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Alternative atrial pacing site to improve cardiac function: focus on Bachmann's bundle pacing

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Pacing from the right atrial appendage (RAA) prolongs the P wave duration and can induce interatrial and especially left-sided atrio-ventricular dyssynchrony. Pacing from Bachmann's bundle closely reproduces normal physiology and has the potential to avoid the electromechanical dysfunction associated with conventional RAA pacing. Interatrial conduction delay is associated with an increased risk of stroke, heart failure, and death. In addition to a reduction in atrial fibrillation, Bachmann's bundle pacing has emerging applications as a hemodynamic pacing modality. This review outlines the pathophysiology of atrial conduction disturbances and their potential remedies and provides the reader with a practical guide to implementing Bachmann's bundle pacing with an emphasis on the recapitulation of normal electrical and mechanical function.

Introduction

The concept of atrial physiologic pacing has, in contrast to the ventricle, received scant attention. Deleterious effects of right atrial appendage (RAA) pacing have long been described, which can be avoided by pacing at the area of Bachmann's bundle (BB). This can be implemented especially in those patients who will receive substantial atrial pacing but also may have advantages as a location for atrial sensing. This review aims to describe the anatomy of this structure, review the derangements of atrial conduction and their consequences, compare the results of RAA and BB pacing, and lastly, provide a practical guide to the implementation of physiologic atrial pacing in clinical practice.

Anatomy of interatrial conduction

The interatrial band, described initially in dogs by Lewis *et al.*,[1] and further characterized by Bachmann,[2] whose name it now bears, is a unique structure of myocardium that runs across the atrial roof from the right to the left atrial appendage and can be grossly visualized spanning the interatrial sulcus. In a series of 12 dissections, the width ranged from 3–14 mm (mean 6 mm), and height 6–18 mm (mean and median 11 mm).[3] Unlike the bundle of His, it is not insulated by a surrounding fibrous sheath but can be distinguished from surrounding fat and connective tissue as a distinct structure in the sulcus, whereas the right and left atrial insertions blend into the surrounding myocardium.[4] The rightward extensions reach the sinus node, terminal crest, and sagittal bundle, whereas the leftward surround the left atrial appendage. The right atrial endocardial projection of the BB is therefore narrow, with a width of 6.3 ± 3 mm (median 4), and centered consistently along a vertical line connecting the fossa ovalis and the superior vena cava, between 2 and 16 mm from the apex of the fossa.[5]

Just as BB can be grossly visualized as a distinct band, it also has a unique histologic appearance with a makeup of abundant Purkinje-like myocytes with characteristically low myofibril content.[6] In dogs, the transmembrane electrical properties of these specialized myocytes are unique and feature a prominent phase 2 plateau, rapid rising velocity (dv/dt), and higher resting membrane and action potentials compared to normal atrial myocytes. This allows for rapid conduction with velocities reaching 1.7 m/s, two to four times faster than surrounding atrial myocardium.[7,8] In addition, parallel longitudinal

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arrangement of myocytes certainly allows for earliest activation of the left atrium over BB, as elegantly demonstrated by electroanatomic mapping studies in patients with atrial fibrillation.[4,5,9] Other routes of interatrial conduction are also present of which the coronary sinus (CS) is the most prominent.[5]

Interatrial conduction delay and atrial myopathy

Given its central role in interatrial conduction, it is no surprise that pathologic processes where fibrosis and inflammation extend into BB are associated with significant delays in left atrial activation. Bachmann produced marked lengthening of the interatrial conduction time by crushing his eponymous bundle in dogs in 1916,[2] although it was not until much more recently that interatrial blocks were formally recognized.[10] Conduction delay in BB results in prolongation of the P wave to 120 ms or greater, along with a notched or bimodal morphology in leads I, II, or III [partial interatrial block (IAB)].[10] When a complete block is present, left atrial depolarization is not only delayed but the activation pattern is also altered, with activation proceeding over the CS and thence in a caudo-cranial direction, producing a terminal negative P wave deflection in the inferior leads. The advanced interatrial block is defined by a P wave with a duration greater than or equal to 120 ms and a biphasic morphology in leads II, III, and aVF.[10] The presence of partial or advanced IAB is associated with an increased risk of stroke, systemic thromboembolism, heart failure, incident AF, and death.[11] Advanced IAB is associated with an increased risk of stroke even in the absence of atrial fibrillation.[12] This observation, along with the finding that while the risk of stroke is transiently higher during and after episodes of AF, many strokes in patients with AF occur temporally remote from the onset of AF,[13,14] suggests that atrial fibrosis is a common cause of both AF and stroke.

The histologic infrastructure of atrial tissue has been well characterized in atrial fibrillation. There is a positive correlation between the severity of AF and extent of fibrofatty replacement of atrial myocardium. Even in BB tissue, there was a significantly greater proportion of fibrosis in permanent AF patients compared to paroxysmal AF ($P = 0.041$).[15] This correlates with other studies which have demonstrated an inverse relationship between degree of IAB and absolute value of strain rate, directly implicating IAB in atrial mechanical dysfunction.[16]

The delayed and abnormal left atrial depolarization sequence seen in advanced IAB also has hemodynamic consequences including overlapping of atrial systole with the onset of the QRS against a closed mitral valve, resulting in reduced stroke volume and maladaptive atrial remodeling (*Figure 1*).[17] This then begs the question, if interatrial conduction could be restored via pacing, could some of these poor clinical outcomes be averted or reversed?

