Fascicular Block within the His Bundle*

José A. Sobrino, M.D.; José F. Sotillo, M.D.;
Alejandro del Río, M.D.; Nicolás Sobrino, M.D.;
and Isabel Maté, M.D.

The findings in a patient with complete atrioventricular block and normal morphology and duration of the QRS complex are presented. A His bundle electrogram was obtained, which led to the location of the atrioventricular block within the His bundle. A careful review of the electrocardiograms obtained during the seven years preceding the onset of complete atrioventricular block showed a QRS complex with the features of left anterior hemiblock and a progressive impairment of atrioventricular conduction. From these data, we inferred that the different degrees of atrioventricular block and the left anterior hemiblock were caused by lesions within the His bundle involving the fibers destined for the left anterior division of the left branch. After the onset of complete atrioventricular block, with the subsidiary pacemaker located in the His bundle distal to the lesions, the QRS complex became normal, indicating the integrity of the bundle branches and fasciculi. The atrial and proximal His potential intervals and those between distal His and ventricular potentials were normal.

One cause of complete atrioventricular block is a lesion of the common His bundle. Since the description of the electrophysiologic features of His bundle block by Narula et al.,¹ the features of the different types of atrioventricular block occurring at this specific site of the conduction system have been reported in several other articles. Bundle branch blocks, fascicular blocks, and bifascicular or trifascicular blocks (or both) are believed to be produced by organic or functional disease of these structures; however, certain lesions of the His bundle involving the fibers going to the main branches or the fasciculi could provoke very similar blocks.² To our knowledge, this possibility has not been proven in electrophysiologic studies.

This report presents a case of complete atrioventricular block with the morphology, axis, and duration of the QRS complex within normal limits and with the block located in the His bundle by means of the His bundle electrogram. The electrocardiograms obtained before the onset of complete atrioventricular block showed the features of fascicular block and different degrees of partial atrioventricular block. This fascicular block was attributed to lesions of the His bundle between its proximal and distal portions.

Case Report

A 62-year-old woman was examined in October 1976 because of persistent bradycardia. She complained of occasional dizziness but had never suffered loss of conscious-

*From the Cardiovascular Surgery Department, Cardiology Unit, La Paz Hospital, Madrid, Spain.
Reprint requests: Dr. Sobrino, Hilatro Eslava 55, Madrid, Spain.

CHEST, 74: 2, AUGUST, 1978

Electrocardiographic Analysis

In 1969, the ECG showed a sinus rhythm of 70 impulses per minute, a P-R interval of 0.20 second, and a QRS complex satisfying the criteria for left anterior hemiblock (QRS axis at −60°, q wave in leads 1 and aVL, and an R S pattern in precordial leads V₆ and V₇).³ In 1970, the P-R interval was 0.24 second, with a similar QRS complex. Since May 1971, periods of partial atrioventricular block (Mobitz type 1) were recorded in alternating periods of 1:1 atrioventricular conduction with long P-R intervals (Fig 1). This same situation was found during 1972 and 1973, without any changes in the QRS complex. In March 1974, the ECG revealed a Mobitz type-2 block, with a ventricular rate between 42 and 45 impulses per minute, while the QRS complex maintained the features of left anterior hemiblock (Fig 1). In 1975, the tracings were identical to the ones from 1974. In September 1976, the first ECG showing complete atrioventricular block and a normal QRS complex was recorded (Fig 2).

His Bundle Electrogram

The His bundle electrogram was recorded in October 1975. The patient had complete atrioventricular block. The tracings were recorded using a commercially available recorder (Mingograf-81), following the technique described by Scherlag et al.⁴ A split of the His potential (H) was recorded. The first His potential was related to the atrial potential (A) with a fixed A-H interval of 80 msec; the other His potential (H') was related to the ventricular potential (V) with an H'-V interval of 50 msec (Fig 3). After administration of atropine, the atrial rate increased without significant change in the ventricular rate.

Discussion

James and Sherf⁵ have proved that the His bundle is characterized by longitudinal fibers (Purkinje's type) that are distinctly partitioned by sheaves of collagen. These fibers go to the branches and fasciculi which finally break up into Purkinje's network. This structural appearance suggests that the different electrocardiographic patterns of bundle branch or fascicular blocks could be produced by lesions of the His bundle selectively affecting the fibers that reach those structures. Several earlier experimental works lacked electrophysiologic data to support the existence of longitudinal dissociation of transmission of the impulse in the His bundle. In fact,
Figure 1. Electrocardiograms recorded in July 1972 and in May 1974. Note Wenckebach phenomenon in lead 1 (left) and Mobitz type-2 atrioventricular block with ventricular rate of 44 impulses per minute (right). In both, QRS complex shows left anterior hemiblock.

Figure 2. Electrocardiogram recorded in September 1975, showing complete atrioventricular block. Features of left anterior hemiblock have disappeared.
neither the selective stimulation nor the partial transection of the His bundle could produce bundle branch blocks. This behavior is explained by the existence of transverse crossover connections between the longitudinal sheaves of fibers. These transverse connections, described by James and Sherf, would allow uniform transmission of the impulse. Scherlag et al and, recently, Fabregas et al obtained patterns of bundle branch and fascicular blocks in dogs by partial transection of the His bundle and by providing rapid stimulation to the proximal aspects of the lesions. These findings were ascribed to a functional failure of the transverse connections with fast rates.

