

Microelectrode and His Bundle Studies on Type I and II Second Degree A-V Block*

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Mobitz classified second degree A-V block into two categories. Mobitz type I, or Wenckebach block, is characterized by a gradual prolongation of the P-R interval preceding the dropped ventricular beat. In Mobitz type II block, the dropped beat occurs without preceding prolongation of the P-R interval. Bundle branch block usually is present in patients with Mobitz type II block. The importance of distinguishing between these two types of A-V block lies in the usual irreversibility and higher mortality of patients with type II A-V block, as contrasted with type I or Wenckebach block. Also, Mobitz type II block frequently progresses to complete A-V block and Adams-Stokes attacks, thus necessitating a cardiac pacemaker (1, 2, 3).

Using only electrocardiographic recordings, it is not possible to define the location within the A-V conduction system where A-V block actually develops. The need to distinguish between conduction failure above or below the bundle of His is important since dropped beats occurring from block within the

A-V node would not carry as serious a clinical prognosis as would block within the ventricular specialized conduction system (VSCS or His-Purkinje system). The following figures are presented to demonstrate the use of the microelectrode technique and His bundle electrogram recording technique to localize the site of A-V block in type I and II block.

Figure 1 is an example of Wenckebach or Mobitz type I second degree A-V block. Electrograms were recorded simultaneously from the right atrial appendage (RA), bundle of His (BH), and right ventricles along with a lead II electrocardiogram. The bundle of His electrogram contains three deflections: an atrial deflection, His bundle spike, and a ventricular septal complex. Each of the His bundle depolarization complexes is designated by a small letter *h*.

In the lead II ECG, it can be noted that the first three P-R intervals progressively increase until the fourth P wave is not followed by a ventricular response. This 4:3 Wenckebach cycle then repeats again. An approximation of the conduction time through the A-V node can be determined by the interval between the atrial deflection in the RA electrogram to the His bundle spike (*h*) in the BH electrogram. The fact that the progressive increase in the P-R interval in this typical example of Mobitz

* Presented by Dr. Moore at the Symposium on Cardiac Arrhythmias, June 10, 1972, at Virginia Beach, Virginia. These studies were supported in part by grants from the American Heart Association 71-787 and USPHS (HE-04885-13).

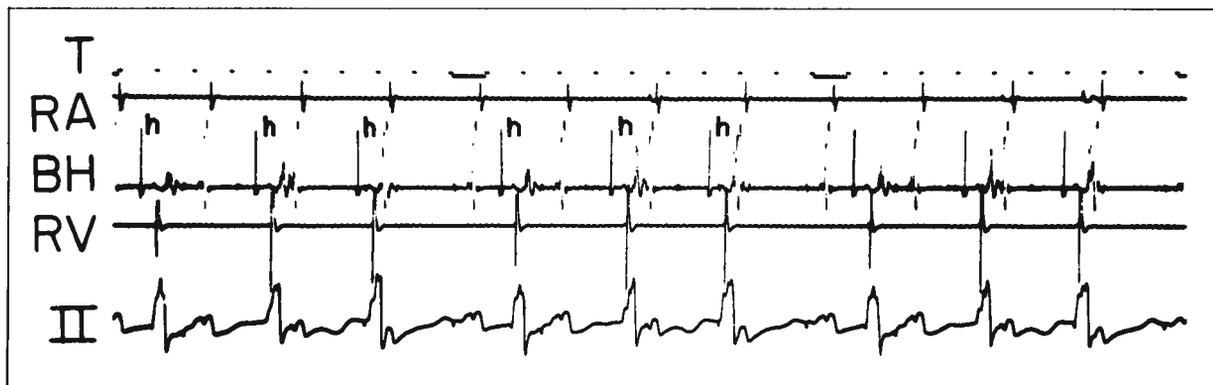


Fig. 1—Mobitz type I block due to conduction failure within the A-V node of the *in vivo* dog heart. Bipolar electrograms were recorded from the right atrium (RA), bundle of His (BH), and right ventricle (RV) together with the lead II electrocardiogram (II). The timing signal (T) denotes 100 msec. The h indicates the His spike in the BH electrogram.

