Mechanisms of A-V Block*

LEONARD S. DREIFUS, M.D.

JOSEPH McMENAMIN, M.D.

DEMETRIS KIMBIRIS, M.D.

From the Departments of Medicine, Physiology, and Biophysics, Hahnemann Medical College, Philadelphia, Pennsylvania

Within recent years an abundance of information has become available concerning the pathology, electrophysiology, anatomy, and clinical significance of disturbances of atrioventricular (A-V) conduction. Interest on this subject apparently began in 1827 with a description by Adams (1) of syncope associated with a slow heart rate and subsequent observations by Stokes (27) in 1846. Wenckebach (36) (1899) and Hay (7) (1906) described atrioventricular conduction block and ushered in the era of eponyms and synonyms in the classification of atrioventricular conduction disturbances. The issue heated up intensely in 1924 when Mobitz (15) classified A-V block according to rather precise criteria. In the following years, numerous clinical and experimental studies appeared in the medical literature. In 1941, Katz (9) attempted to describe the clinical correlation in the presence of various types of A-V block. Uhley and Rivkin (28, 29) first described the ECG pattern following the interruption of the main and peripheral branches of the canine right (1961) and left (1964) bundle branch system, and in 1963, Lenègre (11, 12, 13) and Lev (14) initiated the intense anatomical studies that led to the more recent concepts of intraventricular conduction disturbances. Precise experimental studies by Lenègre (11, 12, 13), Lev (14), Pruitt (22), and Rosenbaum (23) offered a logical classification of block within the fascicles of the Purkinje system. By the mid-1960's, Hoffman and Cranefield (8), Paes de Carvalho (20), Watanabe and Dreifus (30, 31), and others had explored the electrophysiologic mechanisms of atrioventricular conduction delay at the cellular level. Studies in man using His bundle electrograms by Damato and associates (2) and Narula and coworkers (17, 18, 19) confirmed the findings in earlier animal experiments. However, the importance of a more precise classification of A-V block came into sharp focus with the development of electronic pacing and the dramatic lifesaving results which followed. Unfortunately, too little is known about the life expectancy in patients with A-V block, and the medical literature is often distorted by a few scattered cases with unusually long survival or by including cases of A-V block engendered by an acute myocardial process (5). It is our intention to review the present anatomic, electrophysiologic, and clinical knowledge in an attempt to define A-V conduction disturbances. It is probably wise to consider first the classical definitions set forth by Wenckebach (36) and Mobitz (15, 16).

In his original paper in 1899, Wenckebach (36) described a progressive prolongation of the a-c interval (interval between atrial and ventricular contractions) until one ventricular contraction dropped out. Following a pause, the a-c interval was shortest, which suggests improved conductivity. Impairment of conductivity as judged from the increment of the a-c interval was most marked in the second conducted beat and much less in subsequent beats. This resulted in a quickening of the radial pulse. However, the increment of a-c interval was often again greater immediately before the dropped beat in the presence of higher conduction ratios, resulting in a slowing of the pulse. When Mobitz (15) for the first time classified incomplete A-V conduction disturbances in 1924, he termed the above

variety Type I, which subsequently became known by the name of "Wenckebach periodicity."

In contrast, Mobitz (16) called a block Type II when a ventricular complex dropped out without any change in the P-R interval of the electrocardiogram in immediately preceding beats. He also mentioned that, in the Type II variety, often many successive ventricular beats dropped out causing prolonged asystole, despite preceding periods of 1:1 conduction with a normal P-R interval. Hence, it must be reemphasized that the original classification of the two types of "partial heart block" was based entirely on variation or constancy of the A-V conduction time.

Clinical Findings. *First Degree Atrioventricular Block.* First degree block does not itself produce any symptoms. From a physical standpoint the presence of this type block may be suspected by the finding of a soft first heart sound. Levine and Harvey (1949) provided us with an explanation of this finding. These workers reported that the intensity of a first heart sound depended upon the position of the cusps of the A-V valves at the onset of systole. If ventricular systole occurs shortly after atrial systole, the A-V valves are open wide and will be closed violently and abruptly during systole producing a loud first sound. When the interval between atrial and ventricular systole is longer, the cusps will tend to return to their original position so that the sound produced at the time of ventricular systole would be much softer. This suspicion may be confirmed when inspection of the jugular pulse discloses a delay between the A and V waves.

Second Degree and High-Grade Atrioventricular Block. Second degree and high-grade atrioventricular block may or may not produce symptoms. Second degree A-V block with Wenckebach periods must be differentiated from the pause following an extrasystole. This can usually be done with auscultation. In block there is ventricular acceleration before the pause. Difficulty may arise in diagnosis because occasionally a blocked atrial premature beat may cause a pause in the sinus rhythm. If the P wave is hidden in the T wave, interpretation may be quite difficult. From the physical standpoint, however, the extrasystole will produce a cannon wave in the neck. More advanced second degree block with 2:1 A-V ratio produces a marked bradycardia.

