Bachmann’s Bundle
A Key Player in the Development of Atrial Fibrillation?

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Bachmann’s bundle (BB), also known as the interatrial bundle, is well recognized as a muscular bundle comprising of parallel aligned myocardial strands connecting the right and left atrial walls and is considered to be the main pathway of interatrial conduction.1 Disruption of the bundle’s structure causes interatrial conduction block (IAB),2 which is associated with development of various atrial tachyarrhythmias3,4 and with electromechanical dysfunction of the left atrium.5 Technological progress providing sophisticated mapping and imaging techniques in the past decade has increased our knowledge of specific anatomic structures and their role in development of both atrial brady- and tachyarrhythmias. This review outlines the current knowledge of the relation between anatomic and electrophysiological properties of BB and its possible role in initiation and perpetuation of atrial fibrillation (AF).

Macroscopic Anatomy of BB

In 1963, Thomas N. James described 3 pathways connecting the sinus node to the atrioventricular node (AVN), namely the anterior, medial, and posterior internodal pathways.6 Whether these conduction pathways were because of the presence of specialized conduction tissue or because of the anisotropic orientation of the muscle fibers remains controversial. Nevertheless, James described the anterior pathway as leaving the sinus node in anterior direction and giving off a secondary branch at the level of the superior vena cava to form BB.1 BB stretches subepicardially across the interatrial groove (septal raphe). It is at the interatrial groove that the BB can be identified as a discrete bundle (Figures 1 and 2) separated by fatty tissues from the infolded right atrial wall that is the limbus of the oval fossa. Notably, the bundle is not surrounded by a fibrous tissue sheath. Instead, the bundle is comprised of strands of atrial myocardium that are similarly aligned in parallel fashion. Its rightward and leftward extensions bifurcate to pass to either side of the right and left atrial appendages.7 Although they can be traced to varying extents with blunt dissection, both extensions blend into the musculature of the atrial walls. The superior arm of the rightward extension arises in the region of the cavoatrial junction close to the site of the sinus node and in the vicinity of the sagittal bundle. The inferior arm arises in the subepicardium of the right atrial vestibule. Leftward, BB buttressing part of the anterior atrial wall with its thickness (Figure 1) is still traceable to where it encircles the neck of the left atrial appendage and blends in with the lateral atrial wall. The superior part traverses in the infolding of the atrial wall, known to arrhythmologists as the left lateral ridge, to pass in front of the orifices of the left pulmonary veins.8 The inferior part descends toward the atrial vestibule to combine with the circumferentially aligned myocardial strands in the subepicardium of the inferior wall.8

In contrast to the thinner distal extensions, BB’s body across the interatrial groove is a broader band (Figures 1 and 2), with median measurements of 4 mm in thickness and 9 mm in height. It is described as trapezoidal shaped because of its short lower length (3 mm) and longer upper length (10 mm).9

Microscopic Anatomy of BB

BB myocytes are organized into relatively well-aligned myocardial strands or myofibers10 in the subepicardium and differ in orientation and direction from those in the underlying atrial walls. A perpendicular myofiber orientation has been observed at the junction with the superior caval vein, whereas myofibers are oriented more randomly at the level of the interatrial septum.9 BB myocytes are surrounded by thin septa formed of tightly packed collagen fibrils running uninterrupted over distances of 392 µm (ie, 4 times the average myocyte length).10 Over larger distances, those thin septa form several interconnections. Thick septa are present near the surface of BB, where they often encircle groups of myocytes.

