The Bachmann Bundle and Interatrial Conduction

Vignendra Ariyarajah, MD,† and David H. Spodick, MD, DSc, FACC‡§*

Abstract: The cardiac conduction system (CCS) is responsible for generation and systematic conduction of cardiac impulses. The Bachmann Bundle (BB), considered one of its several accessory impulse-conducting pathways, plays a fundamental role in interatrial conduction. Delay in this pathway leads to prolongation of the P wave on the electrocardiogram (interatrial delay or block), which in turn is a precursor for atrial tachyarrhythmias, mainly atrial fibrillation and significant left atrial electromechanical dysfunction. As such, the magnitude of its sequelae has necessitated a flurry of investigations that have been targeted toward its prevention and management. Although current studies on the use of angiotensin-converting enzyme inhibitors and atrial pacing have indeed shown some promise, it would be shortsighted to overlook and circumvent the actual underlying lesion—BB abnormality. Thus, a thorough understanding of the CCS and interatrial conduction is essential. We review current literature on the BB and discuss potential mechanisms that affect its conduction.

Key Words: Bachmann Bundle, cardiac conduction system, accessory conducting pathways, interatrial block

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The cardiac conduction system (CCS)[1,2] is undoubtedly an important focus for all those involved in cardiac care. The sinoatrial (SA) and atrioventicular (AV) nodes along with the Bundle of His branches and Purkinje fibers that make up the bulk of the CCS's highly specialized cardiac tissue groups are responsible for the propagation and rapid conduction of electrical impulses through the heart in a controlled and systematic fashion.[1,2] Any abnormality in this network therefore can result in an arrhythmia. Thus, having a thorough understanding of the CCS is not only essential, but also crucial, especially when dealing with specific arrhythmias.

RIGHT ATRIAL CONDUCTING PATHWAYS

Mahaim fibers[3] and the internodal pathways,[4–7] often considered accessory pathways because they feed or branch off the main CCS, have been described since the discovery of the bundle by Kent in 1893.[6,7] However, because their existence is often disputed, owing to absence of discrete tracts, these pathways often only take precedence when normal AV conduction is impaired, such as during orthodromic tachycardias, AV block, and Wolff-Parkinson-White syndrome.[1,2,6,7] James,[4] and later Treux,[5] described 3 different internodal pathways that traverse the right atrium (RA) from the SA to the AV nodes—anterior, middle (sometimes known as the Bundle of Wenckebach)—and posterior (sometimes known as the Bundle of Thorel) (Fig. 1). Experimental studies by Eyster and Meek[8] demonstrated that these main internodal impulse conduits could be interrupted if surgical cuts were performed along these routes. Jongbloed et al[9] and Viragh and Challice[10] have also described similar communicating pathways in embryos. The anterior internodal pathway, which is the shortest of the 3, gives off a secondary branch as it emerges from the anterior lip of the SA node to form a trapezoid-shaped bundle of parallel fibers.[4,5,7] This secondary branch, first noted by Lewis et al[11] and later described by Bachmann[12] in 1916, courses along the superior quadrant of the interatrial sulcus from the RA to the left atrium (LA) and is today recognized as the Bachmann Bundle (BB) (Fig. 1).

MACROSCOPIC AND MICROSCOPIC ANATOMY

Lemery et al[13] studied dissected postmortem human heart specimens derived from patients without a cardiac disease history to show that the band-like BB was indeed structured from myocardial fibers that were easily demarcated from surrounding fat and connective tissue. The tract traverses the curvature of the atrial walls across the interatrial septal roof as it then tapers across to its LA insertion.[4,12] The median bundle measurements described in that series were 4 mm thickness ×9 mm height with upper and lower bundle lengths of 10 mm and 3 mm, respectively. This wider proximal dimension as compared with its distal counterpart in the LA and longer superior than inferior edges allows for its trapezoid shape as outlined distinctly by Lemery et al.[13] Such geometric measurements could be significant when considering ablation or pacing for atrial tachyarrhythmias. Microscopically, the fibers of this interatrial muscular bridge are sepa-

From the *Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), Veterans Affairs Boston Healthcare System, Boston, Massachusetts; the †Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts; the ‡Department of Medicine, Division of Cardiovascular Medicine, University of Massachusetts Medical School, Worcester, Massachusetts; and the §Department of Medicine, Saint Vincent Hospital, Worcester, Massachusetts.