Electrocardiographic Findings. The traditional classification of atrioventricular block involves three major types. In first degree, there is merely a prolongation of the P-R interval, and every atrial impulse is conducted into the ventricle. Second degree A-V block has been divided into two sub types: Mobitz Type I which is equated with the traditional Wenckebach (15) periodicity, in which the P-R interval is gradually prolonged and eventually the QRS complex drops out (figs. 1 and 2), and Mobitz Type II second degree block, characterized by the sudden dropping out of a QRS complex without progressive prolongation of the P-R interval (figs. 3 and 4). Complete or third degree A-V block is identified by independent activation of atria and ventricles with no conduction seen when physiologic P-R intervals are possible. The ventricles beat independently, each with its own pacemaker. The difficulty with this traditional classification is that the site of the A-V block is not specifically identified. In addition, it cannot be utilized in 2:1 conduction or in any rhythm other than sinus (figs. 5 and 6). Hence, more precise identification of the type and site of conduction block is mandatory, as specific clinical programs must be organized. There is no problem with first degree block; however, with second degree block, classification based on the width of the QRS interval will apply to all conduction ratios including 2:1, high grade A-V block, and in certain instances of atrial fibrillation (figs. 3, 4, 5, and 6).
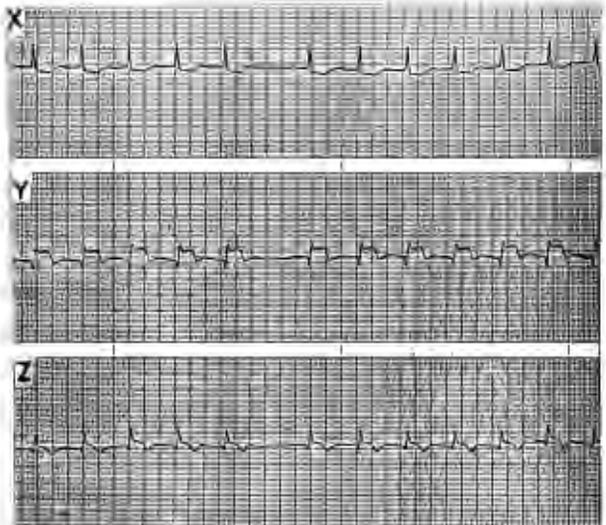

Fig. 1—Orthogonal leads X, Y, Z. A sinus rhythm is present at a rate of 75/min. The P-R interval becomes progressively prolonged with 0.22 sec to 0.32 sec before the 6th P wave fused with the T wave is dropped. This is a Type I block (Wenckebach) associated with an acute inferior wall infarction.
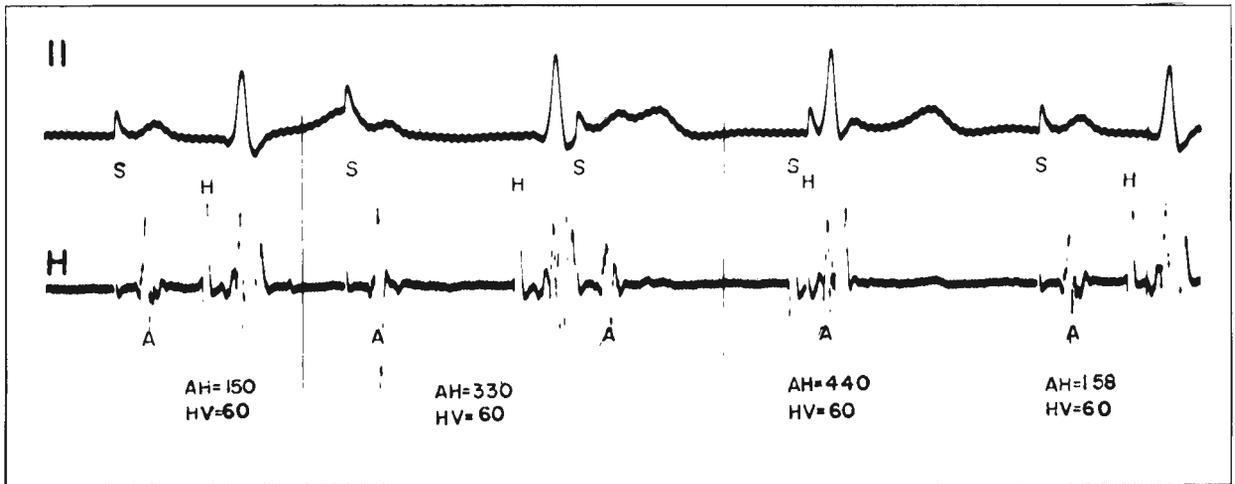
Fig. 2—Second degree block Type I, produced by atrial stimulation, lead 2 and His bundle electrogram shown. The A-H interval increases from 150 to 440 msec before the 4th atrial impulse is not conducted to the ventricles.

