Value of the H-Q Interval in Patients With Bundle Branch Block and the Role of Prophylactic Permanent Pacing

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His bundle electrograms were obtained in 313 patients with chronic bundle branch block who were followed for a mean period of almost 3 years. The infranodal conduction time (H-Q interval) was <55 ms in 97 patients (Group I), 55 to 69 ms in 99 patients (Group II), and ≥70 ms in 117 patients (Group III). There was a higher incidence of organic heart disease in patients in Group III, but the groups were otherwise comparable. On follow-up study, mortality and the incidence of sudden death were similar among the groups, but patients in Group III had a greater incidence of progression to high degree atrioventricular block (HDB) than did those in Groups I and II (14 of 117 [12%] versus 4 of 97 [4%] and 2 of 99 [2%], p <0.01, respectively). High degree block was found in 4 of 17 (24%) patients with an H-Q interval (H-Q) ≥100 ms.

Sixty-two patients underwent permanent prophylactic pacemaker insertion at the discretion of the referring physician and were compared with 231 patients who did not. Paced patients had a higher incidence of transient neurologic symptoms and prolonged H-Q, but the groups were otherwise comparable. On follow-up study, mortality and the incidence of sudden death were similar among the groups, but symptom relief was significantly more common among patients with pacemakers.

In conclusion, in our population (1) H-Q ≥70 ms was an independent risk factor for progression to HDB, (2) H-Q ≥100 ms identified a subgroup at particularly high risk, and (3) prophylactic pacemakers relieved neurologic symptoms but did not prolong life.

Patients with chronic bundle branch block have an increased risk for the development of complete atrioventricular (AV) block.[1-5] The recent introduction of techniques for ready recording of the His bundle potential in man has been applied to patients with bundle branch block.[6,7] Prolongation of the infranodal conduction time in a patient with bundle branch block signifies associated disease in either the unblocked bundle branch or the His bundle. Theoretically, a prolonged H-Q interval suggests advanced conduction system disease and may conceivably identify a subgroup at high risk for developing AV block or sudden death. The clinical significance of H-Q prolongation remains controversial.[8-10] We previously reported short-term prospective follow-up data in patients with chronic bundle branch block who underwent His bundle recordings.[11] In the present report, this experience is updated with a larger cohort to appraise the clinical significance of a prolonged H-Q interval more accurately. In addition, we are updating our experience with prophylactic permanent pacing in these patients.[12]

Methods

The methodology for this study was described in detail previously.[11] In brief, all patients with the electrocardiographic pattern of chronic bundle branch block were included in the present study. Patients from the cardiology clinics of various San Francisco Bay Area hospitals as well as patients referred for electrophysiologic study were included. Patients with bundle branch block and acute myocardial infarction as well as those with second or third degree AV block were excluded from the study.

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A complete history, chest roentgenogram, and physical examination were obtained in all patients. His bundle electrograms were obtained using standard techniques. In brief, a multipolar electrode catheter was inserted into the right femoral vein and positioned across the tricuspid valve. The His bundle electrogram and 3 surface electrocardiographic leads were displayed simultaneously on an oscilloscope and recorded (Electronics for Medicine VR-12 recorder). The AV nodal conduction time was measured from the initial high frequency deflection of the low right atrial electrogram to the first high frequency His bundle deflection (A-H), whereas infranodal conduction time was measured from the initial high frequency deflection recorded from the His bundle to the earliest onset of ventricular activation determined from the surface leads (H-Q). Patients who had second and third degree AV block during the His bundle recording were also excluded from study.

Follow-up study: Patients were followed up every 3 months either in the pacemaker and arrhythmia clinic or by referring physicians. Follow-up information was obtained by a public health nurse from patients who did not keep scheduled appointments. When necessary, home visits were made to obtain additional information including 12-lead electrocardiograms. In the event of progression to a higher degree of AV block, electrocardiographic tracings showing AV block were obtained. The diagnosis of second or third degree infranodal block was made on the basis of typical Mobitz type II AV block or periods of complete AV block with slow idioventricular rhythm associated with a different QRS morphology. Only patients with spontaneous AV block were included for study. Excluded were patients who showed AV block in the course of acute myocardial infarction or those with associated electrolyte disorders. In 6 patients the infranodal site of block was confirmed at the time of repeat electrophysiologic study. Patients requiring pacemaker insertion for sinus node disease or those in whom block developed at the level of the AV node were excluded from the study. In the event of death, hospital or coroner's reports, or both, were reviewed and observers were interviewed to determine if death was sudden.

