to be excitable again. Stimuli 4 and 5 result in NS-HBP because with a longer coupling interval, both the HB and ventricular myocardium are excitable. Modified with permission from Jastrzebski et al. (24). Abbreviations as in [Figures 1, 3, and 4](#).

S-HBP or NS-HBP without correction of the underlying RBBB pattern, sequential RV pacing following HBP may be performed to achieve further QRS narrowing and improved electrical resynchronization ([Figure 13](#)). This is analogous to LV pacing via a coronary sinus lead which results in narrowing of the QRS complex in patients with LBBB.

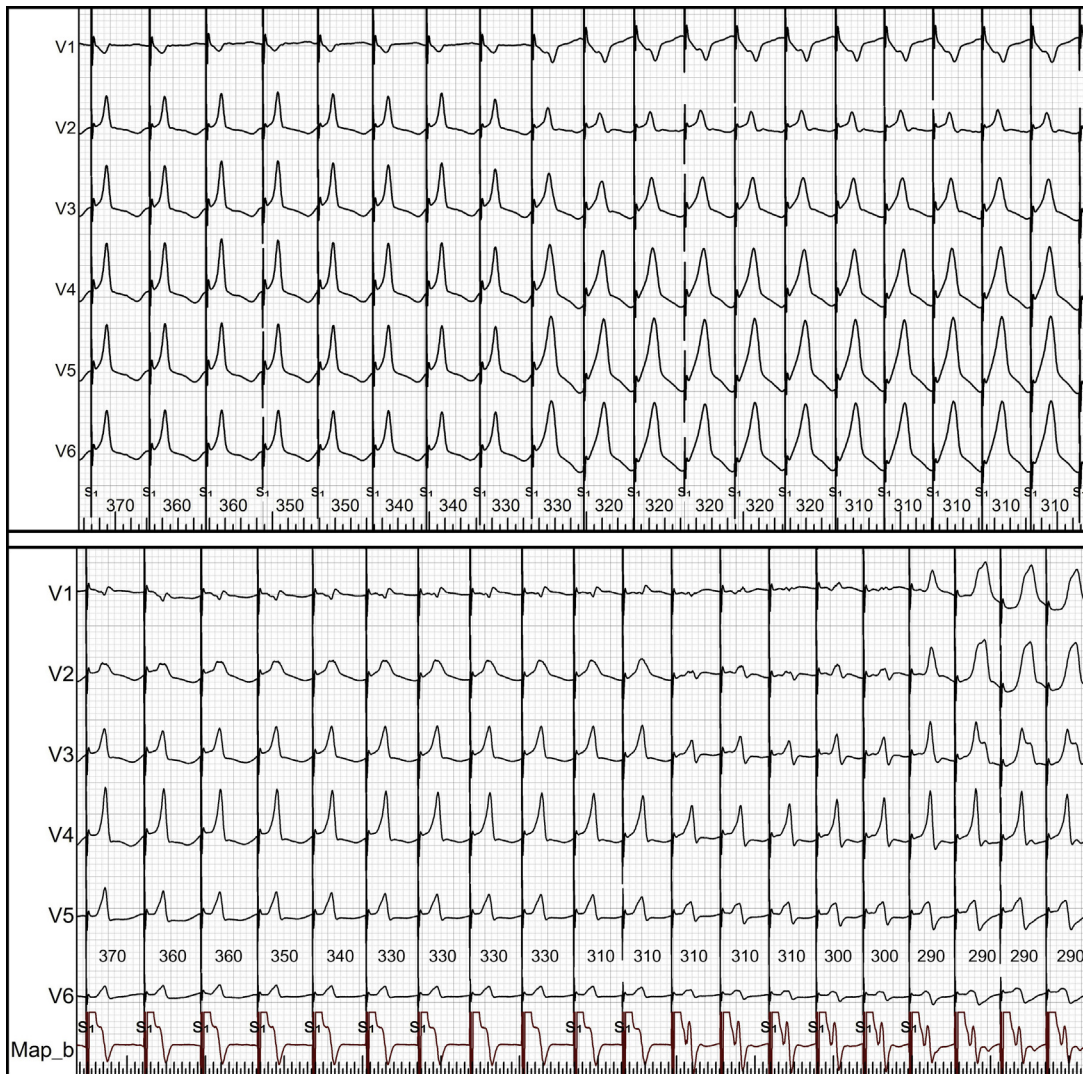
SPECIAL CONSIDERATIONS AT IMPLANTATION

Lead fixation may result in traumatic RBBB in approximately 6% of patients, which should be recognized before starting threshold tests; RBBB

resolves in more than one-half of these patients (28). In approximately 2% of patients, transient traumatic LBBB and second- to third- degree atrioventricular block may also be observed (28).