Confusion occurs when one talks exclusively of the progressive or sudden increase of the P-R interval before the dropped beat in attempting to classify the two types of block, as this criteria can be applied only in the presence of regular supraventricular rhythm and second degree A-V block associated with conduction ratios greater than 2:1 (33) (fig. 7). Furthermore, instances of atrial fibrillation and higher grades of A-V block cannot be considered in this classification (fig. 6).

In figure 7, taken from the same patient within a few seconds, both types of conduction block are demonstrated. In the upper strip (lead 3) sudden dropping out of a QRS complex is evident after beats 1, 6, and 7. The P-R remains constant at 0.20 seconds before the block occurs. This is Type II conduction block. However, in the lower strip, the P-R interval increases from 0.20 to 0.32 seconds before the third and ninth P waves are not conducted. This represents a Type I conduction block.
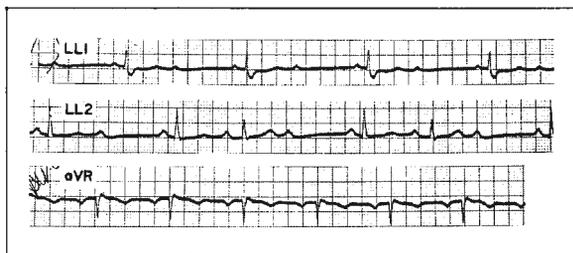

Fig. 3—Second degree block Type II variety. In lead 1, 2:1 conduction is seen; in lead 2, 3:2 conduction is present. AVR shows 1:1 conduction.

However, the QRS complexes are narrow, and block, in both instances, is most likely within the A-V node.

On the other hand, the QRS duration cannot always identify the precise site of conduction block. Type I or the Wenckebach variety can occur in all excitable tissue, and along the entire A-V transmission system (4). However, the nature of conduction delay usually localizes the block in the intranodal region of the A-V node (6). Further problems may arise when more than one region of block may be present in the same patient. From the clinical standpoint, it is the location of the block that largely determines the significance, rather than the variation or constancy of the P-R interval. Mobitz originally described the high incidence of Adams-Stokes attacks as well as complete heart block in cases of Type II variety. Later Katz (9) and Donoso and associates (3) confirmed the sinister prognosis associated with block and wide QRS complexes. Similar observations were made by Lenègre (12), Scanlon (24, 25), and Haiat (6) and their co-workers. In the latter study, major neurologic or cardiac symptoms were present in 86.2% of patients with A-V block associated with wide QRS complexes as contrasted to 37.5% of those patients with narrow QRS complexes. Futhermore, the incidence of sudden cardiovascular death was more than twofold in the group with wide QRS complexes. Second degree and high-grade A-V block offered a similar prognosis.
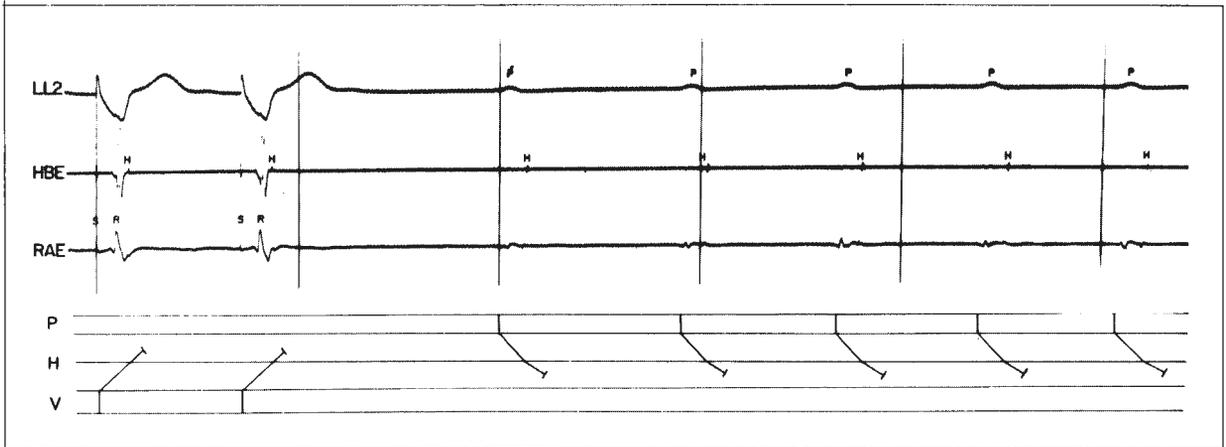
Fig. 4—His bundle electrogram, first two beats are produced by ventricular pacing. P waves are conducted to His bundle but are blocked below His. This type of conduction block is usually associated with Type II conduction.

