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Contemporary Review of Left Bundle Branch Block in the Failing Heart – Pathogenesis, Prognosis, and Therapy

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Cardiac resynchronisation therapy (CRT) is a cornerstone in the contemporary management of heart failure. The most effective way of predicting response to this therapy remains electrocardiographic (ECG) criteria of electromechanical dyssynchrony. The left bundle branch block (LBBB) pattern is currently the most robust ECG criterion in predicting improvement in symptoms and reduction in mortality. However, recent studies using three-dimensional (3D) mapping and cardiac magnetic resonance imaging (CMR) have demonstrated heterogeneous left ventricular activation patterns in patients with LBBB. This has led to intense debate on the activation pattern of "true LBBB" and resulted in the proposal of stricter criteria for defining LBBB. This review will focus on the definitions and implications of LBBB in the CRT era. At a minimum, the use of stricter ECG criteria appears warranted, and adjunctive pre-implant imaging or mapping may further identify patient-specific electrophysiological patterns that determine response to CRT.

Keywords Left bundle branch block • Heart failure • Cardiac resynchronisation therapy

Introduction

Left bundle branch block (LBBB) occurs frequently in the context of cardiomyopathy of various aetiologies [1,2]. It is thought to be secondary to ventricular remodelling from congestive heart failure and/or fibrosis of the conduction system [3]. Left bundle branch block results in dyssynchronous electrical activation of the ventricles [4] and consequently inefficient contraction of the left ventricular failure which often correlates with further progression of the conduction abnormality as well [7,8]. This process may be disrupted by treatment such as cardiac resynchronisation therapy (CRT).

Cardiac resynchronisation therapy has become one of the cornerstones in the management of heart failure. Recent

studies have focussed on predicting response to CRT and these studies have shaped the guidelines for prescribing this therapy [9]. Electrocardiographic (ECG) criteria of electromechanical dyssynchrony have remained the most effective in predicting benefit of CRT [4]. The left bundle branch block (LBBB) pattern is currently the most robust ECG criterion in predicting improvement in symptoms and reduction in mortality [4].

However, recent evidence, using three-dimensional (3D) mapping and cardiac magnetic resonance imaging (CMR), has demonstrated heterogeneous left ventricular activation patterns in patients with LBBB [10,11]. This had lead to stricter criteria for definition of LBBB being recommended [12]. This review will focus on the definitions and implications of LBBB in the CRT era.

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Anatomical and Electrocardiographic Considerations

The electrical impulse from the bundle of His is carried through a conduction network which has three divisions. The right bundle branch courses through the subendocardium on the right side of the interventricular septum and terminates in the Purkinje fibres of the right ventricle [13]. The left bundle branch tracks subendocardially on the left side of the septum and is composed of multiple fascicles grouped into the antero-superior and postero-inferior divisions [13]. The third bundle is the medial division, which supplies the mid-septal area. It can originate from the main left bundle branch or from one of the fascicles and is extensively interconnected with them [13].

In left bundle branch block, the activation of the left ventricle is altered leading to delay in the depolarisation of the left ventricle. Firstly, the activation of the intraventricular septum commences on the right, then propagates inferiorly, to the left and slightly anteriorly [14]. Delay in intrinsic LV activation contributes to LBBB. Early electrical mapping studies in patients with LBBB and heart failure demonstrated that left ventricular activation often occurs in a "U" shaped wavefront which travels around the apex and across the inferior wall [15]. This pathway of activation is due to a line of functional block from the base to the apex of the left ventricle, which may explain the presence of pronounced notching on the ECG of some patients with LBBB and not others (Figure 1). The location of functional block is variable and may occur in the anterior, lateral or inferior walls.

The electrocardiogram in left bundle branch block has been debated over the past century. Carter presented the first series of electrograms in patients with bundle branch block in 1914 but incorrectly switched the diagnosis of right bundle branch block (RBBB) and LBBB [12]. Wilson and Hermann



Figure 1 Left ventricular electrical activation sequence in many patients with left bundle branch block. Activation occurs in a "U" shaped wavefront around the apex due to a line of functional block from the base to the apex. This may explain the presence of pronounced notching on the ECG.

Abbreviation: ECG = electrocardiograph.

presented the first criteria for bundle branch block and observed that the QRS was prolonged (usually much more than 100 ms) and that there was frequent notching within the QRS complex [12]. The diagnosis of RBBB and LBBB continued to be switched until Barker et al. performed electrical stimulation of the epicardium of the human heart in 1930. After precordial leads became widely available, in 1941, Wilson proposed more detailed definitions of bundle branch block using a dog model. He used the QRS duration \geq 120 ms to distinguish complete bundle branch block from incomplete bundle branch block and this continues to be used currently [12].

In 1985, the ECG criteria for intraventricular conduction disturbances and ventricular pre-excitation were reviewed by an ad hoc working group established by the World Health Organization and the International Society and Federation of Cardiology [13]. In 2009, the definition of LBBB was refined by the American Heart Association (AHA), American College of Cardiology Foundation (ACCF) and the Heart Rhythm Society (HRS) [16]. The definition of LBBB according to their recommendations is detailed in Table 1. Specifically, complete LBBB in adults is defined as: QRS duration ≥120 ms; broad notched or slurred R wave in leads I, aVL, V₅ and V₆ and an occasional RS pattern in V₅ and V₆ attributed to displaced transition of QRS complex; absent q waves in leads I, V_5 and V_6 but in the lead aVL, a narrow q wave may be present in the absence of myocardial pathology; R peak time >60 ms in leads V₅ and V₆ but normal in leads V₁, V₂ and V₃, when small initial r waves can be discerned in the above leads; and ST and T waves usually opposite in direction to QRS. The appearance of LBBB may change the mean QRS axis in the frontal plane to the right, to the left, or superiorly, in some cases in a rate-dependent manner [16].

The initial criteria for complete bundle branch block being QRS duration of \geq 120 ms was partly based on observations in canine models. It was also somewhat pragmatic in that it measured 3 mm on the ECG at the conventional paper speed of 25 mm/s [13]. However, the authors stated that the QRS in LBBB usually exceeds 140 ms in most patients with complete bundle branch block. Recently, since the advent of CRT, more attention has been placed on the definition of LBBB. Stricter criteria have been proposed since insights from studies using electrical mapping in the human heart have shown that electrical activation through the intraventricular septum in LBBB requires a minimum of 40 ms [12]. In addition, a proportion of patients with left ventricular hypertrophy and left anterior fascicular block, fulfil the electrocardiographic criteria for LBBB, despite not having true LBBB [15]. It has, therefore, been suggested that the definition for LBBB should include a QRS duration \geq 140 ms in men and \geq 130 ms in women and QRS notching or slurring in two or more contiguous leads of V₁, V₂, V₅, V₆, I and aVL [12].

Nevertheless, the 2009 AHA/ACCF/HRS remains the most commonly used definition for LBBB in clinical practice. The same definition for LBBB was also adopted as a class 1 indication for CRT implantation in the 2012 AHA/ACCF/HRS device implantation guidelines, with an additional

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