An unwanted left bundle branch block: The proteus-unveiling of his bundle pacing

Mirco Lazzeri, MD, Michele Bertelli, MD, Alessandro Carecci, MD, Andrea Angeletti, MD, Matteo Ziacchi, MD, Mauro Biffi, MD

From the Istituto di Cardiologia, IRCCS Azienda Ospedaliero Universitaria di Bologna, Bologna, Italy.

Introduction

His bundle pacing (HBP) has emerged in the last decade as an attractive alternative to conventional right ventricular pacing (RVP), being the most physiologic form of cardiac stimulation by the avoidance of pacing-induced intraventricular and interventricular dyssynchrony.[1] Although the premises to replace customary RVP are sound, evidence is still limited, with only small, randomized trials available. Furthermore, HBP threshold increase leading to unpredictable His capture at follow-up or to excessive battery drain are a concern in young patients, especially when pacemaker dependent.[1,2] These aspects led to broadening the concept of conduction system pacing (CSP) to include the stimulation of the left bundle branch (LBB) area.[2]

We report a case illustrating both the superior benefits and the tricky management of HBP in a patient with progressive atrioventricular block (AVB) and a long-term pacing threshold increase resulting in heart failure.

Case description

A 61-year-old man was admitted to the emergency room because of near syncope. His medical history included chronic obstructive pulmonary disease, obstructive sleep apnea syndrome, and multiple cardiovascular risk factors, namely type II diabetes mellitus complicated by nephropathy, high blood pressure, dyslipidemia, active smoking, and obesity (body mass index, 31). His electrocardiogram (ECG) showed sinus rhythm with second-degree type I AVB (Figure 1A). Transthoracic echocardiography demonstrated hypertensive heart disease with moderate concentric hypertrophy and normal biventricular volumes and function (interventricular septum thickness, 1.4 cm; posterior wall thickness, 1.4 cm; indexed left ventricular (LV) end-diastolic volume, 55 mL/m^2; LV ejection fraction (LVEF), 55%; right ventricle end-diastolic area, 22 cm^2; fractional area change, 45%), atrial enlargement, no significant valvular disease, and normal systolic pulmonary artery pressure (sPAP) (Supplemental material—Video 1). Telemetric recording detected Wenckebach, intermittent 2:1, and sporadic third-degree AVB.

The patient underwent dual-chamber pacemaker implantation, with the aim to achieve CSP. A 3830 Select Secure (Medtronic Inc., Minneapolis, MN) was implanted at the distal His recording site where reliable ventricular sensing was obtained (bipolar sensing, 3.5 mV) (Figure 2A, 2B). During the procedure, nodal AVB was recorded (AH interval, 170 msec) with normal HV interval (42 msec) (Figure 2C). Decremental output test was performed in unipolar configuration (Figure 1B) with nonselective His bundle (NS-HB) capture up to 1 V at 0.75 ms; selective His bundle (S-HB) capture between 0.9 and 0.3 V at 0.75 ms was achieved, whereas loss of capture occurred at outputs $<$ 0.3 V at 0.75 m. Ventricular output was therefore programmed as 2 V at 0.75 ms unipolar (Figure 1C), with a large safety margin ($>$1.5 V). The device was programmed DDD 50–130 beats/min with atrioventricular (AV) delay based on

KEY TEACHING POINTS

- Despite the possibility of avoiding pacing-induced intraventriculat and interventricular dyssynchrony, His bundle pacing long-term reliability is still threatened by unpredictable threshold rise, potentially leading to loss of capture of conduction system or excessive battery drain.
- Knowledge of conduction system pacing electrocardiography is mandatory to aid the clinical diagnosis in common clinical scenarios.
- ECG during decremental threshold test is necessary at follow-up visits to unmask sneaky threshold rise. A patient-specific "EGM template" of selective as well as nonselective His bundle pacing should assist the remote monitoring to confirm the stability of capture by comparison with the reference.

