Permanent, Direct His-Bundle Pacing
A Novel Approach to Cardiac Pacing in Patients With Normal His-Purkinje Activation

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Background—Direct His-bundle pacing (DHBP) produces synchronous ventricular depolarization and improved cardiac function relative to apical pacing. Although it has been performed transiently in the electrophysiology laboratory and persistently in open-chested canines, permanent DHBP in humans has not been achieved.

Methods and Results—A total of 18 patients aged 69 ± 10 years who had a history of chronic atrial fibrillation, dilated cardiomyopathy, and normal activation (ie, QRS ≤ 120 ms) were screened for permanent DHBP using an electrophysiology catheter. In 14 patients, the His bundle could be reliably stimulated. Of these 14, permanent DHBP using a fixed screw-in lead was successful in 12 patients. Radiofrequency atrioventricular node ablation was performed in patients exhibiting a fast ventricular response. All patients received single-chamber rate-responsive pacemakers. Acute pacing thresholds were 2.4 ± 1.0 V at a pulse duration of 0.5 ms. Lead complications included exit block requiring reoperative adjustment and gross lead dislodgment. Echocardiographic improvement in heart function was shown by reductions in the left ventricular end-diastolic dimension from 59 ± 8 to 52 ± 6 mm (P = 0.01) and in the end-systolic dimension from 51 ± 10 to 43 ± 8 mm (P < 0.01), with an accompanying increase in fractional shortening from 14 ± 7% to 20 ± 10% (P = 0.05). The left ventricular ejection fraction improved from 20 ± 9% to 31 ± 11% (P < 0.01), and the cardiothoracic ratio decreased from 0.61 ± 0.06 to 0.57 ± 0.07 (P < 0.01). Despite DHBP, 2 patients died at 8 and 36 months.

Conclusions—Permanent DHBP is feasible in select patients who have chronic atrial fibrillation and dilated cardiomyopathy. Long-term, DHBP results in a reduction of left ventricular dimensions and improved cardiac function. (Circulation. 2000;101:869-877.)

Key Words: bundle of His ■ pacing ■ cardiomyopathy

Tremendous advances have been made in optimizing the modality of permanent cardiac pacing, from initial asynchronous right ventricular (RV) pacing to the physiological dual-chamber rate-responsive pacing modes of today. However, relatively little progress has been made in identifying new methods to restore (or simply maintain) the normal activation of the ventricular muscle during cardiac pacing.

As early as 1925, it was demonstrated that ventricular pacing results in asynchronous delayed activation of the musculature and compromised hemodynamics in mammals.1 More recent studies in canines showed that RV apical pacing causes abnormal contraction patterns2,3 and a negative inotropic effect4–8 and that it has a disadvantageous effect on maximal venous oxygen consumption uptake and cardiac efficiency.9 Moreover, sustained RV pacing has been associated with histological10,11 and structural changes12 that cause left ventricular (LV) function to deteriorate.13 In humans, short-14,15 and long-term16 studies confirmed the adverse effects of RV pacing.

His-Purkinje activation of ventricular myocardium, however, causes synchronous activation and contraction of the ventricles and preserves LV function. Therefore, we hypothesized that permanent, direct His-bundle pacing (DHBP) and atrioventricular (AV) nodal ablation should provide the maximum therapeutic benefit in a group of critically ill patients with a history of chronic atrial fibrillation (AF), dilated cardiomyopathy, and LV dysfunction who had preserved ventricular activation (ie, narrow QRS).

Methods

Study Patients
From April 1995 to November 1997, 18 patients were identified for attempted, permanent DHBP. All had a narrow QRS complex ≤ 120 ms, chronic AF, and dilated cardiomyopathy secondary to ischemia (5 patients), tachycardia (4 patients), or both (6 patients). Three patients had idiopathic dilated cardiomyopathy. All patients had decreased LV function (LV ejection fraction [LVEF] < 0.40), a
history of congestive heart failure, and a New York Heart Association (NYHA) functional status of either Class III or IV.

