Permanent His-Bundle Pacing After Atrioventricular Node Ablation in a Patient With Chronic Atrial Fibrillation and Mitral Regurgitation

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Hemodynamic deterioration because of worsening of mitral regurgitation can occur in a small number of patients undergoing atrioventricular node (AVN) ablation and pacing therapy. Patients with moderate mitral regurgitation before ablation seem prone to this complication. Successful permanent His-bundle pacing after AVN ablation was performed in a patient with chronic atrial fibrillation and moderate mitral regurgitation. Pulmonary capillary wedge pressure V-wave amplitude was markedly diminished and the mitral regurgitation area, calculated from the echocardiogram, was decreased by His-bundle pacing compared with that during right ventricular outflow tract or apical pacing. (Circ J 2005; 69: 510–514)

Key Words: Ablate and pace therapy; Atrial fibrillation; His-bundle pacing; Mitral regurgitation

Atrioventricular node (AVN) ablation followed by permanent pacemaker implantation has proven very effective in controlling ventricular rates in patients with drug-refractory atrial fibrillation1-4 and most patients show a marked improvement in cardiac performance and quality of life. This potential benefit seems to be greatest in patients with depressed left ventricular (LV) function prior to the procedure.2 However, hemodynamic deterioration in relation to worsening mitral regurgitation (MR) can occur in a small number of patients following AVN ablation and pacing therapy5,6 and those with moderate MR prior to the ablation seem prone to this complication.5,7 High right ventricular (RV) septal pacing produces shorter QRS duration and better chronic LV function than RV apical pacing in patients with mild to moderate LV dysfunction and chronic atrial fibrillation after AVN ablation8-11 but direct His-bundle pacing produces a more physiological ventricular contraction and may result in less worsening of MR in comparison.12,13 We describe a case of successful permanent His-bundle pacing after AVN ablation in a patient with chronic atrial fibrillation and MR.

Case Report

A 72-year-old woman with chronic atrial fibrillation and MR was referred because of recurrent congestive heart failure. Her 12-lead electrocardiogram showed atrial fibrillation at a rate of 170 beats/min and nonspecific ST-T changes (Fig 1). Echocardiogram revealed moderate LV dilatation (LV end-diastolic dimension, 58 mm) and decreased LV systolic function (ejection fraction, 33%) associated with moderate MR. After relief of congestive heart failure, 24-h Holter monitoring showed that total ventricular activity was 171,360 beats/day at a mean heart rate of 119 beats/min, even on methylidigoxin and verapamil.

Because of the drug-refractory atrial fibrillation, we decided to use AVN ablation and pacing therapy, but because we were worried that there might be post-procedural deterioration of the MR we elected to use direct His-bundle pacing therapy, which is an advanced but still investigational technique, after written informed consent was given. A hexapolar catheter with 2-mm interelectrode spacing was introduced via a femoral vein and advanced near the AV septum superior to the tricuspid valve. Subsequent mapping and localization of the His-bundle was done in biplane fluoroscopic projections. The catheter was positioned to record the largest bipolar His-bundle potential, and an attempt was made to capture and pace the His-bundle. Next, the ablation catheter with a 4-mm distal electrode (EP Technologies Inc, San Jose, CA, USA) was positioned in the compact AVN area. Radiofrequency energy was delivered, starting posteriorly and advancing anteriorly toward the area of the largest His-bundle potential recording site, until complete AV block was obtained (Fig 2C). After the second radiofrequency application to the compact AVN, complete AV block was produced and junctional escape rhythm emerged at a mean heart rate of 40 beats/min (Fig 2A). The successful ablation site had a large atrial potential, small ventricular potential, and tiny His-bundle potential, which implied compact AVN ablation, not His-bundle ablation.

Acute hemodynamic assessment using a Swan-Ganz catheter and LV cannulation compared the hemodynamic improvement between 3 pacing sites: the RV outflow tract (RVOT), RV apex (RVA), and His-bundle area. Data obtained were as follows. LV end-diastolic pressure (LVEDP) was 14 mmHg before ablation and after AVN ablation, LVEDP decreased to 11 mmHg during RVA pacing, 9 mmHg during RVOT pacing, and 7 mmHg during His-bundle pacing. Mean pulmonary capillary wedge pressure

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Fig 1. Twelve-lead electrocardiogram before and after atrioventricular node (AVN) ablation and His-bundle pacing. Baseline rhythm before ablation is atrial fibrillation with mean heart rate of 119 beats/min. After AVN ablation, direct His-bundle pacing is achieved. Note that the QRS morphology of His-bundle pacing waveforms is identical to that recorded before ablation.