Reprints: David H. Spodick, MD, DSc, Department of Medicine, Division of Cardiovascular Medicine, University Campus, University of Massachusetts Medical School, 55 Lake Avenue North, Worcester, MA 01655. E-mail: spodickd@ummhc.org.

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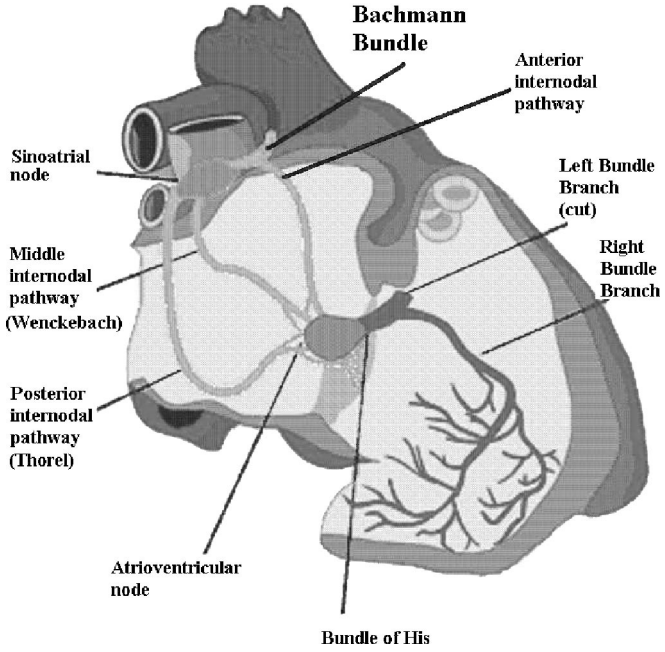


FIGURE 1. The Bachmann Bundle and the cardiac conduction system (modified from http://www.academy.de).

rate in orientation and perpendicular to the transverse atrial myocardial fibers. A more random, noncontiguous subendocardial BB fiber orientation, however, has been noted at the level of the interatrial septum and correlates with the line of functional delay of interatrial impulse conduction and subsequent increased fiber electrical capacity of the LA described later.[13,14]

CORONARY BLOOD SUPPLY

The right coronary artery (RCA), which supplies the SA node in 55% of cases, the posterior third of the septum, and the RA as well as the right ventricle, is thought to be the main blood supply to the internodal pathways.[4,15,16] Its clinically important branch, the SA nodal artery (that can also arise from the left circumflex artery in 45% of cases), is a key blood supply for the BB in humans. Smaller branches from the SA nodal artery fan out and facilitate vascularization of its watershed areas.[15,16]

Kugel's artery (a vascular tributary arising from either the proximal RCA or left coronary artery) courses in the plane of the AV valve ring along the anterior atrial wall behind the aorta to reach the atrial septum.[17] As it nears the junction of the ventricular septum, it then passes posteriorly to the crux of the heart to supply the posterior circulation. During this tortuous passage, it gives collaterals to the AV as well as SA nodal arteries, potentially contributing to BB vascularization.[16,17] In 50% of cases, the conus artery that originates from the RCA's sinus of Valsalva may also supply the proximal portion of the BB.[16,18] Branches from the "arc of Vieussens" that course superiorly toward the roof of the atria may also prove to be contributing vascular tributaries for the BB.[16]

INNERVATION

The sympathetic (arising from postganglionic cardiac fibers and thoracic cardiac nerves) and parasympathetic (arising from cardiac branches of the vagus nerve) nervous systems both play important roles in SA nodal stimulation.[1,2,7,19] Although, theoretically, impulse transmission along the BB could indeed fall under the influence of neurohormones such as epinephrine (through beta-adrenergic receptor binding) and acetylcholine (through muscarinic receptor binding),[19–22] such effects on the BB have been poorly studied.

Sensory input from baroreceptors (in the aorta and internal carotid arteries), chemoreceptors (in the aortic arch, carotid arteries, and medulla oblongata), proprioceptors, and higher brain centers (in the cerebral cortex, limbic system, and hypothalamus) also contribute to SA chronotropic effects. Such input may therefore also affect the rate of impulse relay along internodal pathways as well as the BB, more so in the presence of cardiac disease.[1,2,7,19]

