Permanent His Bundle Pacing (HBP): Recommendations From A Multi-Center HBP Collaborative Working Group For Standardization Of Definitions, Implant Measurements And Follow-Up

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Abstract

His bundle pacing (HBP) prevents ventricular dyssynchrony and its long-term consequences by preserving normal electrical activation of the ventricles. Since the original description of permanent HBP in 2000, the adoption of HBP has increased over the last several years. However the reporting of procedural and clinical outcomes to date is not uniform. This paper is a collaboration between several implanters with significant experience in HBP to establish a uniform set of definitions encompassing the different forms of HBP, as well as define a standardized approach to gathering data endpoints to ensure consistency in reported outcomes.

Criteria for His Bundle Pacing

Baseline	Normal QRS	His-Purkinje Conduction Disease	
		With correction	Without correction
Selective HBP	• S-QRS = H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead with S-V=H-V • Paced QRS = native QRS • Single capture threshold (His bundle)	• S-QRS ≤ H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead • Paced QRS < native QRS • 2 distinct capture thresholds (HBP with BBB correction, HBP without BBB correction)	• S-QRS ≤ or > H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead • Paced QRS = native QRS • Single capture threshold (HBP with BBB)
Non-selective HBP	• S-QRS < H-QRS (usually 0, S-QRS$_{end}$ = H-QRS$_{end}$) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact (local myocardial capture) • Paced QRS >native QRS with normalization of precordial and limb lead axes with respect to rapid dV/dt components of the QRS • 2 distinct capture thresholds (His bundle capture, RV capture)	• S-QRS < H-QRS (usually 0, S-QRS$_{end}$ < H-QRS$_{end}$) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact • Paced QRS ≤ native QRS • 3 distinct capture thresholds (HBP with BBB correction, HBP without BBB correction, RV capture)	• S-QRS < H-QRS (usually 0) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact • Paced QRS > native QRS (see text) • 2 distinct capture thresholds (HBP with BBB, RV capture)

Introduction

From an electrical and hemodynamic standpoint, His bundle pacing (HBP) is desirable in patients who require chronic ventricular pacing. By preserving normal electrical activation of the ventricles, HBP prevents ventricular dyssynchrony and its long-term consequences. However, the technical challenge of achieving permanent HBP (PHBP) has been an obstacle to its reliable application in routine clinical practice. With the advent of improved pacing lead and delivery sheaths, [1,2] several publications showing safety and feasibility, PHBP has been gaining more widespread acceptance in the electrophysiology community. Since the original description of PHBP in patients undergoing AV nodal ablation by Deshmukh et al[3] in 2000, several investigators have reported on the successful implementation of PHBP in patients with normal His-Purkinje conduction, [4,5,6,7,8,9] bundle branch block (BBB), complete nodal and infra-nodal AV block, [10,11] and as an alternative to cardiac resynchronization therapy (CRT). [12,13,14,15] The field however is relatively nascent. The reporting of procedural and clinical outcomes to date is not uniform and is largely based on single-center experiences with differing definitions and variations in data reporting with gaps in data. To improve the adoption of HBP, data will need to be aggregated to demonstrate procedure success, safety, and efficacy in controlled and real-world settings.

This paper is a collaboration between implanters with significant experience (>50 implants) in PHBP to establish a uniform set of definitions encompassing the different forms of HBP as well as define a standardized approach to gathering data endpoints to ensure consistency in reported outcomes. The authors collectively

have performed more than 1500 HBP lead implantations during the last 10 years. Additionally, the authors provide insights and recommendations based on current evidence regarding patient selection, His bundle lead placement, outpatient management, and guidance for technical training.

Definitions:

A lack of uniformity in terminology in the literature regarding PHBP has contributed to confusion regarding the types of capture observed and pacing threshold definitions.[3,7,9,13,16] Furthermore, oft-quoted criteria used to define His bundle capture that were established in the setting of normal His Purkinje tissue are imprecise in some respects when pacing diseased His Purkinje tissue. In order to provide uniformity, the authors propose the following definitions and criteria, building on the original descriptions published by Williams et al[17] and Deshmukh et al[3] (Supplemental Table 1). The authors use the term "His bundle" to denote any portion of AV junction activation, which results in maximal engagement of functional right and left fascicles without significant decremental conduction as would be seen when activating the AV node and/or AV nodal atrial inputs. Broadly there are two forms of HBP capture: Selective capture in which the only tissue that is captured by the pacing stimulus is the His bundle, and Nonselective capture in which there is fusion capture of the His bundle and adjacent ventricular tissues: The authors further refine these definitions in the presence or absence of His Purkinje conduction disease as follows according to 4 basic criteria:

1) Relationship of the His-QRS and stimulus-QRS intervals

2) Presence or absence of direct capture of local ventricular electrogram on the pacing lead
3) QRS duration and morphology
4) Capture thresholds

Electrograms recorded in the HBP lead should generally demonstrate His deflection with HV intervals greater than 35 ms. There may be exceptions where successful HBP is achieved by pace-mapping in dependent patients without actually recording His electrograms.

HBP in Normal His-Purkinje Conduction:

Selective His bundle pacing (S-HBP) is defined by ventricular activation occurring exclusively over the His-Purkinje system (Figure 1). S-HBP is recognized by the following criteria:

(1) The pacing stimulus to QRS (S-QRS) onset interval is equal to the native His-QRS onset interval (H-QRS); S-QRS interval is measured from the end of the stimulus artifact to the earliest onset of the QRS on the 12 lead ECG (S-QRS≅ H-QRS)

(2) The local ventricular electrogram is not directly captured by the pacing stimulus and is discrete on the pacing lead[18] with the stimulus to local ventricular (S-V) activation time on the pacing lead being equal to the His to local ventricular (H-V) activation time (S-V ≅ H-V). The difference between the two intervals is usually less than 10 ms.

(3) The paced QRS morphology is the same as the native QRS morphology since in both cases cardiac activation and repolarization are the result of the same antegrade His Purkinje activation sequence, as evidenced by electrocardiographic (ECG) concordance of QRS and T wave complexes

(4) Usually a single capture threshold (His capture) is observed, although at significantly higher output, capture of adjacent RV myocardium may result in non-selective pacing (fusion of ventricular and His capture). In some cases, there may be atrial capture at higher pacing outputs in addition to S-HBP. Selective-HBP has variably been described in the literature as direct HBP,[3] pure-His pacing,[7] and selective-direct HBP.[19]

Non-Selective His bundle pacing (NS-HBP) is defined as simultaneous capture of local myocardium at the pacing site and the His bundle and is recognized by the following criteria:

(1) S-QRS interval is usually equal to zero as there is no isoelectric interval between pacing stimulus and QRS due to the presence of a pseudo-delta wave, and the stimulus to the end of QRS ($S\text{-}QRS_{end}$) is ≤ $H\text{-}QRS_{end}$. (Figure 2) Occasionally the local myocardial capture may not reach a critical mass to inscribe an instantaneous pseudo-delta wave in which case S-QRS < H-QRS. However, careful analysis of all 12 leads usually reveals evidence of anteroseptal myocardial capture prior to HPS-mediated ventricular capture.