Definitions: The criteria for diagnosis of bundle branch or fascicular block as well as intraventricular conduction delay have been discussed elsewhere.[13]

Congestive heart failure was diagnosed on the basis of physical findings and chest roentgenograms, and the patients were categorized according to the New York Heart Association functional classification. The diagnosis of valvular heart disease was based on physical examination or the results of cardiac catheterization. The diagnosis of cardiomyopathy was made in those patients with myocardial disease without demonstrable cause. For purposes of this study, patients with a history of smoking in excess of 10 cigarettes a day were classified as cigarette smokers. *Sudden death* was defined as unexpected natural death occurring either instantaneously (during sleep) or within 1 hour of the onset of symptoms. *Cardiovascular deaths* were those due to acute myocardial infarction or congestive heart failure.

Sixty-two patients underwent prophylactic pacemaker insertion. None of these patients showed evidence of second or third degree AV block during repeated continuous electrocardiographic monitoring. Pacemaker insertion was performed at the discretion of the referring physician on the basis of neurologic symptoms (syncope, recurrent dizzy spells, or seizures) or electrophysiologic abnormalities, or both. In some patients without symptoms, the nature of their occupation proved to be an important factor in the decision to insert a permanent pacemaker. These patients were seen regularly either by their private physicians or in an arrhythmia and pacemaker clinic. In addition, follow-up study included periodic telephone transmission of the pacemaker rate and stimulus characteristics.

Statistical methods: All data were stored on punch cards and later retrieved using a program written for the IBM model 360/75 computer. All nominal data were tested with the chi-square test or, when numbers were small, with Fisher's exact probability test.[14]

Results

A total of 367 patients with bundle branch block have been entered into the study since March 1972. Twenty-three patients were lost to follow-up study and 19 were excluded either because pacemakers were inserted for sinus node disease (12 patients) or because AV nodal block was demonstrated at repeat study (7 patients). Similarly, 12 patients in whom second or third degree AV block developed as a result of electrolyte abnormality or acute myocardial infarction were not included in the present analysis. The remaining 313 patients form the basis of this report.

TABLE I Clinical and Electrophysiologic Data in Patients With Chronic Bundle Branch Block

Variable	Group I: H-Q 30–54 ms		Group II: H-Q 55–69 ms		Group III: H-Q >70 ms		p Value
	n	%	n	%	n	%	
Patients (n)	97	...	99	...	117	...	
Age (mean ± SD) (yr)	66 ± 13	...	66.6 ± 14	...	66.5 ± 11	...	NS
Total with organic cardiac disease*	85	88	87	88	104	89	NS
Coronary artery disease	63	65	68	67	78	69	NS
Transient neurologic symptoms	58	60	58	59	68	56	NS
Syncope	38	39	40	40	46	39	NS
Congestive heart failure (class III or IV)	18	18.5	22	22	38	32	<0.05†
Follow-up (mo)	34 ± 24	...	34.5 ± 27	...	28 ± 23	...	NS
Progression to 2° or 3° block	4	4	2	2	14	12	<0.001†
Sudden deaths	12/97	14	12/99	12	19/117	16	NS
Cardiac deaths	14/97	14	15/99	15	28/117	24	NS
All deaths	41	42	50	51	57	49	NS

* Organic cardiac disease included patients with coronary artery, primary myocardial, valvular, hypertensive, and congenital heart disease.
† The incidence of organic cardiac disease, severe congestive heart failure, and progression to second (2°) or third degree (3°) AV block was higher in Group III than in either Group I or II.
NS = not significant; SD = standard deviation.

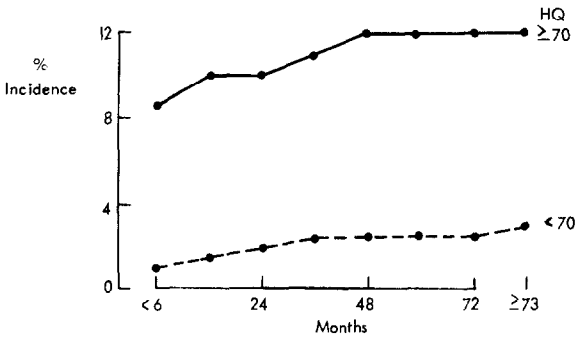

FIGURE 1. Incidence of spontaneous progression to second or third degree infranodal block with in patients an H-Q interval ≥70 ms is compared with that in patients with an H-Q interval <70 ms. The bulk of progression occurs within 6 months of entry into the study.