DRUG EFFECTS

Caffeine (through cAMP breakdown), nicotine (through catecholamine stimulation), cocaine, tyramine, and certain medications that cause metabolic or adrenergic changes such as fluctuations in sodium or calcium often cause chronotropic effects on the SA node.[7,23–26] However, despite their known direct or indirect potential effects on interatrial activity, such factors targeting the BB specifically or as a systemic result of electrolyte alterations from metabolic disorders such as cirrhosis, uremia, or even mucopolysaccharide (lysosomal storage) diseases have not been adequately investigated. On the other hand, quinidin (and potentially other class Ia antiarrhythmics),[27] adenosine,[28] and certain antiarrhythmics as well as diuretics have been reported to affect interatrial conduction and imply BB involvement.[29] In one study of patients with chronic atrial fibrillation who had undergone direct current cardioversion, magnesium sulfate had been implicated to cause delayed interatrial conduction, whereas infusion of glucose, insulin, and potassium reversed these effects.[30] Such information may be of use during electrophysiological (EP) studies pertaining to interatrial conduction assessment and may have a role during atrial lead implantation or pacing in the future.

CLINICAL CORRELATES

Coronary artery disease and its risk factors such as diabetes mellitus and hypertension or vasculitides (proinflammatory conditions) such as rheumatologic, postinfectious, or autoimmune disorders may impair arterial circulation to the BB.[31,32] As such, smoking, hypercholesterolemia, obesity, and physical inactivity, in addition to increasing age and genetic predisposition, may contribute to atherosclerotic plaque formation and endothelial injury, harbingers for ischemia-mediated interatrial delay.[33] Interestingly, Myrianthefs et al[34] had shown that inclusion of P-wave durations $\geq$120 ms during exercise tolerance tests in addition to conventional criteria for diagnosing ischemia would increase its sensitivity from 57% to 75% while only decreasing specificity from 85% to 77%.[35]

Montereggi et al[36] had described changes in the P-wave morphology and duration in hyperthyroidism, whereas Oreto et al[37] reported P-wave artifactual effects of respiratory disease in electrocardiographic tracings that could mimic atrial dissociation. It is possible that any form of back pressure of blood into the atria producing atrial strain such as AV valvular disorders, congestive heart failure, and hypervolemia could cause prolonged conduction or unmask an already slowed impulse transmission in the BB. Stretch and pressure buildup on the superior portion of the atrial septum and the atrial roof where this interatrial conduction tract resides could alter function of the BB.[38–40] Therefore, amyloidosis and hypertrophic cardiomyopathy involving the septum, especially the superior part of its interatrial portion, can also produce similar interatrial conduction delay.[41]

Engelen et al reported a high-grade primary lymphomatous infiltration of the interatrial septum, causing interatrial conduction delay which improved after treatment with chemotherapy and radiation.[42] Atrial tissue sampling from patients with interatrial conduction delay has consistently shown intercellular fibrotic changes as well as intracellular metabolic inclusions causing ultrastructural disarray, particularly in the sarcomere and sarcoplasmic reticulum.[43]

INTERATRIAL CONDUCTION

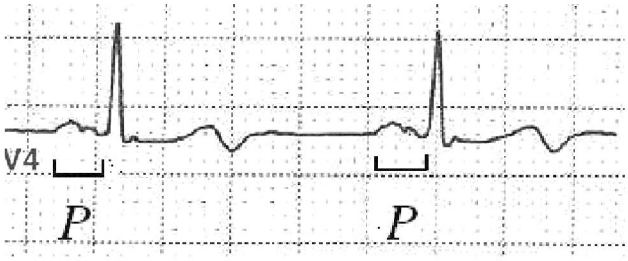
By positioning multiple electrode array catheters in the RA and LA, Lemery et al used a biatrial noncontact isopotential mapping system to demonstrate that atrial endocardial septal activations were indeed separate and asynchronous to each other.[44] Roithinger et al[45] used electroanatomic mapping to describe 3 potentially functional right-to-left atrial conducting pathways—BB projections at the coronary sinus region and transseptal fibers near the limbus of the fossa ovalis, whereas Markides et al[46] described interatrial conduction through posterior interatrial connections. However, because discrete lesions produced experimentally by Drury in 1925[47] and Cohen et al in 1965[48] along the BB had caused significant delay in LA depolarization during sinus rhythm, the BB has long been considered the predominant and preferred route for electrical impulse conduction from the RA to the LA during sinus rhythm[35] (Fig. 1). Harrild et al[49] used a 3-dimensional computer model of realistic anthropoid atrial geometry to simulate normal human atrial activation and found that the differently orientated septal BB fibers mentioned here forces a considerable impulse deceleration (to approximately 50 cm/s) first before transmission velocity regains momentum along the increased electrical capacity of the LA interatrial myofibers. Asynchronous atrial depolarization with such deceleration–acceleration of interatrial impulses produces the distinct P-wave morphology with discernible RA and LA components in normal human hearts.[7,14,35]

