A Prospective Evaluation of Intrahisian Conduction Delay

JOHN H. McANULTY, M.D., EDWARD MURPHY, M.D.,
AND SHAHBUDDIN H. RAHIMTOOLA, M.B., F.R.C.P.

SUMMARY We prospectively evaluated 46 patients who had intrahisian conduction delay. Twenty-three had a split His potential and 23 had a prolonged HV interval with a normal QRS complex. In those with a split His, the interval between the two His potentials averaged 32.7 msec (range 9–90 msec); in nine patients this split His was demonstrated only by atrial pacing. The 20 patients from this group with 1:1 atrioventricular conduction have been followed for an average of 18.1 months (range 2–48 months). All are alive. Three have had syncope, but Holter monitoring revealed no bradyarrhythmias.

In the 23 patients with a narrow QRS and prolonged HV interval, the HV interval averaged 73.7 msec (range 57–180 msec). Twelve of these patients received pacemakers at the time of the His bundle study, six had symptomatic atrioventricular block and five had symptomatic sinus pauses. The 11 patients who did not receive pacemakers have been followed for an average of 15.1 months (range 2–44 months). In three with recurrent syncope and five with dizziness, monitoring has revealed no bradyarrhythmias. One patient died from a myocardial infarction without arrhythmias.

Further prospective evaluation of patients with intrahisian conduction delay without documented bradyarrhythmias is needed, but with follow-up averaging 17 months and up to 4 years, patients with intrahisian conduction delay and without documented bradyarrhythmias appear not to require prophylactic permanent pacemakers to decrease morbidity or mortality.

INTRAHISIAN CONDUCTION DELAY is detected only by an intracardiac electrophysiologic study and has been documented as the site of symptomatic atrioventricular block seen on the surface ECG.1,2 On occasion, however, intrahisian conduction delay is demonstrated in patients with 1:1 atrioventricular conduction by finding a split His potential, or a prolonged HV interval in patients with a normal QRS complex.3,4 Recognition of intrahisian conduction delay (also called His bundle, His bundle delay, or first-degree His bundle block) has raised questions about its clinical significance, and its association with complete atrioventricular block in some select cases has led to recommendation for the use of permanent pacemakers in any patient with intrahisian conduction delay.5 We prospectively evaluated our patients with intrahisian conduction delay and 1:1 atrioventricular conduction in order to better understand the natural history of the disease.

Methods

All patients who underwent intracardiac electrophysiologic studies at the University of Oregon Health Sciences Center from January 1974 to February 1978 and had intrahisian conduction delay have been followed prospectively. Intrahisian conduction delay was considered to be present when the following criteria were met: 1) A split His potential. In such instances, the A (atrial potential) to H (first His potential) time had to increase as the rate of atrial pacing was increased. The time from the second His potential (H') to the ventricular depolarization (V, the earliest deflection of the QRS complexes from leads I, II, III or V1 of the surface ECG leads) had to be >30 msec to be certain that a right bundle potential was not confused with the His potential (normal HV (mean ± 2 sd) 43 ± 12 msec)6 (fig. 1); 2) A prolonged HV interval (>55 msec)7 in patients without surface electrocardiographic evidence of bundle branch block. Patients were entered into the evaluation at the time of the intracardiac electrophysiologic study and were seen every 3–6 months in a special conduction clinic. Particular care was taken during follow-up to identify and evaluate the symptoms of dizziness and syncope, and to determine the incidence and mode of death. Patients with symptoms during follow-up were readmitted for reevaluation and 48–72 hours of ambulatory monitoring. In addition, many patients had electroencephalograms, metabolic workups, exercise stress tests and evaluation of the vestibular system when indicated. In patients in whom symptomatic bradyarrhythmias were documented, permanent pacemakers were inserted.

Results

Forty-six patients with evidence of intrahisian conduction delay were identified; 23 had a split His potential (group 1) and 23 had a prolonged HV interval with a normal QRS width (group 2).