I block is a reflection of a progressive increase in A-V nodal conduction time as demonstrated by the atrial to His conduction time between the first and third beats. The fourth atrial deflection is not followed by a His bundle spike. Therefore, A-V block of this atrial beat occurred at a site above the bundle of His recording site, that is, within the A-V node. Note that the interval between His bundle depolarization and ventricular depolarization did not change in any of the conducted beats, thereby indicating that ventricular conduction in Mobitz type I block is normal. Type I Mobitz block can be a functional type of A-V block when associated with rapid atrial pacing. Most normal human hearts will exhibit Wenckebach type block upon rapid atrial pacing. Again, this type of second degree A-V block is nearly always due to conduction delays and block within the A-V node.

Figure 2 is presented to further demonstrate that the prolongation and block of A-V conduction in type I block occurs within the A-V node. Atrial (RA) and ventricular (RV) electrograms were recorded simultaneously with transmembrane potentials from two single A-V nodal fibers (N and NH) during 3:2 Wenckebach. Ten msec and 100 msec time dots are indicated in the top trace, T. The first atrial response was conducted to the upper A-V nodal fiber (N) with little conduction delay. Conduction time from the upper nodal fiber (N) to the lower A-V nodal (or upper bundle of His) fiber, labeled NH, required 110 msec. Conduction from the NH fiber to the RV extracellular electrode required only 58 msec. The increase in atrial (RA) to ventricular (RV) conduction time of the second beat resulted

from slowed conduction within the A-V node as demonstrated by the increased time for the excitation wave to be transmitted from the upper to lower A-V node, that is, the N-NH interval (A-V conduction time) for the second response is 60 msec longer than for the first beat. Conduction time between the NH fiber to RV extracellular electrogram was 12 msec longer than for the first beat. The third atrial response (RA) failed to be conducted to the ventricles as shown by the absence of a ventricular depolarization complex in the RV electrogram. Block of this atrial beat occurred within the A-V node at a location between the impaled upper and lower A-V nodal recording sites. Clearly, this example of 3:2 Wenckebach resulted from block within the A-V node.

In Mobitz type II second degree A-V block, the P-R interval is usually normal and remains

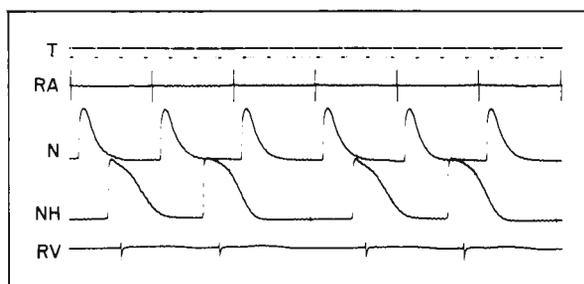


Fig. 2—Mobitz type I block due to conduction failure within the A-V node of the *in vitro* rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV) together with transmembrane potentials from the high A-V node (N) and low A-V node (NH). The timing signal (T) denotes 10 msec and 100 msec intervals.

constant preceding the dropped beat. Figure 3 is an example of Mobitz II block in a patient with left bundle branch block. As mentioned previously, Mobitz type II block is usually accompanied by some form of bundle branch block. In figure 3, electrograms were recorded from the bundle of His, atrium, and ventricles together with ECG leads 1, 2 and V_1 . His bundle depolarization complexes are denoted by "h". The first three beats were conducted normally to the bundle of His; conduction time from the His bundle to the ventricles was prolonged to 70 msec. The fourth atrial response resulted in a bundle of His depolarization complex, but was not accompanied by ventricular depolarization. The atrial to His bundle conduction time for the dropped ventricular beat was normal. Therefore, in this typical case of Mobitz type II block, conduction failure occurred within the ventricular specialized conduction system at a site below the A-V node and bundle of His. The fourth ventricular beat is an idioventricular escape beat. The P-R intervals before and after the "dropped" beat are identical and within normal limits. On a routine ECG, the presence of an intraventricular conduction defect accompanying second degree A-V block would be highly suggestive that block developed below the A-V node.

Figure 4 demonstrates type II Mobitz block in the isolated rabbit heart. In this experiment the heart was paced from the atrium at a constant rate



Fig. 3—Mobitz type II block in a patient with left bundle branch block. Catheters were used to record from the bundle of His (BH), the right atrium (RA), and right ventricle (RV) together with three simultaneous electrocardiographic leads (I, II and V_1). The h in the BH record indicates the His spike. (Reproduced with permission from B. N. Goldreyer and from *Annals of Internal Medicine* 77:132, 1972.)