Conventional right atrial appendage pacing

The earliest transvenous leads were tined, passive fixation leads, which required a trabeculated surface in which to entangle the tines for stability. The RAA provided an ideal location for these leads. With the advent of active fixation leads, almost any site became available for implantation and a pacing site in the RAA was no longer advantageous, however, it remained the default location for atrial lead implantation for most operators. The sinus node, arguably the most physiologic atrial pacing site, presents a smooth surface on the endocardial right atrial free wall, with the attendant risk of perforation. In addition, patients with sinus node dysfunction (SND) may manifest a prolonged sino-atrial conduction time and even sinus node exit block, making this an unattractive site. The high lateral right atrial free wall below the sinus node is highly trabeculated and therefore offers good lead stability, sensing, and pacing performance. However, screw-in lead tips will typically be implanted between the trabeculae where the wall can be paper-thin, similar to the RAA.[18] To date, the RAA remains the conventional site for atrial lead placement, however, pacing from the RAA does not reproduce normal atrial activation.

The RAA-paced P wave is of low amplitude, often measuring less than 0.12 mV in lead II and P wave duration is typically prolonged, as much as 27% greater than the sinus P wave (*Figure 2*). The P wave morphology resembles that of sinus rhythm with an increase of the terminal force of the P wave in V1, a marker for an increased risk of atrial fibrillation.[19,20] RAA pacing has been recognized to increase atrial conduction time since the early days of pacing, leading Furman to recommend programming the paced atrioventricular (AV) delay 25 ms longer than the sensed AV delay.[21] RAA pacing prolongs the P wave duration, interatrial activation time, atrial electromechanical delay, and interatrial mechanical delay, compared with sinus rhythm.[22,23] Especially in patients with intrinsic interatrial conduction delay, RAA pacing further prolongs the P wave and is associated with a particularly high risk of atrial fibrillation.[24]

Deleterious hemodynamic effects of RAA pacing vs. sinus rhythm have been most intensively studied in patients being treated with cardiac resynchronization therapy: Liang *et al.* prospectively examined the impact of atrial pacing mode on echocardiographic hemodynamic measures, and found that atrial pacing compared to atrial sensing not only delayed interatrial conduction and reduced active atrial strain, but also resulted in a significant reduction in trans-mitral inflow velocity time integral (TVI), left ventricular (LV) outflow tract TVI, diastolic filling period, and global strain.[25] Higher percentages of RAA pacing have been proportionately associated with AF incidence and burden in cardiac resynchronization therapy (CRT) recipients.[26] A high burden of atrial pacing (>50%) was associated with worsened atrial strain parameters over baseline and induced significant intra-atrial dyssynchrony in another prospective imaging cohort.[27] Clinically, those with a high atrial pacing burden showed reduced LV reverse remodeling, a higher risk of new-onset or recurrent atrial fibrillation, and more frequent heart failure (HF) readmissions.[27] However, it is not known if these associations represent cause or effect. Adverse clinical consequences of RAA pacing, including time to first atrial high rate episode, were not seen over 12 months follow-up in the PEGASUS randomized trial which compared DDD-70 programming to DDD-40 and DDDR-40.[28]