(2) The local ventricular electrogram is directly captured by the pacing stimulus and is not seen as a discrete component[18]

(3) The paced QRS duration will usually be longer than the native QRS duration by the H-QRS interval. The overall electrical axis of the paced QRS will be concordant with the electrical axis of the intrinsic QRS with the rapid dV/dt components of both QRS morphologies being the same.

(4) There will usually be two distinct capture thresholds – right ventricular and His capture: During threshold testing, narrowing of QRS at higher output due to fusion between RV and His bundle capture and as the output is decreased there is widening of QRS due to loss of His bundle capture (figure 2). Alternately, QRS may be wider at higher pacing output and narrower when output is decreased, due to loss of RV capture (figure 3). Because the difference between RV and His capture thresholds is small, the final programmed output including the safety margin would result in nonselective HBP. The hallmark of NS-HBP is pacing output dependent changes in QRS morphology due to variable capture of RV and His.

HBP in Patients with His-Purkinje Conduction Disease (HPCD):

HBP has been shown to be feasible in patients with underlying BBB and infra-nodal AV block.[10-15] In these patients, the HV interval may be prolonged or absent (as is the case in complete HV block). The final His bundle paced QRS morphology and duration in these patients may be significantly different from the baseline QRS, depending on the degree and extent of recruitment of latent fascicular tissue during HBP, and on whether or not the underlying escape is fascicular or ventricular in origin (figure 4). S-QRS time may also be notably shorter with recruitment of more

distal segments of the His bundle. Finally, patients with cardiomyopathy may have peripheral conduction disease superimposed on proximal His bundle disease, wherein complete normalization of QRS may not be possible. The following are criteria that further refine the patterns of activation observed with HBP in a diseased His Purkinje system.

***S-HBP with correction of HPCD**:*

(1) S-QRS ≤ H-QRS with an isoelectric interval between stimulus to onset of QRS; HV interval is often prolonged in patients with BBB and HBP may shorten the S-QRS interval by output dependent capture of latent fascicular tissue[19] or by virtual electrode polarization effect. [20] In patients with complete or 2:1 HV block, selective His capture can occur with short S-QRS intervals, especially when the HBP lead is located beyond the site of intra-Hisian block, [11,21] or functionally correcting the underlying conduction disease.

(2) The local ventricular electrogram on the pacing lead will be discrete from the pacing artifact. The morphology and timing of the local ventricular electrogram will be different from the baseline due to the change in local activation resulting from the correction of the BBB (Figure 5, panel C)

(3) Paced QRS duration will be narrower than the native QRS with BBB. Bundle branch block may be completely normalized or partially corrected. In patients with HV block, paced QRS will be narrower than the conducted beats or the escape rhythm.

(4) HBP will result in 2 distinct capture thresholds, capture with and without QRS normalization. An example is shown in Figure 6. If the HBP lead is located distal to the site of block, only a single capture threshold may be observed (with QRS normalization). It is important to recognize the different thresholds during follow-up in order to program the optimal output that results in maximal recruitment of the His Purkinje system.

NS-HBP with correction of HPCD:

(1) S-QRS interval is less than H-QRS interval and is most likely to be zero without isoelectric interval due to pseudo-delta wave resulting from ventricular fusion; Occasionally, S-QRS interval is less than H-QRS interval but with isoelectric interval between the stimulus and QRS onset as explained earlier; Because of correction of BBB, the stimulus to the QRS_{end} will be less than the His–QRS_{end}

(2) The local ventricular electrogram is directly captured by the pacing stimulus and is not seen as a discrete component

(3) Paced QRS duration will usually be less than the native QRS (Figure 5, Panel B); however, in some patients with prolonged HV intervals, the duration of ventricular fusion may overshadow the narrowing of the BBB and result in the same or longer paced QRS duration. There will be normalization of precordial and limb lead axes with respect to rapid dV/dt components of the QRS after the initial RV fusion during the HV intervals.

(4) Three distinct capture thresholds will be observed typically, nonselective capture with normalization, non-selective capture without normalization, and finally ventricular capture only. (Figure 7).

S-HBP without correction of BBB:

(1) S-QRS interval is usually equal to the H-QRS interval. However, depending on the location of the HBP lead in relation to the site of conduction disease and capture characteristics of the diseased tissue, sometimes the S-QRS may be shorter or longer than the H-QRS interval.

(2) Local ventricular electrogram in the HBP lead will be discrete from the pacing artifact. The S-V will usually be the same as H-V, as in criterion 1 above.

(3) The paced QRS duration will be equal to the native QRS.

(4) There will be a single His capture threshold (HBP with BBB).

NS-HBP without correction of BBB:

(1) S-QRS interval is 0 or less than H-QRS depending on the amount of pre-excitation present

(2) The local ventricular electrogram is directly captured by the pacing stimulus

(3) The paced QRS duration will usually be longer than the native QRS duration by the H-QRS interval. However, in patients with RBBB, NS-HBP may significantly narrow the QRS even in the absence of BBB correction due to fusion of left bundle activation with early anteroseptal RV activation (figure 7, panel 3).

(4) Two distinct capture thresholds will usually be observed (HBP with BBB, followed by the RV-only capture threshold). The ventricular capture threshold may be higher or lower than the His capture threshold.

The above criteria for HBP are based on the pacing response at a given pacing site: the precise anatomical location at any given site can only be conjectured on the basis of the pacing response since we currently have no means to determine the precise location of the lead tip and its relationship to the His bundle non-invasively. The amplitude of the His bundle electrogram and the presence or absence of His injury current may distinguish physical contact of the lead with the His bundle but in the absence of autopsy and/or animal model data this remains conjectural. Additional nuances may be observed and clarified as we gain more experience with this technique. The most important issue is to clearly document RV and His capture thresholds and BBB correction thresholds for the purposes of follow-up and programming final output settings.

Recommendations for outcomes endpoints:

Despite the initial description of successful HBP by Deshmukh et al, in 2000, HBP did not reach mainstream implementation due to perceived procedural difficulties until recently. Recent reports suggest fluoroscopy and procedure duration for HBP to be only slightly longer than conventional pacemakers, and well within the range of LV lead placement procedure times.[1,2] However, compared to RV pacing site, HBP lead requires detailed mapping and lead fixation in the His bundle region. Despite

improved procedural success rates, anatomical and pathological variations continue to pose technical challenges during the implant process. Review of the current literature provides insufficient insight into the nature and challenges of the procedure given the absence of standardized reporting for procedural details. The authors propose that studies involving HBP should report fluoroscopy and procedural duration times for implantation of the HBP lead – from vascular access to lead sleeve fixation - in addition to the overall fluoroscopy and skin-to-skin procedural duration.