Fig 2. (A) After second radiofrequency application to the atrioventricular (AV) node area, complete AV block is obtained, and junctional escape rhythm emerges at a mean heart rate of 40 beats/min and an His-ventricular (H-V) interval of 38 ms. (B) QRS morphology of His-bundle pacing waveforms is identical to that of the junctional escape beats. The pacing stimulus–ventricular potential interval is 38 ms, which is exactly that of the H-V interval, suggesting that perfect His-bundle capture has been obtained by the screw-in permanent pacing lead. (C) The screw-in lead is located in the AV septum, near the ablation catheter and is adjusted to obtain the largest His potential. Once the site that allowed capture of the His-bundle directly at a reasonably low pacing output was established, the lead screw was anchored. PM, pacemaker; St, stimulus; ABL, ablation catheter; RVA, right ventricular apex.
(PCWP) also changed, from 18 mmHg before ablation to 13 mmHg after ablation during RVA pacing, 12 mmHg during right outflow tract pacing, and 11 mmHg during His-bundle pacing. A large V-wave was present in the PCWP before and after ablation because of moderate MR (Fig 3). After ablation, the peak V-wave amplitude during RVA pacing was 17 mmHg, 15 mmHg during RVOT pacing and 13 mmHg during His-bundle pacing. Af, atrial fibrillation; LVP, left ventricular pressure.

Fig 3. Acute hemodynamic assessment using a Swan-Ganz catheter and left ventricular cannulation compared the hemodynamic change from 3 different pacing sites: the right ventricular apex (RVA), right ventricular outflow tract (RVOT), and His-bundle area. Large V-wave in the pulmonary capillary wedge pressure (PCWP) is present before and after ablation. After ablation, the peak V-wave amplitude during RVA pacing is 17 mmHg, 15 mmHg during RVOT pacing and 13 mmHg during His-bundle pacing. Af, atrial fibrillation; LVP, left ventricular pressure.

Fig 4. 2-D color Doppler echocardiogram recorded 1 month after ‘ablate and pace therapy’. Note that the mitral regurgitation area visualized during His-bundle pacing (Right panel) is dramatically reduced compared with that seen during right ventricular apical pacing (Left panel).

RVA Pacing  Direct His-bundle Pacing

After these measurements were made, separate pacemaker leads were inserted in the RVA and His-bundle area. The His-bundle pacemaker lead, a model 5068 ‘CAPSUREFIX’ bipolar screw-in lead (Medtronic, Inc, Minneapolis, MN, USA), was advanced to the AV septum and positioned near the mapping catheter. Lead position was refined with the aid of a ‘LOCATOR’ steerable stylet (St Jude Medica, Inc, Sylmar, CA, USA) to obtain the largest His-bundle potential. Once the His-bundle could be
directly captured at a reasonably low pacing output, the lead screw was anchored; however, it was hard to engage the screw in the membranous septum and several attempts were made because of repeated gross dislodgement of the lead tip. Finally, this procedure succeeded in permanent, direct His-bundle pacing at a pacing threshold of 1.75 V and 0.4 ms. The ventricular sensing threshold was 2.0 mV. The QRS configuration of His-bundle pacing was identical to that recorded before ablation, and the pacing stimulus—ventricular potential interval was 38 ms, exactly the same as the His-ventricular interval, suggesting that perfect His-bundle capture had been obtained by the screw-in permanent pacing lead (Fig 2A, B). A dual chamber rate-response pacemaker was implanted, and the pacemaker lead anchored to the His-bundle was connected to its atrial output and the RVA lead to the ventricular output. The pacemaker was programmed to DDDR mode so that back-up ventricular apical pacing would occur immediately if His-bundle pacing failed for any reason. During the follow-up period, the direct His-bundle pacing threshold gradually increased to 3.5 V/0.4 ms even though we used a steroid-eluting screw-in lead.

M-mode and 2-dimensional (2-D) color Doppler echocardiograms were recorded at 1 day and 1 month after pacemaker insertion. LV ejection fraction during His-bundle pacing improved dramatically from 41% to 65% at 1 month after the procedure and the LV end-diastolic dimension was reduced from 58 mm to 50 mm. Based on this evidence, we diagnosed tachycardia-induced cardiomyopathy with chronic atrial fibrillation and mild to moderate MR. The color Doppler echocardiogram recorded at 1 month after the procedure showed that the MR area during His-bundle pacing appeared to be smaller than that during RVA pacing (Fig 4). The patient’s New York Heart Association (NYHA) classification changed from III to I after direct His-bundle pacing. And then, we changed the pacing mode from direct His-bundle pacing to RVA pacing tentatively, but consequently the patient’s condition worsened to NYHA II. After returning to direct His-bundle pacing mode, the patient regained NYHA I status.

**Discussion**

Radiofrequency catheter ablation of the AVN followed by permanent pacemaker implantation, so-called ‘ablate and pace’ therapy, is an effective therapy for patients with drug-refractory atrial fibrillation with rapid ventricular response. A meta-analysis showed that ‘ablate and pace’ therapy improves a broad range of clinical outcomes for patients with medically refractory atrial fibrillation and that calculated 1-year mortality rates after this therapy are low and comparable with medical therapy. The greatest benefit of ‘ablate and pace’ therapy may be the ability to achieve reliable and definitive rate control, especially for patients with congestive heart failure and severe LV dysfunction. Several reports have indicated a high success rate and an improvement in LV systolic function in a majority of patients; although others reported that hemodynamic deterioration caused by worsening of MR can occur in a small number of patients. Retrospective analysis shows that patients with an increased LV end-diastolic dimension and moderate MR before ablation are prone to this complication. Because the present patient also had LV dilatation and moderate MR before ablation, conventional ‘ablate and pace’ therapy might have caused hemodynamic deterioration, which is why we planned permanent direct His-bundle pacing, not RVA pacing.