**Study Protocol**

After obtaining consent for participating in this Institutional Review Board–approved protocol, patients were brought to the electrophysiology (EP) laboratory and sedated using propofol, with the addition of morphine and Versed (midazolam) as necessary. A hexapolar catheter with 2-mm interelectrode spacing was introduced via femoral venipuncture and advanced to a point near the AV septum superior to the tricuspid valve. Subsequent mapping and localization of the His bundle was done in the right anterior oblique fluoroscopic projection to best visualize the tricuspid annulus. On positioning the catheter to record the largest bipolar His bundle potential, an attempt was made to pace the His bundle. Successful, direct His bundle capture was defined using the following criteria: (1) His-Purkinje-mediated cardiac activation and repolarization, as evidenced by ECG concordance of QRS and T wave complexes; (2) the pace-ventricular interval being almost identical to the His-ventricular interval (Figure 1); and (3) His bundle capture in an all-or-none fashion, as demonstrated by the absence of QRS widening at a lower pacing output.

Once the proximal His bundle was localized, a model 4269, “Sweet-Tip” bipolar screw-in lead (Cardiac Pacemakers, Inc) that had a fixed, nonretractable helix with quickly dissolving mannitol coating was introduced via the right subclavian vein and advanced to the AV septum. A modified “J”-shaped stylet with a secondary distal curve orthogonal to the J plane allowed the lead to be properly oriented when rotated medially toward the AV septum. Iterative adjustments to the stylet shape were required because of individual patient anatomy. Using the exposed screw as a temporary anchor point, the lead was positioned near the mapping catheter and adjusted to obtain the largest His potential. Slight movements of the hexapolar His bundle catheter and permanent pacing lead were frequently required to achieve optimal positioning. If necessary, the electrode was adjusted slightly to capture the His bundle directly at a reasonably low pacing output (ie, <5 mA at 1.0 ms). Once established, the lead body was rotated 2 to 3 turns, with the screw remaining as parallel to the perceived path of the His bundle as

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Surface ECG leads I, AVF, V1, V5, and V6 and intracardiac electrograms from His-bundle mapping catheter (HBED), pacemaker lead (PPM-L), and temporary catheter in RV apex (RVA). Two leftmost QRS complexes are intrinsically conducted, whereas remaining ones resulted from His-bundle pacing. ECG concordance and equivalence provide evidence of successful DHBP in pace-ventricular (Vp-V) and His-ventricular (H-V) activation intervals.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Right anterior oblique fluoroscopic projection demonstrating final position of His-bundle pacing electrode. PPM-L indicates permanent pacemaker lead; Hx-map, His-bundle mapping catheter; and AbL-cath, ablation catheter.
possible (Figure 2). Occasionally, a slight advancement of the helix by a half-turn at a time was required to achieve consistent capture.

**Electrophysiological Study**

In the final position, His-ventricular and pace-ventricular intervals were measured at a sweep speed of 100 mm/s using a multichannel EP analysis system (Bard Electrophysiology). QRS widths were determined by measuring the widest of ECG Leads I, AVF, and V1 during native rhythm and during pacing.

In those patients in whom ventricular rate was not controlled pharmacologically, radiofrequency ablation was performed. Lesions were applied in a sequential fashion, starting posteriorly and advancing anteriorly to a site 5 mm from the area of the His bundle. AV nodal ablation/modification was deemed successful with the evidence of 100% His-bundle pacing at a rate of 70 pulses/min. Final pacing thresholds and impedances were measured using a model 5311B (Medtronic, Inc) pacing system analyzer. Although sensing of His-bundle capture, and ECG concordance in all 12 leads was considered unimportant, the filtered sensed potential was also measured before hospital discharge. In all patients, the pacemaker was inserted and at various times during follow-up (range, 5 to 38 months). M-mode and 2D imaging were used to assess and measure interventricular septal motion, LVEF, the LV end-systolic dimension (LVESD), the LV end-diastolic dimension (LVEDD), and fractional shortening.