The patients were divided into 3 groups on the basis of the H-Q interval (Table I). Ninety-seven patients had H-Q intervals of 30 to 54 ms (Group I), 99 had H-Q intervals of 55 to 69 ms (Group II), and 117 had H-Q intervals ≥70 ms (Group III). There was no significant difference in age, incidence of syncope, or transient neurologic symptoms or length of follow-up study among the groups. The majority of subjects had organic heart disease; the most common diagnosis was coronary artery disease (67%).

The incidence of spontaneous progression to second degree or complete AV block was significantly higher in Group III (14 of 117 or 12%) than in Groups I (4 of 97 or 4%) and II (2 of 99 or 2%) ($p < 0.001$). The cumulative incidence of progression to second or third degree AV block for patients with H-Q ≥70 ms and those with H-Q <70 ms is detailed in Figure 1. The bulk of progression occurred in Group III patients within 6 months after entry into the study. Thereafter, a more gradual rate of progression was observed until plateau levels were achieved in both groups after approximately 36 months. The "plateau" may reflect the smaller numbers of subjects still alive at this time.

The risk of progression appears to be quantitatively related to the degree of H-Q lengthening once a critical H-Q interval of 70 ms is exceeded. For example, 17 patients with an H-Q interval of ≥100 ms showed a 23.5% progression rate (4 of 17) over a mean follow-up period of 22 ± 17 months. Most of the patients (13 of 17) had transient neurologic symptoms. Four of 17 had spontaneous progression to second or third degree AV block 11, 12, 18, and 56 months after entry and received pacemakers. Two of the 4 died of severe congestive heart failure. Seven of the 17 patients had prophylactic pacing; of the 7, 6 had transient neurologic symptoms. Five patients died. Three deaths were due to cardiac causes and 2 to noncardiac causes; none died suddenly. Three of 6 patients who did not have pacing died suddenly. Although the group of patients with an H-Q interval of ≥100 ms form a small subgroup within the total study group (17 of 313 [5.1%]), the high spontaneous progression rate is a minimal figure because over half of these patients were treated with prophylactic pacing.

The incidence of sudden death was similar among the groups (Table I). Although total cardiac mortality was higher in patients with marked prolongation of the H-Q interval (Group III) than in the other groups, this difference was not statistically significant. The possibly higher cardiac mortality in Group III may be related to the higher incidence of severe congestive heart failure in this group than in the others (Table I).

The surface electrocardiographic pattern proved to be of limited value in predicting either spontaneous progression to second or third degree AV block or the presence of marked prolongation of the H-Q interval. The various electrocardiographic patterns for each group are summarized in Table II. First degree AV block was significantly more frequent in Group III than in Group I, but this finding proved of limited clinical value. Similarly, there was no significant difference in the incidence of left bundle branch block or a bifascicular block pattern among the 3 groups.

These data identify a subset of patients with bundle branch block at increased risk for the development of AV block and in whom permanent pacemaker insertion might conceivably improve neurologic symptoms and survival. To test this hypothesis, we examined the clinical course of patients in whom permanent pacemakers were inserted prophylactically (in the absence of documented high grade AV block) and unpaced patients. Prophylactic pacemakers were inserted at the discretion of the referring physicians on the basis of

TABLE II Electrocardiographic Patterns in Patients With Chronic Bundle Branch Block

Variable	Group I		Group II		Group III		p Value
	n	%	n	%	n	%	
Patients (n)	97	...	99	...	117	...	...
First degree AV block	26	27	45	45	65	56	<0.001*
Right bundle branch block	22	23	34	34	49	42	<0.02*
Left bundle branch block	29	30	17	17	21	18	NS
Right bundle branch block with left anterior hemiblock	33	34	36	35	29	25	NS
Right bundle branch block with left posterior hemiblock	7	7	5	4	7	6	NS
Intraventricular conduction defect	6	6	7	7	11	9	NS

* There was no significant difference between Groups II and III. However, Group III had a higher incidence of first degree AV block and right bundle branch block than Group I.

