Editorial:
Prognosis of Patients with Chronic Bifascicular Block

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"Doctor, I understand that I have three connections to the pumping part of my heart, but two are already closed off. If the third goes, my heart will stop, Doctor. What can you do about it?" — Transcript from patient's history.

OF ALL RECENT conceptual advances in electrocardiography, few, if any, are as popular as the concept of a trifascicular conduction system distal to the bifurcation of the His bundle. The wide appeal of the trifascicular concept is understandable. The distinct morphological electrocardiographic patterns of various fascicular blocks are easily recognizable even to a nonexpert. The schematic diagram depicting the trifascicular system is beguilingly simple, and the electrocardiographic interpretations are relevant to the management and prognosis of patients with acute myocardial infarction. Using well-defined and relatively noncontroversial electrocardiographic criteria, all conduction disturbances in the bundle branches may be subdivided into categories of unifascicular, bifascicular or trifascicular blocks. Of these, the bifascicular types are responsible for the majority of therapeutic dilemmas, particularly in patients without symptoms, or without a clearly established relation between symptoms and bradycardia, or atrioventricular (AV) block.

The electrocardiographic diagnosis of bifascicular block implies that the maintenance of AV conduction hinges on the integrity of the remaining third fascicle. The interest in the function of this fascicle is, therefore, obvious. If the imminent trifascicular block could be predicted by measuring the HQ interval,* then the His bundle electrograms would assume an important role in therapeutic decisions related to temporary or permanent ventricular pacing.

During the past decade, many investigators have studied prospectively the natural history of the bifascicular block, and the predictive value of the prolonged HQ interval in patients with such block. These studies have already generated useful information which may be helpful in answering the following questions:

* How many lives can be saved by prophylactic insertion of a permanent pacemaker in patients with bifascicular block? Table 1 is a summary of the results from several retrospective and prospective studies in patients with chronic bifascicular block. A common feature in these studies is high annual mortality, probably a reflection of advanced age and serious underlying diseases in the studied patient groups. While the total mortality figures are undoubtedly accurate, the information about the mechanism of sudden cardiac death in these studies is not always precise. Scanlon et al. did not report systematic analysis of the mechanism of death in all patients but attributed two sudden deaths to a presumed complete AV block. De Pasquale and Bruno reported 19 deaths but suspected AV block only in one patient who died suddenly at home. Of 68 deaths in the study of Denes et al., sudden cardiac deaths occurred in 30, but cardiac standstill with a failure of escape mechanism was implicated in only one of these victims. Of 50 deaths reported by McAnulty et al., 27 were sudden, but only three were attributed definitely to bradyarrhythmia. In seven other patients, the cause of death was unknown, so the possibility of bradyarrhythmia could not be ruled out. No sudden deaths were attributed to AV block in the population screened by Kulbertus et al., in Belgium. In a recent study of Scheinman et al., sudden death occurred in 15 un paced patients, but the mechanism of death in those who died suddenly was apparently unknown. Ten sudden deaths occurred in un paced patients studied by Narula et al. Of these, complete AV block was documented in one hospital patient, and suspected but not proven in the other nine.

Adding data from the 1242 patients with chronic bifascicular block followed for periods averaging from 1—3 years results in a total of nine patients in whom a complete AV block immediately before sudden cardiac death was documented and an additional 31 patients in whom the block was suspected. Confining this analysis to 950 participants in the prospective studies which included His bundle electrograms, the presence of complete AV block immediately before sudden death was documented in six and suspected in 31 patients.

Can the results of electrophysiologic studies identify potential victims of sudden death due to complete AV block? This is still a debatable issue, pitting skeptics against believers. The skeptics include the investigators from Chicago, Oregon and Belgium. Table 1 shows that these three groups studied prospectively 640 patients. Denes et al. found no significant
differences between the duration of HV intervals in the sudden cardiac death victims and the remaining patients. In the same cohort of patients, Dhingra et al. found no differences in mortality between patients with normal and prolonged AH interval. McAnulty and co-workers concluded that none of the electrocardiographic or electrophysiologic indices could have identified patients at high risk of sudden death. Analogous results were reported by Kulbertus et al. The three groups of investigators concluded that neither His bundle recording nor prophylactic pacing was indicated in asymptomatic patients with chronic bifascicular block. Similar views were expressed earlier by DePasquale and Bruno.

The opposing views favor electrophysiologic studies and prophylactic pacing in patients with prolonged HV intervals. In general, the evidence in support of these procedures suffers from difficulties in documenting the mechanism of sudden death. Narula et al. recommended permanent pacing in asymptomatic patients with HV ≥ 70 msec. They based these recommendations on the observation of sudden cardiac deaths in 10 nonpaced patients in whom the HV intervals averaged 70 msec but ranged from 50–115 msec. Vera et al. reported a study of 50 patients, the majority of whom had dizzy spells or Stokes-Adams attacks. In the discussion of their paper, the authors recommended recording His bundle electrograms in asymptomatic patients with bifascicular block if the PR interval was prolonged or if the bifascicular block had been documented for longer than 3 years. Further, they recommended pacemaker implantation in patients with HV ≥ 65 msec, but did not provide evidence to support this recommendation. The studies of Scheinman et al. suggested that long HQ interval was an independent risk factor and that the "prophylactic insertion of a permanent pacemaker is warranted in those patients with HQ ≥ 70 msec in whom no other cause for neurologic symptoms is found." However, more recent experience from this group showed no significant differences in mortality between patients with a prolonged HQ interval in whom prophylactic pacing was instituted and those who remained unpaced. Moreover, their serial electrophysiologic studies stressed the importance of relating changes in HQ interval to the changes in cardiac status.

