The Natural History of Trifascicular Disease
Following Permanent Pacemaker Implantation

Significance of Continuing Changes in Atrioventricular Conduction

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SUMMARY Seventy-two patients with trifascicular disease were followed for an average of 40 months following permanent pacemaker insertion. The indications for pacemaker insertion were either electrocardiographic evidence of complete heart block with a wide QRS escape complex or a pattern of bifascicular block with either periods of Mobitz type II atrioventricular (A-V) block or a documented history of syncope. The patients were then divided into three groups depending on subsequent change in A-V conduction. There were 31 (43%) patients with no change in A-V conduction, 17 (24%) with increasing A-V block, and 24 (33%) with decreasing A-V block. The characteristics of these three groups, including age and sex distribution, were compared and found to be similar. The incidence of previous transmural myocardial infarction as determined by electrocardiographic criteria was higher in the group with decreasing block. Survival curves showed a significantly decreased probability of surviving for those with decreasing block compared to both those with increasing block and those with no change in conduction ($P < 0.03$).

We conclude that the probability of long-term survival was less in the group with decreasing block. This finding may be related to the greater prevalence of coronary heart disease in these patients.

THE NATURAL HISTORY OF BIFASCICULAR DISEASE has been described and has emphasized the incidence of progression to complete heart block. It is the purpose of this study to follow the natural history of fascicular conduction in patients with trifascicular disease who require permanent pacemaker insertion. Emphasis will be placed on changes occurring in atrioventricular (A-V) conduction and the relationship of these changes to the prognosis of the patient. The survival rates of those patients who showed further change in A-V conduction will be compared to those who showed no change and possible differences in the etiology of the fascicular disease will be discussed.

Material and Method

Seventy-two patients with trifascicular disease requiring permanent pacemaker insertion were followed for an average of 40 months (1 to 133 months) during the period 1966–1975. Ventricular-inhibited pacemaker generators* were used in most cases and frequently replaced fixed-rate generators† that had been implanted initially. An endocar
dial bipolar electrode‡ was used in most instances.

Periodic evaluations using a schedule similar to that suggested by Escher and Furman§ were done in an outpatient pacemaker clinic and occasionally during a period of hospitalization for generator change. The indications for pacemaker insertion were either electrocardiographic evidence of complete heart block with a wide QRS escape complex or a pattern of bifascicular block with either periods of Mobitz type II A-V block or a history of documented syncope with no other obvious explanation. None of the patients had evidence of acute myocardial infarction.

Right bundle branch block (RBBB) and left bundle branch block (LBBB) were defined according to the criteria of the New York Heart Association. Diagnoses of left anterior hemiblock (LAH) and left posterior hemiblock (LPH) were made by the criteria suggested by Rosenbaum. Transmural myocardial infarction was diagnosed by using the New York Heart Association criteria in addition to the criteria suggested by Sodi-Pallares for the electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block and beats of ventricular origin.

Periodic re-examination of the conduction system was obtained by three different techniques. Those with ventricular-inhibited pacemakers with intrinsic heart rates faster than the pacemaker were evaluated by a standard 12-lead electrocardiogram. Some, especially those with fixed-rate pacemakers, were evaluated only when there were episodes of pacemaker failure or during a period of generator change. The technique of chest wall stimulation was utilized to examine the underlying cardiac mechanism in patients with ventricular-inhibited pacemakers. This procedure was done in a manner similar to that described by Samet et al. and involves applying a 0.5 to 2 mA stimulus across the anterior chest wall in the region of the pacemaker generator by two suction cup electrodes connected to a fixed rate pacemaker. The rate of the low amplitude electrical stimulus is increased to a rate sufficient to suppress the QRS blocking pacemaker, which thus allows information to be obtained about the intrinsic rhythm of the heart. The procedure is done under constant electrocardiographic monitoring and is immediately discontinued if high grade block with no escape pacemaker is noted. In all instances the pacemaker artifact with capture immediately returned upon termination of chest wall stimulation.

Survival curves were plotted for each group using the Kaplan-Meier life table procedure and were evaluated using the generalized Wilcoxon W statistic for comparing two survival curves.

*Medtronic Models 5841, 5842, 5942, 5944.
†Medtronic Model 5870C
‡Medtronic Models 5816, 6901

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Results

Seventy-two patients were studied and divided into three groups. There were 31 patients (43%) who showed no change in A-V conduction, 24 (33%) who showed decreasing A-V block, and 17 patients (24%) who showed increasing A-V block. The characteristics of these three groups are shown in tables 1 and 2. None of these differences was statistically significant.

The 31 patients with stable conduction all had complete heart block with a wide QRS complex. The 24 patients with decreasing block had complete heart block initially and improved to first degree A-V block in ten and normal A-V conduction in the remaining 14. The 17 patients with increasing block included nine with periods of Mobitz type II A-V block and eight patients with documented periods of syncope. The electrocardiographic patterns observed in this latter group of patients were first degree A-V block and LBBB in two; first degree A-V block, RBBB, and LAH in two; first degree A-V block, RBBB, and LPH in one; and three with normal A-V conduction, RBBB, and LAH. These patients all progressed to complete heart block with a wide QRS complex. Electrocardiograms and a His bundle recording of a patient with changing conduction are shown in figures 1 and 2. There was no chronological relationship between changes in conduction and patient deaths. Information concerning cause of death or whether death was sudden or not was available in too few patients to be used in the study.