Pacing threshold: There is a lack of uniformity in the HBP literature in reporting of capture thresholds given that the higher capture thresholds sometimes encountered are offset by programming the pulse width at 1ms, which is useful for maximizing battery longevity. The authors suggest that His bundle capture thresholds be reported at a width of 1ms duration to provide uniformity in terms of comparison. Additionally, a 12-lead ECG should be recorded intra-procedurally to assess pacing thresholds at implant. It is extremely helpful in identifying and differentiating NS-HBP and local myocardial capture in addition to assessing bundle branch correction. Historically in patients reported with “para-Hisian pacing” (NS-HBP), operators may have reported the RV threshold rather than the His capture threshold. In cases of NS-HBP, it is critical that investigators report the RV capture threshold in addition to the His capture threshold. In patients with BBB, the output necessary to correct the BBB should be reported as the target threshold.

Sensing: The sensing characteristics of the HBP electrograms can be challenging in a patient where the HBP lead serves as the right ventricular sensing electrode. Because of the location of the lead at the tricuspid annulus often within or immediately adjacent to the fibrous membranous septum, the amplitude of ventricular electrograms is low. The amplitude of the atrial and occasionally the His electrograms can be large enough to cause ventricular oversensing. However in situations where the HBP lead is connected to LV or atrial port, sensing is not an issue but the measured "R" wave should be reported.

Threshold testing during follow-up should be performed using a 12-lead ECG, especially in patients with underlying BBB. Selective or Nonselective HBP should be recorded during follow-up. In patients with NS-HBP, both the His capture threshold and RV capture threshold should be reported. In patients with BBB, His capture threshold required to correct the BBB should be reported.

Lead complications and safety: Better data are needed regarding chronic capture thresholds and lead stability. Any significant and/or sudden increases in His capture threshold and/or need for lead revision should be reported, as would be performed for standard leads. An increase in capture threshold of >1 V in His bundle or RV pacing threshold is considered significant and should be reported. Lead-related complications should be defined as an adverse event due to the presence or performance of the lead for HBP, and which was either resolved by invasive intervention or resulted directly in the death of the patient, explantation of the device or termination of significant device function. Issues such as far-field atrial

over-sensing, and ventricular under-sensing, should be documented. Any interventions required to address sensing issues should be reported. All pacing and sensing parameters should be documented at each in-person follow-up visit, which is presumed to be yearly. The need for an unscheduled visit for reprogramming or troubleshooting should also be reported.

In publications of clinical research in HBP, the total number of patients studied, the number and types of complications and deaths, and the events-over-time data should be recorded with a minimum follow-up of 6 months.

Patient Selection:

Majority of patients in published literature on PHBP were treated for AV block, and demographics reflect a typical pacemaker population. HBP has been shown to be feasible even in the setting of infra-nodal block.[10,11] However, more data are needed to determine the long term outcomes in these patients, and in light of valid concerns regarding the possibility of disease progression and/or lead failure, one must consider the possibility of providing a backup RV lead or intentionally targeting His bundle sites demonstrating nonselective capture with low ventricular capture thresholds (back-up RV capture from the His lead). A similar consideration applies to patients for whom AV node ablation is contemplated.

Several studies involving PHBP have reported on the utility of HBP instead of biventricular pacing to implement CRT[12,13,14,15,19] (Figure 5). This application has garnered great interest, representing an alternative and more physiologic means to implement CRT. However, His bundle pacing in CRT-indicated patients will be

relevant only in the setting of bundle branch disease due to longitudinal dissociation in the AV junction and probably not helpful in patients with distal conduction disease. Much remains to be learned about prospectively distinguishing the type of underlying conduction disease that is present. At a minimum, PHBP by maintaining normal QRS likely prevents pacing induced dyssynchrony. Until long-term data on clinical and echocardiographic outcomes are available, we consider HBP a reasonable back-up option among patients in whom BiV pacing either can't be performed or in whom BiV pacing has failed despite ideal lead placement and optimization attempts. Given the morbidity and poor lead durability in the setting of surgical epicardial lead placement, it may be reasonable to attempt re-synchronization with HBP before sending a patient for surgical LV lead placement.

Lead placement

As defined above, there are two broadly distinct patterns of electrical activation encountered during His bundle lead implantation. Though intuitively one might anticipate selective capture to be preferable over NSHBP, published data indicate there is little hemodynamic and clinical difference between the two forms of capture possibly due to rapid conduction of His-Purkinje system relative to ventricular myocardial conduction.[22,23] The degree of ventricular pre-excitation varies considerably in the setting of nonselective capture and conceivably - especially if there is underlying conduction delay in the HPS - it might result in some dyssynchrony. The preponderance of published data suggests that adequate pacing

threshold for His capture should be the primary determinant at the time of implantation, irrespective of the presence or absence of ventricular fusion.

There is no absolute threshold cutoff defining an adequate His bundle pacing threshold. However, observations from experienced implanters suggest that a high threshold (>3V @ 1 msec) and a significant difference between unipolar and bipolar pacing thresholds at the time of implant are likely to demonstrate worsening capture thresholds at follow up, sometimes requiring lead revision. The presence of His bundle injury current at the time of implantation, conversely, is associated with stable thresholds at follow up. [24] While this is a desirable observation, it is not clear that it is critical, i.e. there may be sites demonstrating excellent threshold with little or no current of injury that remain stable at follow-up. It is reasonable to accept His bundle pacing thresholds that are less than 2.5V @ 1ms in non-dependent patients and lower thresholds in dependent patients, pending more outcomes data: Until more data are available, and pending the development of His-specific pacing systems, one must make a clinical decision balancing the output required to maintain ideal capture, the anticipated pacing burden, the relative importance of maintaining synchrony, and the calculated battery longevity.

Outpatient management/Device Clinic:

Often device clinics are run by ancillary staff for whom many of the concepts associated with His bundle pacing will be novel. It is therefore critical that device interrogation is done with clear documentation of HBP thresholds and that the staff

is well educated to recognize the different thresholds. Patients with HBP leads should have a simultaneous 12-lead EKG (long rhythm strips during threshold testing) at the time of in-office interrogations. This is especially true in leads demonstrating nonselective HB capture, where there is a risk of setting pacing outputs that capture local myocardium only without recruitment of the His Purkinje system. This is also true for patients with BBB in whom appropriate pacing outputs need to be selected to maximally implement conduction system capture. HB paced QRS duration and morphology during follow-up should be carefully measured and reported.

The authors recommend that patients have standard pacemaker and/or ICD interrogation scheduling at 1 month, 3 months and 6 months post implant, and every 6 months thereafter.

Recommendations for training:

His bundle pacing requires a thorough understanding of fundamental aspects of electrophysiology and cardiac anatomy, and as such it would be expected that most operators performing this procedure would be trained in electrophysiology and be high volume device implanters familiar with all aspects of complex device management and implantation techniques. On the basis of the combined experience, the authors think it is reasonable to expect that any well trained implanting electophysiologist could learn to perform HBP with focused didactic training and case observation and/or the presence of an experienced proctor, with an approximate learning curve of 10 cases.