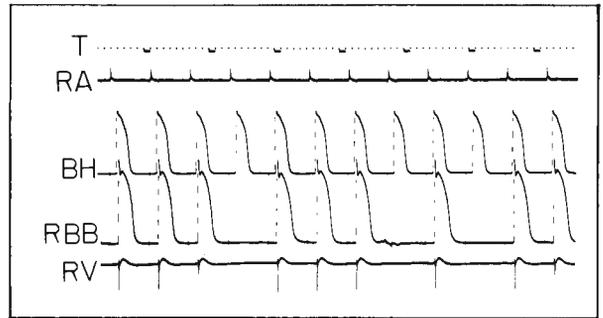


Fig. 4—Mobitz type II block occurring in the His-Purkinje system of the *in vitro* rabbit heart. Bipolar electrograms were recorded from the right atrium (RA), and right ventricle (RV) together with transmembrane potentials from the bundle of His (BH) and right bundle branch (RBB). The timing signal (T) denotes 100 msec and 1 sec intervals. (Reproduced by permission of The American Heart Association, Inc. from J. F. Spear and E. N. Moore, "Electrophysiologic Studies on Mobitz Type II Second Degree Heart Block," *Circulation* 44:1090, 1971.)

of 99 per minute; this rate caused some beats to be dropped abruptly. Electrograms were recorded from the right atrium (RA) and right ventricle (RV) simultaneously with transmembrane action potentials recorded from a bundle of His fiber (BH) and right bundle branch fiber (RBB). Time marks (T) denote 100-msec and 10-sec intervals. Notice that each right atrial electrogram was accompanied by an action potential recorded from the bundle of His, and that the atrial-to-His bundle conduction time (RA-BH) and His-to-right ventricular conduction time (BH-RV) remained constant. The bundle of His action potential was not accompanied by a right bundle branch action potential when the atrial impulse failed to be conducted to the ventricles (fourth, eighth, and tenth RA responses). Therefore, in this example of Mobitz type II block, conduction failure occurred below the bundle of His somewhere above the impaired bundle branch fiber. Figures 3 and 4 confirm studies completed in man where the sudden dropped beat in type II second degree block results from block within the ventricular specialized conduction system.

Figures 3 and 4 demonstrate the usual site of conduction failure in Mobitz type II block, that is, block within the VSCS below the A-V node and bundle of His. However, in some rare instances a constant P-R interval with sudden dropped beats may result from block within the A-V node. These cases of Mobitz type II block are usually associated

with a prolonged P-R interval and a normal QRS complex (4). In these rare cases of Mobitz type II block where block develops within the A-V node, one would not expect as grave clinical consequences as those associated with A-V block below the bundle of His within the VSCS.

Figure 5 presents an example of a constant P-R with a sudden dropped beat developing due to A-V nodal conduction block. The data was recorded in an *in vivo* dog preparation in which electrograms were recorded from the right atrium (RA), bundle of His (H), and left endocardial Purkinje fiber (LPF) simultaneously with the lead II electrocardiogram (II). Time marks denote 100 msec intervals. The right atrium was paced at a basic cycle length of 258 msec (heart rate of 234 per min). At this rapid rate, conduction time through the A-V node was somewhat prolonged, but the P-R interval of 0.14 sec is still within the normal range in the dog (0.06 to 0.15 sec). Every fourth atrial beat in figure 5 was made premature by 20 msec. In the standard electrocardiographic tracing, this would mean that every fourth response would have a variation in the P-R interval of 0.5 mm, that is, a sinus arrhythmia was present which would be barely perceptible in the routine ECG tracing. This small variation in the P-R interval resulted in 4:3 second degree block; it can be observed in the lead II ECG that the fourth atrial response is blocked. As so commonly occurs in clinical cases of type II block in man, the P-R interval following the blocked beat was slightly shorter than that for the responses preceding the blocked beat. The fact that bundle of His and left Purkinje electrograms were not recorded

