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ScienceDirect

Journal of Electrocardiology 48 (2015) 380 – 384

JOURNAL OF Electrocardiology

www.jecgonline.com

Review

Electrocardiographic right and left bundle branch block patterns in athletes: Prevalence, pathology, and clinical significance☆

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Abstract Differentiating benign electrocardiographic (ECG) patterns in athletes from those representative of underlying cardiac pathology is both clinically relevant and challenging. Complete right (RBBB) and left (LBBB) bundle branch block are relatively rare in asymptomatic athletic populations, and current expert consensus guidelines recommend further clinical investigation upon detection of either ECG pattern. However, present data suggest that typical RBBB is not associated with structural cardiac pathology and may alternatively represent an ECG marker of exercise-induced right ventricular remodeling. In accordance with current guidelines, the presence of asymptomatic LBBB in athletes is not associated with normal exercise physiology and more likely indicative of underlying cardiac pathology. While long-term outcomes for asymptomatic athletes with RBBB or LBBB remain unknown, current evidence regarding these ECG patterns should be considered to improve the specificity of future athlete-specific ECG interpretation guidelines.

Keywords: Bundle branch block; Athlete; ECG; Screening

Introduction

Tragically, the first manifestation of cardiac disease in athletes is often sudden cardiac death (SCD). The 12-lead electrocardiogram (ECG) may enhance the ability to detect occult structural and electrical cardiac pathology in athletes. Accordingly, various governing bodies within sport and the European Society of Cardiology have recommended mandatory utilization of 12-lead ECG during the pre-participation evaluation of young athletes [1–3].

Accurately differentiating benign ECG patterns from those indicative of underlying cardiac pathology in athletes may be challenging. Despite several expert consensus documents carefully outlining "training related" versus "training unrelated" athletic ECG patterns [2,4,5], false positive rates, defined as the absence of direct ECG correlation with underlying structural cardiac pathology, remain between 4% and 15% [4–7]. While the American Heart Association endorses a targeted pre-participation evaluation comprised of only medical history and physical examination [8], a significant number of US professional sporting teams and universities employ ECG-inclusive pre-participation protocols. In addition, a growing number of private organizations offer screening ECGs to community-based athletes. As such, refining the specificity and decreasing the rate of false positive ECG interpretations in athletes remains a high priority.

Recent data have advanced our understanding of what constitutes both adaptive ECG patterns and those that are unrelated to athletic training [9], and therefore emphasize the need for critical reappraisal of contemporary athlete ECG interpretation criteria. Currently, the specific ECG patterns of complete right bundle branch block (RBBB) and left bundle branch block (LBBB) in athletes are considered training unrelated patterns that warrant further clinical investigation [2,4,5]. This review provides an overview of the anatomy and physiology of bundle branch block and an up-to-date summary of the data evaluating both RBBB and LBBB in athletes and their differential relationships with underlying cardiac pathology.

Anatomy and physiology of the bundles

In the early 1900s, Eppinger and Tothberger first discovered the classic bundle branch block ECG patterns upon injecting canine myocardium with silver nitrate [10]. At present, the electrogenesis of RBBB and LBBB continue

☆ Disclosures: The authors have no relevant disclosures or conflicts of interest to report.

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http://dx.doi.org/10.1016/j.jelectrocard.2015.03.015
0022-0736/

to be further elucidated in humans. Anatomically, it is known that electrical conduction originates from the sinoatrial (SA) node, spreads through both atria and specialized tissue at the atrioventricular (AV) junction (including the AV node and His bundle), and then separates into the bundle branches activating both the right (RV) and left ventricle (LV). The bundle that supplies the LV further separates into a narrow anterior fascicle, broad posterior fascicle, and a third septal segment that originates from smaller branches from each of the fascicles [11]. Ultimately, conduction spreads within the ventricular myocardium via specialized Purkinje fibers.

Normal electrical activation of the ventricles originates from the left side of the interventricular septum, which under normal physiology manifests as a small septal r wave in lead V_1 and Q-wave in lead V_6. Propagation of this stimulus then travels through both ventricles with the LV maintaining electrical predominance on surface recordings of electrical conduction. In RBBB, only the third sequence of ventricular depolarization (RV depolarization) is delayed with septal and LV activation unaffected. In the presence of LBBB, ventricular depolarization initiates on the right side of the interventricular septum with subsequent delayed activation of the LV. Although the exact electrical activation sequence in LBBB is highly complex with variability in the location and length of block, LV depolarization propagates in a non-homogeneous and dyssynchronous fashion, previously described as a "U-shaped" activation pattern [12].