Recommendations for Technology Enhancement

Currently there are a limited set of commercially available tools specific for His bundle pacing and there are no pacing algorithms that specifically address the unique aspects of pacing the His bundle. Improvements need to be made in delivery sheaths, mapping systems, lead and electrode design, pacemaker battery longevity, and power output. HBP-specific pacing algorithms need to be created that can accommodate the lower sensing and higher pacing thresholds, distinguish multiple electrograms, and that can sense QRS morphology to insure maximal conduction system capture.

Conclusions

The concepts underlying HBP have been around nearly as long as clinical EP became a distinct discipline. However, PHBP has only recently gained broad interest, in part due to the advent of tools making the technique more feasible, but also in large part due to an increased awareness of the detrimental effects on cardiac function of dyssynchrony. Currently, we have increasing data from largely independent centers bringing into focus the need for a homogenous way to report acute and chronic aspects of HBP that have been agreed upon by several experienced implanters in the field and presented herein. With increased awareness and interest in this pacing technique, the authors have also provided opinions regarding various aspects of HBP that are of central concern, namely recommendations for recognizing adequate lead placement, patient selection, and training required to competently perform the

423 procedure. The overarching objective is to provide a starting point to initiate larger
424 studies and considerations regarding optimization of the procedure in its various
425 contexts, and to codify the procedure as a unique additional tool in the
426 armamentarium of pacing options.
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Figure Legends

Figure 1: Selective His bundle pacing in a patient with no HPCD: Twelve lead ECG and intracardiac electrograms from the HBP lead at baseline and during HBP are shown at a sweep speed of 100 mm/sec. His-QRS and the stimulus-QRS intervals are identical at 40 ms. The QRS morphology during HBP is same as baseline. The local ventricular electrogram (arrow) is discrete from the pacing stimulus suggesting absent local ventricular capture.

Figure 2: Nonselective HBP in a patient with no HPCD: Twelve lead ECG and intracardiac electrograms from the right atrial and HBP lead at baseline and during HBP at decreasing output are shown at a sweep speed of 100 mm/sec. During HBP at 1.2V, the paced QRS duration is 120 ms due to fusion between conduction via the His-Purkinje system and ventricular capture. There is no isoelectric interval between the stimulus and QRS onset. There is no discrete local ventricular electrogram noted following the pacing artifact (arrow) in the HBP lead. As the pacing output is decreased to 1V, the paced QRS duration is wider at 160 ms with the stimulus to atrial interval prolonging from 150 to 250 ms confirming loss of His bundle capture.

Figure 3: Nonselective HBP to Selective HBP in a patient with no HPCD: Twelve lead ECG and intracardiac electrograms from the HBP lead at baseline and during HBP at decreasing output are shown at a sweep speed of 100 mm/sec. During HBP at 1.5V, the paced QRS duration is 140 ms due to fusion between conduction via the His-Purkinje system and ventricular capture. There is no isoelectric interval between the stimulus and QRS onset (S-QRS = 0). There is no discrete local

ventricular electrogram noted following the pacing artifact (arrow) in the HBP lead. As the pacing output is decreased to 1V, there is selective His bundle capture and loss of ventricular capture (see the discrete local ventricular electrogram in the HBP lead - arrow) resulting in QRS morphology identical to baseline QRS (90 ms).

Figure 4: Nonselective HBP in a patient with complete intra-Hisian AV block: The left panel demonstrates complete HV block and an escape rhythm with RBBB morphology. Pacing from the HBP lead at 1.5V results in narrowing of the QRS to 110 ms due to nonselective capture of the His bundle and RV. At 1.2V, there is selective capture of the left bundle (distal to the site of block in the His bundle) resulting in paced QRS morphology (RBBB) identical to the escape rhythm explained by longitudinal dissociation in the His bundle. Local electrogram discrete from the pacing artifact in the HBP lead suggests loss of RV myocardial capture (arrow).

Figure 5: Cardiac resynchronization during HBP: The top panels show the standard surface precordial leads (25mm/sec sweep speed), and the bottom panels show the 12 surface leads and intracardiac electrograms for each respective condition (200mm/sec sweep speed). Panel A is native conduction with LBBB, QRS duration 166 msec. Conduction time from His to lateral LV wall is 225 msec. Panel B shows pacing from the HBP lead at high output (8V/1.5ms): The change in morphology –more rapid dV/dt, axis normalization are consistent with NS-HBP. No local ventricular egm on the octapolar mapping catheter indicates local ventricular capture expected with nonselective capture. The LV timing is advanced by 100 msec. Note that due to local ventricular capture the total QRS duration as measured is

similar to native conduction (160 msec) Panel C: The HBP output has been decreased to 4.25V/1.5ms: the surface ECG is now isoelectric from stimulus artifact to QRS onset, and the QRS measures 125 msec. Septal ventricular activation now occurs well after the local stimulus artifact from the HBP lead, indicating S-HBP. Timing from His to LV activation remains advanced by 100 msec, proving re-engagement of the left fascicles. Panel D: Further decrease in output (4V/1.5ms) results in QRS morphology identical to native conduction, coincident with which the His- LV timing returns to baseline (225 msec). Labels: His d to His 4: Bipolar leads on the octapolar His mapping catheter in the His position (anteroseptal). HBP lead: The actively fixed His bundle pacing lead from which pacing is being performed in panels B-D. LV: The LV lead placed in a lateral coronary vein. (Modified from: Lustgarten et al. Heart Rhythm 2015;12:1548-1557)

Figure 6: Selective HBP in Right Bundle Branch Block: Twelve lead ECG and intracardiac electrograms from the HBP lead at baseline and during HBP at decreasing output are shown at a sweep speed of 100 mm/sec. Baseline ECG shows RBBB with QRS duration of 180 ms with HV interval of 65 ms as shown in the HBP lead. During HBP at 1.2V there is selective capture of the His bundle with resultant QRS duration of 100 ms and correction of RBBB. Note the discrete local electrogram in the HBP lead with a different morphology from baseline and stimulus to ventricular interval of 65 ms. At a pacing output of 1.0V, there is loss of right bundle capture and resultant QRS morphology identical to baseline on surface ECG and local ventricular electrogram (arrow).

Figure 7: Nonselective HBP in RBBB: Twelve lead ECG and intracardiac electrograms from right atrial (RA) and the HBP lead at baseline and during HBP at decreasing output are shown at a sweep speed of 100 mm/sec. Baseline ECG shows RBBB with QRS duration of 160 ms. During HBP at 2V, the paced QRS duration is 120 ms due to fusion between conduction via the His-Purkinje system (both right and left bundles) and ventricular capture. There is no isoelectric interval between the stimulus and QRS onset. There is no discrete local ventricular electrogram noted following the pacing artifact (arrow) in the HBP lead. As the pacing output is decreased to 1.5V, the paced QRS duration is wider at 130 ms due to loss of right bundle capture as evidenced by the terminal R waves in V1 and V2 (circle). At pacing output of 1V, there is only right ventricular capture with QRS duration of 170 ms with lengthening of the stimulus to atrial interval from 110 to 210 ms confirming loss of His bundle capture.