His bundle recordings were obtained in five of the 31 patients with stable conduction, nine of the 24 patients with decreasing block, and nine of the 17 patients with increasing block. No significant difference was noted between these

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<th>Table 1. Male-Female Frequencies</th>
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$\chi^2 = 0.29 \text{ (N.S.)}$

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<th>Table 2. Age of Patients</th>
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Using the ANOVA statistical technique these differences are not significantly different.
three groups, i.e., all patients showed either H-V prolongation or block below or within the bundle of His.

The incidence of previous transmural myocardial infarction as determined by electrocardiographic criteria alone was examined in all three groups. Evidence of previous myocardial infarction was found in one of the 31 patients with stable conduction (3%), one of the 17 patients with increasing block (6%), and eight of the 24 patients with decreasing block (33%) ($P < 0.005$). The total incidence of documented prior myocardial infarction was 10/72 (14%).

Survival curves were constructed for the three groups and are shown in figure 3. Comparison of these survival curves shows that the probability of survival was significantly less in those with decreasing block when compared with those with either increasing block or no change in conduction ($P < 0.03$).

**Discussion**

The cause of chronic complete heart block within or distal to the bundle of His includes coronary artery disease, Lenegre's disease, and Lev's disease. Lenegre's disease is an idiopathic sclerodenerative process involving both bundles. Lev's disease is similar but occurs secondary to fibrosis and calcification of the cardiac skeleton related to aging and is, therefore, more commonly seen in the elderly. Other postulated etiologies include healed myocarditis and syphilis.

The frequency of coronary artery disease as the etiology of bifascicular block and chronic complete heart block remains unclear. In studies limited to patients with RBBB-LAH, we found clinical evidence of angina pectoris or myocardial infarction in 16/65 (25%) patients while DePasquale and Bruno found evidence of coronary disease in 61/83 (73%) patients. In a review of 64 patients with conduction disturbances requiring permanent pacemaker, McConahay et al. found associated ischemic heart disease in 19/64 (30%) while Codini, Caralis, and Voight noted coronary artery disease in 45/100 (45%). It is probable that differences in criteria of diagnosing coronary artery disease explain, in part, the differing incidences in various studies.

Our study shows that the changes in conduction separate this large group of patients into three groups with different survival rates. Those with decreasing A-V block have a decreased survival rate which we feel may be related to the higher incidence of coronary heart disease in this group. It is possible that the groups with increasing block and stable conduction are comprised mostly of those with the sclerodervative process.

The concept that coronary artery disease is a more likely etiology when changing conduction is present is consistent with our preliminary findings concerning a group of patients with LBBB. In this study we found that 91% (20/22) of patients with unstable LBBB had evidence of angina or acute myocardial infarction as opposed to only 27% (6/22) of a group with stable LBBB.

We have reached the following conclusions from this study: 1) Changes in A-V conduction are fairly common in patients with trifascicular disease following permanent pacemaker insertions. 2) The probability of survival over an extended period of time was less in the group with decreasing A-V block. This finding may be related to the higher incidence of coronary heart disease in these patients.
HEMODYNAMIC EFFECTS OF SVT/Goldreyer, Kastor, Kershbaum

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Acknowledgment

The secretarial assistance of Mrs. Edith Erick and Mrs. Frances Schlesinger is appreciated.

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The Hemodynamic Effects of Induced Supraventricular Tachycardia in Man

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SUMMARY The circulatory effects of supraventricular tachycardia (SVT) were studied in eight patients who reported disabling symptoms during paroxysms of the arrhythmia. Supraventricular tachycardia was induced in each patient by rapid atrial pacing or with atrial premature stimuli. Hemodynamic parameters in sinus rhythm and following the initiation of SVT were recorded and compared.

The following mean values were observed in sinus rhythm (SR) and SVT. Heart rate (beats/min): SR 79, SVT 183; P-R interval (msec): during SR, 154; during SVT, 256; ratio of mean P-R intervals to mean R-R cycle lengths: SR 20%, SVT 76%; brachial artery pressures (mm Hg): SR 141, SVT 99; cardiac index (l/min/m²): SR 3.6, SVT 2.2; pulmonary artery pressures (mm Hg): SR 18/7, SVT 26/15; peak right atrial pressures (mm Hg): SR 4, SVT 17. Large waves appeared in the right atrium during SVT due to atrial contraction against closed tricuspid valves. Pulsus alternans were observed in each case during SVT. Despite the presence of chest pain during SVT, the coronary arteries were normally patent in four patients who underwent coronary arteriography.

SUPRAVENTRICULAR TACHYCARDIA (SVT) may severely disable afflicted patients who are otherwise in good health and without apparent cardiac disease. Dyspnea, weakness, lightheadedness, and even syncope may be produced by SVT at heart rates generally well tolerated when the rhythm is of sinus origin. The hemodynamic basis of these symptoms during SVT has never been fully explained.

Previous hemodynamic observations during SVT have been obtained fortuitously when the arrhythmia developed during cardiac catheterization. Since the usual mechanism of SVT is atrioventricular (A-V) nodal re-entry it may be predictably initiated and terminated by atrial stimulation. As a result, comparative evaluation of the hemodynamic consequences of SVT may be assessed with the patient in sinus rhythm serving as his own control. We report here a combined electrophysiologic and hemodynamic study in eight highly symptomatic patients with A-V nodal re-entrant SVT. Our findings help to explain why this arrhythmia produces such discomfort in affected patients.

Methods

Eight patients with a clinical history consistent with the presence of recurrent paroxysmal supraventricular tachycardia were evaluated in the Cardiac Clinical Electrophysiology Laboratory (table 1). Each patient gave informed consent to the performance of both hemodynamic and electrophysiologic studies. The following venous catheters were inserted percutaneously or by cut-down through the right or left basilic veins or the right femoral vein:
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