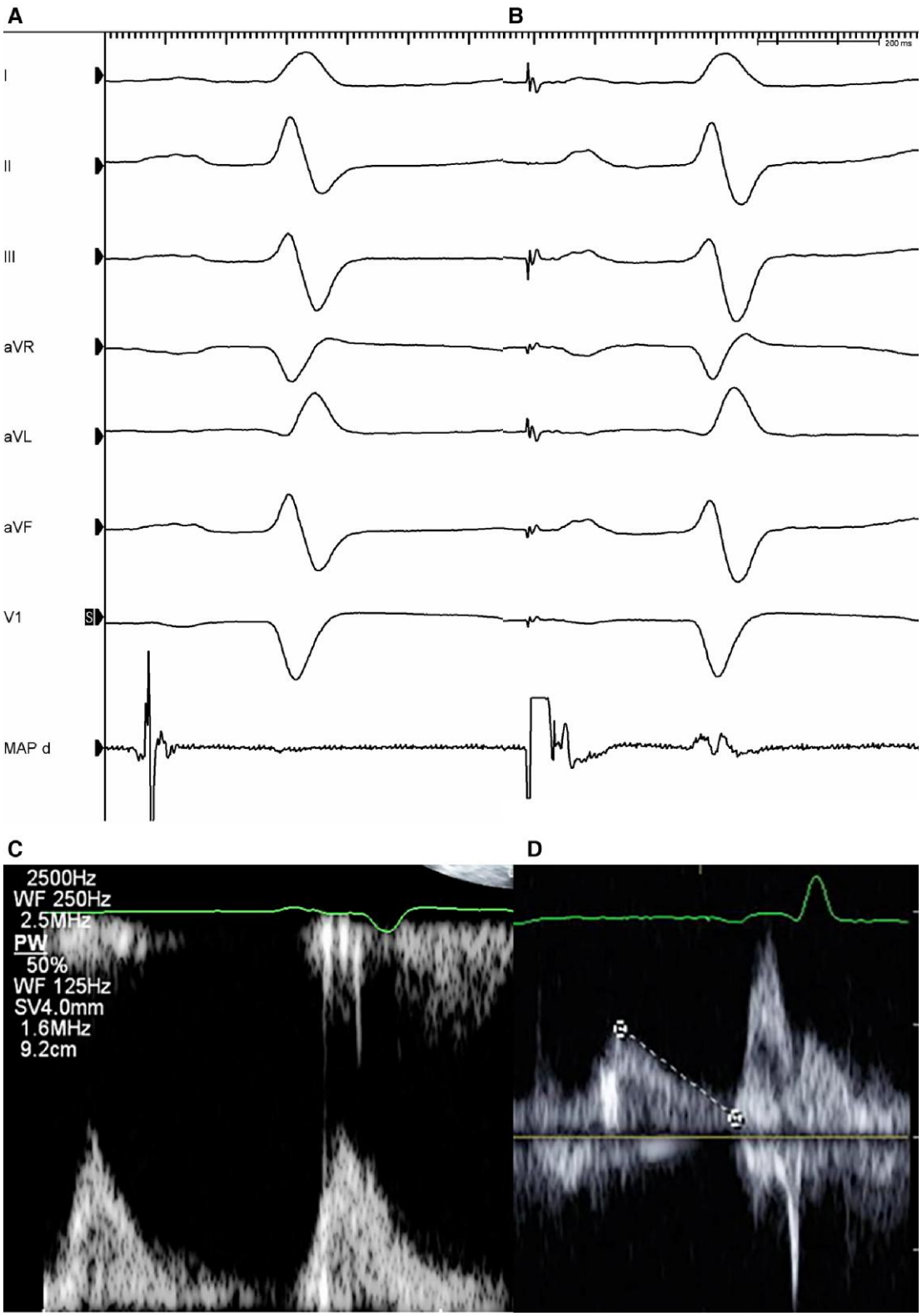

Figure 1 Electrocardiogram (ECG), intra-cardiac electrogram, and echo trans-mitral Doppler before and after implantation of a Bachmann's bundle pacing lead. (*A*) Sinus rhythm P wave duration is prolonged at 129 ms. (*B*) Bachmann's bundle pacing shortened the P wave duration to 86 ms while preserving the PR interval (204 vs. 208 ms). (*C*) Separation of E and A waves with the A wave occurring late and truncated by ventricular systole (heart rate 54 bpm). Diastolic mitral regurgitation is also seen. (*D*) With Bachmann's bundle pacing, the A wave is complete before ventricular systole (heart rate 60 bpm).

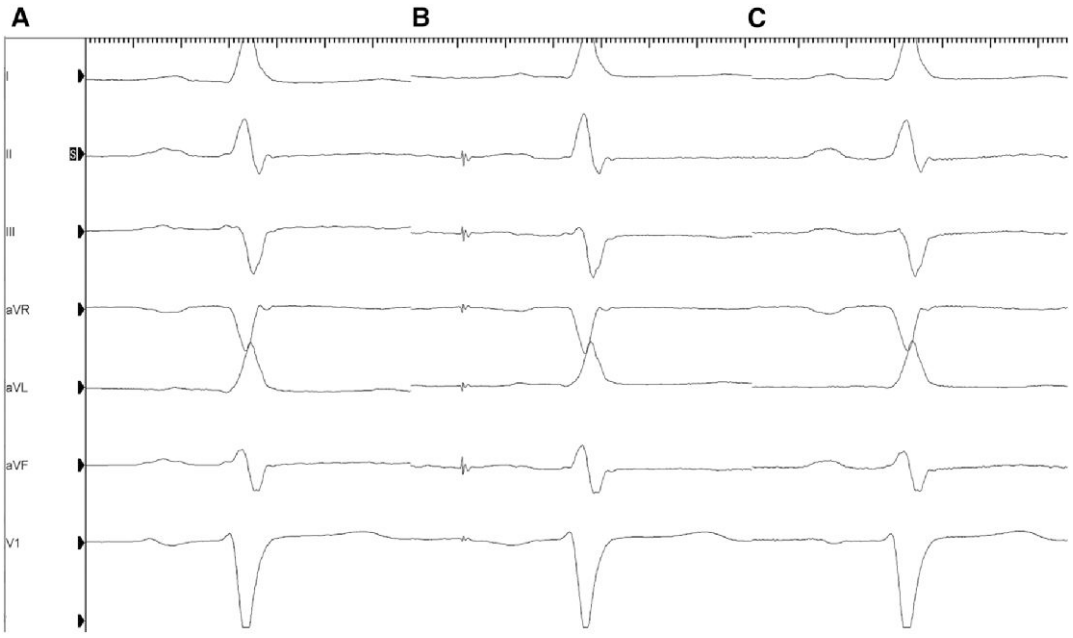

Figure 2 Sinus rhythm, right atrial appendage paced, and Bachmann's bundle-paced P wave morphologies in a patient with non-ischemic cardiomyopathy undergoing primary prevention implantable cardioverter defibrillator implant. Compared with sinus (*A*), right atrial appendage pacing (*B*) produces a low amplitude, prolonged P wave (150 vs. 116 ms) with prominent negative forces in V1, and increases the PR interval (198 vs. 178 ms), whereas Bachmann's bundle-paced P wave (*C*) is similar to but narrower than sinus (95 ms), with a shorter PR interval (159 ms).

Alternative atrial pacing sites to improve electromechanical function

Whether the association of RAA pacing with incident AF is a matter of causation or association, interest has grown in alternative pacing sites that might avoid the interatrial conduction delay and atrial dyssynchrony caused by pacing at this site (*Table 1*). Given that non-physiologic activation of the atria by RAA pacing creates conditions that facilitate re-entry as discussed above, AF has been the major clinical endpoint explored.

Several anatomic locations have been studied as alternate pacing sites, chiefly the interatrial septum (IAS) (high, which includes the BB region; mid; and low), the proximal and distal CS, and the area around the CS-os; as well as pacing from two sites in the right atrium (dual site pacing), and pacing the right and left atria (biatrial pacing). The latter two require additional leads and adaptors, and are therefore technically more complex. Proximal CS pacing may result in unfavorable hemodynamics as evidenced by delayed onset right atrial (RA) wave and short RA wave duration, as well as short PR intervals and early closure of the mitral valve, resulting in both right and left AV dyssynchrony.[41,42] Pacing from the distal CS may avoid this, and indeed biatrial pacing from the right IAS and distal CS resulted in the most favorable acute hemodynamic result compared with the RAA, proximal CS, and IAS (although not compared with BB).[42] Here, we will focus on septal pacing sites, as summarized in *Table 1*.