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<th>Vp-V Interval, ms</th>
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<th>Voltage at 0.5 ms, V</th>
<th>Resistance, ohms</th>
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SD 30 13.4 10.8 10.3 14.3 0.8 1.0 86 8.3 13.3 0.8 2.5 0.2 73

Electrophysiological data collected at the time of pacemaker implantation and later during most recent follow-up visit. Follow-up data is not available in patient No. 3 as the lead acutely dislodged into the RV apex. Sensed potentials were not present in all patients at the time of the most recent follow-up. † indicates no escape rhythm >30 bpm. Pre-ablation heart rate was measured upon initial presentation to electrophysiology lab.

H-V indicates His-ventricular; Vp-V, pace-ventricular activation; PW, pulse width.

**Radiographic Study**

The cardiothoracic ratio was calculated using an anterior-posterior radiographic image and the standard equation (d R +d L)/W T , where d R and d L are, respectively, the measured distances from the right and left lateral-most margins of the cardiac silhouette to a midline drawn through the spinous processes of the vertebras, and W T is the maximum transverse width of the thoracic cavity.

**Statistical Analyses**

All results were analyzed using paired 2-tailed t tests. Significance was defined as P<0.05.

**Results**

**His-Bundle Pacing**

Of the 18 patients who were screened for permanent DHBP, 14 were prequalified on the basis of the ability to achieve reliable His pacing using a temporary EP mapping catheter. Of these 14 patients, insertion of the permanent screw-in lead was subsequently attempted. In 12 patients (86%), all of the criteria for acceptance were met, including pace-ventricular measurements being approximately equal to His-ventricular ones (61.8±10.3 versus 65.8±14.3 ms, P=NS), all-or-none His bundle capture, and ECG concordance in all 12 leads [Figure 3]. The paced-driven surface ECG QRS durations were relatively narrow, and they did not differ significantly from baseline (nonpaced) values (92.8±10.8 versus 95.0±13.4 ms, P=NS).

Permanent DHBP was unattainable in 2 patients in whom the His bundle was localized with the EP catheter. Patient 18 had a QRS of 140 ms at a pacing threshold of 1.8 V and 0.5 ms. Because this patient seemed to have at least partial activation of the His-Purkinje system at a site slightly distal to the His bundle region, the lead was not repositioned. In

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- **Echocardiographic Study**
  - M-mode 2D echocardiograms were obtained before pacemaker insertion and at various times during follow-up (range, 5 to 38 months).
  - M-mode and 2D imaging were used to assess and measure interventricular septal motion, LVEF, the LV end-systolic dimension (LVESD), the LV end-diastolic dimension (LVEDD), and fractional shortening.
another failed attempt (patient 17), right bundle-branch pacing was achieved with a QRS duration of 120 ms, but at a pacing threshold of 8 V. The lead was subsequently repositioned to the outflow tract region, as was done in the 4 patients who were screened and disqualified from DHBP. Permanent pacing was performed in these patients with follow-up at regular intervals.

Overall, the mean procedure duration to achieve permanent DHBP was 3.7 ± 1.6 hours, which included AV node ablation in 10 of the 12 patients. Importantly, all patients who had their AV node ablated exhibited an escape rhythm of >30 bpm. Moreover, 1:1 His-ventricular conduction was exhibited at pacing rates ≤120 bpm, the programmed maximum pacing rate for these patients. Acute pacing thresholds for His-bundle pacing ranged from 0.6 to 3.9 V (2.4 ± 0.9 V) at a pulse width of 0.5 ms. The mean pacing impedance was 494 ± 88 ohms. Sensed potentials were acutely present in all 12 DHBP patients, and they measured 1.6 ± 0.7 mV.

