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EDITORIAL

Right bundle branch block: are we looking in the right direction?

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This editorial refers to 'Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study'[†], by B.E. Bussink *et al.*, on page 138

The right bundle branch is a long, thin, and discrete structure composed of high-velocity conduction Purkinje fibres. It is located in the right side of the interventricular septum and occupies a subendocardial position in its superior and inferior thirds and deeper in the middle third. There are no ramifications in most of its course, but it starts to branch as it reaches the base of the anterior papillary muscle. The appearance of a right bundle branch block (RBBB) alters the ventricular activation sequence, produces a QRS prolongation, and changes the orientation for R- and S-wave vectors, thus generating a typical electrocardiogram (ECG) pattern (*Figure 1*).

The prevalence of RBBB in the general population is estimated at between 0.2% and 0.8%, and it clearly increases with age.[1] It may be associated with different cardiac structural diseases such as ischaemic heart disease, myocarditis, hypertension, congenital heart disease, cor pulmonale, and pulmonary embolism. Its prognosis depends on the type and severity of the associated heart condition; for example, in patients with ischaemic heart disease the presence of RBBB is a well-established mortality predictor.[2–4] The same is true for patients with heart failure where at least two different studies showed a worse prognosis for patients with RBBB hospitalized with this condition.[5,6]

Nevertheless, all previously published data suggest an excellent prognosis in patients free of heart disease. Previous studies of athletes and aeroplane pilots with long follow-up show a favourable prognosis with a very low rate of cardiovascular events or indication for pacemaker implantation.[7–9]

Several epidemiological studies analysed the prognosis of RBBB in individuals without heart disease. The Reykjavik Study found 126 cases of RBBB in 9135 males and 67 cases in 9627 females, with a greater incidence with increasing age.[10] A higher mortality from heart disease ($P < 0.01$) was found in men with RBBB compared with the control population, but this difference was not significant when risk factors of heart disease were taken into account by multivariate Cox analysis.

In 1996 Fahy *et al.* published a 9.5-year follow-up study of 310 healthy individuals with RBBB that were identified from 110 000 participants in a cardiovascular screening programme.[11] Isolated RBBB was more prevalent than isolated left bundle branch block (LBBB) (0.18% vs. 0.1%, $P < 0.001$), and the prevalence of both abnormalities increased with age ($P < 0.001$). Survival was no different for those with LBBB or RBBB. However, the prevalence of cardiovascular disease and cardiac mortality was greater in the LBBB group ($P = 0.01$).

A Swedish study monitored 855 patients who were 50 years old in 1963 for 30 years. The prevalence of BBB increased from 1% at 50 years of age to 17% at 80 years, resulting in a cumulative incidence of 18%.[1] There was no significant relationship between BBB and the development of ischaemic heart disease, and no significant increase in mortality during follow-up.

In a community-based study (Olmsted County), 706 RBBB patients were identified from a population of 123 700 individuals.[12] Of those, 12% had LBBB with left axis deviation (LAD); 20% had LBBB without LAD; 26% had left anterior hemiblock; and 42% had RBBB. At 9-year follow-up, the presence of RBBB did not alter the prognosis.

The most recent study is a Finnish study that evaluated the 12-lead ECGs of 10 899 Finnish middle-aged subjects from the general population (52% were men; mean age 44 $\pm$ 8.5 years) and followed them for 30 $\pm$ 11 years.[13] A prolonged QRS duration was defined as QRS $\geq$ 110 ms and an intraventricular conduction delay as QRS $\geq$ 110 ms, without the criteria of complete or incomplete BBB. Prolonged QRS duration predicted all-cause mortality [relative risk (RR) 1.48; 95% confidence interval (CI) 1.22–1.81; $P < 0.001$], cardiac mortality (RR 1.94; 95% CI 1.44–2.63; $P < 0.001$), and sudden arrhythmic death (RR 2.14; 95% CI 1.38–3.33; $P = 0.002$). LBBB also predicted arrhythmic death ($P = 0.04$), but RBBB was not associated with increased cardiovascular or all-cause mortality.

Based on these data, the position generally accepted is that individuals with isolated, chronic RBBB that are asymptomatic do not

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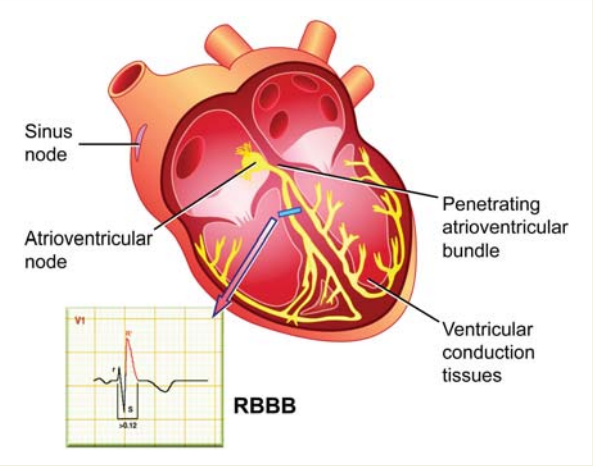

Figure 1 Right bundle branch block produces a change in the normal activation of the heart.

require further diagnostic evaluation or implantation of a pacemaker or any other specific therapy.