It is useful to initiate threshold tests by pacing at high output (e.g., 10 V/1 ms) in the unipolar pacing configuration to have a template for identifying transitions in QRS morphology with decrementing output. However, an artefact at this high output may distort the ECG signal directly after the spike and should not be confounded as being part of the QRS complex ([Figure 14A](#)). In the interest of time, the output may then be reduced to 5 V/1 ms and then in decrementing steps of 1 V to quickly check for the

FIGURE 10 Two Cases of Incremental Pacing From the His Lead to Confirm NS-HBP



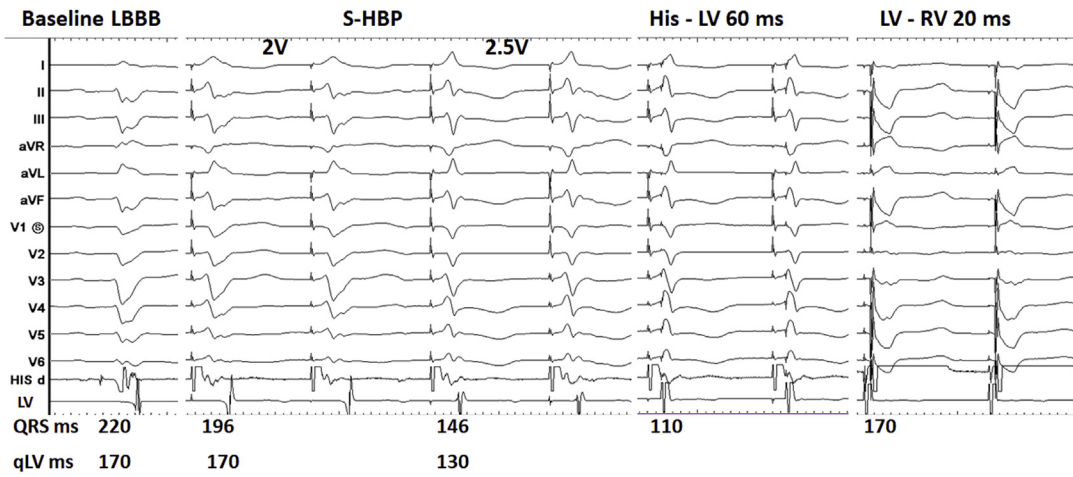
(Top panel) At a cycle length of 330 ms, NS-HBP suddenly transitions into myocardial only capture (due to a longer refractory period of the HS compared with myocardial tissue). There is no intermittent His capture thereafter, probably due to retrograde conduction into the His-Purkinje system. Note QRS notching in lead V₁ and the RWPT of >100 ms in lead V₆ with myocardial capture only. **(Bottom panel)** At a cycle length of 310 ms, NS-HBP suddenly transitions into selective His bundle pacing (note the loss of the pseudo-delta wave in lead V₃ that implies a longer refractory period of local myocardial tissue compared with the HB) and at cycle length of 290 ms, a RBBB aberration appears (which should not be mistaken for myocardial capture only). Note also the changes in electrogram morphology recorded from the His pacing lead. Abbreviations as in [Figures 1, 3, and 4](#).

presence of transitions in QRS morphology. More precise measurements in thresholds can then be performed in the identified ranges of interest, with smaller decrements in amplitudes and with narrower pulse widths if necessary.

HB and myocardial thresholds can significantly improve during the minutes following lead fixation.