Of the multiple studies investigating atrial septal pacing, most have focused on the low IAS, and many have not specified criteria for specific sites. Perhaps unsurprisingly, results have been mixed, with reductions in AF burden and AF episodes in a meta-analysis of the available studies, but no overall effect on progression to permanent AF.[43,44] It should be noted that significant heterogeneity was present, although the most recent, largest study with the longest follow-up did not show a reduction in the progression to persistent AF either with, or without, an atrial overdrive pacing algorithm.[45]

It is clear, therefore, that merely pacing the septum is not sufficient to prevent AF. Because of the near physiologic atrial activation, improved P wave and hemodynamic parameters, and decreased risk of post-pacing AF, BB pacing is an attractive and feasible alternate pacing site. In a multi-center prospective randomized study, patients undergoing dual-chamber pacemaker implantation were randomized in a 1:1 manner to receive either an active fixation lead in the RAA or anterior superior IAS in the BB area.[19] In contrast to other studies, this area was specifically targeted using an anatomic/fluoroscopic approach but also attention to P wave characteristics. At implantation and during the follow-up period, device threshold, impedance, and sensing were similar between the two groups. BB pacing not only demonstrated a shorter P wave duration compared to RAA pacing but also compared to sinus rhythm (123 ± 21 ms vs. 132 ± 21 ms). No adverse events related to lead placement, including lead dislodgement, were reported. There was a marked increase in survival free of chronic AF in those with BB pacing (75%) compared to those with RAA pacing (47%) ($P = 0.01$).[29] These results were not reproduced in a small randomized controlled trial of RAA vs. BB pacing in patients with muscular dystrophy. However, this trial did not describe whether P wave or electrogram (EGM) characteristics were used to distinguish BB location from other areas of the IAS, and the small number of patients, lack of reported power calculations, and specific population question whether it was adequately powered

Table 1 Randomized controlled trials comparing different atrial pacing sites

Study	Design	Study population	Intervention group (s) (N^a)	Comparator group (N^a)	Mean or cumulative follow-up period (intervention)	Mean or cumulative follow-up period (comparator)	Primary outcome	Other results
Bailin *et al.*, 2001[29]	Multi-center RCT	Class I or II indication for permanent dual-chamber pacemaker and recurrent PAF	Active fixation leads into the anterior superior IAS (BB area) ($N = 63$)	Active fixation leads into the RAA ($N = 57$)	12.6 ± 7.4 months	11.8 ± 8.0 months	BB area pacing had a higher percent survival free from chronic AF (75%) than RAA pacing (47%) ($P = 0.01$).	BB area pacing resulted in decreased PWD compared to RAA pacing and sinus rhythm.
Padeletti *et al.*, 2001[30]	Multi-center RCT with 2 × 2 factorial design	Sinus bradycardia and symptomatic PAF	IAS pacing (±CAP algorithm) ($N = 22$)	RAA pacing (±CAP algorithm) ($N = 24$)	6 months[b]	6 months[b]	IAS pacing had a higher percent survival free from symptomatic recurrence (91%) than RAA pacing (83%) ($P =$ NS).	In both IAS and RAA pacing, the CAP algorithm had no effect on the time to the first episode of PAF and frequency of symptomatic episodes of PAF.
Padeletti *et al.*, 2003[31]	Multi-center RCT with 2 × 2 factorial design	Sinus bradycardia requiring pacemaker and symptomatic PAF	RF ablation via RALL or NRALL followed by IAS pacing	RF ablation via RALL or NRALL followed by RAA pacing	6 months[b]	6 months[b]	IAS pacing had a lower burden of AT (70 ± 150 min/day) compared to RAA pacing (219 ± 317 min/day) ($P < 0.016$).	There was no difference in AF burden between RALL and NRALL approach.
Padeletti *et al.*, 2003[32]	Multi-center RCT with 2 × 2 factorial design	Class I or II indications for permanent dual-chamber pacemaker and symptomatic PAF/AT	RAS pacing (±AT/AF prevention algorithm) ($N = 138$)	NS pacing (±AT/AF prevention algorithm) ($N = 139$)	6 months[b]	6 months[b]	AT/AF prevention algorithms did not reduce device-classified AT/AF burden in either pacing groups ($P =$ NS for both).	No difference in AT/AF burden between pacing groups. Pacing prevention was associated with reduced PAC frequency and reduced symptomatic burden in the septal pacing group.
Hermida *et al.*, 2003[33]	Single center RCT	Arrhythmia requiring permanent dual-chamber pacemaker	Mid-atrial septum pacing ($N = 32$)	RAA pacing ($N = 29$)	140 ± 87 days	159 ± 88 days	In atrial septal pacing, PWD was not prolonged, PR interval was shortened and RAEMD was prolonged compared to RAA pacing.	There was less recurrence of AF in the septal pacing group (7%) than in the RAA pacing group (25%) ($P = 0.05$); although, the time to first recurrence was similar.