Interruption in the relay of interatrial impulses further separates the RA and LA components and is thus captured on the surface electrocardiogram (ECG) as prolonged, often notched, P waves.[35] Although normal duration of the P wave as classified by the World Health Organization/International Society and Federation of Cardiology Task Force is <110 ms,[50] P waves in BB conduction block are ≥ 110 ms and commonly bifid, depicting the lag in LA depolarization.[51,52] This has been described as interatrial block (IAB; 120 ms, which is also the mode duration, commonly cited for added specificity) and implies subsequent delayed LA contraction[35,47,48,51,52] (Fig. 2) (on the ECG, time = duration of conduction; excessive time or delay = block).

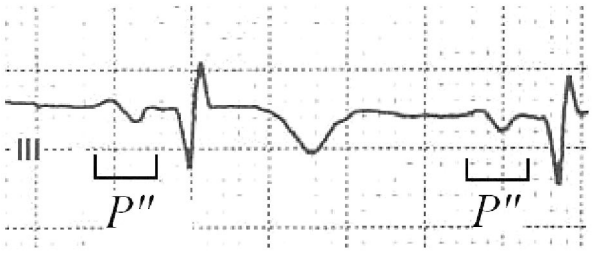
INTERATRIAL BLOCK

Recent observations in 2 separate but comparable general hospital populations of 1000 patients each have individually shown a $>40\%$ prevalence of IAB, especially in those >59 years.[35] This, along with the implication of IAB as a potent predictor for serious atrial tachyarrhythmias, mainly atrial fibrillation (AF),[35,53,54] has renewed interest in BB conduction abnormality. Prolongation of interatrial conduction is now believed to be the main operative mechanisms in AF. The antidromic reentrant circuit ultimately forces wavefront circulation in the region of the atrial isthmus between the annulus and the inferior vena cava playing critical importance in perpetuation of this arrhythmia.[1,2,7]

Jongbloed et al[9] were able to reproduce atrial tachyarrhythmias in embryos by adversely stimulating foci in and around the BB. The arrhythmogenic propensity of IAB for AF was demonstrated in a 16-month follow up of 308 age- and gender-matched patients in which its prevalence was



A Partial block of the Bachmann Bundle



B Advanced block of the Bachmann Bundle

FIGURE 2. Interatrial block occurring with delayed Bachmann Bundle conduction. (A) In partial interatrial block (IAB), impulses continue to pass from the right atrium (RA) to the left atrium (LA) through the Bachmann Bundle (BB) but are delayed (≥ 110 ms). (B) In advanced IAB, overwhelming delay of impulse conduction across the blocked BB forces impulses in the RA to be directed toward the atrioventricular node first before progressing caudocephalically to depolarize the LA. This results in biphasic ($\pm$) P waves (≥ 110 ms) in leads II, III, and aVF.

52% among those who subsequently developed AF compared with 18% among controls in sinus rhythm ($P < 0.0001$).[54] IAB is also a specific marker for LA enlargement (LAE; in >88% of patients with IAB) in which 2-dimensional long axis diameter has been shown to be significantly correlated with its P-wave duration (LA size [mm] = 2.47 + 0.29 [P-wave duration {ms}]).[39] However, delay in interatrial conduction can in fact occur independent of increase in atrial size.[39] In a series of patients matched for LA size, patients with IAB had lower LA emptying fraction (8.5%; $P < 0.0001$), LA stroke volume (17.3 mL; $P < 0.0001$), and LA kinetic energy (19.8 Kdyne/cm/s; $P < 0.0001$).[52] Such associations of a weak and enlarged LA certainly heighten the risk of IAB for LA thrombosis and subsequent arterial embolism.[52,55] Although minimal, secondary effects from delayed left ventricular (LV) active filling and reduced atrial "kick" (atriogenic LV preload) in IAB[52] may also precipitate heart failure, especially when acute and severe or when LV ejection fraction is severely impaired, ie, after a massive myocardial infarction. Furthermore, if sufficiently delayed, mistimed ineffective LA contraction could occur against a closing mitral valve, further contributing to changes in cardiovascular equilibrium. Nevertheless, IAB remains largely underappreciated and often mislabeled as an indolent, insignificant epiphenomenon on the ECG.[56]