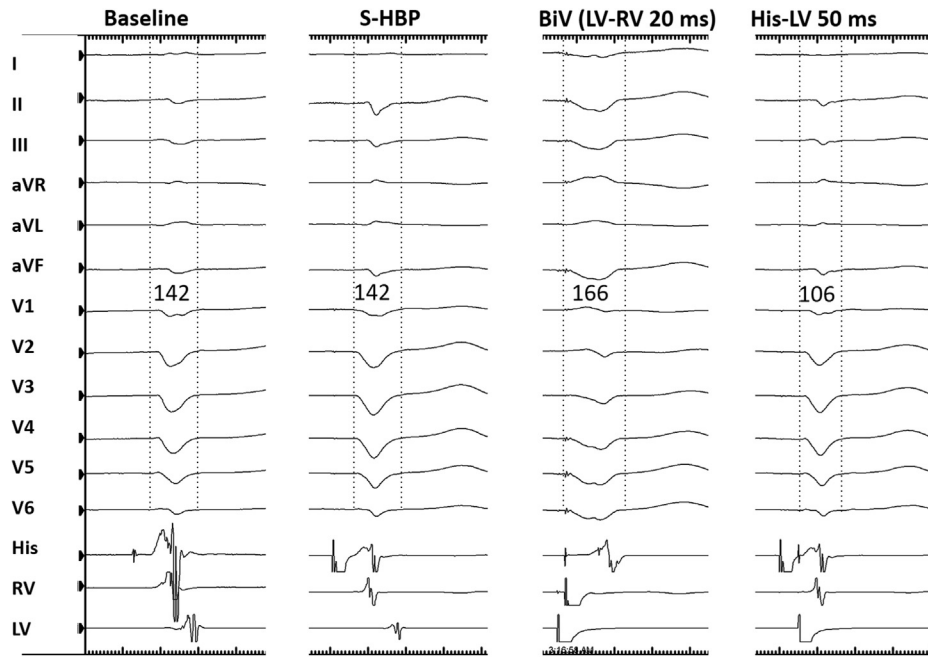
Acute traumatic injury to the HB not only affects acute capture thresholds, but also HV conduction time, and thereby, paced QRS morphology ([Figure 4C](#)). If the HB threshold is initially higher than the myocardial threshold, a transition from NS-HBP to myocardial capture only will be observed. If the HB threshold then falls below that of the myocardium

FIGURE 11 HOT-CRT in Left Bundle Branch Block



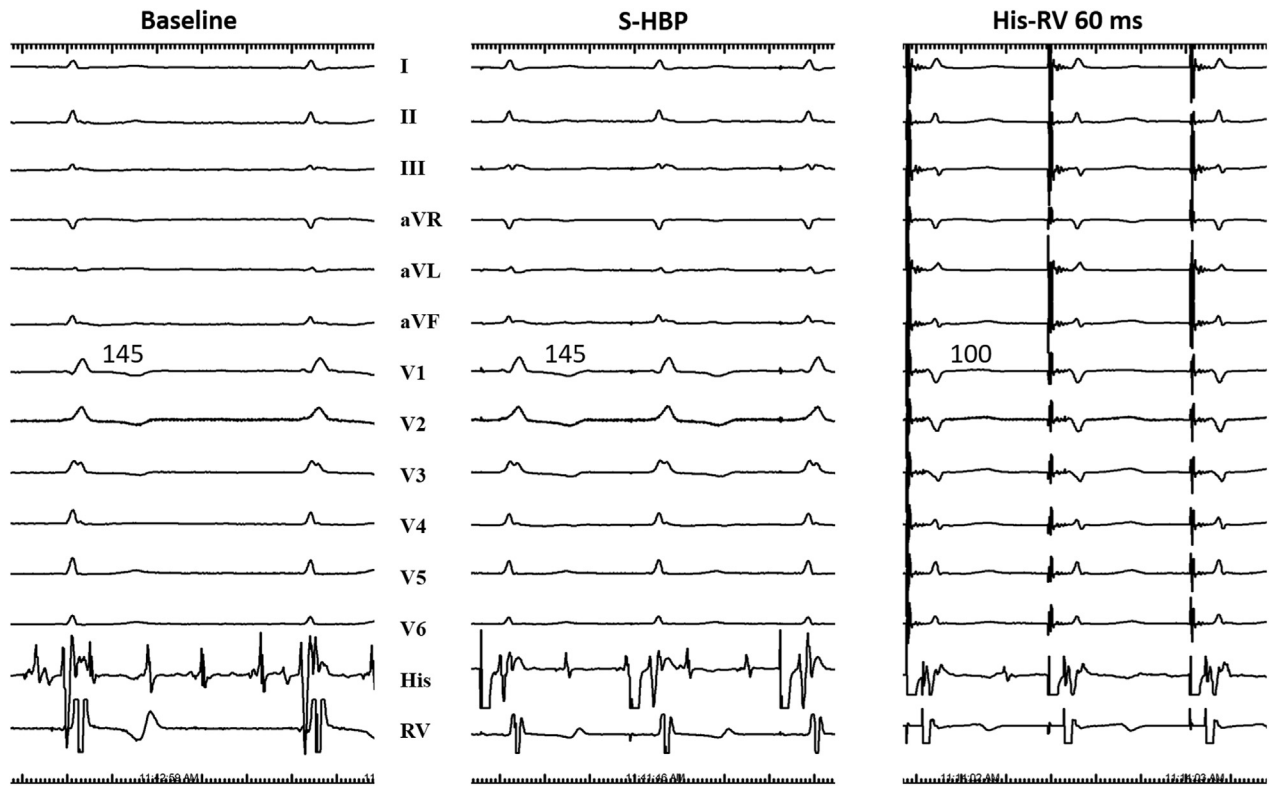
Surface ECG with intracardiac electrograms from HBP and LV leads are shown. Baseline ECG shows LBBB with QRS width of 220 ms and Q-LV of 170 ms. During S-HBP at 2.5 V, there is QRS narrowing to 146 ms and Q-LV of 130 ms, which suggests partial correction and peripheral conduction delay. With His-optimized cardiac resynchronization therapy (HOT-CRT) at a delay of 60 ms, QRS duration is significantly narrowed to 110 ms. Traditional biventricular pacing at LV-RV delay of 20 ms resulted in a QRS duration of 170 ms. Abbreviations as in [Figure 1](#).