KEYWORDS His bundle pacing; conduction system pacing; rise of capture threshold; left bundle branch block; heart failure
(Heart Rhythm Case Reports 2025;11:20–24)

Address reprints and correspondence: Mirco Lazzeri, MD, IRCCS University Hospital of Bologna S Orsola-Malpighi Polyclinic Cardiothoracic-vascular Department, Italy. E-mail address: mircolazzeri@gmail.com.

https://doi.org/10.1016/j.hrcr.2024.09.020

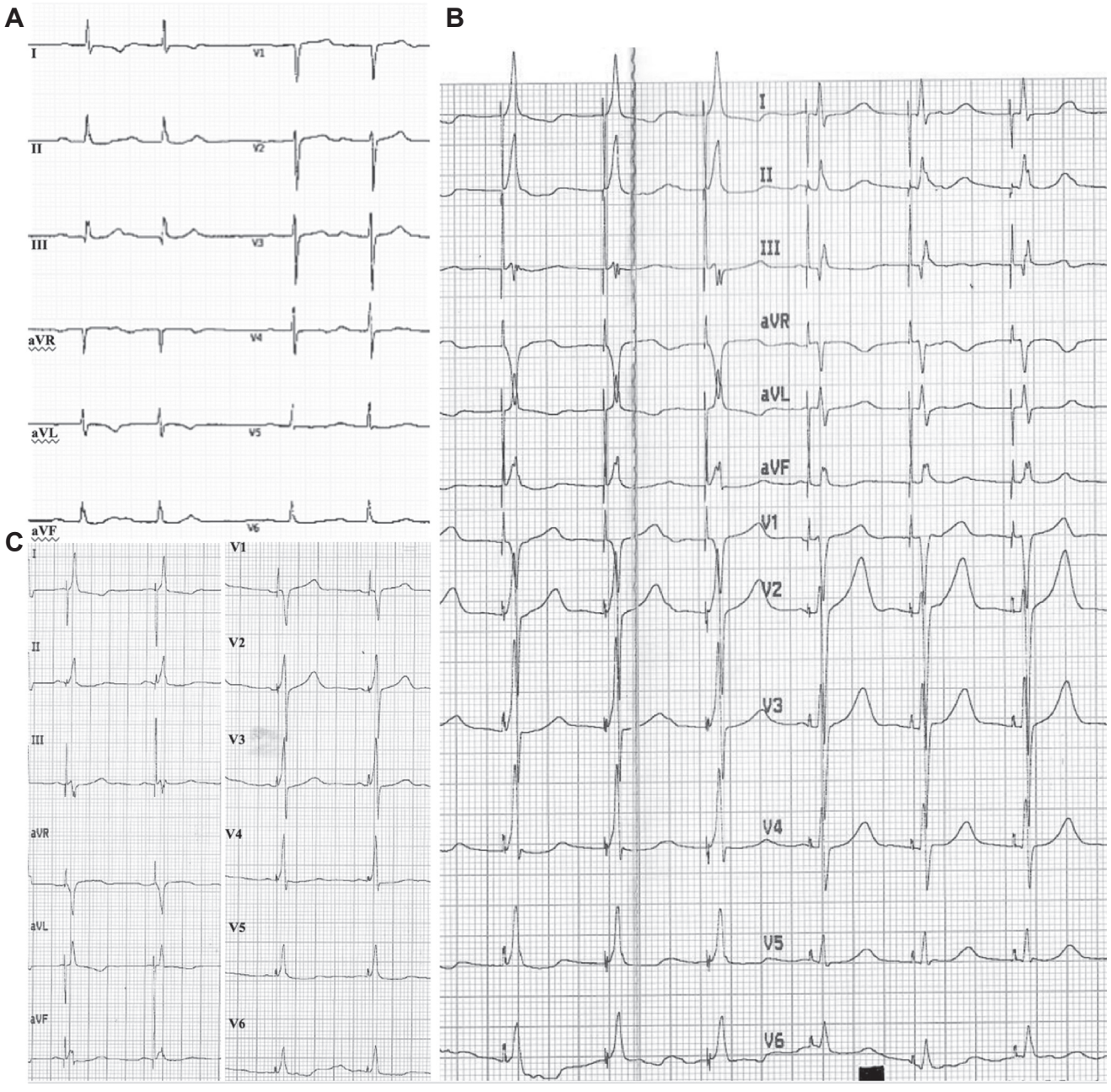

Figure 1 Pre-implantation ECG shows sinus rhythm with second-degree type I AV block (**A**). Decremental test shows the transition from NS to S-HB pacing for output lower than 0.9 mV at 0.75 ms (**B**). After the implantation, the pacemaker is programmed with DDD mode with HB lead programmed at 2 mV at 0.75 ms, obtaining NS-HB pacing (**C**).