What is the role of clinical examination in the evaluation of patients with bifascicular block? Of 388 patients with chronic bundle branch block, Dhingra et al. singled out 18 patients in whom HV intervals were ≥ 80 msec. While this finding was associated with high mortality and morbidity in symptomatic (class II–IV) patients, the clinical course was benign in five asymptomatic (class I) patients. In the study of Scheinman et al., a significantly greater percentage of patients with HQ ≥ 70 msec were in functional class II or IV than patients with HQ ≤ 70 msec. These investigators found that an HQ ≥ 70 msec was associated with an increased incidence of sudden death only in those patients with moderate or severe congestive heart failure. These observations document close association between prolonged HV interval and advanced heart disease, and suggest that in some cases the progression from bifascicular to trifascicular block may be precipitated by deteriorating myocardial function.

The study of Dhingra et al. in this issue of Circulation contributes to our knowledge of the natural history of chronic bifascicular block. This prospective study shows that of 452 patients with bifascicular block, 19% had no clinical evidence of organic heart disease. For these patients, the suggested diagnosis "primary conduction disease" is appropriate, though the more precise term "primary AV conduction disease" may be preferable, because it excludes patients with a more proximal conduction disease such as sick sinus syndrome. Dhingra et al. have shown that in their patients with primary conduction disease, the incidence of cardiovascular morbidity, sudden cardiac death, associated electrophysiologic abnormalities, and spontaneous AV block was significantly lower than in patients with organic heart disease. These results support the concept that the prognosis of patients with chronic bifascicular block is strongly influenced by the underlying clinical condition.

Are electrophysiologic studies needed to clarify the neurologic symptoms in patients with chronic

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**Table 1. Selected Long-term Studies of Patients with Chronic Bifascicular Block**

<table>
<thead>
<tr>
<th>Authors</th>
<th>No. of patients</th>
<th>Approx. average duration of follow-up (years)</th>
<th>No. of deaths Total</th>
<th>Possibly caused by complete AV block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seanlon et al.</td>
<td>209</td>
<td>2</td>
<td>31</td>
<td>3†</td>
</tr>
<tr>
<td>DePasquale and Bruno</td>
<td>83</td>
<td>3</td>
<td>19</td>
<td>1‡</td>
</tr>
<tr>
<td>Denes et al.</td>
<td>277</td>
<td>1</td>
<td>68</td>
<td>1</td>
</tr>
<tr>
<td>Scheinman and Peters</td>
<td>227</td>
<td>2</td>
<td>79</td>
<td>15‡</td>
</tr>
<tr>
<td>McAnulty et al.</td>
<td>257</td>
<td>2</td>
<td>50</td>
<td>3 + 7†</td>
</tr>
<tr>
<td>Narula et al.</td>
<td>83</td>
<td>3</td>
<td>32</td>
<td>1 + 9†</td>
</tr>
<tr>
<td>Kulbertus et al.</td>
<td>106</td>
<td>3</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

*Retrospective studies.† Only partial analysis of the mechanism of death reported.‡ Mechanism unknown. Abbreviation: AV = atrioventricular.
bifascicular block? It has been suggested that cardiac electrophysiologic studies may be helpful to the clinician in identifying the possible causes of syncope, dizziness, and transient neurologic symptoms in patients with chronic bundle branch block. However, the role of the His bundle studies in unraveling the pathogenesis of confusing neurologic symptoms is doubtful. Although the patients with chronic bifascicular block are frequently similar to those with transient ischemic attacks and other symptoms of cerebrovascular disease because of advanced age and frequent association with hypertension and atherosclerosis, careful history and neurologic examination can usually help to differentiate symptoms caused by transient focal cerebrovascular ischemic attacks from those caused by Stokes-Adams attacks. Reed et al. have pointed out that the neurologic symptoms in 251 patients requiring implantation of permanent pacemaker in the Mayo Clinic consisted predominantly of syncope, presyncopal sensation and generalized convulsions. Focal motor and sensory defects which represent the typical manifestations of transient ischemic attacks were present only in four of these 251 patients. However, these differences between the neurologic symptoms in patients with Stokes-Adams attacks and those with transient ischemic attacks do not resolve the vexing clinical problems of documenting the relation between the symptoms and the critical conduction disturbance. The role of clinical electrophysiologic studies in those patients with syncope or presyncopal sensation who have no evidence of trifascicular block in their surface ECG merits further study. In such patients, the documentation of a prolonged HV interval, or a block distal to the His bundle recording site induced by rapid atrial pacing may be useful in making the decision to implant a permanent pacemaker.

In conclusion, the cumulative evidence from several studies suggests that the His bundle electrogams play a very small role in therapeutic decisions in patients with chronic bifascicular block, and that the prognosis of these patients can be more accurately assessed by a thorough clinical evaluation than by the measurements of the HQ intervals.

References
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