FIGURE 12 HOT-CRT in NIVCD in a Patient With Atrial Fibrillation



Surface ECG, RV, LV, and HBP (His) lead electrograms at baseline, during HBP, biventricular (BiV) pacing and His-synchronous LV pacing are shown. Although there is no change in QRS duration or morphology with selective HBP, and even QRS widening during BiV pacing, His-optimized LV pacing at 50-ms delay resulted in significant narrowing of QRS from 142 to 106 ms. NIVCD = nonspecific intraventricular conduction defect; other abbreviations as in [Figures 1 and 11](#).

FIGURE 13 His-Optimized RV Pacing in RBBB in a Patient With Atrial Fibrillation



Surface ECG, His lead, and RV lead electrograms are shown at baseline, during selective HBP without RBBB correction and His-optimized RV pacing at His-RV delay of 60 ms with narrowing of QRS from 145 to 100 ms. Abbreviations as in [Figures 1 and 3](#).

(mostly in patients in whom significant HB injury current is initially observed), the transition will change from NS-HBP to S-HBP. The inverse scenario is also possible in patients with significant myocardial injury current at implantation.

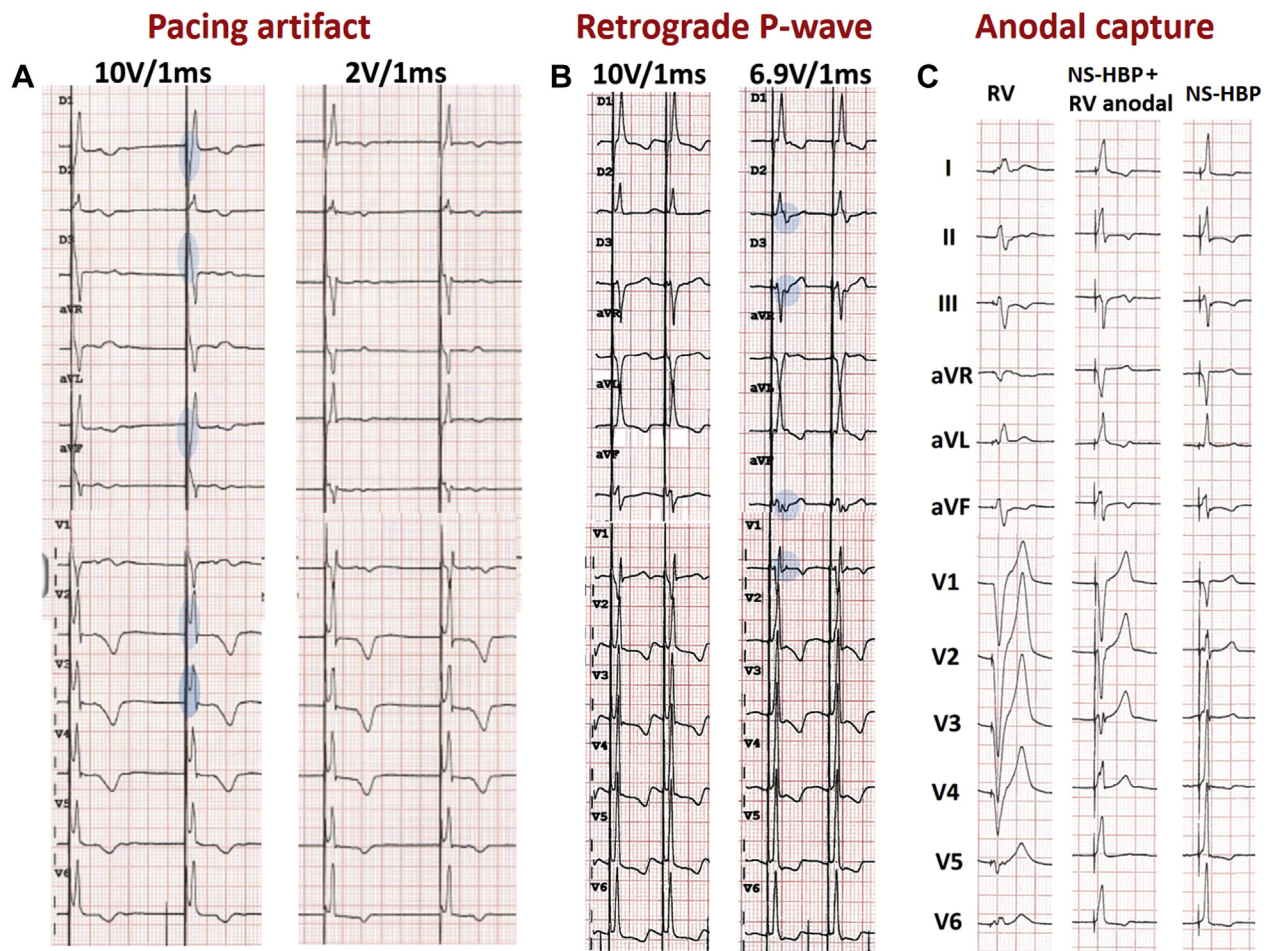
PITFALLS IN ECG ANALYSIS OF HBP

FUSION AND PSEUDO-FUSION BEATS. In patients with intrinsic atrioventricular conduction, it should be borne in mind that the QRS complex in HBP may be due to fusion or pseudo-fusion. This may also be an explanation for isolated changes in QRS morphology during asynchronous pacing (e.g., during threshold tests), in which scrutiny for a preceding P-wave can be helpful. Ventricular premature beats may also masquerade as transitions in morphology with HBP.

ATRIAL CAPTURE. The right atrium may be captured if the His lead is positioned in a proximal position (usually with a large A-wave in the endocavitary

electrogram of the His lead). With simultaneous capture of the atrium and the HB (also sometimes with the ventricular myocardium), the P-wave may not be visible. If the atrial threshold is higher than the HB and/or myocardial threshold, loss of atrial capture by the His lead may result in a modification of the morphology of the terminal part of the QRS due to the retrograde P-wave ([Figure 14B](#)). If the atrial threshold is lower than the HB and/or myocardial threshold, atrial capture only will result. If atrioventricular conduction is preserved, this may masquerade as S-HBP with a long HV interval ([Figure 15](#)). However, the P-wave deflection is usually visible, and pacing at faster rates will result in decremental atrioventricular conduction, which is usually absent with HBP. However, decremental HV conduction may occur in case of HB capture and infranodal conduction disorders.

ANODAL CAPTURE. The His lead may be connected to the LV port of a BiV pacemaker with a backup RV lead, which allows programming of an extended