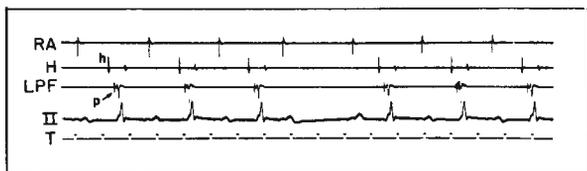


Fig. 5—Mobitz type II block due to conduction failure within the A-V node of the *in vivo* dog heart. Bipolar electrograms were recorded from the right atrium (RA), bundle of His (H), and left Purkinje fiber (LPF) together with a lead II electrocardiogram (II). The timing signal (T) denotes 100 msec intervals. The h in the H electrogram indicates the His spike, and the p in the LPF electrogram indicates the Purkinje spike. (Reproduced by permission of the American Heart Association, Inc. from J. F. Spear and E. N. Moore, "Electrophysiologic Studies on Mobitz Type II Second Degree Heart Block," *Circulation* 44:1091, 1971.)

during the dropped response demonstrates that in this instance of type II Mobitz second degree block, conduction failure occurred above the bundle of His, rather than within the VSCS as is the usual case in man. This finding is important since it points out that type II block can occur above the bundle of His as well as within the VSCS, and that small variations in cycle lengths can determine whether an atrial response is or is not conducted to the ventricles. Similar findings of type II block above the His bundle occurred when A-V conduction was depressed by vagal stimulation or digitalis toxicity (6).

Recent studies indicate that concealed His bundle extrasystoles or echo beats can cause pseudo-type II block (5, 6). Such findings have been dependent upon the chance occurrence of an extrasystole or echo beat blocking a regularly conducted beat. In our experiments we were able to demonstrate pseudo-type II block consistently by evoking extrasystoles in the bundle of His using a technique for intracellularly stimulating and recording through the same microelectrode. This technique allows precise localization of the site of stimulation as well as direct verification of activation of the same cell.

In figure 6, simultaneous atrial and ventricular electrograms are shown as well as a transmembrane potential recording from the bundle of His in an isolated rabbit heart. The ladder diagram is included

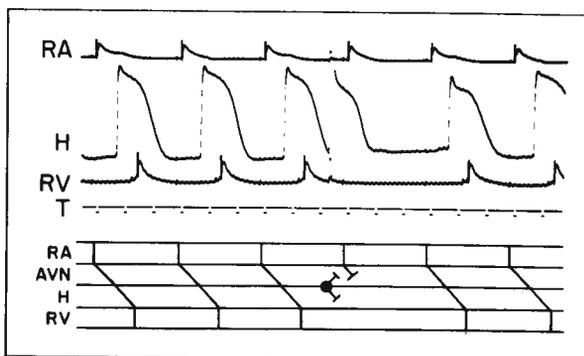


Fig. 6—Pseudo-Mobitz type II block due to a premature concealed impulse arising in the bundle of His in the *in vitro* rabbit heart. Bipolar electrograms were recorded from the right atrium (RA) and right ventricle (RV) together with the transmembrane potential from the bundle of His (H). The timing signal (T) denotes 100 msec intervals. The ladder diagram below demonstrates the conduction sequence through the right atrium, A-V node (AVN), His bundle and right ventricle. (Reproduced by permission of The American Heart Association, Inc. from J. F. Spear and E. N. Moore, "Electrophysiologic Studies on Mobitz Type II Second Degree Heart Block," *Circulation* 44:1093, 1971.)

TABLE 1. PATTERNS OF MOBITZ TYPE I AND TYPE II HEART BLOCK

DESCRIPTION	CLINICAL APPEARANCE		CAUSE OF CONDUCTION FAILURE		
	P-R interval	QRS duration	block in AVN	block in VSCS	"pseudo-block"
Mobitz I (increasing P-R interval)	increased increased	increased normal	possible likely	likely possible	possible possible
Mobitz II (constant P-R interval)	normal increased	increased normal	possible likely	likely possible	possible possible

below the analog tracings as an orientation to the sequence of conduction. Notice that after the third conducted beat a premature action potential is evoked in the bundle of His by stimulation through the recording microelectrode. The premature action potential is concealed both antegradely and retrogradely but has the effect of blocking conduction of the subsequent atrial activation (fourth atrial response). The electrocardiographic pattern that this intervention produces is "pseudo" Mobitz type II block with the site of block occurring within the A-V node.

Table I summarizes various possibilities for Mobitz types I and II second degree A-V block. Mobitz type I, or Wenckebach block, with progressive P-R prolongation is usually associated with a normal QRS complex and block within the A-V node. At rapid atrial rates, this can be a functional type of block without any pathology being present in the A-V node. Mobitz type I block following myocardial infarction in which block develops within the A-V node usually is a reversible arrhythmia. When block occurs below the A-V node within the ventricular specialized conduction system, a graver prognosis would usually be given.