Follow-up

Lead Complications

Lead-related complications occurred in 2 patients; 1 patient (patient 8) was brought back to the EP laboratory the following day to readjust the lead to overcome an unacceptably elevated pacing threshold (ie, >7.5 V and 1.0 ms). Reattainment of an acceptable pacing threshold of 3.5 V at 0.5 ms was achieved by further advancing the screw into the AV septum by 1 to 2 turns. In another patient (patient 3), lead dislodgment into an electrically stable position in the right ventricular apex was observed 2 months after implantation.

Electrophysiological Data

All patients were followed-up in the pacemaker clinic on a regular basis. Electrophysiological data as measured via the pacemaker at the time of their most recent follow-up are summarized in the Table. The mean duration of follow-up for the His-paced patients was 23.4 ± 8.3 months, with a range of 8 to 35 months. Maintenance of His-bundle capture was demonstrated in 11 patients who had continued 12-lead ECG axis concordance in their pacemaker ECG. QRS durations were measured manually using calipers (25 mm/s) on ECG recordings, and they remained relatively narrow (104 ± 13.8 ms); however, these durations were significantly higher than the mean preimplant intrinsic (P < 0.05) or initial paced values (P < 0.05). Sensed potentials during follow-up were measured in 6 patients; they ranged from 1.0 to 3.2 mV. In the remaining patients, an underlying escape rhythm was not observed during temporary programming of the pacemaker rate to 30 bpm. Importantly, AF oversensing by the bipolar pacemaker lead at the pacemaker nominal sensitivity of 0.5 mV was not observed in any of the patients.

Echocardiographic Data

M-mode and 2D echocardiograms showed a significant reduction of LVESD (59 ± 8 to 52 ± 6 mm; P < 0.01) and LVEDD (51 ± 10 to 43 ± 8 mm; P < 0.01) and an increase in fractional shortening (from 14 ± 7% to 20 ± 10%; P = 0.05) (Figure 4). LVEF improved significantly from 18.2 ± 9.8% to 28.6 ± 11.2% (P < 0.05) (Figure 5). Overall, improvement in LV function was seen in 9 of the 11 patients with sustained His bundle pacing. Two patients demonstrated only a slight decrease in LVEF and fractional shortening (patients 9 and 11). Interestingly, patient 9 showed an initial LVEF improvement from 25% to 35% at 2 months; this subsequently declined to 20% at 16 months. Echocardiography revealed normal septal motion in 9 of the 11 patients. As expected, the 2 remaining patients had previous coronary artery bypass surgery and had abnormal septal motion before and after pacemaker implantation.
**Functional Class**

With the exception of patients 2 and 5 who died, the remaining 9 patients with sustained DHBP improved by ≥1 functional class at follow-up. Remarkably, 1 patient (patient 1) improved from class IV to class I; improvement from class IV to class II occurred in 2 others (patients 4 and 6). Overall, functional status changed significantly from a baseline value of 3.6 ± 0.5 to 2.2 ± 0.7 at follow-up (P < 0.002).

**Radiographic Data**

In all but 2 patients (patients 4 and 10) in whom no change was detectable, heart size (as measured by cardiothoracic ratio) decreased significantly (Figure 6). At a mean follow-up interval of 19 ± 11 months, the mean value of the cardiothoracic ratio, 0.57 ± 0.07, was significantly smaller than it was when measured before implantation (0.61 ± 0.06; P < 0.01).

**Patient Deaths**

Two patient deaths occurred in the permanent DHBP series. They were not related to the procedure. The 2 deaths (patients 2 and 5) were due to worsening heart failure and occurred 8 and 36 months after pacemaker implantation.

**Patients Without DHBP**

Seven patients who had a mean LVEF of 18 ± 9% were excluded from the DHBP group (including patient 3 whose lead dislodged into the RV). Of these, 5 patients were alternatively paced via the RV outflow tract (RVOT), and the remaining patient (patient 18) had a septally placed lead with partial His-Purkinje activation. With the exception of patient 18, whose NYHA functional class improved from class III to I and whose LVEF improved from 25% to 50%, the remaining non-DHBP–paced patients showed no improvement in either NYHA class or LVEF over a mean follow-up period of 21 ± 15 months. Ultimately, 4 deaths occurred in this group: 3 of the RVOT–paced patients died at intervals of 5, 6, and 36 months, whereas the RV apically paced patient (patient 3) died 36 months after AV node ablation and pacemaker insertion.