FIGURE 14 Traps With ECG Analysis of HBP

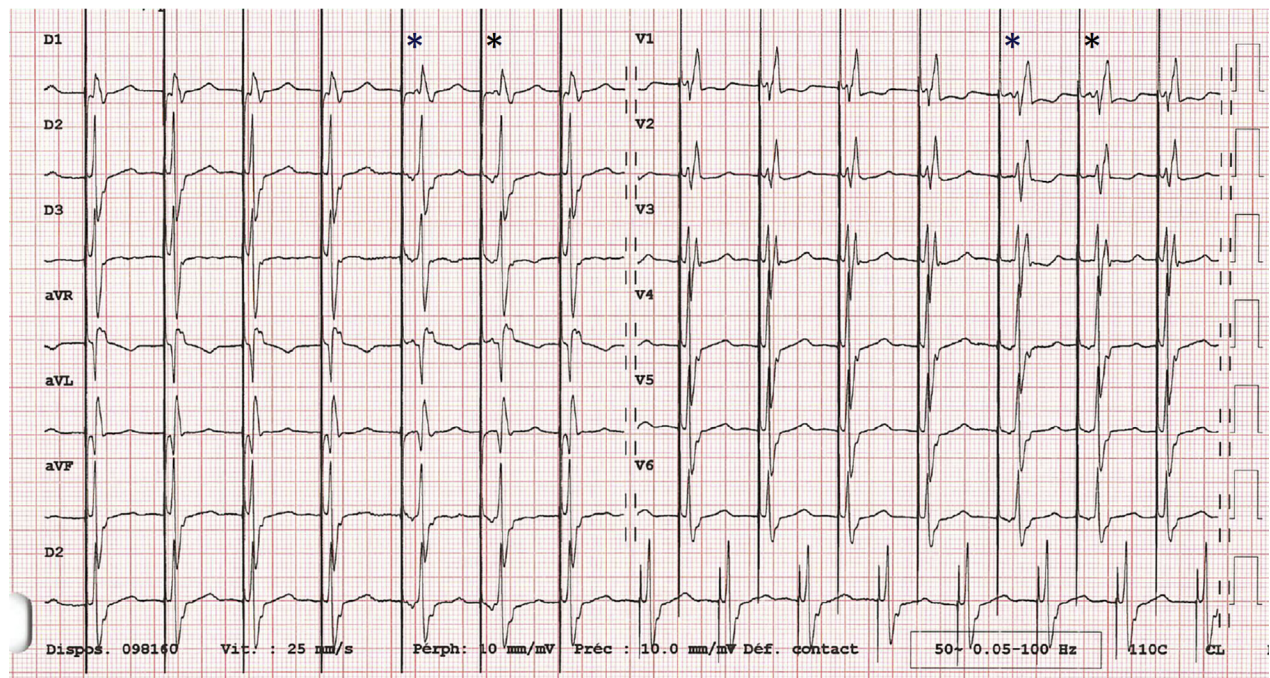
(A) Effect of unipolar output on the pacing artefact. The artefacts that are particularly well visible are highlighted (note the slurring of the waveform following the pacing spike). **(B)** Loss of atrial capture during pacing by the HB lead at 6.9 V/1 ms masquerading as a change in morphology of the terminal portion of the QRS due to the presence of retrograde P waves, which are readily visible in the inferior leads and in lead V₁ (highlighted). **(C)** Illustration of anodal capture during extended bipolar pacing in a patient with a narrow intrinsic QRS using the His lead as the cathode and the RV pacing lead ring as the anode. Note that the resulting QRS is a fusion of morphologies observed during pacing from the RV septal lead only and NS-HBP, recorded while pacing in the unipolar configuration. Abbreviations as in [Figure 1](#).

bipolar configuration (i.e., His tip to RV ring electrodes) (29). This may result in anodal capture from the RV ring electrode in 25% of patients ([Figure 14C](#)) (30). Simultaneous capture from the His lead (either S-HBP, NS-HBP, or myocardial capture only) and anodal capture from the RV lead, will result in a fusion complex and a QRS transition during threshold testing when there is a loss of anodal capture. This should not be confounded with a transition in QRS morphology that indicates successful His capture in patients with myocardial capture only. In case of loss of capture from the His lead with persisting anodal

capture, the QRS complex will resemble that of pacing from the RV lead. In general, His tip to RV anodal pacing configuration is avoided to prevent unnecessary RV pre-excitation (unless it is desirable, e.g., for backup myocardial capture or in case of uncorrected RBBB).

EFFECT OF PACING RATE. Rate-dependent BBB may be observed with S-HBP and NS-HBP. It may also confound results of the previously described pacing maneuvers, which are used to distinguish NS-HBP from myocardial capture only. At high pacing rates

FIGURE 15 Atrial Capture With the His Lead and Loss of HB Capture



Transient loss of HB capture (**asterisk**) with persistence of atrial capture and atrioventricular conduction in a patient with selective HBP and uncorrected RBBB. Note the captured P-wave morphology, which is narrow (due to simultaneous bi-atrial activation) with a short PR interval (due to proximity to the atrioventricular node). Abbreviations as in [Figure 1 and 3](#).