With the development of cardiac pacemakers as well as the expanding knowledge in precise localization of the pharmacologic action of antiarrhythmic and cardiotonic agents, the clinician must acquire a firm understanding of the nature of A-V transmission. Digitalis, acetylcholine, and ischemia appear to slow intranodal conduction, while procainamide, quinidine, propranolol, potassium salts, and lidocaine slow conduction above the A-V node and in the subjunctional region (21, 34, 35). For practical purposes, the site of block can be identified by the duration of QRS in most instances, and His bundle electrocardiography will add little to clinical management. If progressive P-R prolongation is seen with a wide QRS complex, two levels of block may be present, but therapy is predicated on the lowest level of block. Conduction delay of the Wenckebach variety is most common in the "N" region of the A-V node but can be seen in the subjunctional region of the A-V transmission system (19) and even between contiguous ventricular fibers (4), but offers little as a prognostic sign alone.
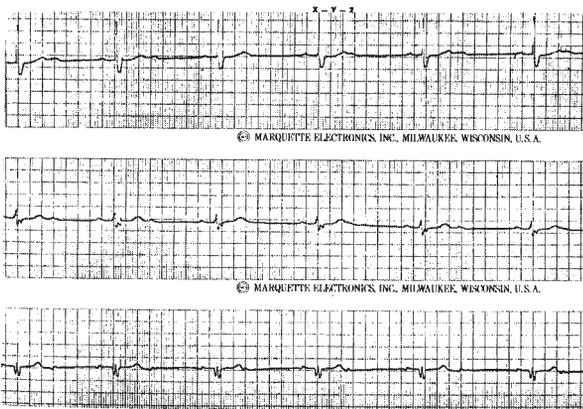

Fig. 5—Orthogonal leads X, Y, Z. 2:1 conduction is present and QRS complexes are wide. This type of conduction cannot be classified, block probably exists in the fascicles and is subjunctional in location.
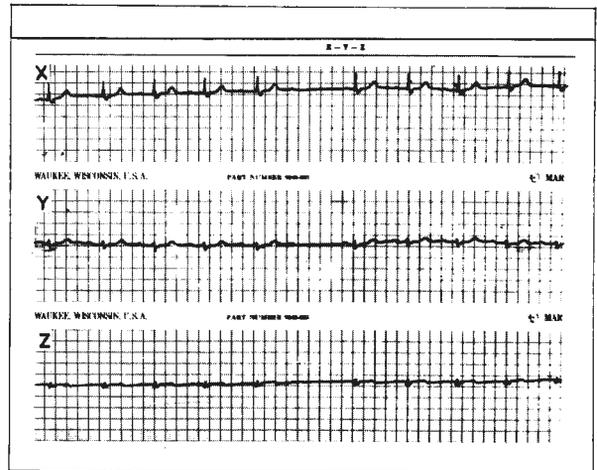

Fig. 6—Orthogonal leads X, Y, Z. QRS complexes are wide and show a right bundle branch system block configuration with an S wave appearing in lead X. A QRS complex fails to appear following the 6th P wave. P-R interval had become progressively longer from 0.18 sec to 0.32 sec before dropping out of QRS. Two levels of block may be postulated, the first within the A-V node, causing Wenckebach periodicity, and second, a subjunction block in the fascicles of the right bundle branch system.
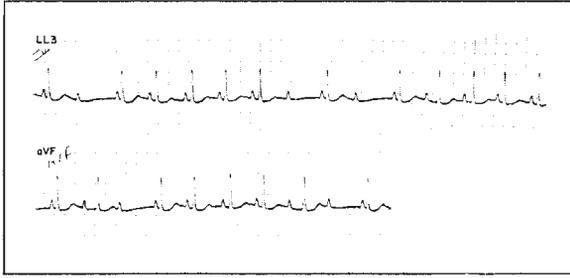
Fig. 7—Second degree block showing both Type I and Type II conduction block (see text).

Determination of the varieties of A-V block is predicated on the precise identification of the site(s) of conduction delay as prognosis, and therapy must follow on this basis. Further electrophysiologic and pharmacologic studies will undoubtedly reveal other mechanisms on the nature of A-V transmission.

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