TABLE III Comparison of Clinical and Electrophysiologic Findings for Paced and Unpaced Patients

Clinical Variable	Paced Patients: Prophylactically n	%	Paced Patients: For Progression to Second or Third Degree AV Block n	%	Unpaced Patients n	%	p Value
Patients (n)	62	...	20	...	231	...	...
Age (mean ± SD) (yr)	67 ± 10	...	65 ± 13	...	66 ± 14	...	NS
Congestive heart failure (class III or IV)	16	26	6	30	61	26	NS
Syncope	41	66	4	20	80	35	<0.02
Transient neurologic symptoms	47	76	8	40	134	55	<0.01
H-Q (mean ± SD) (ms)	72 ± 24	...	78 ± 25	...	59 ± 16	...	<0.001
Follow-up period (mo)	29 ± 22	...	39 ± 34	...	32 ± 24	...	NS
Relief of syncope	36/41	88	4/4	100	43/80	53	<0.001
Relief of any neurologic symptoms	32/47	70	7/8	87	68/133	50	<0.01
Sudden deaths	12/62	19	1/20	5	30/231	13	NS
Cardiac deaths	12/62	19	7/20	35	44/231	19	NS
Total deaths	31	50	11	55	108	47	NS

neurologic symptoms or abnormal electrophysiologic findings, or both. No evidence of AV block was found despite repeated 24-hour continuous dynamic electrocardiographic monitoring or observation in a coronary care unit. Clinical and electrophysiologic data for paced and unpaced patients are shown in Table III.

The 62 patients who underwent prophylactic pacemaker insertion were similar with respect to age and cardiac diagnosis compared with the 231 patients who were not paced. Twenty patients who required permanent pacemaker insertion because of progression to second or third degree AV block were analyzed separately. As expected, the incidence of syncope or transient neurologic symptoms as well as an abnormal H-Q interval were greater in the group with prophylactic pacing (Table III). Patients treated with pacemakers had a significantly higher incidence of relief of any neurologic symptoms or syncope than did the unpaced group.

Figure 2 shows follow-up findings in symptomatic prophylactically paced and unpaced patients. Of note was the finding of a high incidence of spontaneous disappearance of symptoms for the unpaced group. In addition, follow-up evaluation revealed the probable causes of symptoms in approximately half of those patients who had persistent transient neurologic symptoms. The causes included episodes of tachyarrhythmia (8 patients), cerebrovascular disease (4 patients), orthostatic hypertension (1 patient), drug use including alcoholism (5 patients), and hypoglycemia (1 patient). Seven of the 8 patients with transient neurologic symptoms who underwent pacemaker insertion because

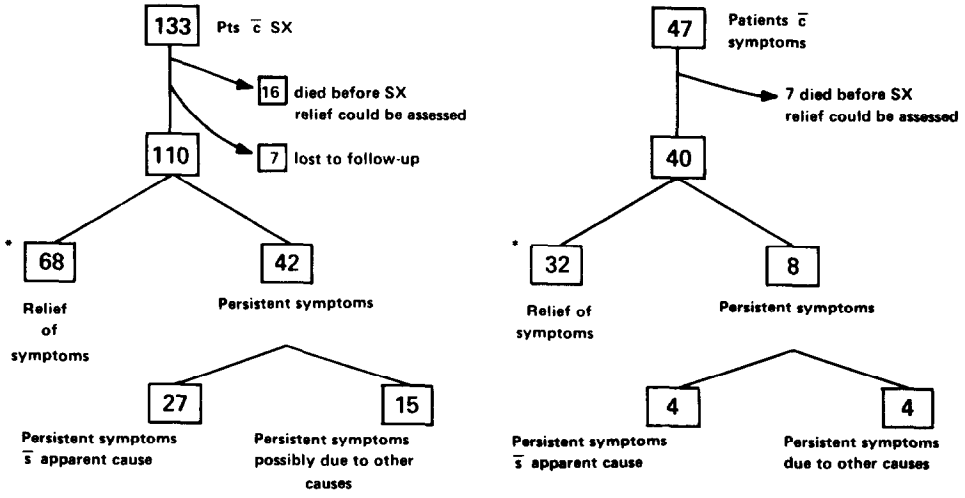

FIGURE 2. Follow-up chart of symptomatic patients who were either not paced (**left**) or paced (**right**). Excluded were 20 patients who underwent pacemaker insertion because of documented spontaneous trifascicular block. The incidence of relief of all transient neurologic symptoms was significantly higher in the paced group. SX = symptoms. **Asterisk** indicates $p < 0.05$.

