



Review

His bundle pacing: Initial experience and lessons learned

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Abstract

Direct His bundle pacing provides the most physiologic means of artificial pacing of the ventricles with a preserved His-Purkinje system and may play a role in patients with a diseased intrinsic conduction system. We describe our initial motivations and experience with permanent direct His bundle pacing and important lessons learned since that time.

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Motivation and first experience

The relationship between the activation sequence of the ventricles and its contractile function was first described by Wiggers in 1925 [1]. In this pioneering study, it was demonstrated that artificial stimulation of the ventricles resulted in altered cardiac activation and impaired systolic function when compared to the rapid multisite excitation coordinated by the native His-Purkinje system [1–3].

That the alteration in the sequence of ventricular activation influences ventricular performance was clearly demonstrated by the experimental study by Kosowsky et al. compared His bundle versus RV apical pacing at different AV intervals. They demonstrated that at any P-R interval, ventricular function (LV peak pressure, LV dp/dt and aortic flow) were significantly greater with His bundle pacing than with pacing from the RV apex [4]. These differences were well understood by cardiac physiologists and was summarized as such by Drs. N Levy and PJ Martin in their chapter titled “Cardiac excitation and contraction” in their cardiac physiology textbook, edited by Dr. AC Guyton (1974) [5]. They cite numerous other papers to bolster their conclusion and to point out the practical significance of this information to the then “widespread” use of artificial pacemakers. For example, in 1971 a study demonstrated biventricular pacing improved force generation of the ventricles when compared to right ventricular pacing [6].

At the time when we first performed permanent selective or direct His-bundle pacing (DHBP), evidence was also mounting that long term right ventricular pacing induced altered myocardial perfusion and contractile dysfunction [7,8]. In order to avoid the deleterious consequences of right

ventricular pacing, it was our practice to prolong programmed atrioventricular delays and avoid rate responsive pacing so as to minimize ventricular pacing and utilize the native His-Purkinje activation system. However, in patients with atrioventricular nodal disease this approach could compromise atrioventricular synchrony [9]. Pacemaker modes to directly minimize ventricular pacing were only developed several years later [10,11].

Therefore the impetus for avoiding artificial pacing or developing a more physiologic mode of pacing was clear. DHBP clearly qualified as the most physiologic ventricular pacing mode and was first demonstrated in canines by Scherlag et al. [12,13]. Subsequently, temporary transcatheter DHBP was demonstrated in humans and stable models of transcatheter DHBP were developed in canines [14,15].

In our initial experience reported at the Heart Rhythm Society in 1995, we were presented with a patient who had atrial fibrillation and severely decompensated cardiomyopathy. At this time tachycardia induced cardiomyopathy was a well described entity [16,17]. In addition, rate control of AF via AV node ablation and pacemaker placement had been associated with improved functional outcomes and improvement of cardiomyopathy [18–22]. Therefore, DHBP was attempted with AV node ablation in order to achieve rate control with preservation of the native conduction system. The patient had acute improvement in hemodynamic status with liberation from inotropes and had reversal of cardiomyopathy in longer follow up. We subsequently published the first series of permanent DHBP in a cohort of similar patients [23].

Procedural experience

Our initial experience in permanent DHBP was guided by anatomy, prior animal studies, and standard techniques for mapping and temporary pacing of the His bundle. As a single

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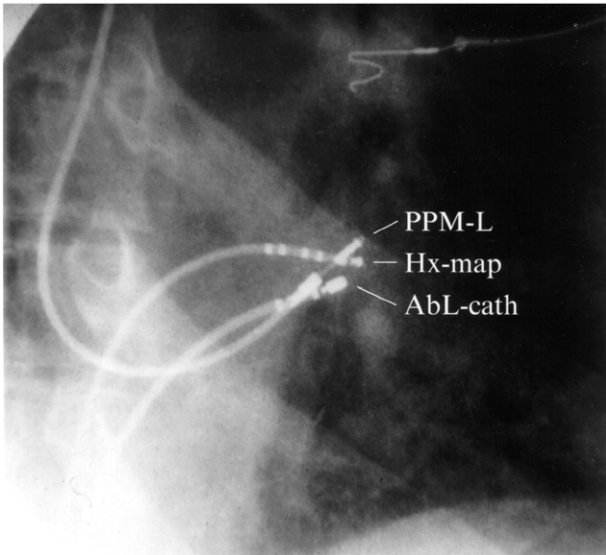


Fig. 1. Fluoroscopic visualization of initial approach to direct His-bundle pacing. Right anterior oblique projection of right femoral access hexapolar mapping catheter and right subclavian access permanent pacemaker lead. Abbreviations: PPM-L, permanent pacemaker lead; Hx-map, mapping hexapolar catheter; Abl-Cath, ablation catheter.

tricuspid annulus and position the catheter superior to the AV septum (Fig. 1).

Once the His bundle was localized and a pacing site which satisfied the criteria below was identified in proximity to the mapped location, lead placement was performed. In order to optimize anatomic selectivity during lead placement, a “Sweet-Tip” non-retractable screw in lead was utilized. In order to minimize the turns required within the pacing lead, it was preferentially introduced via the right subclavian vein. Targeting of the His bundle was further optimized via modification of a preformed J-styler in which the short and distal limb of the J was modified with an out of the plane bend oriented superiorly and anteriorly. In patients with an enlarged right atrium, the width of the turn in the J-styler was widened so as to facilitate placement at the His bundle (Fig. 1).

Criteria for DHBP

The criteria developed for DHBP in humans were based on those set out by Scherlag et al. based on studies in canines and the known electrophysiologic properties of the His bundle. [23–25]:

operator and based on the anatomy of the His bundle, access was obtained via right femoral and right subclavian venipuncture. This approach minimizes the need for repositioning the patient and the number of turns made by the pacing lead. Mapping of this His bundle was performed by a hexapolar catheter with 2 mm inter-electrode spacing using the distal electrode pair. Mapping was performed in the right anterior oblique projection to best visualize the

1. His-Purkinje mediated cardiac activation as evidence by a concordance with native QRS and T wave complexes on 12-lead electrocardiogram.
2. An identical pace-ventricular and His-ventricular interval over a wide range of range of pacing rates.
3. All-or-none capture as demonstrated by the absence of QRS widening with sequentially lower pacing output with abrupt loss of capture (Fig. 2).



Fig. 2. Criteria 3 – Direct His-bundle pacing demonstrates all or none capture. Pacing at the His bundle demonstrates all or none capture. Pacing amplitude is decreased until the 5th stimulus which leads to complete loss of capture.

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