In our case the electrocardiographic patterns of left anterior hemiblock with different degrees of disturbances in atrioventricular conduction, which were present in all of the tracings recorded during seven years, disappeared after the development of complete atrioventricular block within the His bundle; and after administration of atropine, the rate of A-H complex increased without significant change in the rate of H-V complex. These facts indicate that the lesions producing the left anterior hemiblock and the disturbances in atrioventricular conduction were located proximal to the subsidiary pacemaker in the distal portion of the His bundle (H'). There were no disturbances in conduction at this or more distal levels, as is readily inferred from the normal H-V interval and the normal morphology and duration of the QRS complex during complete atrioventricular block.

It would be tempting to speculate on the behavior of left anterior hemiblock as being a rate-dependent block that disappeared with the slow rate attending complete atrioventricular block; however, the ventricular rate of the subsidiary pacemaker during complete atrioventricular block was similar to those existing in several previous tracings that showed a Mobitz type 2 block with the features of left anterior hemiblock.

The other possibility for normalization of the QRS complex could be the existence of a delay in conduction in the left posterior division, in the presence of an incomplete left anterior hemiblock and discordance between conduction and refractory periods of both fascicles. In our case, this possibility would be difficult to justify because of the persistence of left anterior hemiblock during seven years, and a pattern of incomplete left bundle branch block was never seen.

Patients with bifascicular blocks are considered to be at risk of developing complete atrioventricular block because of the potential extension of the disease to the other fascicle. So, it is reasonable to assume the existence of similar risks when the fascicular blocks are located within the His bundle.

In our case the existence of left anterior hemiblock with first-degree and second-degree atrioventricular blocks indicated severe damage of the fibers of the His bundle between its proximal H and distal H' portions. Atrioventricular conduction was maintained by the fibers of the His bundle going to the posterior division and by the ones going to the right bundle. Nevertheless, the atrioventricular transmission gradually deteriorated (first-degree and second-degree atrioventricular block) to complete atrioventricular block because of the total damage to the fibers. It is possible that the electrocardiographic features of bundle branch and fascicular blocks resulting from specific lesions within the His bundle are not exceptional. Their usual combination with associated damage in the bundle branches would mask the clinical diagnosis, since the QRS complex would not return to normal when complete ventricular block is established.

REFERENCES

Recurrent Mediastinal Bronchogenic Cyst* 

Cause of Bronchial Obstruction and Compression of Superior Vena Cava and Pulmonary Artery

D. Craig Miller, M.D.;** John P. Walter, M.D.;† 
Diana F. Guthamer, M.D.;‡ and 
James B. D. Mark, M.D., F.C.C.P.§

The recurrence of a benign mediastinal bronchogenic cyst 20 years after partial excision precipitated potentially serious vascular and pulmonary complications. Aggressive total surgical excision should be feasible in the majority of cases. An approach via a median sternotomy offers distinct advantages in certain cases and should be considered. Computerized axial tomographic scanning promises to provide improved definition of mediastinal anatomic features and should be a valuable noninvasive diagnostic method in selected cases.

 Mediastinal bronchogenic cysts are no longer considered to be rare.1-5 Although usually asymptomatic in adults, such cysts can compress adjacent vital structures. When local anatomic considerations preclude total excision of the cyst, partial excision with or without surgical destruction of the remaining cystic mucosa has been accepted as an alternative technique.1-4,5 This case shows that cysts may recur and become symptomatic when treated in this manner.

CASE REPORT

A 55-year-old woman was referred for evaluation of a possible aneurysm of the ascending aorta. Her past history was germane in that she had undergone surgery for a mediastinal mass with acute superior vena cava syndrome in 1958. At that time the superior vena cava and azygos vein were found to be displaced and compressed by a large mediastinal cyst, which was partially excised. The entire cyst could not be removed due to its extension across the midline. Histopathologic examination of the wall of the cyst revealed bronchial epithelium. The patient’s obstruction of the superior vena cava resolved, and she convalesced without incident.

During the summer of 1977, the patient noticed increasing fatigability, a vague sensation of orothostatic substernal pressure, a new persistent nonproductive cough associated with moderate orthopnea, and one episode of minor hemoptysis. She denied fever, infection of the upper respiratory tract, paroxysmal nocturnal dyspnea, dependent edema, and angina pectoris. A chest x-ray film taken in August 1977 revealed a new 6 x 8 cm mass occupying the right anterior and middle regions of the mediastium (Fig 1).

---

*From the Departments of Cardiovascular Surgery, Surgery (Division of Thoracic Surgery), and Radiology, Stanford University Medical Center, Stanford, Calif.
**Assistant Professor of Cardiovascular Surgery.
† Resident in Radiology.
‡ Acting Assistant Professor of Radiology.
§ Professor and Head, Division of Thoracic Surgery.

Reprint requests: Dr. Miller, Cardiac Surgery A246, Stanford University Medical Center, Stanford 94305