echocardiogram diastolic pattern, to guarantee the most physiological LV diastolic filling (sensed AV/paced AV 105/150 msec). Automated threshold verification and ventricular output adjustment was not feasible, because it often occurs in the HBP setting.[2,3]

The patient did very well; sensing and pacing threshold were unchanged at 1- and 6-months follow-up; paced ECG showed NS-HBP and S-HBP capture threshold stability. Surprisingly, 11 months after implantation, the patient underwent urgent reevaluation in the emergency department because of new-onset fatigue, weight gain (+6 kg in 3 weeks), progressive exertional dyspnea (New York Heart Association class III), and lower limb edema. Blood tests showed brain natriuretic peptide of 654 pg/dL. An ECG was performed (Figure 3A), which showed sinus rhythm and S-HB stimulation with left bundle branch block (LBBB) QRS morphology. Transthoracic echocardiography documented ventricular dyssynchrony with "apical rocking," increase in ventricular volumes, and reduction in ejection fraction (LV end-diastolic volume indexed: 75 mL/m^2, LVEF: 37%), as well as pulmonary hypertension (sPAP = 47 mm Hg) (Supplemental material—Video 2).

All the data were consistent with new-onset congestive heart failure attributable to LV systolic dysfunction

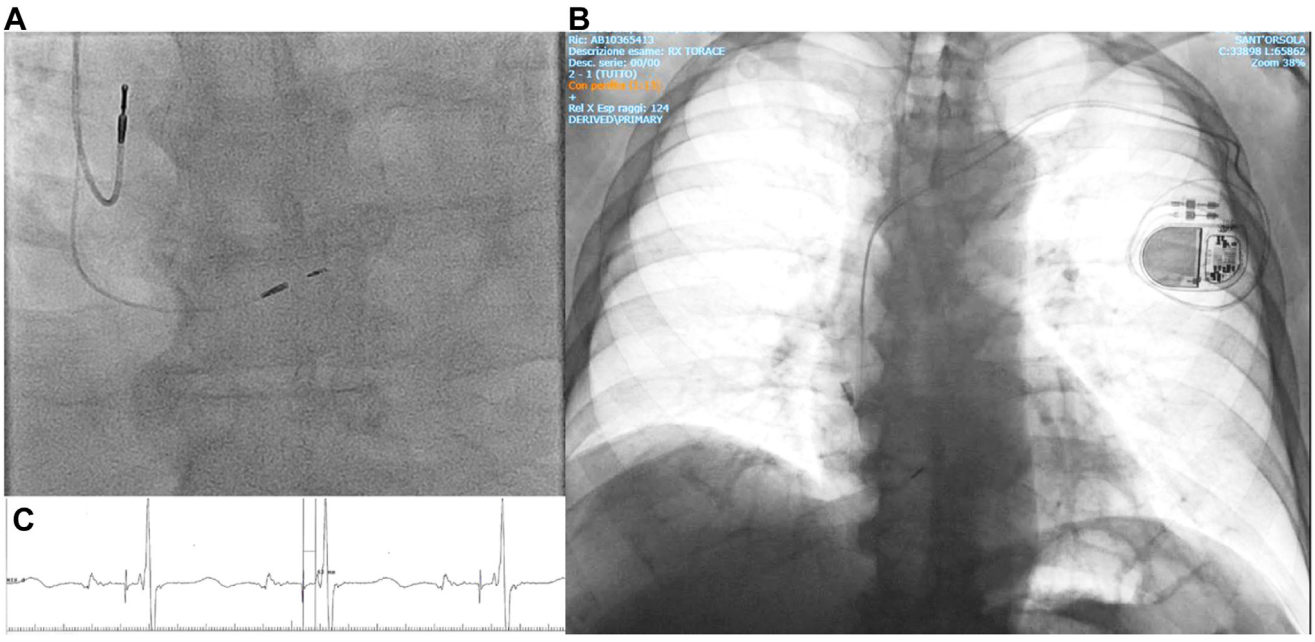

Figure 2 **A:** Fluoroscopic and **B:** chest radiograph view of the HB lead after implantation. **C:** The intracardiac electrogram from the HB lead before fixation shows prolonged AH interval (170 msec) with normal infranodal conduction (HV, 42 msec).

associated with new-onset LBBB. The patient was discharged with loop diuretic therapy on top of optimized medical therapy for heart failure (carvedilol, losartan, and empagliflozin). Because of the suspicion of underlying coronary artery disease, an elective coronary angiography was scheduled.