Group 1 — Split His Potential

In group 1 there were nine men and 14 women with a mean age of 54.2 years (range 25–85 years). Sixteen were studied as participants in a prospective evaluation of chronic bi- and trifascicular disease,8 and the others because of symptoms of syncope or dizziness. Ten patients had left bundle branch block, six had right bundle branch block and axis deviation, and seven patients had normal ECGs. Before the study,
nine had syncope, five dizziness and nine were asymptomatic. Associated heart disease included coronary artery disease in five, congenital heart disease in three, valvular heart disease in two and hypertensive heart disease in one. Twelve patients with no clinically recognizable associated heart disease by physical examination, chest x-ray, echocardiography, or in some cases cardiac catheterization and angiography, were considered to have primary conduction system disease.

The HH' interval from each patient was based on an average of four to 10 beats, and for the group averaged (mean ± SEM) 32.7 ± 4.4 msec (range 9–90 msec). The split His potential was observed in the control tracing in 14 patients. In these 14 the HH' interval was 26.9 ± 4.8 msec. In one of these patients the interval between the His potentials gradually increased with faster atrial pacing rates (as did the AH interval) until Mobitz type I atrioventricular block occurred between the His potentials (atrial rate 140). In another patient, the HH' interval gradually increased with progressively more premature atrial beats, and the atrial effective refractory period was reached before atrioventricular block was achieved (fig. 1).

A split His potential was not observed in the control state in seven patients, but was elicited by atrial pacing at 110–160 beats/min. Two of the seven had a His bundle potential width of 35 msec in the control state; the HH' interval with pacing was 38 msec in one and 70 msec in the other. Normal values for the His bundle width, however, are not known. A split His potential was demonstrated only with atrial extrastimuli in two patients. In each of these two the interval between the His potentials remained constant as the atrial beats became progressively more premature.

The H'V time averaged 53.3 ± 2.4 msec (range 35–90 msec) for the group. Seven patients had an HV interval >55 msec, indicating trifascicular or additional His bundle disease.

Three of these 23 patients received pacemakers at the time of the His bundle study because of documented transient symptomatic atrioventricular block at or near the time of the study, and were followed separately. One of these three patients has died, a 25-year-old woman with associated idiopathic hypertrophic subaortic stenosis in whom complete atrioventricular block with symptoms was demonstrated at the time of the study.

Twenty patients did not receive a pacemaker at the time of their entry into the study and they were followed prospectively (table 1). Seven presented with a history of syncope and another four had had dizziness. None of these had documented bradyarrhythmias or tachyarrhythmias, although 48–72-

---

**Figure 1.** A recording from a patient with a split His potential (paper speed 100 mm/sec). The top four recordings are from leads I, II, III and V₁ from the surface ECG. The fifth line is a recording from the high right atrium (HRA) and the bottom two recordings are from a tr. His bundle catheter (HBE). The initial two beats are sinus in origin with the split His potential best seen in the upper HBE tracing. With a paced atrial extrastimulus (P) the A-to-H time increases, clearly delineating the atrial (A) and His potential. The HH' interval also increased from 23 msec in the nonpaced beats to 70 msec in the paced beat. The H'V interval remained constant at 50 msec. In this patient, with further prematurity of the electrical stimulus, the atrial effective refractory period was reached.
hour ambulatory monitoring was performed in those with symptoms. These 20 patients have been followed for an average of 18.1 months (range 2-48 months). Three have had recurrent syncope since being studied. Holter monitoring revealed no arrhythmias in two, though neither had syncope during monitoring; in the third, syncope occurred and was demonstrated to be caused by paroxysmal atrial fibrillation. None received pacemakers and all three are alive. Another six had recurrent dizziness; four were symptomatic during monitoring and the tracings revealed premature atrial or ventricular beats in three and paroxysmal supraventricular tachycardia in one.

Three patients received pacemakers, one 14 months after entry into the study for control of supraventricular tachyarrhythmias, and two at the insistence of their personal physicians (at 3 and 7 months after entry into the study), despite the lack of further symptoms or documented bradyarrhythmias. All 20 patients are alive.

There was no significant difference ($p > 0.05$) between the nine patients whose split His potential was demonstrated only with atrial pacing techniques and those with a split His on baseline recordings when compared by age (56.7 ± 3.3 years vs 53.5 ± 2.6 years, mean ± SEM); sex (33% male vs 43% male); intrahisian time (40.7 ± 6.5 msec vs 26.9 ± 4.8 msec); or HV time (53.7 ± 2.4 msec vs 53.1 ± 3.8 msec). Of these nine patients, three had syncope and two had dizziness on entry into the study. Two had syncope at follow-up (19.7 ± 4.2 months). Holter monitoring was negative in both patients even though one had syncope during the recording period. The third patient with a history of syncope had no recurrence; he is the patient mentioned above who received a pacemaker at three months.