Table 1: Criteria for His Bundle Pacing

Baseline	Normal QRS	HPCD#	
		With correction*	Without correction
Selective HBP	• S-QRS = H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead with S-V=H-V • Paced QRS = native QRS • Single capture threshold (His bundle)	• S-QRS ≤ H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead • Paced QRS < native QRS • 2 distinct capture thresholds (HBP with BBB correction, HBP without BBB correction)	• S-QRS ≤ or > H-QRS with isoelectric interval • Discrete local ventricular electrogram in HBP lead • Paced QRS = native QRS • Single capture threshold (HBP with BBB)
Non-selective HBP	• S-QRS < H-QRS (usually 0, S-QRS_{end} = H-QRS_{end}) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact (local myocardial capture) • Paced QRS >native QRS with normalization of precordial and limb lead axes with respect to rapid dV/dt components of the QRS • 2 distinct capture thresholds (His bundle capture, RV capture)	• S-QRS < H-QRS (usually 0, S-QRS_{end} < H-QRS_{end}) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact • Paced QRS ≤ native QRS • 3 distinct capture thresholds (HBP with BBB correction, HBP without BBB correction, RV capture)	• S-QRS < H-QRS (usually 0) with or without isoelectric interval (Pseudodelta wave +/-) • Direct capture of local ventricular electrogram in HBP lead by stimulus artifact • Paced QRS > native QRS (see text) • 2 distinct capture thresholds (HBP with BBB, RV capture)

SV stimulus to QRS onset; HV His- ventricular; V ventricular; BBB bundle branch block; RV right ventricle; HBP His bundle pacing; HPCD His-Purkinje Conduction disease
* Narrowing of QRS; # including bundle branch block and infra-nodal AV block