Meanwhile, a device follow-up was recommended. On interrogation, we observed progression of the conduction system disease, with total AV block (Figure 3B) and pacemaker dependence (ventricular pacing rate, 99% vs 70% at the 6-month visit). Sensing and impedance of the His bundle (HB) lead were still acceptable (bipolar sensing, 2.7 mV; unipolar impedance, 290 ohm). Decremental output test (Figure 3C–3D) showed a striking threshold increase of both the myocardial tissue and the fibers for the LBB: NS-HB stimulation was achieved with voltages $>$ 3.5 V at 0.75 ms and complete S-HB capture for voltages between 3.4 and 2.6 V at 0.75 ms. Between 2.5 and 2.2 V partial loss of

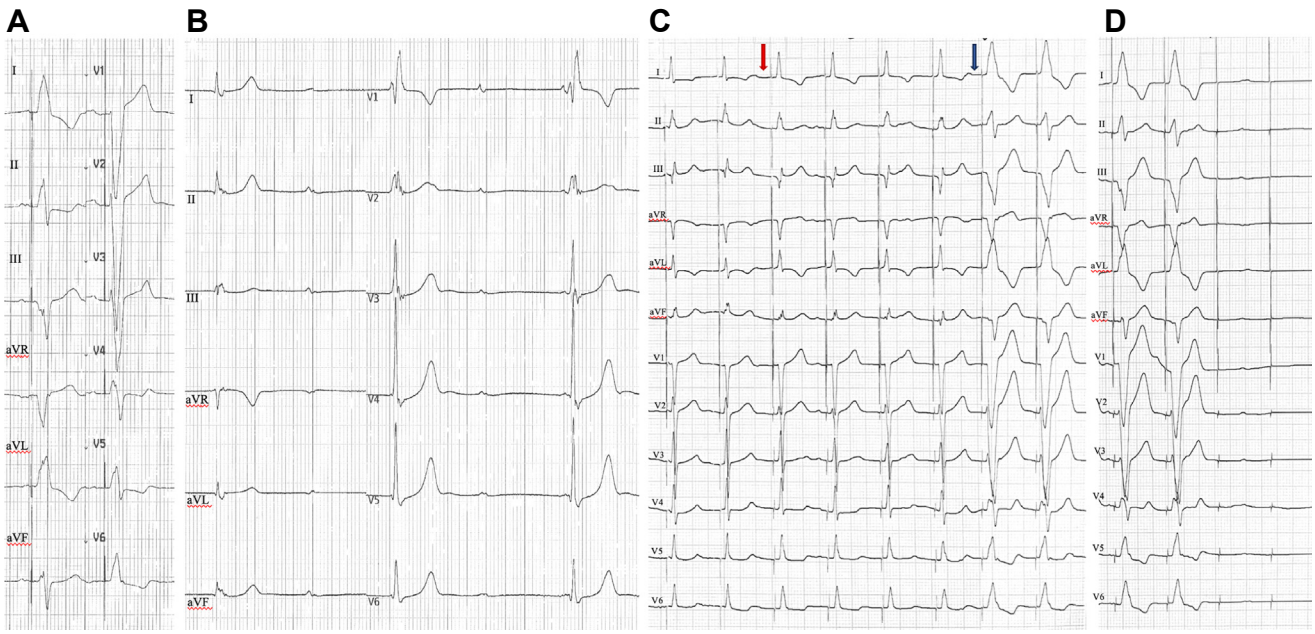

Figure 3 **A:** ECG showing sinus rhythm with S-HB pacing and LBBB QRS morphology. **B:** Pacemaker inhibition revealing total AV block with ventricular escape rhythm at RBBB morphology. **C:** Decremental threshold test demonstrating partial loss of capture of left bundle fibers at 2.5 mV at 0.75 ms (*red arrow*) followed by loss of capture of the left bundle branch at 2.1 mV at 0.75 ms (*blue arrow*). **D:** Loss of right bundle fibers capture at 1.3 V at 0.75 ms.

capture of fibers destined to the LBB was observed, whereas complete loss of the LBB capture occurred below 2.2 V at 0.75 ms. Loss of capture of the fibers destined to the right bundle occurred below 1.4 V at 0.75 ms. Thus, it was understood that the patient had received HBP with loss of LBB capture for several weeks, being ventricular output programmed at 2 V at 0.75 ms. This latter was hence reprogrammed as 3.5 V at 0.75 ms, obtaining an NS-HB stimulation. Coronary angiography was not indicated.