Sherf et al10 suggested that BB contained myocytes specialized for rapid conduction. These myocytes differed from ordinary atrial myocytes by a relative paucity of organized myofilaments and by their lack of a transverse-axial tubular system. Based on electron microscopy, 5 different types of myocardial cells in BB were distinguished. Dependent on their cytological features, these myocardial cells were labeled as myofibril-rich, myofibril-poor, broad transitional cells, slender transitional cells, and P-cells. Myofibril-rich cells in BB...
did not differ from myocytes in other parts of the atrial myocardium. Myofibril-poor cells are Purkinje-like cells that are not only numerous in BB but also in other interatrial conduction pathways. P-cells in BB are very similar to P-cells present in the sinoatrial node (SAN) and AVN. Slender transitional cells are shorter and narrower than broad transitional cells, but both broad and slender transitional cells show similarities with myofibril-rich and myofibril-poor cells. They possess many myofibril-dense zones and also intracellular spaces filled with cytoplasm. However, other investigators were not able to find specialized myocytes.

**Embryology of the Cardiac Conduction System and BB**

To determine whether BB belongs to the specialized cardiac conduction system, the embryological origin of BB was studied. The primary heart tube is known as the embryonic origin of chamber myocardium. By expressing chamber differentiation genes, the primary heart tube myocardium differentiates into chamber myocardium. An in vitro test showed that the expression of these genes can be repressed by T-box transcription factor Tbx2, preventing the chamber from differentiation and enabling the primary heart tube cells to contribute to other parts of the human heart, including cardiac conduction tissue. Given the discussion whether or not BB is part of the cardiac conduction system, it is not yet clear if BB is also a derivate of the primary heart tube myocardium.

By using immunologic markers, a joint embryonic origin of different parts of the human heart can be demonstrated, as how Blom et al did by using HNK1 and how Jongbloed et al did by using CCS-lacZ. In the study of Blom et al, HNK1 staining the developing atrioventricular conduction system — was detected in the right venous valve (corresponding to the posterior pathway as described by James), the left venous valve, and in the anterior pathway. This suggests that the posterior pathway, the left venous valve, and possibly BB have a joint embryonic origin. Jongbloed et al detected CCS-lacZ expression (lacZ stains developing and mature conduction system) in BB, SAN, His bundle, and many other CCS- and non-CCS–related structures. Noteworthy is that all detected lacZ- and HNK1-positive tissues, including BB, are known for their relation with arrhythmias. This supports the hypothesis that the occurrence of arrhythmias preferentially arises from areas that are related to the developmental pathway of CCS and makes attractive the hypothesis of BB belonging to the cardiac conduction system.

**Vascularization of BB**

Vascularization of BB has been studied by using computed tomography imaging in both healthy subjects and patients with structural heart disease. The SAN artery invariably supplied BB. Even in patients in whom BB could not be visualized, small branches originating from the SAN artery could be detected in the anatomic area of BB. In 55% of patients, BB was vascularized by the right sinoatrial node artery originating from the right coronary artery, in 40% by the left sinoatrial node artery originating from the ramus circumflexus, and in 5% by both sinoatrial node arteries. Figure 3 shows a coronary angiogram demonstrating vascularization of BB by branches of the sinoatrial node artery originating from the right coronary artery.