**Discussion**

**His-Bundle Pacing**

DHBP was first described for open chest surgery in dogs in 1968 by Scherlag et al.17 They used wire “plunge” electrodes to continuously stimulate the His bundle by means of an epicardial approach. Soon thereafter, transvenous His stimulation was demonstrated in canines (1969)18 and in humans (1970)19 using multipolar catheters positioned at the AV junction above the septal leaflet of the tricuspid valve. Nonselective para-Hisian stimulation of atrial and/or ventricular tissue and catheter instability during cardiac contraction provided early technological challenges.20–22 However, in a recent (1995) canine study,23 stable and selective His-bundle pacing was demonstrated using multipolar catheters located in the aortic root and/or under the septal tricuspid leaflet using average stimulus intensities of 6 to 16 mA.

In 1992, Karl et al24 described a permanent approach to His-bundle pacing in open-chested canines whereby a specifically designed screw-in lead having a 4.5-mm-long exposed helix was introduced through a custom-mapping introducer delivered via a right atrial cardiectomy and inserted into the septum above the tricuspid valve. We used a similar approach such that the helix was inserted into the AV septum above the tricuspid valve annulus and oriented such that it

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**Figure 4.** M-mode echocardiographic image showing reduction in LVEDD and LVEDD before (left) and after (right) sustained DHBP. Ds indicates systolic dimension; Dd, diastolic dimension; EF, ejection fraction; JR, patient identifier.
extended into the proximal intraventricular septum along the long axis of the His bundle. Important differences included an entirely transvenous approach and the use of a conventional pacing lead having a 1.5-mm helix. The most significant challenge we encountered was the inability to precisely direct the stylet-controlled lead tip to the small (≤2 mm in diameter) target, especially during cardiac contraction and relaxation. Moreover, difficulty was frequently encountered when attempting to engage the screw into the membranous septum. Gross dislodgment of the lead tip requiring multiple reattempts was a common occurrence.

The observation that a slight advancement of the pacing helix into the septum often yielded significantly lower pacing thresholds suggests that the 1.5-mm helix is inadequate in its ability to sufficiently penetrate the membranous septum. Therefore, permanent His bundle pacing should be attempted in all patients, regardless of the ability to demonstrate successful His capture, using the temporary mapping catheter (this was the case in 4 patients in this series).

Although acute and chronic pacing thresholds were relatively high, the pacing thresholds remained clinically acceptable (≥50% below the maximum deliverable [energy] output of the permanent pacemaker). No statistical comparison with implant values could be made because the pacing thresholds were measured using alternative methods at follow-up according to the programming capability of the pacemaker.

Bipolar intrinsic sensed potentials acutely measured 1.8±1.3 mV (range, 0.6 to 3.4 mV) and were not present in all patients at follow-up. Because the measured potentials typically coincided with ventricular depolarization, it seems that they consisted of the summation of far-field ventricular potentials (rather than the actual His potential). From a practical standpoint, the origin and amplitude of these potentials was considered insignificant because the intrinsic (escape) rate in these patients was typically ≤30 bpm and, therefore, unlikely to interfere with a demand rate-responsive pacemaker having a programmed lower-rate limit ≤70 bpm.

The observation of a widened mean QRS duration at follow-up compared with implant values cannot be readily explained. Three of the 11 patients showed a >20% increase in paced QRS width from the implant value. One possibility is that the higher pacing outputs required during chronic follow-up resulted in a greater degree of para-Hisian pacing compared with implant measurements taken at the pacing threshold. Close analyses of the follow-up 12-lead ECGs in 3 patients with slightly widened QRS durations seemed to show slurred depolarization onset, consistent with muscle-muscle activation before His-Purkinje activation. Because an improvement in heart function was seen in these patients, it seems that the slight increase in QRS width may not be physiologically or clinically significant.