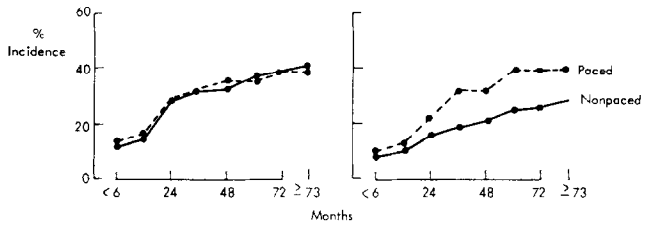

FIGURE 3. Cumulative incidences of cardiac deaths (**left**) and sudden deaths (**right**) for paced and unpaced patients. Although the incidence of sudden death was higher in paced patients at each follow-up interval, the differences were not significant.

of documented AV block had relief of symptoms (not shown in Figure 5).

Similar causes of transient neurologic symptoms were discerned in paced patients who had persistent symptoms after pacemaker insertion. We found no significant difference in the cumulative incidence of either cardiac or sudden deaths between paced and unpaced patients (Fig. 3). Sudden death was greater in the paced groups, but this difference was not significant.

It might reasonably be argued that the incidence of sudden death in the paced group might have been substantially higher had they not undergone prophylactic pacemaker insertion. We therefore compared all nonpaced patients with transient neurologic symptoms and an H-Q interval ≥70 ms with the paced group (Table IV). The 2 groups were comparable with regard to all clinical and electrophysiologic variables. The incidence of relief of syncope (88 versus 55%) and relief of all neurologic symptoms (89 versus 59%) was significantly greater in the paced group, but there was no difference in the incidence of sudden death or cardiac death between these 2 groups. Thus, we found no evidence tht prophylactic pacing increased longevity even when such patients were compared with a group with an expected increased risk for progressive conduction system disease.

Discussion

This report confirms and extends our previous observations relative to the importance of the H-Q interval in patients with bundle branch block. A markedly prolonged H-Q interval represents a clear-cut risk factor for spontaneous progression to second or third degree infranodal block. This increased risk was confined to those patients with an H-Q interval ≥70 ms, who had a 4-fold increase in progression compared with subjects who had either normal or slightly increased H-Q intervals (35 to 69 ms). A new and potentially important finding was the extremely high risk of progression to second or third degree AV block (23.5%) among patients with an H-Q interval ≥100 ms, suggesting a graded increment of risk dependent on the H-Q interval. It is pertinent to compare our data with those of 2 other similarly designed larger prospective studies of patients with bundle branch block.

Our findings confirm the recent report of Dhingra et al[15] involving 517 patients followed for a mean of 3.4 years. They also found an increased risk for spontaneous trifascicular block in patients with prolonged H-Q intervals. The incidence of spontaneous AV block in their study was lower than that in our series (that is, 0.6% for those with a normal H-Q interval versus 4.5% for those with H-V interval prolongation. In addition, our data differ with respect to the cumulative incidence of AV block. In our series, the bulk of spontaneous progression occurred within 6 months of study with a gradual increase to the 36th month of follow-up study followed by a plateau. In contrast, Dhingra et al[15] found a gradual increase in the cumulative incidence of AV block throughout the course of their study. Our results also differ from those of McAnulty et al,[16] who found that the H-Q interval failed to define a high risk subgroup (in terms of progression to complete AV block or sudden death) in 257 patients followed up over an average of 25 months. We believe that differences between studies are best reconciled by the characteristics of the respective

TABLE IV Prophylactically Paced Patients Versus Symptomatic Patients With an H-Q Interval ≥70 ms

Variable	Prophylactically Paced Patients		Unpaced Patients (H-Q ≥70 ms)		p Value
	n	%	n	%	
Patients (n)	62	...	37	...	...
Sex (male)	51	...	32	...	NS
Age (mean ± SD) (yr)	66 ± 11	...	64 ± 13	...	NS
Coronary artery disease	42	68	27	73	NS
Hypertension	23	37	15	41	NS
Diabetes	9	15	4	11	NS
Congestive heart failure (class III or IV)	16	26	13	35	NS
Syncope	41	66	20	54	NS
All transient neurologic symptoms	47	76	37	100	...
Relief of syncope	36/41	88	11/20	55	<0.05
Relief of all symptoms	32/40	59	20/34	59	<0.05
H-Q (mean ± SD) (ms)	72 ± 24	...	81 ± 12	...	<0.05
Follow-up period (mo)	29 ± 22	...	24 ± 23	...	NS
Sudden deaths	12/62	19	6/37	16	NS
Cardiac deaths	12/62	19	6/37	16	NS
Total deaths	31	50	14	38	NS

