His Bundle Pacing

Is It Ready for Prime Time?

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KEYWORDS

• His bundle pacing • Ventricular pacing • Pacing strategies • Bundle branch block

KEY POINTS

- His bundle pacing is emerging as a viable pacing strategy in daily clinical practice. Several investigators have shown both feasibility and positive clinical outcomes with His bundle pacing.
- Clinical benefits include lack of pacing induced dyssynchrony, correction of bundle branch blocks, improvement in heart failure symptoms and left ventricular systolic function.
- With improvement in delivery tools and lead designs, His bundle pacing is likely to become a common pacing strategy in the near future.

INTRODUCTION

Long-term right ventricular apical pacing (RVAP) has been associated with detrimental effects, including increased risk for heart failure (HF), atrial fibrillation, (AF), and death.[1–4] Most of these adverse effects result from ventricular dyssynchrony related to perturbed ventricular depolarization. In addition, biventricular pacing (BiVp) has limited benefits in patients with non–left bundle branch block (LBBB) and severely reduced ejection fraction (EF); 20% to 30% of patients seem to not respond to cardiac resynchronization therapy (CRT).[5,6] Consequently, alternative pacing strategies that mimic natural physiology are desired. Recently, permanent His bundle pacing (PHBP) has emerged as a true physiologic form of ventricular pacing that has been shown in recent years to be safe and feasible in clinical practice. Because it induces ventricular contraction by exciting the intrinsic conduction system, it has the benefit of reducing or eliminating both interventricular and intraventricular dyssynchrony.

BACKGROUND

The bundle of His was first described in 1893 by Wilhelm His Jr, a Swiss anatomist and cardiologist.[7] He described it as a muscle bundle connecting the auricular and ventricular septal walls. Damage to the conducting cells forming the bundle of His caused asynchrony in the beat of the auricle and ventricle, referred to as heart block. His bundle recordings were first performed by Alanis and colleagues[8] on isolated perfused hearts of dogs and cats and were published in 1958. In 1969, Scherlag and colleagues[9] published the first His bundle recordings performed in human beings using intravascular catheters. A year later, Narula and colleagues[10] demonstrated the possibility for achieving temporary HBP in humans using a multipolar catheter positioned at the atrioventricular (AV) junction above the septal leaflet of the tricuspid valve.

The bundle of His is a chordlike structure that traverses from the compact AV node through the membranous interventricular septum and measures around 20 mm in length and 4 mm in diameter. The membranous septum is formed between the junction of 2 valve commissures. On the left side, it is the commissure between the right and noncoronary cusps of the aortic valve; on the right side, it is the commissure between the septal and anterior leaflets of the tricuspid valve. The bundle of His traverses at the junction between these commissures and is covered by fibrous annular tissue.[11] In 1919, Kaufman and Rothberger[12] first proposed the concept of

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functional longitudinal dissociation of the His bundle. Purkinje cells were found to conduct predominantly in a longitudinal rather than transverse direction, and predestined fibers within the His bundle conducted to each fascicle. James and Sherf[13] studied the structure of the human His bundle under both light and electron microscopy and found that the AV node is made of conducting cells organized by twisting collagen strands that funnel into a more parallel orientation in the His bundle. The Purkinje cells were also longitudinally oriented and separated by collagen septa. These longitudinal bundles had sparse interspersed transverse connections. This concept of longitudinal dissociation was redemonstrated by Narula14 in 1977. Patients with LBBB and baseline prolonged HV intervals were paced slightly distal in the proximal His bundle, resulting in narrowing of the QRS. The following year, El-Sherif and colleagues[15] noted that distal HBP, in patients with acute right bundle branch block (RBBB) after a myocardial infarction and patients with chronic LBBB, resulted in normalization of the QRS complex with a shorter stimulus to the ventricular interval compared with the intrinsic HV interval.[15] This theory was also demonstrated in canine experiments in which conduction delay within the His bundle was associated with the appearance of a BBB following anterior septal artery ligation.