(e.g., >120 beats/min), infra-Hisian block may be mistaken for an elevation in capture thresholds because it manifests as complete loss of capture in patients with S-HBP and as QRS prolongation in patients with NS-HBP. Conduction via the His-Purkinje system at higher rates such as 120 to 150 beats/min should be checked for at implantation in patients with infranodal block (31). If the His lead is placed proximal to the site of block (threshold for correction of BBB is higher than the threshold for HBP without correction of BBB), then pacing thresholds should generally be tested at rates close to the programmed upper rates to ensure continued correction of BBB during follow-up.

MISCELLANEOUS FINDINGS. A number of device troubleshooting issues can result in ECG findings (e.g., oversensing of atrial or His potentials by the His lead with inhibition of pacing). HBP troubleshooting and programming is covered in detail elsewhere (29,32).

CONCLUSIONS

With HBP, the standard 12-lead ECG is a useful tool for performing threshold tests, for identifying the type of capture, to evaluate whether conduction disease has been corrected, and also for optimizing device settings (e.g., with HOP-CRT). Although ECG interpretation with HBP is straightforward in most cases, it may be complex in some instances, with a number of pitfalls. Physicians should not be deterred by the occasional puzzling tracing and should strive to elucidate its interpretation. This will ultimately enable them to understand the physiology of HBP and better manage their patients.