After AVN ablation, all patients require permanent pacemaker implantation to maintain sufficient rate control. The permanent pacemaker lead is usually inserted into the RVA. RV apical pacing changes the sequence of myocardial contraction and relaxation, producing abnormal sepal motion and reducing LV function. In patients with severely decreased LV function, this asynchronous delayed activation of the ventricular musculature occasionally causes a deterioration in hemodynamics. RV apical pacing may also disturb mitral valve apposition, which has been associated with the development or worsening of MR. Vanderheyden et al studied 108 patients undergoing ‘ablate and pace’ therapy, 3 of whom developed severe MR after AVN ablation and required mitral valve replacement to control progressively worsening heart failure. Permanent pacing using a screw-in lead located near the His-bundle or in the high RV septum or RVOT may contribute to better LV performance than does RVA pacing, and may minimize the worsening of MR.

Amitani et al reported successful experimental His-bundle pacing using a conventional screw-in lead with no histological influence on the conduction system during the chronic phase. Permanent direct His-bundle pacing in humans was recently reported by Deshmukh et al. His-bundle pacing produces synchronous ventricular depolarization and obviously preserves cardiac function in comparison with RVA or high septal pacing. Furthermore, His-bundle pacing does not appear to disturb mitral valve apposition or worsen MR in comparison with RV pacing. Deshmukh et al reported that permanent direct His-bundle pacing was feasible in selected patients with chronic atrial fibrillation and dilated cardiomyopathy and that long-term direct His-bundle pacing resulted in a reduction of LV dimensions and improved cardiac function. However, they did not evaluate the beneficial hemodynamic effects of this therapy on MR compared with RV pacing. In the present patient with chronic drug-refractory atrial fibrillation and moderate MR, we found that direct His-bundle pacing markedly reduced MR in both the acute and chronic phases in comparison with RVA pacing. DeCock et al suggested that RVOT pacing conferred the hemodynamic benefit of an overall increase in cardiac index of 17% compared with standard apical pacing. Karpawich et al found that RV inlet pacing delivered from above, on, or below the annulus of the septal/anterior tricuspid valve leaflets may be hemodynamically superior to pacing from the RVOT because pacing close to the bifurcation of the His-bundle results in normalized biventricular activation and a relatively narrow QRS complex and normal axis. Twidale et al reported the notable finding that when MR developed after AVN ablation, MR was less pronounced when the site of pacing was moved to the RVOT; which might be attributable to the more normalized ventricular activation sequence (cephalad to caudal direction). The results of our investigation are similar to those observed by Twidale et al. Furthermore, our results show that PCWP V-wave amplitude was reduced and that cardiac output increased when the site of pacing was moved from the RVOT to the His bundle itself. This implies that direct His-bundle pacing is hemodynamically superior to RVA or RVOT pacing, especially in patients with medically refractory atrial fibrillation and moderate to severe MR undergoing radiofrequency ablation of the AVN.
Several limitations exist in direct His-bundle pacing with a screw-in lead. It is difficult to anchor the screw-in lead into the membranous septum because the tissue of the tricuspid valve annulus is hard. Acute and chronic pacing thresholds are relatively high despite the use of a steroid-eluting lead. Because biventricular pacing may be more beneficial than direct His-bundle pacing in patients with a wide QRS complex before ablation, candidates for direct His-bundle pacing are confined to those with a narrow QRS complex and without interventricular conduction disturbance.

When Karpawich et al first described a permanent approach to His-bundle pacing in canine models in 1992, they used a specially designed screw-in lead with a 4.5-mm-long exposed helix. The lead was introduced through a custom mapping introducer delivered via right atrial cardiomyotomy and was inserted into the septum above the tricuspid valve. In the present case, although we similarly inserted the helix into the AV membranous septum above the tricuspid valve annulus, an important difference was that ours was an entirely transvenous approach with the use of a conventional screw-in lead with 1.8-mm-long helix. Because the target site of His-bundle direct capture was so small, it was relatively hard to engage the screw into the membranous septum with the conventional screw-in lead. Because this conventional lead is not specialized for direct His-bundle pacing, there is a risk of lead dislodgement in the acute and chronic phases, which might cause deterioration of hemodynamics or unexpected events. Direct His-bundle pacing is still an investigational technique and so routine use of this technique cannot be recommended. Further studies concerning lead designs are needed to improve the success of lead insertion and the long-term stability of the pacing threshold. Once these weak points are overcome by the design of a deflectable stylet with a narrow QRS complex and medically refractory atrial fibrillation and MR.

References