PHYSIOLOGY

Broadly speaking, PHBP can be divided into 2 subgroups: selective HBP (SHBP) and nonselective HBP (NSHBP) (Fig. 1). SHBP occurs when the pacing stimulus to the QRS onset is equal to

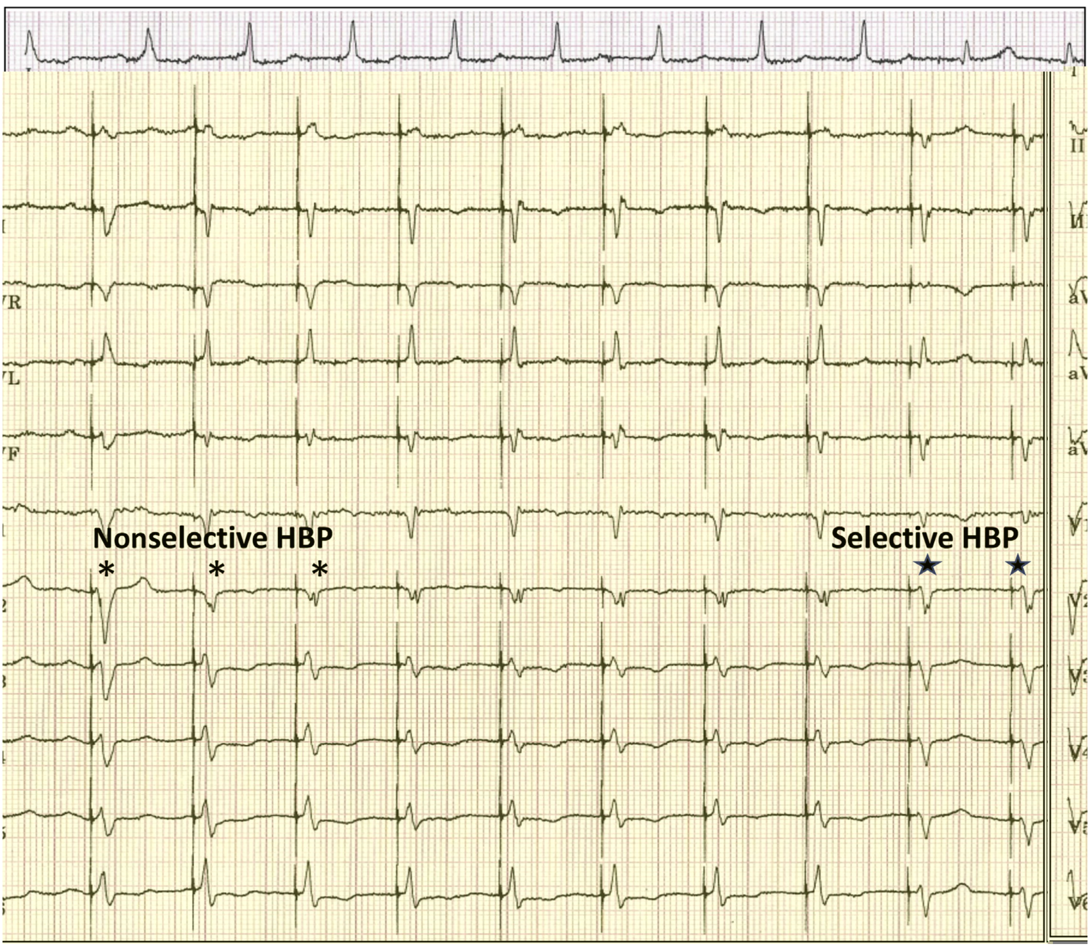

Fig. 1. A 12-lead electrocardiogram demonstrating recruitment of the His bundle as the pacing output is reduced gradually. The first 8 paced complexes demonstrate NHBP. In lead V2, subtle differences between the first 3 paced complexes (*asterisk*) can be seen because of variable fusion. The final 2 paced beats are SHBP (*star*). Note the isoelectric segment following the pacing stimulus and a narrow QRS complex with no fusion.