Figure 1

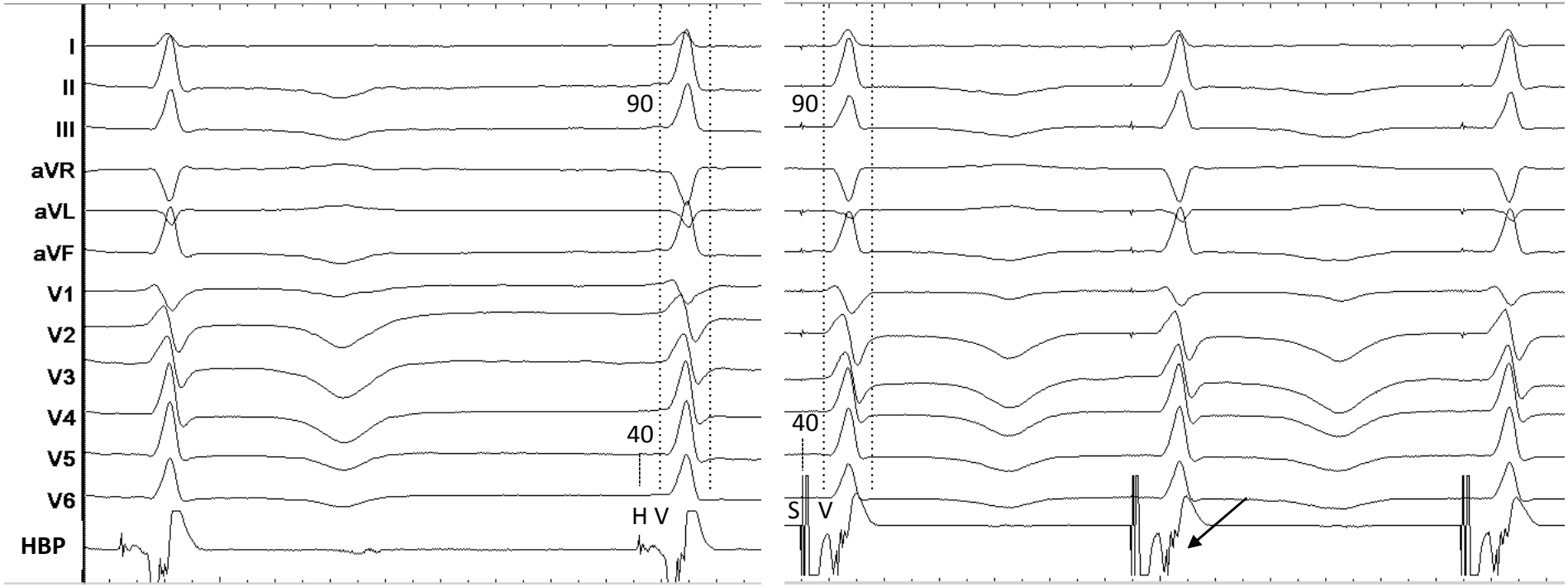


Figure 2

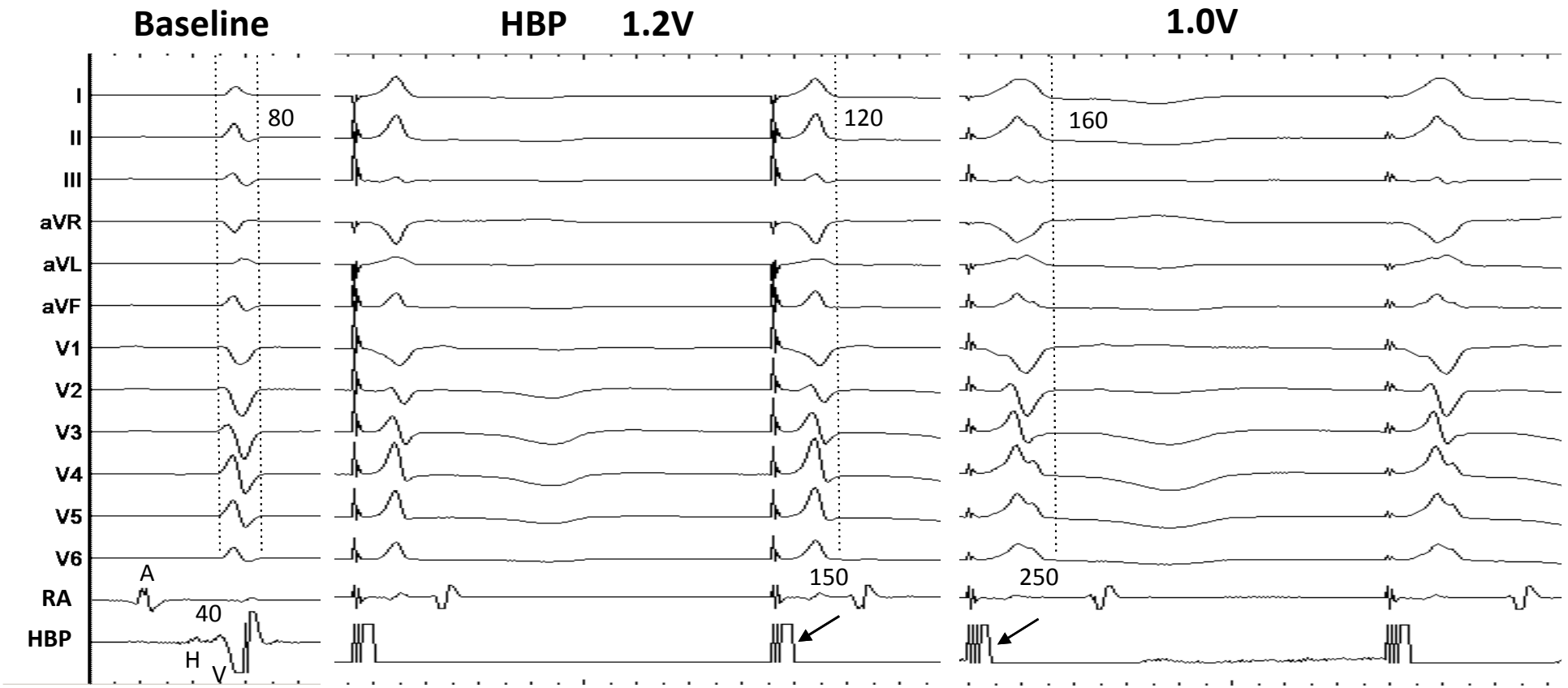

Baseline
HBP 1.2V
1.0V
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RA
HBP
80
120
160
A
40
H
V
150
250

Figure 3

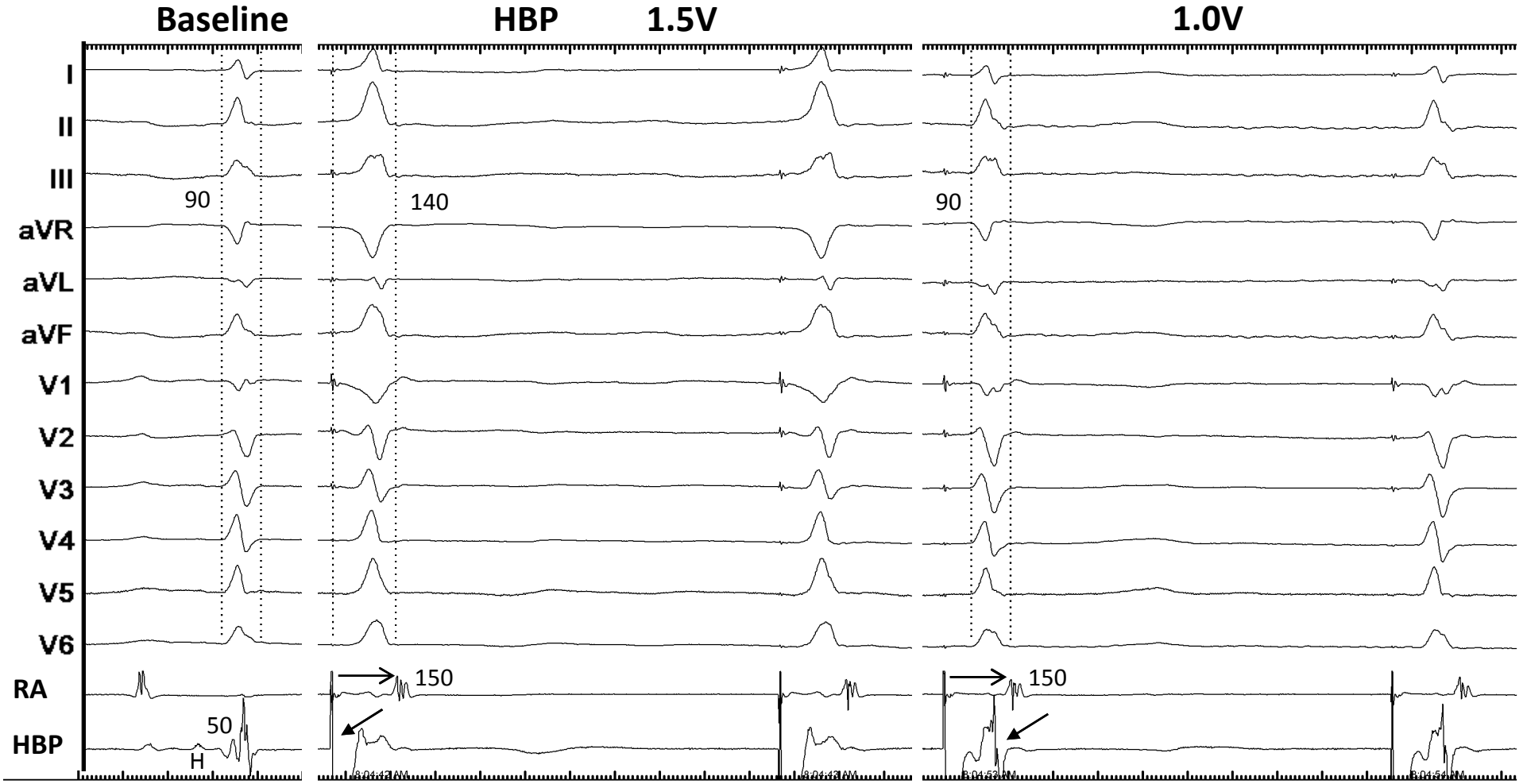

Baseline
HBP 1.5V
1.0V
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RA
HBP
90
140
90
150
150
50
H