**Electrophysiological Properties of BB**

Changes in the normal anatomy of BB, such as disruption of the parallel orientation of muscle fibers, may predispose to development of atrial tachycardias. Some studies have demonstrated preferential conduction corresponding to the internodal pathways. Whether this preferential conduction is because of the presence of specialized conduction tissue or because of the anisotropic orientation of the muscle fibers is still a matter of debate.
In vivo and in vitro canine experiments revealed that during hyperkalemic atrial arrest, fibers of the interatrial band remained excitable. Sinus node impulses were still able to propagate to the AVN, suggesting the existence of specialized conduction tissue between the sinus node and the AVN. In addition, electric activity in the interatrial band persisted when both atria were inexcitable, indicating the presence of specialized atrial fibers in this region. Evidence of specialized interatrial band fibers was also provided by Hogan et al. who demonstrated that pacemaker activity of these fibers could be induced by catecholamines. In 1963, Horiba described BB membrane action potentials in mongrel dogs, with an average action potential duration of 345±40 ms during sinus rhythm compared with atrial and ventricular muscle action potential durations varying between 200 and 300 ms. In addition, the author demonstrated that BB action potentials were characterized by a steep upstroke followed by a pointed overshoot and a short plateau phase (Figure 4). Similar characteristics of BB membrane potentials were found in subsequent studies. Wagner et al. recorded transmembrane potentials in superficial canine BB fibers during an in vitro experiment. They measured a resting potential up to −95 mV, dv/dt up to 225 V/second, and an action potential amplitude of 130 mV, a prominent overshoot (up to 40 mV), and a distinct plateau. Those transmembrane potentials differed from recordings from left atrial muscle fibers that showed a resting potential up to −80 mV, dv/dt up to 225 V/second, action potential amplitude up to 98 mV, and a less prominent overshoot (up to 30 mV). No distinct or a very short plateau phase was present. At that time, the 5 different types of myocytes had not been described yet, but Wagner et al. noticed that when the measuring microelectrode was penetrating deeper than 1 to 5 cells into BB, ordinary atrial potentials with lesser plateaus and lower upstroke velocities (1.3 m/second in crest versus 0.9 m/second in deeper layers) were obtained. Similar results were found by Childers et al. except for a lower maximum upstroke velocity. In summary, BB fiber action potentials show both similarities and differences with action potentials that had been obtained from the Purkinje system, which is part of the CCS, and those that had been obtained from atrial myocardium. Action potentials recorded from BB fibers show a distinct overshoot and convex shoulder to the repolarization phase, whereas ordinary atrial cells and Purkinje cells have a smaller overshoot (up to 30 mV in atrial cells versus 40 mV in BB myocytes) and a concave or a straight repolarization.

BB’s plateau resembles the Purkinje prominent phase 2 plateau. Compared with ordinary atrial muscle fibers, transmembrane action potentials of BB myocytes are characterized by a higher resting membrane potential and higher dv/dt max. Unlike Purkinje fibers, BB’s action potential durations are significantly abbreviated by acetylcholine.

In vivo assessments of interatrial conduction velocity in canine hearts demonstrated that, like Purkinje fibers, BB fibers conduct impulses at a significantly higher velocity (1.7 m/second) than by the surrounding myocardium (0.4 m/second). Moreover, Wagner et al. discovered that the conduction velocity even varied within BB. Assessment of conduction velocity of superficial fibers demonstrated velocity values up to 1.3 m/second, whereas in the deeper layers, conduction velocities of only 0.9 m/second were recorded. From these observations, the authors concluded that impulse spread must occur through multiple, relatively independent, linear paths, without transverse conduction. However, most remarkable is the presence of a supernormal phase of excitability, which occurred when the coupling interval of premature beats was shortened during pacing. A similar phase of supernormal excitability has also been observed in Purkinje fibers.

In conclusion, we can say that BB shares electrophysiological properties of both Purkinje and atrial fibers.

BB Block

Because BB is the preferential pathway of interatrial conduction, structural abnormalities of BB may cause IAB. This was demonstrated by Waldo et al. who produced surgical lesions in left and right atrial portions of BB in canine hearts and studied alterations in the ECGs. After creating surgical lesions, significant changes in P-wave morphology (both in duration and polarity) were seen during pacing from different directions. Conduction delays in BB resulted in partial IAB, whereas complete block in BB resulted in advanced IAB. Normal atrial conduction is reflected on the surface ECG by P-waves with

![Figure 3: Coronary angiogram of the RCA demonstrating vascularization of Bachman’s bundle by branches of the SAN originating from the RCA. BBA indicates Bachman’s bundle artery; RCA, right coronary artery; and SAN, sinus node artery.](http://circep.ahajournals.org/)

**Figure 3.** Coronary angiogram of the RCA demonstrating vascularization of Bachman’s bundle by branches of the SAN originating from the RCA. BBA indicates Bachman’s bundle artery; RCA, right coronary artery; and SAN, sinus node artery.