Bussink *et al*. have now completed the largest study of the prevalence and prognosis of RBBB in the general population.[14] They conduct a 20.5-year follow-up of 18 441 participants included in the Copenhagen City Heart Study examined between 1976 and 2003, all free from previous myocardial infarction, chronic heart failure, and LBBB. They found a greater prevalence of complete RBBB and incomplete RBBB in males than in females (1.4%/4.7% in men vs. 0.5%/2.3% in women, $P < 0.001$) and, in contrast to previous studies, the presence of RBBB was associated with significantly increased all-cause and cardiovascular mortality in both genders, with age-adjusted hazard ratios (HRs) of 1.31 (95% CI 1.11–1.54) and 1.87 (95% CI 1.48–2.36). RBBB was also associated with a significantly greater rate of myocardial infarction (HR 1.67; 95% CI 1.16–2.42) and pacemaker implantation (HR 2.17; 95% CI 1.22–3.86). On the other hand, the incidence of chronic heart failure (HR 1.37; 95% CI 0.96–1.94), atrial fibrillation (HR 1.10; 95% CI 0.73–1.67), or chronic obstructive pulmonary disease (HR 0.99; 95% CI 0.60–1.62) was not different for the RBBB group when compared with normal individuals. In accordance with previous studies, the presence of incomplete RBBB was not associated with any adverse outcome.

This is a very solid study as the National Danish Registry has been validated in several previous studies[15–18] where it was proven to be a robust tool for epidemiological research. Its methodology is very accurate and it must be emphasized that spirometry is performed in each follow-up visit and the ECG tracings are classified by the Minnesota Code Classification System for Electrocardiographic Findings. Follow-up was almost complete, with only loss to follow-up due to emigration (<0.5%). These aspects give a strong consistency to the results so this study might change the paradigm of RBBB benignancy in individuals without heart disease.

Nevertheless, this study does not clarify all doubts and may open up a series of new questions without an outright answer. The first one is the large difference in the prevalence of complete RBBB and incomplete RBBB between men and women (1.4%/4.7% vs. 0.5%/2.3%, $P < 0.001$). This difference cannot be explained by a different prevalence of cardiovascular risk factors or any other clinical parameter analysed.

Also there is a significant association between RBBB and myocardial infarction in women (HR 2.79; 95% CI 1.50–5.22), but not in men (HR 1.37; 95% CI 0.87–2.16). Although this difference is statistically significant (*P*-value for interaction 0.01), the authors ascribe it to chance. This may be true, but it still is an intriguing finding.

Also, it is not clear by which mechanism RBBB confers a worse prognosis, especially since this association seems stronger in younger patients.

Our final conclusion is that after publication of this study we no can longer underestimate the presence of RBBB in the asymptomatic individual. It is unclear how we should change our clinical practice. Finding a predictor of greater risk during follow-up does not imply that we can perform a medical intervention able to diminish that risk. The current 2010 ACCF/AHA Guidelines for Assessment of Cardiovascular Risk in Asymptomatic Adults recommend a resting ECG for cardiovascular risk assessment in asymptomatic adults with hypertension or diabetes[19] (level of evidence: C). In asymptomatic adults without hypertension or diabetes, the recommendation is IIb (level of evidence: C). Until we encounter new evidence on how to manage the asymptomatic patient with RBBB, we should, as the authors of this study suggest, be alert to the patient's cardiovascular risk factors.

Conflict of interest: none declared.

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CARDIOVASCULAR FLASHLIGHT

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Winding without twiddling of a pacemaker wire

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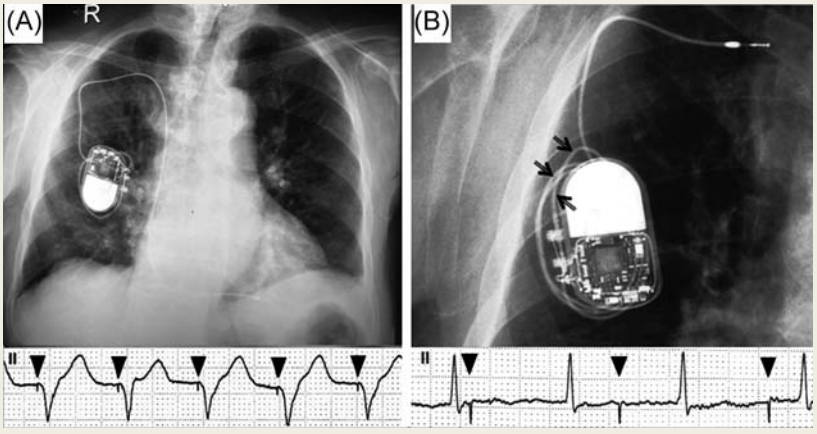

A 82-year-old obese woman with a history of permanent atrial fibrillation with slow ventricular response and syncope underwent pacemaker implantation using a single passive-fixation lead. Before discharge, a chest X-ray revealed a pacemaker lead placed in the right ventricular apical region, and an ECG recording showed a proper function of the pacemaker (*Panel A*—arrowheads indicate pacemaker artefacts).

Three months later, the patient was readmitted to the hospital for dizziness and near-syncope episodes. As shown in *Panel B*, in the chest X-ray detail, a displacement of the ventricular lead into the right subclavian vein along with three windings of the lead around the pulse generator was noticed (arrows). Moreover, the ECG recording disclosed pacemaker dysfunction with complete undersensing and pacing failure (arrowheads). Pacemaker twiddler's syndrome (i.e. pacemaker malfunction due to the patient's conscious or unconscious manipulation of the pulse generator) was taken into consideration, but the patient and its relatives denied any manipulation of the device. Instead, since the patient had congenital hip luxation and used a stable aluminium orthopaedic walker, the repetitive rotational movement of the shoulders could have contributed to a spontaneous lead dislodgement and coiling. Accordingly, a new active fixation lead was implanted and the patient was advised to use a rolling walker.

Twiddler's syndrome is an uncommon cause of pacemaker failure and this report suggests that, besides twiddling, additional mechanisms might be involved. Female gender, obesity, older age, and dementia constitute risk factors. Active-fixation leads should be probably preferred to prevent the syndrome.