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REFERENCES

- Narula OS. Longitudinal dissociation in the His bundle. Bundle branch block due to asynchronous conduction within the His bundle in man. *Circulation* 1977;56:996-1006.
- Vijayaraman P, Herweg B, Ellenbogen KA, Gajek J. His-optimized cardiac resynchronization therapy to maximize electrical resynchronization. *Circ Arrhythm Electrophysiol* 2019;12:e006934.
- Vijayaraman P, Dandamudi G, Zanon F, et al. Permanent His bundle pacing: Recommendations from a multicenter His bundle pacing collaborative working group for standardization of definitions, implant measurements, and follow-up. *Heart Rhythm* 2018;15:460-8.
- Nagarajan VD, Ho SY, Ernst S. Anatomical considerations for His bundle pacing. *Circ Arrhythm Electrophysiol* 2019;12:e006897.
- Saini A, Serafini NJ, Campbell S, et al. Novel method for assessment of His bundle pacing morphology using near field and far field device electrograms. *Circ Arrhythm Electrophysiol* 2019;12:e006878.
- Gu M, Hu Y, Hua W, et al. Visualization of tricuspid valve annulus for implantation of His bundle pacing in patients with symptomatic bradycardia. *J Cardiovasc Electrophysiol* 2019;30:2164-9.
- Zhang J, Guo J, Hou X, et al. Comparison of the effects of selective and non-selective His bundle pacing on cardiac electrical and mechanical synchrony. *Europace* 2018;20:1010-7.
- Beer D, Sharma PS, Subzposh FA, et al. Clinical outcomes of selective versus nonselective His bundle pacing. *J Am Coll Cardiol EP* 2019;5:766-74.
- Lustgarten DL, Calame S, Crespo EM, Calame J, Lobel R, Spector PS. Electrical resynchronization induced by direct His-bundle pacing. *Heart Rhythm* 2010;7:15-21.
- Lustgarten DL, Crespo EM, Arkhipova-Jenkins I, et al. His-bundle pacing versus biventricular pacing in cardiac resynchronization therapy patients: a crossover design comparison. *Heart Rhythm* 2015;12:1548-57.
- Ajjola OA, Upadhyay GA, Macias C, Shivkumar K, Tung R. Permanent His-bundle pacing for cardiac resynchronization therapy: initial feasibility study in lieu of left ventricular lead. *Heart Rhythm* 2017;14:1353-61.
- Teng AE, Massoud L, Ajjola OA. Physiological mechanisms of QRS narrowing in bundle branch block patients undergoing permanent His bundle pacing. *J Electrocardiol* 2016;49:644-8.
- Barba-Pichardo R, Morina-Vazquez P, Fernandez-Gomez JM, Venegas-Gamero J, Herrera-Carranza M. Permanent His-bundle pacing: seeking physiological ventricular pacing. *Europace* 2010;12:527-33.
- Sharma PS, Dandamudi G, Herweg B, et al. Permanent His-bundle pacing as an alternative to biventricular pacing for cardiac resynchronization therapy: a multicenter experience. *Heart Rhythm* 2018;15:413-20.
- Upadhyay GA, Cherian T, Shatz DY, et al. Intracardiac delineation of septal conduction in left bundle-branch block patterns. *Circulation* 2019;139:1876-88.
- El-Sherif N, Amay YLF, Schonfield C, et al. Normalization of bundle branch block patterns by distal His bundle pacing. Clinical and experimental evidence of longitudinal dissociation in the pathologic his bundle. *Circulation* 1978;57:473-83.