**Group 2 — Narrow QRS and Prolonged HV Interval**

These 23 patients included 13 men and 10 women with an average age of 60 years (range 28–86 years). Eleven were studied because of syncope, seven because of dizziness, one because of fatigue, and four asymptomatic patients had His bundle studies as part of other procedures. Associated heart disease included coronary artery disease in seven, valvular heart disease in four, congestive cardiomyopathies in three, and hypertension in three. Six patients had primary conduction system disease. The presenting ECGs revealed evidence of the sick sinus syndrome in six (atrial pauses >2 seconds or a heart rate <50 beats/minute with no apparent cause), transient atrioventricular block in six, atrial fibrillation in one, and paroxysmal supraventricular tachycardia in three.

The HV interval averaged 73.7 msec (range 57–180 msec). Four patients had transient atrioventricular block that occurred spontaneously or could be readily induced with atrial pacing. The block was below H in three of the four patients.

Twelve patients received pacemakers within 1 week of the electrophysiologic study: Atrioventricular block with a slow ventricular response was demonstrated in six and symptomatic prolonged sinus pauses were documented in five. One patient with syncope and no documented bradyarrhythmias received a pacemaker. Because they received pacemakers, these patients have been followed separately for an average of 30.4 months. One died a non-sudden death 23 months after pacemaker insertion, and three have been lost to follow-up.

Eleven patients did not receive pacemakers, although three had syncope and five had dizziness before inclusion in the study. These 11 have been followed for an average of 15.1 months (range 2-44 months) (table 1). During this period, three have had recurrent syncope and five have had recurrent dizziness. Hospitalization and intense ambulatory monitoring have failed to reveal bradyarrhythmias as the cause of symptoms in any of these patients. Three had dizziness and one had syncope during monitoring, and premature atrial or ventricular beats were associated with symptoms in each case. One patient received a pacemaker 1 year after entering the study for persistent dizziness, though a bradyarrhythmia was not documented. One year after entering the study, one patient died from a myocardial infarction without associated arrhythmias. One patient has been lost to follow-up.

**Groups 1 and 2 — Patients Who Did Not Receive Pacemakers on Entry into the Study**

The 31 patients with intrahisian conduction delay who did not have symptomatic bradyarrhythmias at the time of the His bundle study and did not receive permanent pacemakers (20 with a split His and 11 with a narrow QRS and prolonged HV) have been followed an average of 17.1 months (range 2-48 months). Six have had recurrent syncope and 11 have had persistent dizziness; bradyarrhythmias have not been documented as the cause in any, although nine had their symptoms during Holter monitoring. Four of the 31 patients have received pacemakers, none because they had documented bradyarrhythmias.
Only one patient has died, a non-arrrhythmogenic death caused by a myocardial infarction. No patients have died from arrhythmias and, except for the transient neurologic episodes, there has been no morbidity.

**Discussion**

Patients with intrahisian conduction delay who do not have symptomatic bradyarrhythmias at the time of an intracardiac electrophysiologic study appear not to be at risk of sudden death. This is based on follow-up averaging over 17 months, although some patients were followed up to 4 years after identification of the conduction disturbance. As the patient population described here was studied because of symptoms or evidence of other conduction system disease, it may not be representative of all patients with intrahisian conduction delay, but it is probably the group of patients with intrahisian conduction delay who are at highest risk.

Most of our patients were studied because of associated conduction and rhythm abnormalities, and intrahisian conduction delay was an incidental finding. Nine had evidence of the sick sinus syndrome or other atrial arrhythmias, and 16 had evidence of bi- or trifascicular disease. Intrahisian conduction delay is a relatively common finding in the group of patients we study. It occurs in almost 5% of a large group of patients being followed at this institution for chronic distal conduction disease with a similar prevalence in patients being evaluated for supraventricular dysrhythmias. It is likely that the underlying disease causing these other conduction and rhythm abnormalities is also the cause of the intrahisian conduction delay. In 18 of the 46 patients there was no evidence of associated heart disease, indicating that primary conduction disease can affect the His bundle. Twelve patients — three with evidence of a previous myocardial infarction — had coronary artery disease. Ligation of the anterior septal artery in dogs has resulted in well-documented, progressive degrees of intrahisian conduction delay, and the coronary artery disease was a likely cause of intrahisian conduction delay in some of our patients. Complete atrioventricular block during myocardial infarction in man has been shown to occur at the level of the His bundle. The intrahisian conduction delay was associated with a number of other diseases in our study, and has been documented by others to be a congenital defect, to be caused by cardiac surgery, to be secondary to a penetrating stab wound to the heart, to be caused by an intracardiac fibroma and by catheter manipulation within the heart.