Continued

Table 1 Continued

Study	Design	Study population	Intervention group (s) (N^a)	Comparator group (N^a)	Mean or cumulative follow-up period (intervention)	Mean or cumulative follow-up period (comparator)	Primary outcome	Other results
Hermida *et al.*, 2004[34]	Single center RCT	SND requiring permanent dual-chamber pacemaker	Mid-atrial septum pacing ($N = 66$)	RAA pacing ($N = 68$)	16 ± 13 months	16 ± 13 months	There was no difference in the occurrence of first episode of AF between the pacing groups.	AF-free survival was higher in the septal pacing group (70 ± 18%) than in the RAA pacing group (40 ± 19%) ($P = 0.018$) in patients with ≥1 AF episode(s) in the 3 months before implantation.
Hakacova *et al.*, 2007[35]	Single center RCT	Class I or II indication for permanent dual-chamber pacemaker and recurrent PAF	Mid-atrial septum pacing ($N = 22$)	High atrial pacing ($N = 21$)	6 months or 12 months[b]	6 months or 12 months[b]	There was no difference in ventricular pacing between the two groups.	There were more mode switches and longer durations of AF in the high atrial pacing group compared to the septal pacing group; however, this was not statistically significant.
Verlato *et al.*, 2007[36]	Multi-center RCT with 2 × 2 factorial design	SND and bradycardia-related symptoms requiring pacemaker and at least one episode of AF in 3 months before enrollment	Low IAS pacing (in patients with $\Delta CT_{OS} \geq 60$ ms vs. $\Delta CT_{OS} <$ 60 ms +/− AT/AF prevention algorithms) ($N = 20$)	RAA pacing (in patients with $\Delta CT_{OS} \geq 60$ ms or $\Delta CT_{OS} <$ 60 ms +/− AT/AF prevention algorithms) ($N = 22$)	6 months[b]	6 months[b]	In the presence of $\Delta CT_{OS} \geq$ 60 ms, rate-adaptive pacing with prevention algorithms in the IAS group demonstrated a trend toward a reduction in AF episodes/day compared to DDD only.	
Spitzer *et al.*, 2009[37]	Multi-center RCT	SND or SND with AV block	Free RA wall pacing ($N = 33$), CS-Os pacing ($N = 41$) or dual site (RAA + CS-Os) pacing (N = 32)	RAA pacing ($N = 36$)	24 months[b]	24 months[b]	No difference in survival free of AF >1% burden or AF >10% burden among all groups ($P =$ NS).	No difference in the number of patients who developed permanent AF among all groups.
Nigro *et al.*, 2010[38]	Single center RCT	Myotonic dystrophy type I and indication for permanent dual-chamber pacemaker	Bachmann's bundle pacing ($N = 14$)	RAA pacing ($N = 16$)	12 months[b]	12 months[b]	There was no significant difference in number of AF episodes and total duration of AF between the two groups.	

Continued

Table 1 Continued

Study	Design	Study population	Intervention group (s) (N^a)	Comparator group (N^a)	Mean or cumulative follow-up period (intervention)	Mean or cumulative follow-up period (comparator)	Primary outcome	Other results
Verlato *et al.*, 2011[39]	Multi-center RCT	SND and indication for permanent dual-chamber pacemaker	IAS pacing (in patients with $\Delta CT_{OS} \geq 50$ ms vs. $\Delta CT_{OS} <$ 50 ms) plus CAP algorithm (N = 47)	RAA pacing (in patients with $\Delta CT_{OS} \geq 50$ ms vs. $\Delta CT_{OS} <$ 50 ms) plus CAP algorithm (N = 50)	15 ± 7 months	15 ± 7 months	Among patients with $\Delta CT_{OS} \geq 50$ ms, IAS pacing was superior to RAA pacing in preventing persistent or permanent AF ($P = 0.047$)	There was no difference in time to the first recurrence of AF among groups and sub-groups.
Lau *et al.*, 2013[40]	Multi-center RCT with 2 × 2 factorial design	SSS and PAF in whom permanent dual-chamber pacemaker was indicated	Low IAS pacing (±CAP algorithm) (N = 188)	RAA pacing (±CAP algorithm) (N = 197)	3.1 years[b]	3.1 years[b]	There was no significant difference in time to develop persistent AF between the two pacing sites (or if CAP algorithm was on or off).	RA septal pacing significantly reduced PWD compared with RAA pacing.

[a]Number of study participants were randomized.
[b]Cumulative follow-up duration.

AT, atrial tachyarrhythmia; AV, atrioventricular; BB, Bachmann's bundle; CAP, consistent atrial pacing; IAS, interatrial septum; NRALL, non-right atrial linear lesions; NS, statistical non-significance; PAC, premature atrial contraction; PAF, paroxysmal atrial fibrillation; PWD, P wave duration; RAA, right atrial appendage; RAEMD, right atrial electromechanical delay; RALL, right atrial linear lesions; RAS, right atrial septum; RCT, randomized clinical trial; RF, radiofrequency; SND, sinus node dysfunction; SSS, sick sinus syndrome; ΔCT_{OS}, difference between basal and incremental conduction times from the right atrial appendage to the coronary sinus ostium

and generalizable. More recently, Infeld *et al*. questioned whether an anatomic approach alone would be sufficient to ensure BB capture and hypothesized that P wave analysis could provide incremental information on BB vs. right atrial septal (RAS) pacing which may have clinical implications.[40] A large cohort of patients with baseline inter-atrial conduction delay; sinus P wave duration (PWD) > 120 ms) and a minimum atrial pacing burden of >20% was divided into BB (134), other high right atrial sepal (107), and RAA (108) pacing sites based on paced P wave morphology. While the burden of AF/atrial tachyarrhythmia (AT) increased over follow-up in the patients with RAS pacing and RAA pacing, the AF/AT burden remained unchanged in those with BB pacing. In multivariate Cox proportional-hazards models, the risk of AF/AT with BB pacing was nearly half of that with RAS pacing (hazard ratio 0.43; 95% confidence intervals 0.29, 0.66; $P < 0.001$), and both *de novo* and recurrent AF were similarly affected.[40] Although these data are retrospective, they concur with the randomized controlled trial of Bailin *et al*.[29] and the dramatic difference in clinical outcomes among patients with anatomically similar lead placement (BB and high IAS) suggests a value of P-wave analysis to guide optimal atrial lead placement.

Right atrial appendage pacing may also increase the risk of atrial fibrillation indirectly through an increase in the right ventricular (RV) pacing burden. In a prospective randomized study of 385 patients with paroxysmal AF and sick sinus syndrome (SSS), RAA pacing was associated with a significantly increased RV pacing burden compared to low IAS pacing.[17] While a potential solution may be to program longer AV delays to reduce RV pacing burden, this creates its own limitations including constricting the maximum tracking rate, inducing unfavorable hemodynamics, and causing pseudo-pacemaker syndrome. On the contrary, BB pacing results in shorter AV conduction time and a significantly decreased burden of ventricular pacing compared to RAA pacing (21% vs. 4%, $P < 0.01$).[46] Suzuki et al. compared parameters of LV performance among heart failure patients with CRT devices and BB atrial lead in DDD vs. VDD mode.[47] Compared with RA-sensed bi-ventricular pacing, BB-paced bi-ventricular pacing demonstrated a shorter AV delay, in addition to shorter PWD and PQ interval. BB pacing in DDD mode appeared to improve LV hemodynamics as evidenced by a significant increase in left ventricular outflow tract velocity time integral (14.5 ± 4.6 cm in VDD compared with 15.7 ± 4.2 cm in DDD). A similar observation was noted in a patient with significant interatrial conduction disease which was reversed with temporary BB pacing and resulted in reductions in left atrial (LA) and left ventricular end-diastolic pressure suggestive of restored AV synchrony.[48]

