

Syncope and Bifascicular Block: Who Needs a Pacemaker?

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Infra-hisian conduction delay is a well-known phenomenon. It was studied extensively in the early seventies when invasive recording of the His potential and measurement of the interval from the His bundle to the right ventricle (HV interval) became widely available.¹ Studies showed that infra-hisian conduction delay is associated with progression to trifascicular (that is, complete atrioventricular [AV]) block. On the assumption that conduction through the His bundle, unlike the AV node, follows an “all or nothing” rule, a maximum value for a normal HV interval has been suggested. By means of the measurement of the HV interval in 517 patients with bifascicular block, Dhingra et al found a value of >55 ms to be associated with an increased risk of progression to complete AV block.² In contrast, Scheinman et al studied 313 patients with bundle branch block³ over 3 years and found a progression to complete AV block in only 2% of the patients with an HV interval <55 ms, compared to 4% for an HV interval of 55 to 69 ms and 12% when it was ≥70 ms. In these patients, syncope had occurred before the electrophysiological study in only 40%. Interestingly, progression to complete AV block was observed in 24% of the individuals with very long HV intervals (≥100 ms).

Once the enthusiasm about the recording of the His potential had faded away, there has been no major study on this topic over the last 25 years. Therefore, current guidelines on the indication for permanent pacing in bifascicular block are still based on these data: pacemaker therapy is indicated if the HV interval exceeds 70 ms in syncope, or if an electrophysiologic study performed for other reasons happens to find an HV interval ≥100 ms.⁴

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Unfortunately, these old studies^{2,3} had several weaknesses:

1. Detection of progression to AV block required electrocardiogram (ECG) documentation. A potentially large number of patients with intermittent complete AV block very likely remained undetected.

2. Only a minority of patients had syncope before HV interval measurement. The percentage of patients with an HV interval in the range of 55 to 70 ms with progression to trifascicular block may be higher among patients with syncope of otherwise unknown origin.

3. No statistical methods were used to define an optimal value for the maximum “normal” HV interval; the value of 70 ms was chosen rather arbitrarily.

The most important study that questions the use of an HV interval ≥70 ms as the indication for pacemaker therapy in bundle branch block has been published by Brignole et al.⁵ Among 52 patients with syncope, bundle branch block and a negative electrophysiologic study, recurrence of syncope or presyncope with documentation of AV block occurred in 20 patients (38%) within a mean follow-up period as short as 48 days—despite a normal HV interval during the electrophysiologic study. The remarkable discrepancies between these study results are explained by the method used for detection of AV block: surface ECG recording with AV block is available only in the small fraction of patients with a persistent conduction disturbance. The majority of the patients with syncope and bifascicular block very likely has only infrequent spells of complete AV block, with or without syncope or presyncope, that are too short to be detected by resting ECG and too infrequent to be detected by 24-hour Holter. An implantable continuous monitor is incomparably more sensitive for detecting intermittent (or “paroxysmal”) high degree AV block as the cause of syncope in bundle branch block, as demonstrated in the ISSUE (International Study on Syncope of Uncertain Etiology) studies (eg, Brignole et al⁵).

In this context, the study by Martí-Almor et al in this issue of *Revista Española de Cardiología* is of particular interest⁶: reporting on 249 patients with bifascicular block, it confirms previous observations that a history of syncope is predictive of complete AV block. The association of structural heart disease, renal failure and a wide QRS complex with an increased incidence of complete AV block is interesting, while not completely surprising. The definition of a maximum “normal” HV interval by using a ROC curve with an optimal sensitivity and specificity relationship is welcome: a maximum value of 64 ms may be more effective than 70 ms in this respect.

In this study, the different types of bifascicular block had differing rates of progression to complete AV block: the incidence was 44/124 (35%) in left anterior hemiblock plus right bundle branch block (LAHB+RBBB), 47/102 (46%) in complete left bundle branch block, and 13/23 (57%) in left posterior hemiblock plus RBBB. The higher risk of AV block in bifascicular block involving the left posterior fascicle is of special note and has been described before. The association of LAHB and RBBB is certainly more “benign” in this respect. However, the incidence of AV block (35% over 4.5 years) was much higher than expected in this study. This has not been studied sufficiently, potentially because bifascicular block consisting of LAHB and RBBB has generally been looked upon as the most “physiologic” bifascicular block. Data from this study suggest that this may not be the case in patients with structural heart disease.

The most interesting point in the study by Martí-Almor et al is the method used to detect AV block: all patients with a positive electrophysiologic study (ie, HV interval >60 ms, more recently >70 ms) received a pacemaker programmed to respond at a lower rate limit of 40 beats per minute—the activation of which would lead to a strong suspicion of severe bradycardia. It was a brilliant idea to use the necessity of pacing at this rate as an indicator of AV block. Pacemaker counter data suggest an occurrence of complete AV block of 44% in these patients with bifascicular block and HV interval >60-70 ms, with syncope in 68% and structural heart disease in 44%. Whether or not the 10% proportion for ventricular pacing is a reasonable threshold for ventricular pacing is a matter of debate—it might be less or

more. It may also be argued that ventricular pacing may have occurred after post-extrasystolic pauses unrelated to AV block. Therefore, this threshold of 10% may have been reached in frequent premature beats, such as bigeminy. However, this method may significantly improve the sensitivity for the detection of intermittent complete AV block and should be refined in future studies using pacemaker devices with specific functions for electrogram storage, eg, bradycardia <40 bpm, sudden bradycardia, or electrogram storage triggered by the patient in case of syncope.

In conclusion, the study by Martí-Almor et al⁶ provides data in a large population of patients with bifascicular block. The results suggest that the incidence of progression to trifascicular block is higher than previously reported, particularly if syncope, renal disease or structural heart disease is present. Implantation of a pacemaker that is programmed to respond to a very slow “back-up” rate may be an interesting option for the continuous monitoring of the heart rate and detection of significant bradycardia with a much higher sensitivity. These and additional data may improve our understanding of infra-hisian conduction block and the treatment of affected patients.

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