His-Bundle Pacing: An Alternative Physiologic Site of Cardiac Pacing

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KEY WORDS: His-bundle pacing (HBP), pacemaker, right ventricular apical pacing

IMAJ 2017; 19: 657–658

Conventional apical right ventricular pacing has been the standard of practice for patients requiring permanent ventricular pacing. However, this method of pacing can lead to asymmetric ventricular hypertrophy and dilatation, myofibrillar disarray and ventricular dyssynchrony, as well as heart failure and persistent atrial fibrillation [1].

His-bundle pacing (HBP) has been proposed as a more physiological alternative pacing site and has been shown to prevent the detrimental effects of right ventricular apical pacing in long-term follow-up [2].

Despite the evidence supporting HBP, the procedure has not gained widespread acceptance in clinical practice because technical difficulties, such as the necessary use of a mapping catheter for locating the His-bundle, implantation of an additional right ventricular lead in the apex for backup, and the complexity of positioning the lead in a proper and stable position.

Recently, Sharma and colleagues [3] showed the feasibility and safety of HBP without using either a mapping catheter or backup right ventricular lead in the apex.

We decided to perform HBP using the technique proposed by Sharma et al. [3]. To the best of our knowledge, this is the first report of this particular procedure performed in Israel.

PATIENT DESCRIPTION

The patient was a 77 year old female who required a permanent pacemaker due to sick sinus syndrome. She did not have any evidence of atrioventricular node or infranodal disease and had normal left ventricular systolic function. According to the latest 2013 European Society of Cardiology guidelines on cardiac pacing and cardiac resynchronization therapy, a dual chamber pacemaker with preservation of spontaneous atrioventricular conduction is preferred [4].

We accessed the left subclavian vein and inserted a short 9Fr sheath (SafeSheath®, Pressure Products, CA, USA). Next, a dedicated delivery sheath (C315HIS, Medtronic Inc., MN, USA) with a double curve, which enables pointing the sheath to the superior atrioventricular septum, was inserted through the short sheath into the right ventricle over a guide wire (EMERALD™ Guidewire, 150 cm, 0.035 inch, Standard J-Tip, Cordis, FL, USA). Subsequently, the pacing lead (Select Secure™ 3080-69 cm, Medtronic Inc.) was advanced through the sheath so that only the distal part extended beyond the sheath. A unipolar electrogram was recorded from the lead tip and displayed on a Medtronic pacing system analyzer (gain 0.2 mv/mm, sweep speed 50 mm/s).

The delivery sheath was pulled back from the right ventricle with minimal counterclockwise rotation while recording a unipolar electrogram from the lead tip until a His-bundle electrogram was recorded at the superior atrioventricular septum [Figure 1A and 1C]. The lead was then screwed in with 4–5 clockwise rotations.

We did not use a mapping catheter for locating the His-bundle nor did we use a backup right ventricular lead in the apex.

Measured R wave amplitude was 5.9 mV, pacing threshold was 0.8V@0.5ms, and lead impedance was 854ohm (all in unipolar).

We used a slitter (Universal II Slitter 6230, Medtronic Inc.) to slit the long delivery sheath (C315HIS) after advancing the lead to create minimal slack.

The next step was to implant an atrial lead (Model 5076-52 cm, Medtronic Inc.) using a standard technique. The two leads were then connected to a dual chamber pacemaker (Sensia SEDR01, Medtronic Inc.).

A follow-up examination in our pacemaker clinic was conducted 4 weeks after the procedure. R-wave amplitude was 8.0 mV. Pacing threshold was 0.5V@0.4ms. Lead impedance was 481 ohms (all in bipolar). Follow-up of 12 lead electrocardiograms in the pacemaker clinic are shown in Figure 1B. Figure 1C presents fluoroscopy in right anterior oblique and left anterior oblique views 4 weeks post-implantation and confirms that both leads continue to be in their original locations.