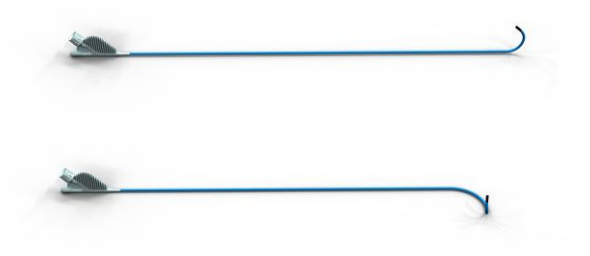

Figure 3 Pre-shaped delivery catheters (C315-J and C315-S5, Medtronic, Minneapolis, MN, USA) for delivery of the 3830 lead (Medtronic, Minneapolis, MN, USA) to the Bachmann's bundle area. Courtesy of Medtronic.

Bachmann's bundle pacing

The traditional approach to BB area pacing is based primarily on fluoroscopy. Any active fixation pacing lead can be used: either fixed or extendable-retractable helix stylet-driven leads, or a sheath-delivered, lumenless lead (SelectSecure model 3830, Medtronic, Minneapolis, MN, USA) delivered via a pre-shaped or deflectable sheath (C315 delivery catheter, Medtronic, Minneapolis, MN, USA; *Figure 3*). Stylet-driven leads can be placed using a hand-modified J stylet or a deflectable stylet (Locator Plus, Abbott, Plymouth, MN, USA).[49] In normal-sized right atria a smaller radius J is ideal. In dilated right atria, a larger radius J stylet is used, and can be modified by bending the straight part of the stylet 1–1.5 cm before the start of the curve to 150°, which extends the reach. The lead is advanced into the RA. In the right anterior oblique (RAO) view, the lead is positioned so that it is facing anterior (*Figure 4*). Switching to the left anterior oblique

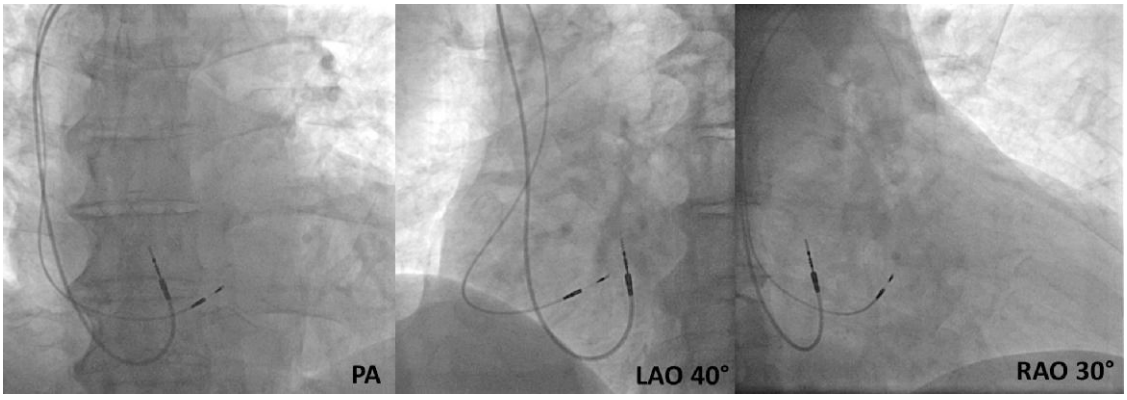

Figure 4 Final position of a stylet-driven atrial lead at Bachmann's bundle in postero-anterior (PA, left), LAO 40° (center), and RAO 30° (right) projections. In the PA view, the site of the atrial lead tip resembles a position in the right atrial appendage. However, there is no windscreen wiper movement. In this very large right atrium, the corner between the right atrial roof and the right side of the atrial septum is leftward (posterior) to the His bundle lead which serves as an approximate landmark for the plane of the interatrial septum. In RAO, the atrial lead at Bachmann's bundle points perpendicularly to the roof of the right atrium.