Intrahisian conduction delay can be missed if not carefully looked for during a His bundle study. Schuilenburg and Durrer stressed the importance of slow pullback recordings and suggested that intrahisian conduction delay may be more common than recognized. A split His potential was demonstrated only with atrial pacing or the extrastimulus technique in nine of our patients, indicating the need to stress the conduction system to recognize the abnormality in some cases. It is unclear whether a split His potential demonstrated only with atrial pacing has more or less clinical significance than that found in the control state. From our data there is no apparent difference, but further follow-up may eventually allow a better understanding of the significance of intrahisian conduction delay.

Further refinement of techniques may also allow us to be more certain about the incidence of His bundle disease. In this evaluation of split His potentials, only patients in whom the components of the His potential were distinct and reproducible were included. It is possible that patients in whom there is an occasional suggestion of a split His potential (not included in this study) may also have His bundle disease, and that it is simply catheter position or movement that prevents a more certain diagnosis. In other patients, again not included in this study, the HV interval recorded at one time during a study can clearly differ from an HV interval recorded later in the study, frequently with a different His bundle configuration. Though there are other explanations for this, such as a true change in the HV interval during the study, it is possible that this too could reflect a split His potential, with the recording catheter observing one portion of the His potential at one time during the study and another portion later in the study. Two of our patients had a relatively wide His potential in the control state, which may, though we are uncertain about normal values for His bundle width, reflect intrahisian conduction delay. Some investigators have found attempts to pace at the site of the His bundle a useful way to evaluate and document intrahisian conduction delay. We have been unable to pace from that region reliably.

Ambulatory monitoring did not reveal bradyarrhythmias in our patients. Clearly, there are limitations to this technique, as it can be used for only a brief period and bradyarrhythmias could easily be missed. It is important to note, however, that even though we are most concerned about bradyarrhythmias, tachyarrhythmias and premature beats were invariably the cause of symptoms in the nine of 17 patients with recurrent symptoms that occurred during the Holter monitoring. It is possible, however, that these nine, or the eight who did not have symptoms with monitoring, could have had bradyarrhythmias at other times. Because of limitations in arrhythmia detection, it seems appropriate to ask whether it is “safe” to conclude that prophylactic permanent pacemakers are not indicated in these patients with conduction abnormalities. The major concern about bradyarrhythmias is that they may cause increased morbidity and mortality. In following our group of patients for an average of over 17 months, and in some cases for longer than 4 years, these concerns have not been realized.

The management of patients with symptomatic bradyarrhythmias is not a clinical problem; most require permanent pacemakers. This is true in patients with intrahisian conduction delay as well. The finding of intrahisian conduction delay in patients with bradyarrhythmias, however, has raised the consideration...
that all patients with this finding may be at high risk of atrioventricular block or death. It has led some to suggest prophylactic pacemaker insertion in all patients with intrahisian conduction delay. Further prospective follow-up of our patients will be necessary before the true significance of intrahisian conduction delay can be fully evaluated. In no study, however, have patients with this finding and without documented bradyarrhythmia been shown to be at high risk of atrioventricular block or sudden death. None of the patients without a pacemaker followed by us developed documented bradyarrhythmias or died from an arrhythmia. Until there is more evidence that patients with intrahisian conduction delay are at risk, prophylactic insertion of permanent pacemakers seems unwarranted.

Acknowledgment
The authors greatly appreciate the assistance of Helen Fischer in the preparation of this manuscript.

References
Prospective evaluation of intrahisian conduction delay.
J H McAnulty, E Murphy and S H Rahimtoola

Circulation. 1979;59:1035-1039
doi: 10.1161/01.CIR.59.5.1035

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/59/5/1035