patient populations. Patients in the studies by McAnulty et al[16] and Dhingra et al[15] were recruited primarily by mass electrocardiographic screening. In contrast, most of our patients either were referred for study or consented to participate in the study because of symptoms. The ages of their patients as well as the incidence of organic cardiac disease, transient neurologic symptoms, and severe congestive heart failure were less than those in our studies. Earlier reports by Dhingra et al[17] and Denes et al,[18] for example, failed to show an increased risk for progression in those with a prolonged H-Q interval versus subjects with a normal H-Q interval. We hypothesize that as their patients aged, the respective population cohorts developed more similar characteristics which may help to explain the concordance in findings between our most recent studies. It is still a matter of conjecture whether or not the same will prove true for the patients followed up by McAnulty et al.[16] Similarly, the difference in the cumulative incidence of spontaneous AV block in our series compared with that of Dhingra et al[15] probably results from the higher incidence of syncope in our patients. In some of our patients, syncope was probably related to episodic high grade AV block that was eventually diagnosed relatively soon after entry into the study. Of special clinical importance was the finding of a very high incidence of spontaneous progression to complete AV block in patients with atrial pacing-induced AV block.[19]

In the present study, although prophylactic permanent pacing (in a subgroup with either symptoms or a prolonged H-Q interval or both) resulted in a significant decrease in the incidence of syncope, the incidence of sudden death or death was unchanged compared with that in the unpaced group. Similarly, the incidence of sudden death was comparable in the prophylactically paced group and in symptomatic patients with an H-Q interval ≥70 mm who were not treated with pacemakers. We interpret these findings as suggesting that the cause of sudden death was largely related to malignant ventricular arrhythmias, because no evidence of pacemaker malfunction was found in follow-up evaluation. In 2 paced patients undergoing continuous electrocardiographic monitoring, death was documented as due to ventricular fibrillation. These findings are in accord with the observations of Denes et al,[20] who concluded that death in patients with bifascicular block were largely due to ventricular arrhythmias; however, somewhat different results have been reported by other groups.[21,27]

Clinical implications: Conclusions from our data must be interpreted in the light of our study population: almost all had organic cardiac disease and most had transient neurologic symptoms. Therefore, our findings are not comparable to data of epidemiologic surveys of asymptomatic subjects with bundle branch block. In this study group, those with an H-Q interval ≥70 ms had a definite increased risk of progression to spontaneous trifascicular block compared with patients with an H-Q interval <70 ms. The overall risk of progression in patients with an H-Q interval ≥70 ms is still small, and routine prophylactic pacing in asymptomatic patients is not warranted. Transient neurologic symptoms alone may be an unreliable guide in determining the role of prophylactic pacing, because over half the patients with bundle branch block will have spontaneous remission of symptoms and a significant number have symptoms due to cardiac arrhythmias or neurologic, metabolic, or drug-related causes. These findings are in accord with the observations of Dhingra et al,[23] who found a high rate of spontaneous remission of symptoms. In patients with an H-Q interval ≥70 ms with recurrent transient neurologic symptoms (of no known cause), pacing appears to be indicated and will likely result in relief of symptoms, but there is no evidence to suggest that this intervention will prolong life. This conclusion is supported by our follow-up observations in 20 patients who received pacemakers because of documented progression to second or third degree AV block in that cardiac mortality was identical to that in unpaced or prophylactically paced patients; however, a trend toward a lower incidence of sudden death appeared in patients with documented AV block. The ultimate prognosis appears to be related to the severity of the underlying cardiac disease.[12] A new finding in our study is the extremely high incidence of spontaneous progression in those with an H-Q interval ≥100 ms. These patients, when symptomatic, should clearly undergo prophylactic pacemaker insertion.

Available data suggest 2 subgroups of patients with bundle branch block at very high risk for spontaneous trifascicular block. One group includes those with atrial pacing-induced infranodal block as described by Dhingra et al,[19] and the other is of patients with an H-Q interval ≥100 ms. Either of these findings is of great clinical value but unfortunately is only found in a miniscule percentage of patients with bundle branch block. Clearly, more effective means are required to identify patients with bundle branch block at high risk for second or third degree AV block or sudden death. For example, if sudden death is related to malignant ventricular arrhythmias, then perhaps Holter monitoring or ventricular stimulation studies[24] or both will prove of value in the detection of a subset of patients with bundle branch block and transient symptoms who are at increased risk for this complication.

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