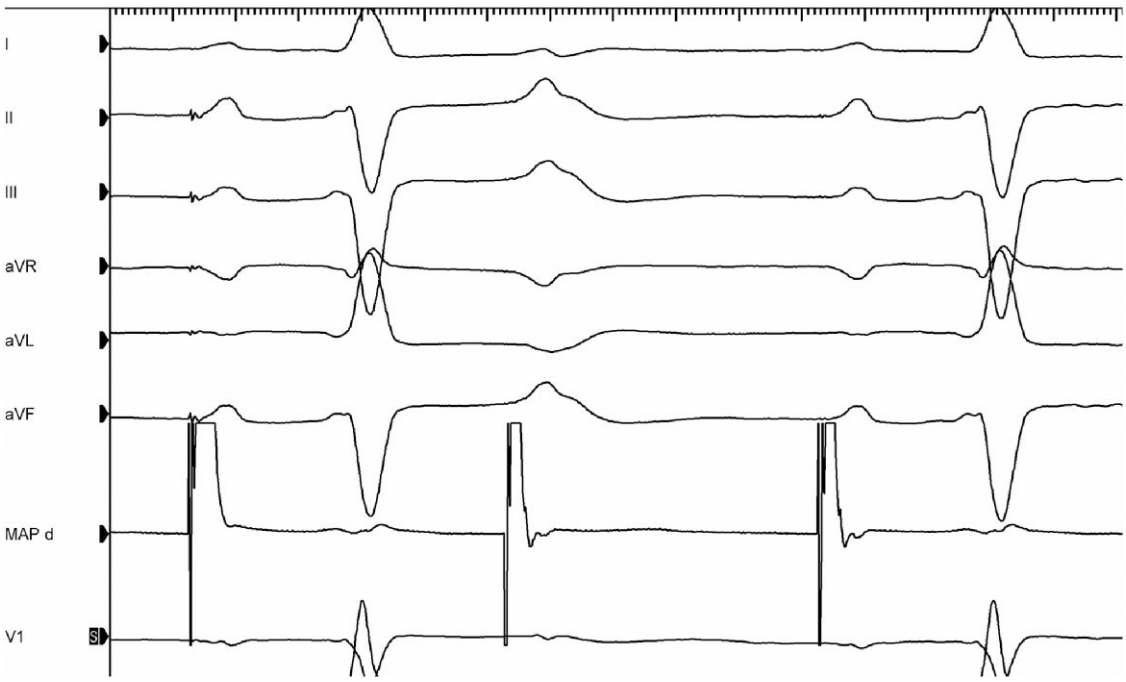

Figure 5 Pacing at 5 V/0.5 ms (first P wave) and 1 V/0.5 ms (third P wave; the second is superimposed on the T wave) showing an inferior axis P wave consistent with capture of Bachmann's bundle area but without significant change in P wave morphology or latency.

(LAO) view, the lead is directed towards the septum (*Figure 4*). A previously placed right ventricular septal, His, or left bundle lead can serve as a marker of the plane of the ventricular septum, which approximates the plane of the IAS. The lead is withdrawn until the 'J' begins to straighten out, indicating that the lead tip has reached the cranial aspect of the right atrium, and then fixated.[50] Difficulty may be encountered reaching the septum in larger atria, requiring modification of the standard J stylet or the use of a larger pre-shaped or deflectable sheath. Care must be taken not to inadvertently enter the left atrium via a patent foramen ovale, and, similar to RAA lead implantation, aortic perforation should be very rare but possible.[18] Injury current confirms adequate tissue contact as with other lead locations, and sensing and threshold values are similar to those seen at the RAA.[29] In the largest series of patients undergoing BB pacing, the success rate was 75%, and implantation and fluoroscopy times were slightly longer (15.7 and 1.9 min).[51] Pacing should produce shortening of the P wave by 10–20 ms vs. sinus, and a more positive P wave in lead II.[50] However, few investigators have reported on the reduction in P wave duration, or P wave morphology, or used these to guide the exact placement of the lead (*Table 1*).

The electrogram-guided approach to BB pacing was first described by Infeld *et al.*[40] This utilizes distinct electrogram and P wave characteristics which suggest capture of BB itself, rather than purely anatomic placement of the lead. Experience with this approach has largely been with the 3830 lead delivered via a pre-shaped or deflectable sheath, but is also feasible with stylet-driven leads (*Figure 3*). The lead is navigated to the area of BB as described above. Mapping is then performed using the tip of the lead connected in unipolar fashion to an electrophysiology recording system and to the pacemaker system analyzer to seek the right atrial endocardial electrogram which is posited to represent BB activation. Biphasic or multiphasic potentials at this location can be found in the majority of patients, rather than the simple monophasic EGM which is seen at most atrial sites, including the RAA. A distinct electrogram is often seen, separated from the local right atrial electrogram by up to 30 ms. Depending on electrode orientation, this may be more appreciable in unipolar or bipolar (after lead fixation) electrograms. Response to pacing is then assessed, both before and after lead deployment.

With high output pacing (5–10 V at 0.4–0.5 ms), local potentials are captured, and the P-wave deflection begins immediately or shortly after the pacing stimulus, as seen at most other pacing sites. With a decrement in the output during threshold testing, one of two responses is seen:

1. In approximately 50% of cases a narrow P wave results without significant change in P wave morphology with decrementing pacing output (*Figure 5*).
2. In other cases, with decrementing output, an increase in latency (isoelectric stim-P wave interval) is seen and P wave duration remains constant or shortens further. This is often accompanied by the emergence of a separate EGM that times (approximately) with the onset of the narrowed-paced P wave, and likely represents local right atrial activation in the setting of selective capture of BB, perhaps analogous to the local RV potential seen during selective His bundle capture (*Figure 6*).

The proposed origin of these potentials is supported by the observation of premature atrial contractions from this

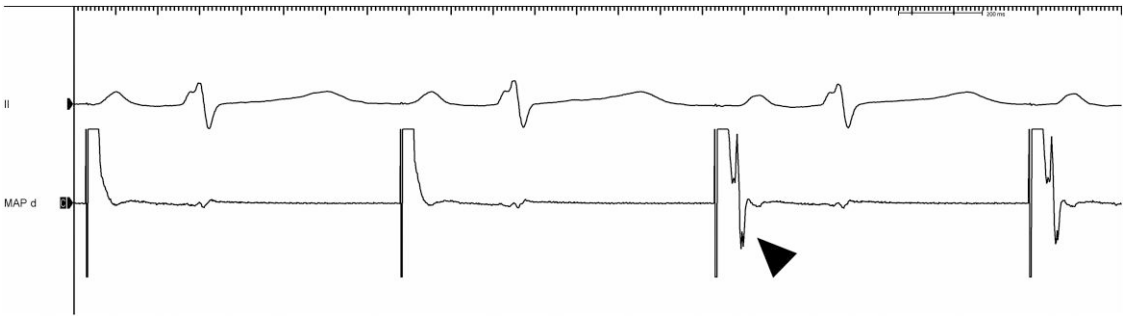

Figure 6 Decremental threshold testing from 1 V/0.5 ms to 0.9 V/0.5 ms results in increased latency and the emergence of a separate EGM (arrowhead) representing local RA activation and timing with the start of the P wave. This suggests Bachmann's bundle is selectively captured. We observe this phenomenon in a minority of cases.