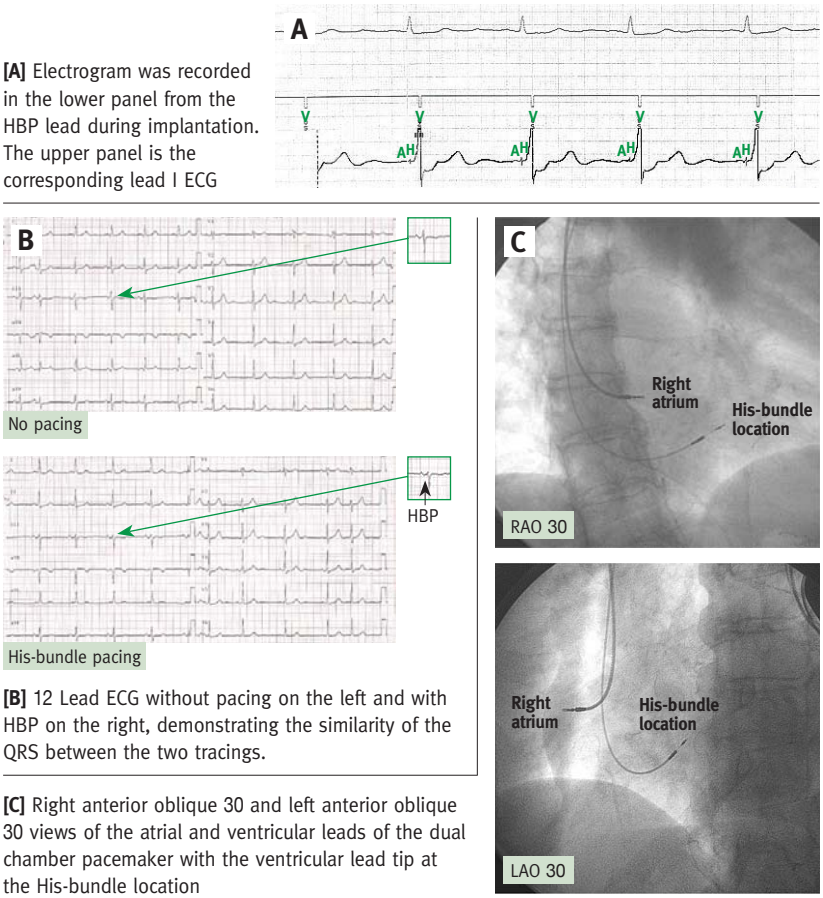
COMMENT

Right ventricular apical pacing may potentially cause adverse outcomes such as heart failure and atrial fibrillation.

HBP has been known for many years to be an alternative site of pacing to avoid the deleterious effect of right ventricular apical pacing. The success rate of HBP has been reported in the literature to be between 44–95% using various implant techniques, including a mapping catheter to identify the His-bundle location. A second backup right ventricular lead is often implanted as well.

According to a recently published study of patients with more than 40% ventricular pacing, heart failure hospitalization was significantly reduced with HBP compared to right ventricular apical pacing after 2 years of follow-up (2% vs. 15%; $P = 0.02$) [3].

Figure 1. His-bundle pacing



[A] Electrogram was recorded in the lower panel from the HBP lead during implantation. The upper panel is the corresponding lead I ECG

[B] 12 Lead ECG without pacing on the left and with HBP on the right, demonstrating the similarity of the QRS between the two tracings.

[C] Right anterior oblique 30 and left anterior oblique 30 views of the atrial and ventricular leads of the dual chamber pacemaker with the ventricular lead tip at the His-bundle location

HBP = His-bundle pacing, ECG = electrocardiogram, RAO = right anterior oblique, LAO = left anterior oblique

Furthermore, Lustgarten et al. [5] showed that it is feasible to normalize the QRS in patients with bundle branch block disease with a permanently implanted HBP lead, and that HBP can elicit a 6 month cardiac resynchronization therapy (CRT) response that is comparable to that of biventricular pacing.

We used the technique of Sharma and colleagues [3], who recently showed a success rate of 80% with acceptable feasibility and safety without using a mapping catheter or backup right ventricular lead.

Using the specified lead and sheath we found the procedure feasible and safe without significantly increasing the fluoroscopy time or procedure duration (17 minutes and 90 minutes, respectively).

CONCLUSIONS

We hypothesize that HBP will become more prevalent and used more often due to its high feasibility and advantages over the standard right ventricular apical pacing, in particular in patients who will require frequent ventricular pacing or for whom CRT therapy is not possible.

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Capsule

A clue to a drug's neurotoxicity?

The drug BIA 10-2474 (BIAL, Portugal) inhibits fatty acid amide hydrolase (FAAH), a lipase that degrades a specific endocannabinoid. On the basis of this activity, BIA 10-2474 was being developed as a potential treatment for anxiety and pain. In a phase 1 trial of the drug, one subject died, and four others suffered brain damage. As an initial step in investigating whether inhibition of off-target proteins by BIA 10-2474 might contribute to its clinical neurotoxicity, van Esbroeck et al. used activity-based proteomic assays to identify proteins targeted by the drug. Studying human cells and brain samples from subjects not associated with the trial, they found that BIA 10-2474 targeted several different lipases in addition to FAAH. It also substantially altered lipid metabolism in cultured neurons.

Science 2017; 356: 1084

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