![Figure 4: Differences between an action potential recorded from Bachmann’s bundle and that from atrial myocytes. See text for description.](http://circep.ahajournals.org/)

**Figure 4.** Differences between an action potential recorded from Bachmann’s bundle and that from atrial myocytes. See text for description.
a duration of <110 ms. Prolonged interatrial conduction, or IAB, is, therefore, defined as a P-wave of >110 ms. Both partial and advanced IAB produce anomalous P-waves. Partial IAB gives rise to wide, usually biphasic waves because the left and right atria are not activated simultaneously. In advanced IAB, impulses traverse the right atrium toward the AVN, after which it continues its pathway in superior direction to end in the LA. This results in a biphasic (+/-) P-wave in the inferior leads. Atrial disruption caused by fibrosis has been suggested as the mechanism underlying IAB. Fibrosis was associated with both ischemic heart disease and nonischemic heart disease, including systemic sclerosis, dilated cardiomyopathy, hypertrophic cardiomyopathy, lymphoma, and increased atrial strain because of valvular heart disease.

Histological studies demonstrated that right atrial tissue (including BB tissue) of patients with IAB shows apparent fibrosis, though fibrosis of BB alone has not been described.

It has also been suggested that increased atrial strain, irrespective of the underlying heart disease, causes delayed conduction over BB.

Clinical Importance of BB in Pathophysiology of AF

In a general hospital population, the prevalence of IAB in patients with AF is 52% (in contrast to 18% in patients without AF), suggesting a relationship between IAB and development of AF. Whereas, in a prospective study including 16 patients with advanced IAB, 15 patients (94%) developed atrial arrhythmias over time. In a follow-up period of 30 months in which no interventions were committed, patients developed AF (n=7; 44%), atrial flutter (n=4; 25%), or atrial flutter and AF (n=4; 25%). Patients were compared with a control group of 22 patients without advanced IAB of similar age, sex, clinical findings, degree of heart failure, left atrial size by M-mode echocardiography, and duration of follow-up. In the control group, only 5 patients developed AF and 1 patient developed atrial flutter during follow-up. Patients in both groups had structural heart disease.

In a larger and more recent prospective study performed on 118 patients who were admitted to a general department of a tertiary care general hospital, 29% of 41 patients with partial IAB developed AF during a 12-month follow-up period. In contrast, in patients with sinus rhythm without IAB (n=44), the incidence of AF was only 9%. Seventy-five percent of patients who developed AF during the follow-up period showed IAB in electrocardiographic registrations at baseline. Furthermore, Ariyarajah et al reported a patient with advanced IAB who developed atrial flutter over time. Abe et al studied the progression of paroxysmal AF to persistent AF. In 122 patients during a follow-up period of 26±12 months. At the onset of the study, P-wave–triggered, signal-averaged ECG was abnormal in 23 patients. Ten of them developed persistent AF, whereas only 4 patients with a normal P-wave–triggered, signal-averaged ECG had persistent AF. Hence, all these studies support the hypothesis that IAB, presumably occurring in BB, reflects the presence of electrophathological alterations throughout the atria that may predispose to the development of atrial tachyarrhythmias in humans.

Role of BB in Initiation and Perpetuation of AF

It is widely accepted that re-entry is an important mechanism in the initiation of atrial tachyarrhythmias, but whether or not BB is involved in this process has not yet been elucidated.

Ogawa et al demonstrated in canine hearts that premature impulses generated in the high LA initiated re-entry. The premature beat conducted slowly to the right atrium and resulted in echo beats in the LA. Starting from the circus movement concept of Allessie et al, the authors suggested that their observations were because of longitudinal dissociation in conduction across BB that resulted in re-entry. Hence, BB may be involved in initiation of paroxysmal supraventricular tachycardias. Epicardial and endocardial mapping during induced AF in a canine sterile pericarditis model showed that unstable re-entry circuits with very short cycle lengths maintained AF. A large number of these unstable re-entrant circuits used BB as part of their re-entrant pathway. Based on these findings, the authors hypothesized that a lesion in BB would prevent induction of stable AF.