- Khaykin Y, Exner D, Birnie D, Sapp J, Aggarwal S, Sambelashvili A. Adjusting the timing of left-ventricular pacing using electrocardiogram and device electrograms. *Europace* 2011;13:1464-70.
- Jastrzebski M, Moskal P, Bednarek A, Kielbasa G, Vijayaraman P, Czarnecka D. His bundle has a shorter chronaxie than does the adjacent ventricular myocardium: implications for pacemaker programming. *Heart Rhythm* 2019;16:1808-16.
- Hirao K, Otomo K, Wang X, et al. Para-Hisian pacing. A new method for differentiating retrograde conduction over an accessory AV pathway from conduction over the AV node. *Circulation* 1996;94:1027-35.
- Ganière V, Domenichini G, Niculescu V, Cassagneau R, Defaye P, Burri H. A new electrocardiogram algorithm for diagnosing loss of ventricular capture during cardiac resynchronisation therapy. *Europace* 2013;15:376-81.
- Jastrzebski M, Kukla P, Fijorek K, Czarnecka D. Universal algorithm for diagnosis of biventricular capture in patients with cardiac resynchronization therapy. *Pacing Clin Electrophysiol* 2014;37:986-93.
- Surawicz B, Childers R, Deal BJ, et al. AHA/ACC/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society: Endorsed by the International Society for Computerized Electrocardiology. *J Am Coll Cardiol* 2009;53:976-81.
- Jastrzebski M, Moskal P, Curila K, et al. Electrocardiographic characterization of non-selective His-bundle pacing: validation of novel diagnostic criteria. *Europace* 2019;21:1857-64.
- Jastrzebski M, Moskal P, Bednarek A, Kielbasa G, Vijayaraman P, Czarnecka D. Programmed His bundle pacing. *Circ Arrhythm Electrophysiol* 2019;12:e007052.
- Trucco E, Tolosana JM, Arbelo E, et al. Improvement of reverse remodeling using electrocardiogram fusion-optimized intervals in cardiac resynchronization therapy: a randomized study. *J Am Coll Cardiol EP* 2018;4:181-9.
- Boczar K, Slawuta A, Zabek A, et al. Cardiac resynchronization therapy with His bundle pacing. *Pacing Clin Electrophysiol* 2019;42:374-80.
- Sharma PS, Naperkowski A, Bauch TD, et al. Permanent His bundle pacing for cardiac resynchronization therapy in patients with heart failure and right bundle branch block. *Circ Arrhythm Electrophysiol* 2018;11:e006613.
- Vijayaraman P, Dandamudi G, Ellenbogen KA. Electrophysiological observations of acute His bundle injury during permanent His bundle pacing. *J Electrocardiol* 2016;49:664-9.
- Burri H, Keene D, Whinnett Z, Zanon F, Vijayaraman P. Device programming for His bundle pacing. *Circ Arrhythm Electrophysiol* 2019;12:e006816.
- Starr N, Dayal N, Domenichini G, Stettler C, Burri H. Electrical parameters with His-bundle pacing: considerations for automated programming. *Heart Rhythm* 2019;16:1817-24.
- Vijayaraman P, Naperkowski A, Ellenbogen KA, Dandamudi G. Electrophysiologic insights into site of atrioventricular block: lessons from permanent His bundle pacing. *J Am Coll Cardiol EP* 2015;1:571-81.
- Lustgarten DL, Sharma PS, Vijayaraman P. Troubleshooting and programming considerations for His bundle pacing. *Heart Rhythm* 2019;16:654-62.

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