THE BACHMANN BUNDLE AND PHARMACOLOGIC THERAPY

Although potential causes and risk factors for IAB have yet to be adequately studied, owing to the foregoing associations, IAB has spawned several investigations aimed at correcting electrical impulse delay between the atria, primarily through the BB. Zaman et al[57] and Madrid et al[58] studied the significance of angiotensin-converting enzyme inhibitors (ACEIs) in AF and prolonged P-wave durations with promising preliminary findings. Indeed, the causal role of hypertension in worsening BB conduction and its effects on renin–angiotensin–aldosterone pathways may be multifarious. However, these studies raise questions whether ACEIs may have a direct effect on the BB by either altering its refractoriness, possibly through suppression of atrial fibrosis by cytokine modulation,[59] or through unloading of a pressure- and stretch-overloaded atria.[38] Thus, it is imperative to keep in mind that use of medications like ACEIs[57,58] and certain antiarrhythmics[29] could potentially have a double-pronged effect of treating both the potential cause, IAB, and also the potential sequela, AF. Besides lowering the risk of CAD, 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors, or commonly "statins," have also shown some promise of countering the tumor necrosis factor alpha-induced proinflammatory effects of matrix-degrading metalloproteinases in atrial fibrosis.[60]

THE BACHMANN BUNDLE AND NONPHARMACOLOGIC THERAPY

Investigators have studied pacing during interatrial conduction delay as an adjunct to atrial tachyarrhythmia treatment through controlled and multicenter trials.[61–63] It has been demonstrated that prolonged P-wave duration could indeed be corrected and that RA and LA activation times could be coupled closer to human physiological state with pacing.[61] Moreover, several investigations have been done on the actual location of atrial pacing, direct impulse delivery performed at the atrial appendage or along the BB. Atrial pacing at the BB site has shown considerable improvement in the frequency of sinus rhythm among patients with recurrent paroxysmal AF (75% vs. 47% survival free from AF at 2 years; $P < 0.01$) compared with RA appendage pacing.[61] Also, BB pacing significantly improved IAB (123 ± 21 ms from 132 ± 21 ms), whereas RA appendage pacing paradoxically worsened such delay 2 weeks postimplantation (148 ± 23 ms from 123 ± 23 ms). Similarly, synchronous biatrial pacing ("dual-site"; one atrial lead placed in the high right atrium and the other into the mid or distal portion of the coronary sinus) has also been shown to moderately reduce P-wave duration (187 ± 30 ms to 106 ± 14 ms) among patients with histories of recurrent drug-refractory atrial tachyarrhythmias.[62] However, the SYNBIAPACE (SYNchronous BIAtrial PACing) study that ultimately compared the various modes of atrial pacing, ie, standard RA "dual-chamber pacing, sensing, and response" (DDD; at 70 beats/min), backup demand DDD pacing (programmed at 40-beats/min), and synchronous biatrial pacing (at 70 beats/min), failed to show significant benefit among these modes with respect to time of first AF recurrence.[63] Although the benefits of pacing as secondary prevention for prolonged BB conduction and possible subsequent atrial tachyarrhythmias are indeed promising, the absence of clear predictors of which patients would benefit most greatly necessitates further investigation.

SUMMARY

The BB, often perceived as an accessory impulse-conducting pathway, is truly a fundamental unit of the CCS.[7,12] It is responsible for the uninterrupted conduction of SA node-generated impulses from the RA to the LA to synchronize atrial activation.[7,45,46] Thus, abnormality of this preferred interatrial bundle of collimated fibers results in delay of interatrial conduction. Such delay is known as IAB and is depicted on the ECG as prolonged, often notched P waves (≥110 ms).[7,35,47,48] IAB is highly prevalent[35] and is associated with the pathogenesis of atrial tachyarrhythmias, especially AF,[35,53,54] LAE,[39] LA electromechanical dysfunction,[52] and possibly embolism.[55] Therefore, a thorough understanding of the CCS and the BB cannot be overemphasized and is indeed essential for optimal patient care. Knowledge of the coronary blood supply and innervation of the BB as well as the mediators that dynamically affect its function can help medical caregivers risk-stratify patients and anticipate the associated sequelae of IAB.[35] The clinical magnitude of such sequelae certainly justifies further investigation in both prevention and management of BB abnormality such as currently studied use of ACEI and antiarrhythmic therapy[29,57,58] as well as atrial pacing techniques.[61–63]

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