Figure 4

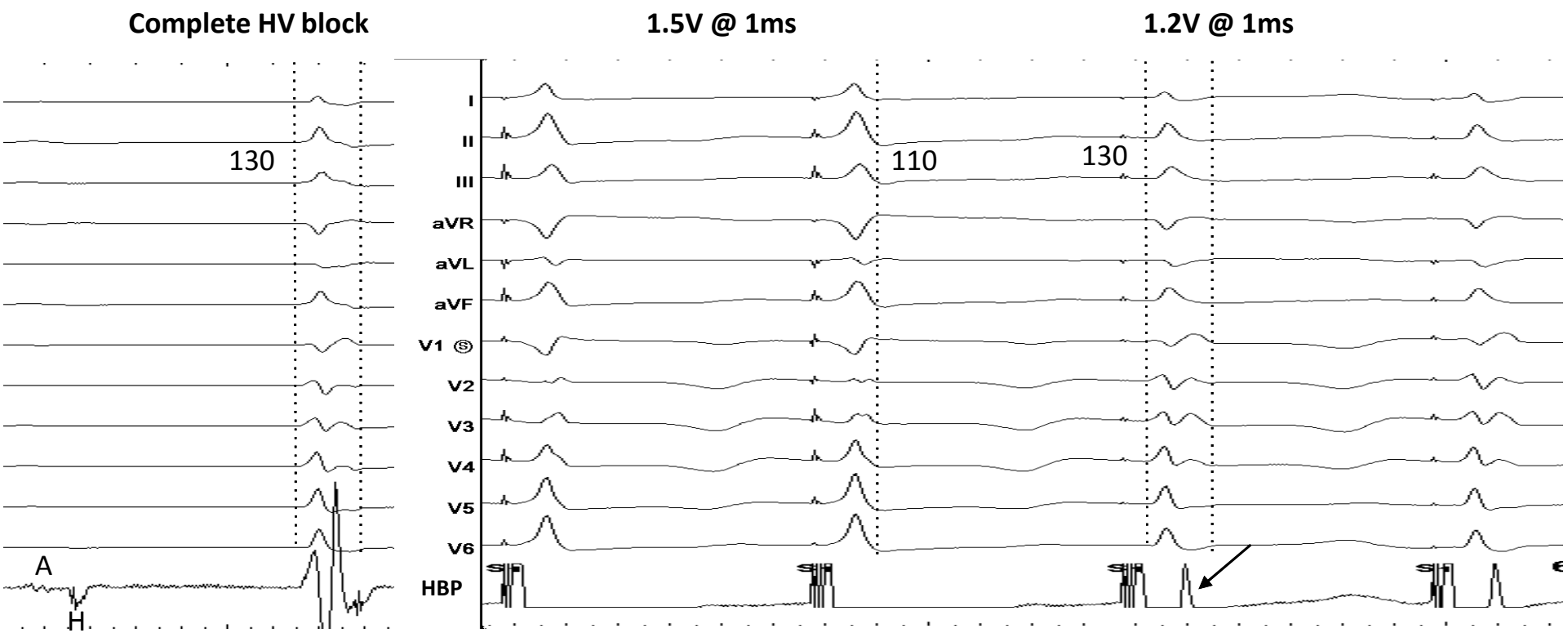

Complete HV block
1.5V @ 1ms
1.2V @ 1ms
130
110
130
I
II
III
aVR
aVL
aVF
V1 Ⓢ
V2
V3
V4
V5
V6
HBP
A
H

Figure 5

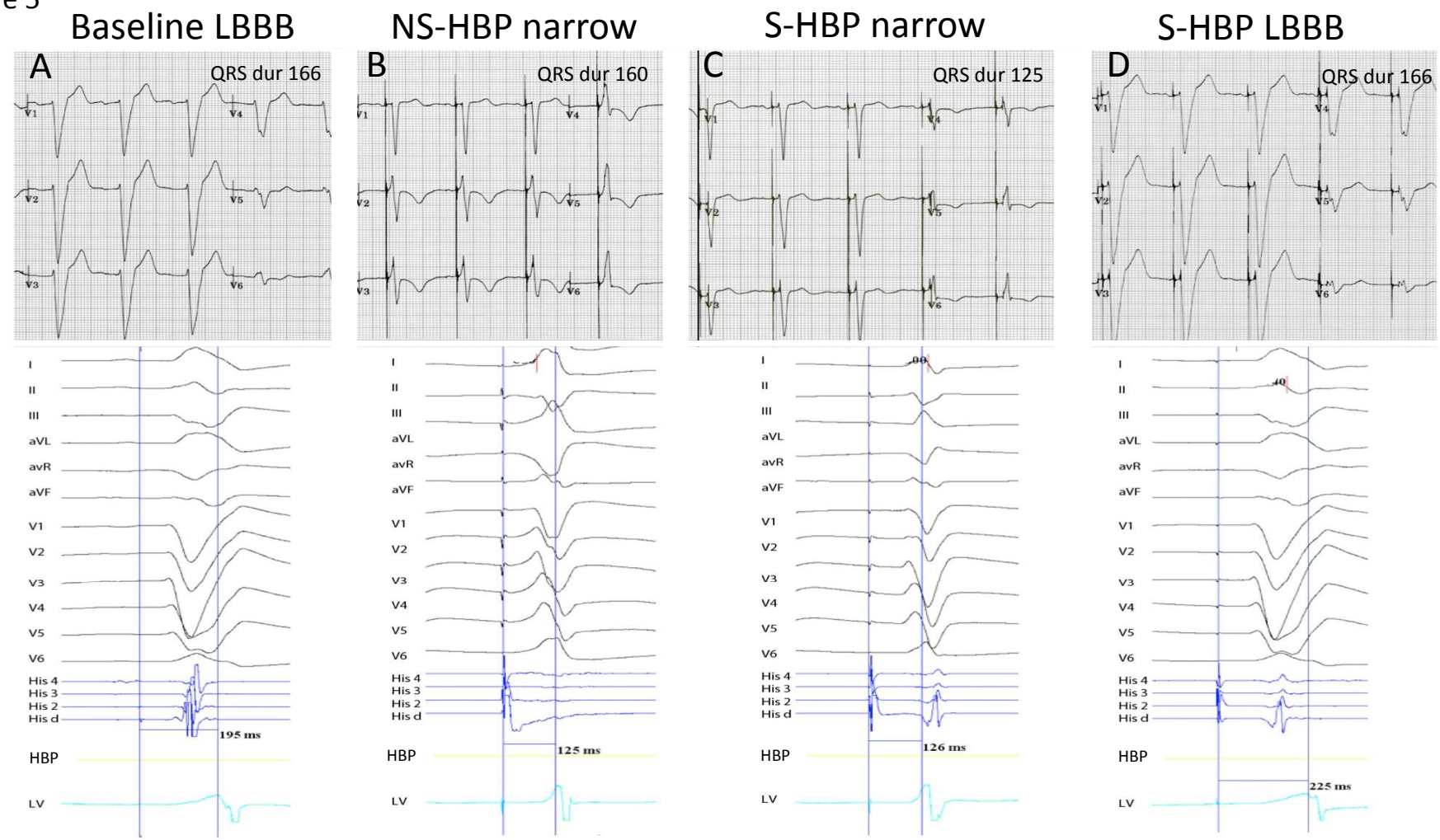

Baseline LBBB
NS-HBP narrow
S-HBP narrow
S-HBP LBBB
A
QRS dur 166
B
QRS dur 160
C
QRS dur 125
D
QRS dur 166
I
II
III
aVL
avR
aVF
V1
V2
V3
V4
V5
V6
His 4
His 3
His 2
His d
HBP
LV
195 ms
125 ms
126 ms
225 ms