Two months later, the patient was reevaluated; he reported weight loss, exercise tolerance improvement (New York Heart Association class I), and resolution of lower limb edema. Transthoracic echocardiography demonstrated recovery of LVEF, normalization of LV volumes, and sPAP. ECGs confirm HB capture threshold stability. Loop diuretic was discontinued. Clinical status, echocardiogram, and pacing parameters remained stable 12 months afterward.

Discussion

This case highlights a potentially correctable cause of heart failure. The patient had HBP because of age and >20% RVP expected at follow-up.[4] Indeed, progression of AVB occurred, and he became pacemaker dependent. Although complete AV block is a rare complication during implantation of an HBP lead,[4] in this case it manifested as the late evolution of a preexisting milder conduction disease. The remarkable observation in this patient is the appearance of LBBB because of a threshold increase with loss of capture of the fibers destined to the LBB,[5] which, being undetected, caused a significant LV reverse remodeling (20 mL/m^2 increase of LV end-diastolic volume indexed, 18% decrease of LVEF) with an overt congestive heart failure syndrome. This phenomenon represents a novel complication of HBP, owing to the complexity of using automatic capture verification algorithms to maintain complete HB capture, and to the hidden challenges in detecting the shift from complete HB capture to loss of capture of any of the two branches.[3,6] In this setting, pacing output should have been increased as soon as the appearance of LBBB, to maintain full HB capture; regretfully, an algorithm to detect a morphologic EGM shift from complete HBP to loss of capture of 1 of the branches is not available.[6] The complete recovery of both LV function and of the heart failure syndrome by increasing the pacing output to maintain capture of the LBB confirms the clinical relevance of HBP as the most physiological pacing modality.[1,2]

The rise of capture threshold is still a "sword of Damocles" hanging over HBP: beyond higher thresholds at implantation, delayed increase in up to 15% of patients is reported.[7–9] This may lead to lead revision in the event of HB capture loss, or to earlier device replacement because of a huge current drain. The cause of threshold increase is still debated, fibrosis at the implantation site possibly resulting of micro-dislodgement caused by repeated septal leaflet interaction with the lead. The variability of the HB anatomy is very likely the main reason of threshold changes, fibrotic tissue growth being more relevant when HB fibers are intercepted at the membranous septum site or between a thick muscle layer, with respect to a purely subendocardic HB course.[10] Although the occurrence of this phenomenon was predicted by cornerstone studies of the conduction system,[5,11] we found a significant gap in the capture voltage of the right vs left branch only after the 6-month follow-up. Whether micro-dislodgement of the HB lead (very unlikely, given the time elapsed since the implantation) or thick overlaying muscle was the cause of the HBP increase, this case sheds light on the "2 sides of the coin" of HBP. In our opinion, patients with HBP pacemaker, especially when threshold capture rise is detected during the follow-up, need to be reassessed at each device interrogation with 12-lead ECG and decremental testing to unmask sneaky HB capture threshold elevations (NS-HBP vs S-HBP vs branch capture loss) that the device is unable to detect with currently algorithms. Moreover, the use of a patient-specific "EGM template" of selective as well nonselective complete HBP should assist the remote monitoring of CSP recipients to confirm stability of HB capture along follow-up by comparison with this reference.