Figure 5. Preimplant and follow-up echocardiographic examinations measured (A) LVEDD, (B) LVESD, (C) percent of fractional shortening (%FS), and (D) LVEF for patients with sustained DHBP. Baseline LVEDD, LVESD, and percent of fractional shortening were not available for patient 11. *P≤0.05; **P≤0.01.
Biventricular Synchrony and Septal Motion

Robert Schlant first described the improved systolic function that results from coordinated myocardial segment activation as “idiodynamic kick” in 1966. He attributed the phenomenon to the greater stretch and increased contractility (by Starling’s law) of later contracting areas that is imparted by earlier contraction of other (e.g., apical) areas. Echocardiographic studies performed in the present series indicated that during His pacing, the heart contracts in a normal fashion. With the exception of the 2 patients who had an expected preexisting septal motion abnormality from prior coronary bypass surgery, all patients exhibited normal septal motion. Paradoxical septal excursion, which was previously described by others during RV apical pacing and in left bundle-branch block, was not observed.

Tachycardomyopathy Reversal

Dilated cardiomyopathy with ventricular dysfunction is a known consequence of chronic cardiac supraventricular tachyarrhythmia that can often be reversed by restoring a normal rhythm using cardioversion or ablative techniques. Prior studies of AV node ablation followed by sustained RV apical pacing in chronic AF by Heinz et al., Brignole et al., and Natale et al. demonstrated an improvement in mean fractional shortening of 44% (range, 21.3% to 30.7%), 34% (range, 23% to 31%), and 17% (range, 24% to 28%), respectively, in patients with depressed LV function. The latter study by Natale et al and 3 other clinical studies in similar patients, including the recently completed multicenter Ablate and Pace Trial (APT), uniformly demonstrated LVEF improvements of 30%, 41%, 31%, and 32%, respectively, from mean baseline values of 30% to 32%. By comparison, a single study of rate control alone with a maintained normal ventricular activation via AV node modification in less functionally impaired hearts improved mean LVEF by 16%, from 44% to 51%.

In the present study of sustained DHBP in patients with severe LV dysfunction, mean fractional shortening and LVEF improved by 36% (range, 14% to 19%) and 61% (range, 18% to 29%), respectively. Cardiomyopathy reversal was also demonstrated by the significant reductions in mean LVEDD (by 12%) and LVESD (by 14%), with an accompanying 12% decrease in cardiothoracic ratio. Importantly, no patients exhibited immediate hemodynamic deterioration, as was reported after postablation RV apical pacing in 7% to 9% of patients in 2 studies. Because the patients in this series had much worse baseline LV function compared with patients in any of the previously referenced studies and all patients presumably benefited from rate and rhythm control, the additive effect of His pacing on LV functional improvement cannot be assessed.

Our findings suggest that the presence of a narrow QRS duration in patients with chronic AF and severe LV dysfunction may be a marker for the potential reversal of cardiomyopathy. Improvement of LV function in 2 patients with a relatively slow, yet irregular, ventricular response who did not require AV node ablation suggests that rhythm control does contribute to reversing cardiomyopathy. The observed benefit of permanent DHBP seems to be derived from both rate and rhythm control, combined with the preservation of normal LV contraction by His-Purkinje activation of the ventricular myocardium.

Clinical Implications

Chronic AF

Although RV apical pacing after AV node ablation in symptomatic patients with AF has become an often-employed procedure, questions remain as to whether it causes early exacerbation of LV dysfunction in some patients. Prior investigations have demonstrated that patients with dilated cardiomyopathy and left bundle-branch block activation seem to be at an increased risk of premature death. Therefore, one cannot exclude the possibility that left bundle-branch block pattern activation via RV apical pacing is harmful in this patient group.