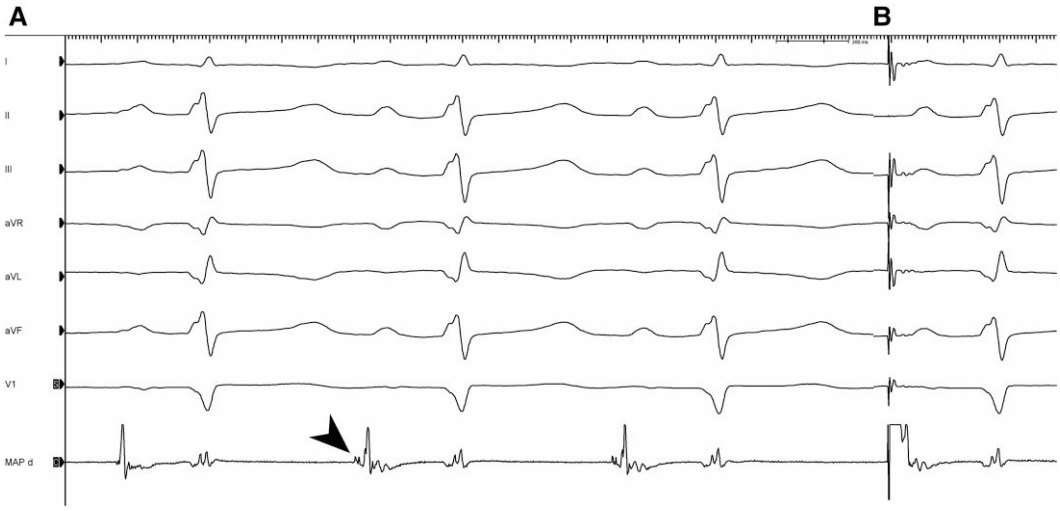

Figure 7 Surface ECG and unipolar EGM from the lead tip after implantation of a Bachmann's bundle lead (*A*). The first beat is a sinus beat. The EGM consists of a large right atrial component followed by a low amplitude but sharp component, followed by far-field left atrial EGM. The second and third P waves are premature atrial complexes, in which the small sharp EGM is earliest (arrowhead), followed by the RA and LA components. The P wave, EGM sequence, and stim-P wave time are strikingly similar to that produced by pacing from the lead tip (*B*), suggesting that pacing selectively captures the low amplitude sharp component and that this represents activation of Bachmann's bundle.

area (*Figure 7*). It is not yet clear if one pattern is more favorable than the other, nor whether each response is possible in any given patient. In general, a higher impedance (>1000 ohms acutely during unipolar pacing) is noted with output-dependent changes in P wave morphology, suggestive of deeper penetration into the tissue.

Device programming is similar to other atrial lead locations, but special attention must be paid to AV delays. Bachmann's bundle pacing site leads to a shorter P wave duration and shorter or preserved PR interval, reducing unnecessary ventricular pacing. Paced P wave latency is longer than seen at the RAA, and therefore the paced AV delay should be programmed to account for this and avoid promoting ventricular pacing, if not desired. While the paced AV delay should be programmed 25–50 ms longer than the sensed AV delay for leads in the RAA, pacing at BB typically results not only in a short interatrial delay but also in a shorter AV conduction. In our clinical experience the difference is typically at 10–20 ms. Far-field R wave oversensing is seen less often at BB than the RAA, even with the use of leads with a 10–15 mm interelectrode distance,[52] let alone with shorter distances (e.g. Optisense 1999, Abbott, Plymouth, MN, USA). We have not observed double-counting due to the more complex electrogram, such as can be seen near the crista terminalis. Medium-term follow-up of BB area leads has shown similar sensing, threshold, and impedance to RAA leads in a randomized trial.[29]

Future directions

Cardiac pacing does not merely restore chronotropic and dromotropic function but can induce dyssynchrony if normal physiology is not emulated as closely as possible. This is well established in the field of ventricular pacing, but, after a long period of relative neglect, BB pacing has increasingly demonstrated this in the atrium as well.

Bachmann's bundle pacing is currently performed using available pacing leads and a very small number of specific components. Although this is largely satisfactory, future

developments may increase the ease of implant, or reduce the need for fluoroscopy, for instance by substituting echocardiographic guidance. The optimal EGM and P wave characteristics remain to be defined and integrated into the implant workflow.

In addition to reducing AF in patients with SND, a number of other populations may benefit. Patients with advanced IAB may derive hemodynamic and symptomatic benefit from correction with BB and/or left atrial pacing.[53] Normalization of the AV delay in patients with heart failure with a reduced left ventricular ejection fraction (LVEF) and prolonged PR interval significantly improved quality of life in the recent HOPE-HF trial.[54] Maintaining or correcting atrial synchrony with BB pacing may add further benefits in this population and in those with a normal LVEF.

The resting heart rate provides a novel therapeutic target in patients with HFpEF. In the recent myPACE clinical trial, treatment with a moderately accelerated, algorithm-derived personalized pacing rate improved quality of life, NT-proBNP levels, physical activity, and atrial fibrillation compared with the conventional 60 bpm in patients with HFpEF and pre-exisiting pacemakers.[55] Notably, most patients in this study had BB leads, which likely avoided the potential negative consequences of non-physiologic atrial pacing. Whether such benefits would be seen with RAA pacing is unclear, suggesting that BB pacing be considered for patients undergoing pacemaker implants in whom this strategy might be used in the future.

Whether the implantation of completely (atrial and ventricular) physiologic pacemakers for the modulation of heart rate and AV delay in patients without a conventional indication for pacemaker implant will deliver a net benefit remains to be tested, but presents an opportunity to move cardiac pacing beyond palliative treatment of bradyarrhythmias into a holistic paradigm of restoring cardiac electrical and hemodynamic function.

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Data availability

No new data were generated or analysed in support of this research.

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