LBB area pacing appears to have overcome the HBP issue of long-term threshold rise.[2] Nonetheless, the superiority of either against each other as well as against conventional RVP remains to be proven.[12]

A clinically important aspect is the need to spread awareness and knowledge of the electrocardiography of cardiac pacing among the medical community: a paced broad QRS complex observed in symptomatic patients should prompt counseling about the implanted system in the view of a pacing-induced cardiomyopathy. When it comes to CSP, a careful evaluation must be undertaken,[13] because a cause of heart failure potentially correctable by simple reprogramming can be the concealed culprit of the clinical picture, as in our case. Beyond providing the patient's care, the electrophysiological reevaluation also prevented a futile and possibly risky coronary angiography. To ease paced patients' evaluation and streamline their clinical management, an updated classification of the pacing modality being delivered should be adopted, as proposed by Marcantoni et al.[14] This would help clinicians to diagnose whether cardiac stimulation is delivered according to clinical intention, or whether device follow-up should be undertaken to fix an "underperformance."

Conclusion

This case report shows an unusual heart failure cause in a patient with HBP-pacemaker, easily fixed by device reprogramming. CSP, and HBP as its highest expression, is the most promising cardiac stimulation modality to prevent and treat electromechanical cardiac dysfunction. The development of dedicated tools and pacing technologies will reduce device-related complication and improve its broad adoption in clinical practice; meanwhile, a parallel growth of knowledge in the electrocardiography of cardiac stimulation is mandatory to detect the hints of changes along follow-up that may undermine its clinical efficacy. A universally accepted

classification coding can help to guide the evaluation of the implanted system to streamline problem identification and patient management.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: The authors have no conflicts of interest to disclose.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.09.020.

References

1. Biffi M, Capobianco C, Spadotto A, et al. Pacing devices to treat bradycardia: current status and future perspectives. Expert Rev Med Devices 2021; 18(2):161–177. https://doi.org/10.1080/17434440.2021.1866543.
2. Burri H, Jastrzebski M, Cano Ó, et al. EHRA clinical consensus statement on conduction system pacing implantation: endorsed by the Asia Pacific Heart Rhythm Society (APHRS), Canadian Heart Rhythm Society (CHRS), and Latin American Heart Rhythm Society (LAHRS). Europace 2023;25(4):1208–1236.
3. Massaro G, Lorenzetti S, Statuto G, Biffi M. Automatic verification of capture in the scenario of His bundle pacing: still an unmet need? J Cardiovasc Electrophysiol 2019;30(10):2110–2112.
4. Kiehl EL, Makki T, Kumar R, et al. Incidence and predictors of right ventricular pacing-induced cardiomyopathy in patients with complete atrioventricular block and preserved left ventricular systolic function. Heart Rhythm 2016; 13(12):2272–2278.
5. Narula OS. Longitudinal dissociation in the His bundle: bundle branch block due to asynchronous conduction within the His bundle in man. Circulation 1977; 56(6):996–1006. https://doi.org/10.1161/01.cir.56.6.996.
6. 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(2):e006878.
7. Vijayaraman P, Naperkowski A, Subzposh FA, et al. Permanent His-bundle pacing: long-term lead performance and clinical outcomes. Heart Rhythm 2018; 15(5):696–702. https://doi.org/10.1016/j.hrthm.2017.12.022.
8. Teigeler T, Kolominsky J, Vo C, et al. Intermediate-term performance and safety of His-bundle pacing leads: a single-center experience. Heart Rhythm 2021; 18(5):743–749. https://doi.org/10.1016/j.hrthm.2020.12.031.
9. Zanon F, Abdelrahman M, Marcantoni L, et al. Long term performance and safety of His bundle pacing: a multicenter experience. J Cardiovasc Electrophysiol 2019;30:1594–1601.
10. Vijayaraman P, Dandamudi G. How to perform permanent His bundle pacing: tips and tricks. Pacing Clin Electrophysiol 2016;39(12): 1298–1304.
11. James TN, Sherf L. Fine structure of the His bundle. Circulation 1971;44(1):9–28. https://doi.org/10.1161/01.cir.44.1.9.
12. Vijayaraman P, Chelu MG, Curila K, et al. Cardiac conduction system pacing: a comprehensive update. JACC Clin Electrophysiol 2023;9(11):2358–2387. https://doi.org/10.1016/j.jacep.2023.06.005.
13. Burri H, Jastrzebski M, Vijayaraman P. Electrocardiographic analysis for His bundle pacing at implantation and follow-up. JACC Clin Electrophysiol 2020; 6(7):883–900.
14. Marcantoni L, Pastore G, Biffi M, Zanon F. The weakest point of cardiac resynchronization therapy: new technologies facing old terminology. Front Cardiovasc Med 2023;10:1236369.