Mobitz type II block, in which the P-R interval is constant preceding the dropped beat, is usually associated with bundle branch block, prolonged QRS complex, and a high instance of Adams-Stokes attacks. In these cases, the site of A-V block is nearly always below the A-V node within the ventricular specialized conduction system. In rare instances, a variation of Mobitz type II block in which the P-R interval is fixed but prolonged and the QRS complex is normal may be encountered where the site of block is within the A-V node. Thus, second degree A-V block is one instance where His bundle electrocardiography may be indicated. This is true since from the clinical standpoint it is predominantly the site of block rather than the P-R interval

which determines the significance of the block; block above the bundle of His is usually benign while block below His bundle is usually malignant.

REFERENCES

1. DREIFUS, L. S., WATANABE, Y., HAIAT, R., AND KIMBIRIS, D. Atrioventricular block. *Amer. J. Cardiol.* 28:371, 1971.
2. LANGENDORF, R., COHEN, H., AND GOZO, E. G. Observations on second degree atrioventricular block, including new criteria for differential diagnosis between type I and type II block. *Amer. J. Cardiol.* 29:111, 1972.
3. NARULA, O. S. AND SAMET, P. Wenckebach and Mobitz type II A-V block due to block within the His bundle and bundle branches. *Circulation* 41:947, 1970.
4. ROSEN, K. M., LOEB, H. S., GUNNAR, R. M., AND RAHIMTOOLA, S. H. Mobitz type II block without bundle branch block. *Circulation* 44:1111, 1971.
5. ROSEN, K. M., RAHIMTOOLA, S. H., AND GUNNAR, R. M. Pseudo A-V block secondary to premature nonpropagated His bundle depolarizations. Documented by His bundle electrocardiography. *Circulation* 42:367, 1970.
6. SPEAR, J. F. AND MOORE, E. N. Electrophysiologic studies on Mobitz type II second degree heart block. *Circulation* 44:1087, 1971.

PANEL DISCUSSION

Dr. Baird: Dr. Bigger, if a patient presents with a Mobitz type II block and syncope, although you have not documented that the patient had third degree block, what would you recommend with regard to permanent pacemaker therapy, or would you recommend pacemaker therapy without documentation of third degree A-V block?

Dr. Bigger: With Mobitz type II block or even

with left axis deviation, right bundle branch block, and even one nonconducted beat and the history of syncope, I think there is sufficient indication for a pacemaker. I think there is almost unanimous agreement on that. There are groups now, however, who would put pacemakers in those patients with a vague history of dizziness, light-headedness, fatigue, and marked left axis deviation with right bundle branch block. I want to tell this audience that this is a highly experimental approach and not of proven benefit. People can carry the pattern of marked left axis right bundle branch block and, if they have never dropped a beat, they may carry that pattern for 20 years without experiencing difficulty. In my view, it has by no means been proven that you should put in a pacemaker unless you have seen dropped beats. However, it is very clear in several prospective studies that once you have seen one drop beat in a patient with marked left axis deviation and a right bundle branch block type pattern on the electrocardiogram, then a very high percentage of those patients will be in complete heart block within one year to 18 months.

Dr. Moore: Would you put a pacemaker in a patient with Mobitz type I block in which a His bundle electrogram demonstrates that type I block is due to progressive delay in the His-Purkinje system?

Dr. Bigger: I have studied only three such patients myself. All of them had marked left axis with right bundle branch block and subsequently showed Wenckebach phenomenon with the H-V getting longer and longer in the His bundle study. One characteristic of the body surface electrogram is that the increments in P-R interval prolongation are not large. The entire Wenckebach cycle tends to be just two or three beats; the beat drop is more abrupt. It is strikingly different to the eye than the usual type I A-V block. I think these patients do deserve pacemakers.