In a prospective study, the authors demonstrated the effect of catheter ablation of BB on AF in the same model. AF (defined as a sustained episode of AF lasting >2 minutes) was induced with rapid atrial pacing, after which the midportion of BB was transected by radiofrequency ablation. AF terminated during ablation, and afterward it was not possible to induce sustained episodes of AF by rapid pacing. In 2001, Goyal and Spodick speculated on a possible relationship between conduction block in BB and induction of atrial tachyarrhythmias. They hypothesized that IAB, being a marker for left atrial dysfunction, may predispose for AF.

By using echocardiography, left atrial functional parameters of patients with and without IAB were compared. The groups were matched for left atrial size. The extent of left atrial dysfunction was larger in patients with IAB and related to the degree of conduction delay across BB. However, a cause-and-effect relationship cannot be extracted from this study, and the data should be interpreted with caution.

Role of BB Pacing in Prevention of AF

Given the numerous indications that BB plays a role in initiation and perpetuation of atrial tachyarrhythmias, it has been tested whether AF could be prevented by BB pacing. One of the first studies comparing pacing at various atrial sites in the prevention of AF was performed by Yu et al in 2000. In this study, P-wave duration of 15 patients with paroxysmal AF was measured during pacing at 6 different anatomic sites, including right atrial appendage (RAA), BB, right posterior interatrial septum (RPS), distal coronary sinus (CSd), RAA plus CSd simultaneously (DSA), and RAA plus CSd simultaneously (BiA). The P-wave duration was longest during RAA pacing and decreased in the order from RAA pacing to CSd pacing, to pacing involving septal components (BB, RPS, or DSA), finally to BiA pacing. At that time, it had already been observed that pacing at the right atrial septum reduced total atrial activation time. Later studies demonstrated that pacing at BB could also shorten atrial activation time. BB-, CSd-, and RPS-driven pacing was more effective than DSA- or BiA-driven pacing in reducing P-wave duration. Additionally, these investigators did not succeed in initiating AF by BB-, RPS-, or CSd-driven pacing when coupled with RAA extrastimulation.
whereas they did succeed during RAA-, BiA-, and DSA-driven pacing. It was, therefore, concluded that BB, RPS, and DCS pacing may be successful in preventing AF. Duyschaever et al. provided evidence that the optimal site to prevent atrial re-entry is at the middle portion of BB.

In 2002, Gozolits et al. performed single-site and dual-site pacing after ablative therapy of supraventricular tachycardia in 15 patients without structural heart disease who were not using antiarrhythmic drugs. BB pacing resulted in the shortest endocardial atrial activation time of all single-pacing sites (81±15 ms) and in the shortest P-wave duration (96±12 ms). Shorter P-wave duration during BB pacing was also observed by others. Bailin et al. compared RAA pacing with pacing from the anterior superior interatrial septum (BB region; n=63) in patients with recurrent paroxysmal AF who did not use antiarrhythmic drugs. In the BB group, P-wave duration was shorter than in sinus rhythm (BB, 123±23 versus 132±21 ms; P<0.05). One year after implantation, patients who received BB pacing were less likely to develop persistent AF (25%) compared with those who received RAA pacing (53%).

Conclusions
BB is the preferential pathway of interatrial conduction because of its electroanatomic properties. Structural changes of BB may cause longitudinal dissociation in conduction of adjacent muscle fibers, thereby facilitating re-entry and hence development of AF. Data obtained from clinical studies suggest a relationship between electrophatological alterations of BB and the development of AF. However, areas of conduction block may not be confined to BB alone. Conduction abnormalities in BB may merely reflect more general electrophathological alterations occurring everywhere in the atria. Further studies are still needed to examine the exact role of BB in initiation and perpetuation of AF and to determine whether therapy targeting BB may contribute to preventing the development of AF.

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None.

References

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