Permanent pacing via the RVOT to achieve more synchronous ventricular contraction has been attempted in this patient subgroup; a number of clinical trials are underway to assess the long-term benefit of such treatment. Preliminary acute studies indicate that RVOT pacing may be beneficial, perhaps to a greater degree in patients with compromised LV function. However, a recent inpatient comparison study...
of sustained RVOT versus RV apical pacing after RF ablation in chronic AF patients failed to demonstrate either significant QRS narrowing or improvement in LV function.47

Our results are the first to demonstrate that long-term pacing at the His-Purkinje origin is not only feasible in symptomatic chronic AF patients, but when successful, hemodynamic improvement of severe LV dysfunction can be demonstrated. This approach may offer a more effective and/or safer alternative to other procedures that maintain normal ventricular excitation, including AV node slow-pathway modification and catheter or surgical Maze procedure.

AV Conduction Disorder

The relative importance of atrial filling to normal ventricular function in patients with delayed, intact conduction often leads to dual-chamber pacemaker programming conflicts. This can occur when trying to optimally program a long AV delay while simultaneously maintaining an adequately high atrial tracking rate during elevated sinus rates. Whether a patient is better served by an optimized AV filling time ending with a paced ventricular contraction or by an extended AV filling time culminating in a normal ventricular contraction is probably highly individualized and depends on many factors, including the existence and nature of structural heart disease. Rosenqvist et al27 and Leclercq et al28 demonstrated significantly higher cardiac output and LVEF with normal versus paced ventricular activation in pacemaker patients with intact AV conduction, even in those with delayed AV timing. In a similar patient group with intact but more variable AV conduction times, Jutzy et al29 argued that optimized AV pacing is preferred in patients with significantly long PR intervals (>220 ms).

The dilemma of whether to optimize AV timing or to allow a normal ventricular contraction is best illustrated in patients with delayed AV conduction and LV dysfunction secondary to dilated cardiomyopathy. Marked improvements in cardiac function have been shown by DDD/R pacing with a short AV delay to prevent mitral valve backflow.49 To accomplish this, however, a conflict arises: RV apical pacing produces a left bundle-branch block activation pattern that has been linked to increased mortality in these same patients.42,43 Dual-chamber pacing using a strategically placed His-bundle lead would provide complete optimization of pacing therapy by allowing proper titration of the AV interval while maintaining normal ventricular activation. Moreover, the long-term detrimental effects of RV pacing would be averted.

A final interesting and less understood potential application of DHBP is based on preliminary work by Scherlag,50 who showed that rapid, subthreshold stimulation in the area of the His bundle using direct or alternating current at 80 to 90 Hz can restore 1:1 AV conduction in animals exhibiting intermittent conduction through a diseased His bundle. This is possibly due to local stimulation of sympathetic nerves with facilitated conduction due to catecholamine release.

Study Limitations

Because we had no control group, the beneficial effect of permanent DHBP on LV function could not be assessed. Moreover, the safety of this technique, especially in patients undergoing AV node ablation, remains a major concern. The routine use of this technique in these patients cannot be recommended.

Future Directions

Although direct His bundle pacing was possible in a high number of patients, future studies with better lead designs are warranted to improve the pacing thresholds and success rate of this therapy. Possible improvements might include a longer helix with a steroid-eluting tip, modifications to the shape of permanent pacing leads for easier placement, or the development of lead delivery systems, such as specifically shaped (or multiplane steerable) guiding catheters that have distal mapping electrodes.

Conclusions

Recent investigations showing the hemodynamic benefits of synchronized ventricular activation accompanied by the growing body of evidence that sustained RV apical pacing can cause long-term harm has led to a resurgence in alternate57 and combined site pacing.52,53 The present study demonstrates that permanent, direct His bundle pacing is attainable in many patients and that this type of pacing provides sustained hemodynamic improvement in a subset of patients with chronic AF and LV dysfunction. If the approach to permanent DHBP can be refined to achieve greater success, randomized, controlled comparison studies are needed to conclusively determine whether DHBP offers physiological benefits over the cardiac pacing approaches used today.

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