Dr. Scherlag: Dr. (Onkar) Narula and Dr. (Philip) Samet have published a paper on Wenckebach not only below the His bundle but also within the His bundle itself. I think one uses the well-accepted clinical criteria that if one sees a drop beat and one knows it is in the His-Purkinje system, certainly that is a strong indication for pacemaker therapy. The problem, and I think this is an important one that others have not really dealt with, is that the natural history of such cases is not well known. I do not know if any physician would want to chance

just following a case of that type because of the possibility that Stokes-Adams might occur a short time afterwards. So, in general, most people want to be safe and would put a pacemaker in under those circumstances. I believe that is what happened to those patients who were described by Narula, *et al.*

Dr. Bigger: I would have to agree with that last statement except that I think it is possible to follow natural history such as when using a demand pacemaker. You can see when the pacemaker starts to stimulate or how often it is active by the usual ambulatory monitoring techniques. If the pacemaker activates, you could bring them in and with temporary transvenous catheters inhibit the demand pacemaker for study. I do not think we completely lose our opportunity for studying the natural history of a patient by introducing therapy.

Dr. Moore: I personally would want a pacemaker just to be sure.

Dr. Bigger: I do not think it is reasonable to implant pacemakers in left axis right bundle branch block. No such indication is evident in second degree A-V block. Yet in some places every patient with that pattern gets a pacemaker. I am not sure all of those physicians are carefully following the patients as a study to see what happens in real life. This is clearly a very experimental type of program to be carrying on.

Dr. Moore: Recently, in a patient with a right bundle branch conduction delay, we demonstrated that slight variations in the P-P interval could result in a sudden dropped beat and a Mobitz type II electrocardiogram. In this patient we did the same thing we did in the dog; that is, we drove the atrium rapidly so that the A-V conduction time was slightly prolonged, but still giving a normal P-R. The QRS was abnormal because of the underlying bundle branch conduction problem. By bringing in one P wave 20 msec early we were able to have this atrial response block within the A-V node. Again, this would look like a Mobitz type II with a sudden drop beat due to the slight prematurity of the atrial response. However, the dropped beat was blocked within the A-V node and, thus, probably should not require a pacemaker.

Dr. Baird: Dr. Bigger, how would you manage a patient with left bundle branch block and syncope that was presumably a recent onset?

Dr. Bigger: How old is this man?

Dr. Baird: He is sixty-seven.

Dr. Bigger: Well, this electrocardiogram, as I see

it of course, is left bundle branch block, and the P-R is probably 19 or 20, clearly prolonged. Did the left bundle branch block just come on about the time of the syncope?

Dr. Baird: It was a persistent left bundle.

Dr. Bigger: Of known duration or just encountered?

Dr. Baird: It was at least two months in duration. A previous tracing was taken two months before he had the syncope and, at that time, during his routine physical examination, the electrocardiogram appeared to be completely within the normal limits.

Dr. Bigger: It is difficult to be sure syncope had anything to do with his heart. It would require a work-up involving the extracranial vascular system and possible CNS causes of syncope.

Dr. Baird: He was lying down one Sunday night and suddenly developed seizure-like activity. He then awoke in about 10 or 15 seconds and resumed normal activity for the evening. We did just as you suggested and had the neurology service evaluate him. They found nothing neurological. We did Holter monitoring, and there was no evidence of A-V block or any dropped response. The patient felt, like Dr. Bigger, that there was possibly no justification for a pacemaker. The question is, would a His bundle recording be of any benefit in a patient of this type in determining whether he deserves consideration for demand pacing, particularly in the presence of the history of recurring syncope?

Dr. Bigger: This is a question Dr. Ken Rosen has been particularly interested in, the H-V time in left bundle branch block. The probability of developing complete heart block may relate to whether or not the H-V interval is long when the body surface cardiogram shows complete left bundle branch block. I think his hope is that H-V time will become a criteria that will be helpful in pointing to those who should and those who should not have pacemaker therapy. I have not been entirely convinced, but it seems a fruitful area for study.

Dr. Scherlag: I can appreciate the perplexity of the problem, and it is rather important because of the studies in Miami. There they have done several hundred cases, and they feel that with an H-V time of 70 or more and with this kind of history, such a patient is possibly going to have another seizure. I think that the H-V time would be an important objective measure to get.

Dr. Baird: Our patient refused pacemaker therapy, so we taught his wife cardiopulmonary resuscitation. However, with recurrent attacks he agreed to have a pacemaker and while waiting in the clinical center, he again had syncope. On this occasion, his wife successfully resuscitated him. The following day during elective pacemaker surgery, he developed complete A-V block. At this time, he has done relatively well, but A-V block was finally documented.