The disruption in the conduction system imparted by both RBBB and LBBB manifests on the 12-lead ECG. The diagnosis of RBBB is established based on the following findings: (1) QRS duration >120 ms in the presence of normal sinus or other supraventricular rhythm, (2) R-wave or RSR′ complex in lead V_1, and (3) an R-complex with a prolonged, shallow S-wave in lead V_5, V_6, aVL or I [13]. Incomplete right bundle branch block (IRBBB) is defined by a QRS duration <120 ms and an R′ or r′ wave in either lead V_1 or V_2[13].

The benefits of cardiac resynchronization therapy in clinical heart failure management have led to renewed interest in defining the exact electrical activation patterns of morphologic LBBB [14]. Consequently, controversy has arisen in accurately defining electrocardiographic LBBB [14]. Although several criteria in the literature now exist [14], previous studies focused on athletic ECG interpretation of LBBB have utilized: (1) QRS-complex duration >120 ms in the presence of normal sinus or other supraventricular rhythm, (2) QS- or RS-complex in lead V_1, (3) broad or notched R-waves in leads V_5 and V_6, or an RS pattern, and (4) the absence of Q-wave in lead V_5, V_6 or I [13]. Incomplete left bundle branch block is defined by a QRS duration ≥100 ms and <120 ms in leads I, aVL, and V_5 or V_6[13].

Right bundle branch block

Prevalence

Although mostly cross-sectional in design, there have been many studies evaluating the presence of ECG abnormalities in athletes [7,15–27]. It has been well established that IRBBB is common, particularly among endurance athletes [21]. More recent studies inclusive of large cohorts of athletes have demonstrated IRBBB in 10–20% of athletes [16,20]. In the largest observational cohort of 32,652 amateur Italian athletes, Pelliccia *et al.* reported that IRBBB was present in 7% of the cohort [28].

Complete RBBB is far less common and has a reported incidence ranging from 0.2% to 3% of athletes [7,15–27]. Compared to healthy members of the general population, the prevalence of RBBB in athletes appears increased. In the largest study evaluating the prevalence and outcomes of those in the general population with RBBB and free of cardiovascular disease, Bussink *et al.* reported that RBBB was present in approximately 1% of a cohort of 18,441 subjects. On closer inspection, in the 1866 subjects who were 30 years or younger, RBBB was observed in approximately 0.5% [29].

Significance

RBBB in endurance athletes may be a marker of physiologic adaptations that accompany the repetitive volume challenge inherent in isotonic/dynamic exercise. We conducted a study analyzing 510 collegiate, primarily endurance, athletes with both 12-lead ECG and 2-D echocardiography with Doppler and found that athletes with IRBBB and RBBB demonstrated increased biventricular chamber dimensions, relative reductions in RV systolic function, and interventricular dyssynchrony compared to those athletes with normal QRS duration [21]. Moreover, the degree of change in RV size and function correlated with increasing QRS duration and previously quantified exercise exposure. It is therefore plausible that exercise-induced RV enlargement may lead to stretching of RV Purkinje fibers, resultant delayed RV depolarization, and the development of RBBB as an ECG marker of physiologic right ventricular adaptation. Although recent data have challenged asymptomatic RBBB as a benign finding in the general population [29], prior evidence has supported that asymptomatic RBBB is not associated with future cardiovascular events and does not require further diagnostic evaluation [30,31]. Specific to athletes, McClaskey *et al.* recently reviewed prior longitudinal studies (1966 to present) of outcomes among athletes with ECG abnormalities [32]. Although adverse cardiovascular outcomes were noted in two studies inclusive of athletes with bundle branch block (combined total of 279 athletes at longitudinal mean follow-up of 5 years; 4 athletes with angina and 1 with acute myocardial infarction) [33,34], it was not specified in either study if the respective adverse outcomes occurred in the athletes with bundle branch block [33,34].

Pathologic correlations

In the absence of clinical symptoms or associated right precordial repolarization abnormalities suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC), the presence of IRBBB should not be considered representative of underlying cardiac pathology [29]. No previous work examining asymptomatic athletes with RBBB has