Figure 6

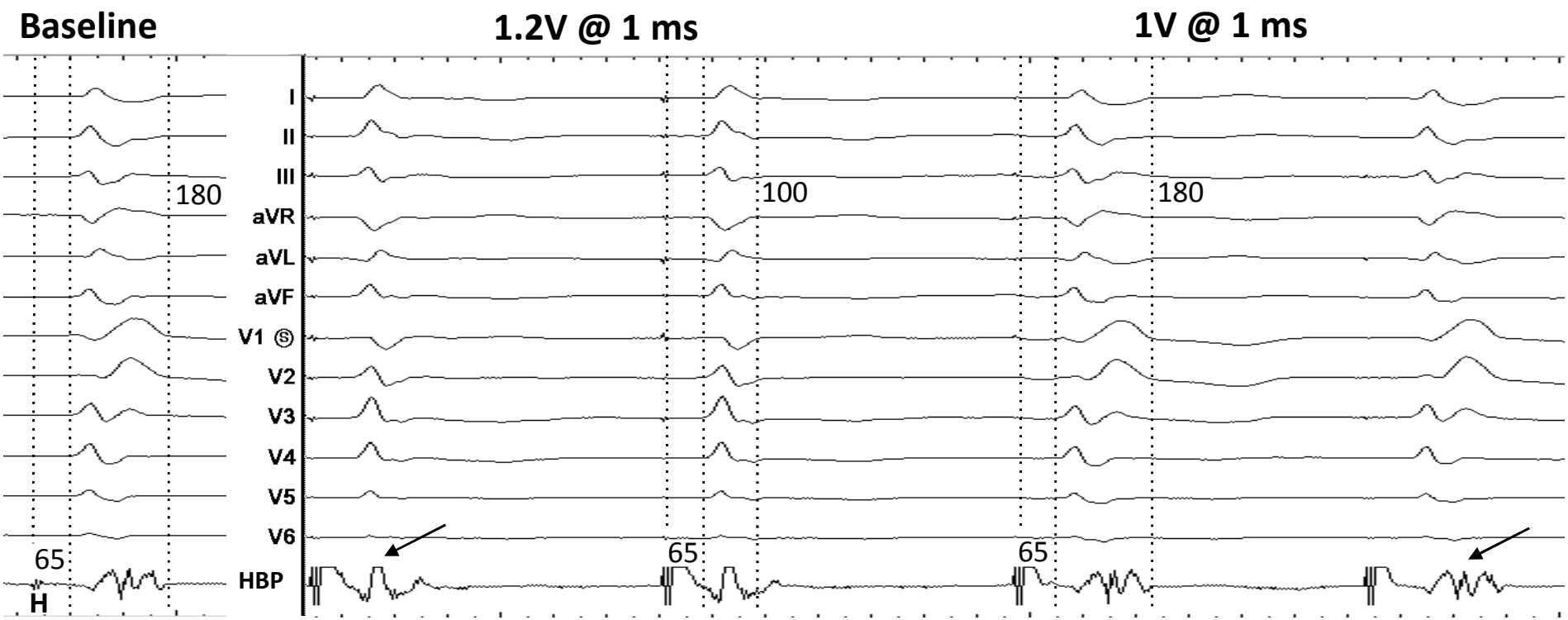
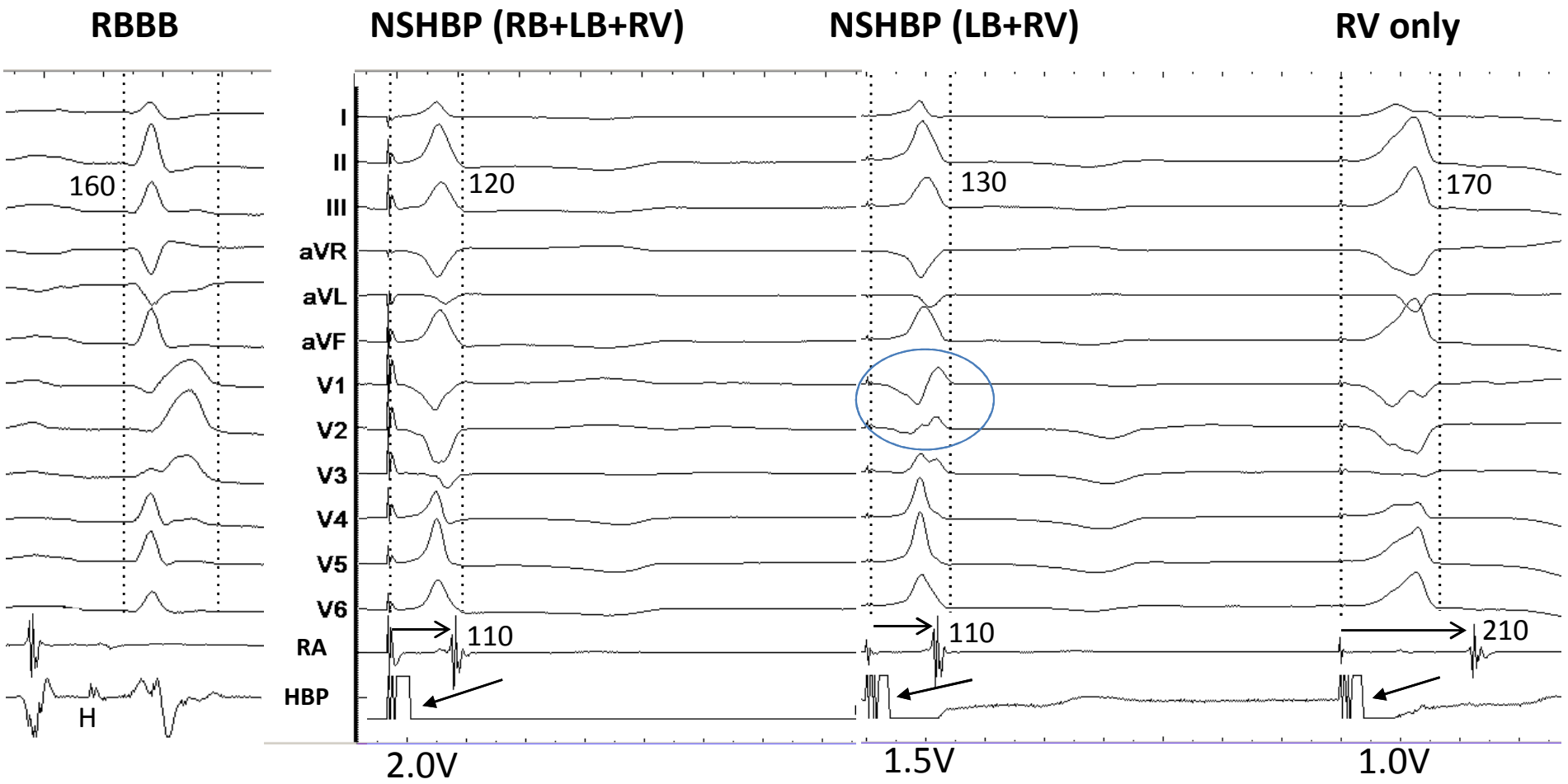


Figure 7


RBBB
NSHBP (RB+LB+RV)
NSHBP (LB+RV)
RV only
160
120
130
170
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RA
HBP
110
110
210
H
2.0V
1.5V
1.0V