

Left is worse than right: the outcome of bundle branch block in middle-aged men

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Online publish-ahead-of-print 7 October 2005

This editorial refers to ‘Bundle branch block in middle-aged men—risk of complications and death over 28 years. The primary prevention study in Göteborg, Sweden’[†] by P. Eriksson *et al.*, on page 2300

As opposed to right bundle branch block, left bundle branch block has been associated with organic heart diseases caused by high blood pressure, coronary artery disease, aortic valve stenosis, and cardiomyopathy since its first description.¹ It is also known that degeneration of the specific conduction system increases with advancing age, resulting in a rise of the prevalence of bundle branch block in older individuals. In contrast, current imaging techniques and ischaemia detection sometimes rule out virtually any cardiac abnormality except abnormal regional left ventricular (LV) wall motion, specifically of the interventricular septum. Recently, resynchronization therapy with biventricular pacing has refreshed our interest in bundle branch block patterns. It has been shown that reduction of left intraventricular conduction delay with biventricular pacing contributes to improved functioning of heart failure patients by ameliorating several systolic function parameters. Despite this new knowledge, our insight into the pathophysiological relationship between left bundle branch block and organic heart disease remains superficial. For example, it is unknown whether LV dysfunction precedes left bundle branch block or whether the reversed course is the case.² Long-term prospective observations can support our insight into this pathophysiological relationship.

The long-term follow-up (>25 years) of a large cohort of Swedish males of 45 years or more without major cardiovascular history at the time of baseline investigations has been reported.³ This prospective study demonstrated that men with left bundle branch block showed both a much higher risk for developing high degree atrioventricular block and a markedly higher hazard ratio for all-cause mortality than individuals with right bundle branch block. Surprisingly, men with a left bundle branch block had high risk to die from fatal out-of-hospital arrhythmias. This information confirms our appreciation of the significance of a bundle

branch block: the left one has to be characterized as an unfavourable marker for the development of cardiac diseases, whereas the right one appears more innocent.

One may question whether this study extends our knowledge of the pathophysiological relationship between bundle branch block and cardiac disease, in particular, myocardial dysfunction. Because this epidemiological study was started in 1970, one cannot expect that echocardiographic baseline data were already collected. Therefore, the important relationship between bundle branch block or delayed ventricular conduction and LV function could not be examined. However, the study shows an increased risk of heart failure in the presence of left bundle branch block. Unfortunately, the contribution of the frontal electrical axis in bundle branch block, in terms of anterior or posterior hemiblock, to these late outcomes could not be addressed because of the small number of individuals with various axis patterns.

The fact that bundle branch block progresses to high degree atrioventricular block requiring chronic pacing and that bundle branch block is strongly related to the development of aortic valve stenosis will not astonish any clinician. Progressive degeneration of the specific conduction system with fibrosis and scarring in the atrioventricular region is the underlying pathology of this development. However, it is striking that the risk for atrioventricular block was much more pronounced in left block than in right bundle branch block. Although this finding disagrees with previous observations of progression of bundle branch block to atrioventricular block,⁴ this very long observation of Swedish middle-aged men might alter our concept of the natural course of bundle branch block and its progression to atrioventricular conduction block.

Finally, this study also showed that the presence of bundle branch block was not related to future risk of non-fatal acute myocardial infarction (MI), and as the authors suppose, coronary artery disease does not contribute to the pathogenesis of bundle branch block. Their theory requires further studies, because in clinical cardiology the presence of a bundle branch block in acute MI heralds an unfavourable outcome. This condition reflects a large infarction, including the septal area and frequently a low LV ejection fraction with poor outcome. In other words, coronary artery disease can produce bundle branch block by damaging myocardial tissue. The question arises whether a proximal bundle branch block or a more diffuse conduction disturbance is operational in scarred areas.

The opinions expressed in this article are not necessarily those of the Editors of the *European Heart Journal* or of the European Society of Cardiology.

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[†] doi:10.1093/eurheartj/ehi580

Will this information affect our policy in the individual middle-aged man without cardiovascular symptoms in whom accidentally a bundle branch block is recorded, for example, at the examination for a driving license? This study does not urge to consider preventive pacing measures to avoid atrioventricular block. It has to be kept in mind that paced patients do not have a lower risk for sudden death, because it is the degree of the LV systolic dysfunction that eventually determines the prognosis. Although in this study the hazard for fatal ventricular arrhythmias in the setting of acute myocardial ischaemia and infarction was clearly enlarged in middle-aged men in whom left bundle branch block was recorded, this study does not permit to consider preventive ICD implantation in those individuals. Identification of the precise causes of death in this condition is extremely difficult, specifically in a retrospective manner, as was done in this epidemiological study. Of more importance, of all currently available markers for freedom of arrhythmic death in the ischaemic patient, the LV function is the most crucial one.

The major contribution of this large and long epidemiological study, deserving our admiration, to clinical cardiology is the confirmation that left bundle branch block heralds a much more unfavourable cardiovascular prognosis than the right one. Because the risk of developing heart failure was

three-fold higher in left bundle branch block than in right bundle branch block, careful examination at the time of recording of left bundle branch block and regular follow-up visits are warranted. It offers the opportunity to follow the course of the relationship between the ventricular conduction delay and the possible mechanical LV asynchrony and timely detection of the onset of systolic heart failure. If right bundle block in an asymptomatic middle-aged man is detected, a one-time examination might suffice to rule out cardiovascular abnormalities. Whether such policy applies to middle-aged women with bundle branch block needs further studies.

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