Second-degree Atrioventricular Block

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WENCKEBACH	extsuperscript{1, 2} AND HAY,	extsuperscript{3} by analyzing the A, C and V waves in the jugular venous pulse, described two types of second-degree atrioventricular (AV) block. After the introduction of the electrocardiograph, Mobitz	extsuperscript{4} classified the two types of block as type I and type II. Electrocardiographically, typical type I second-degree AV block is characterized by progressive PR prolongation culminating in a nonconducted P wave, while in type II second-degree AV block, the PR interval remains constant before the blocked P wave. In both instances, the AV block is intermittent, generally repetitive and may involve several P waves in a row. Usually, the eponym Mobitz type I and Mobitz type II AV block is applied to the two types of block, while the term “Wenckebach block” refers to type I AV block only.

The purpose of this paper is to provide a brief review of clinically relevant electrophysiologic and electrocardiographic concepts of AV conduction and block, and a critical evaluation of the present classification of second-degree AV block.

Electrophysiological Characteristics of the AV Node and His-Purkinje System

Since the difference between type I and type II AV block, in most instances, probably relates to the site at which the block occurs, it is important to review the electrophysiological properties of the tissues involved.

Transmembrane action potentials recorded from cells within the AV node differ markedly from those recorded in the His-Purkinje system. AV nodal cells display reduced action potential amplitude, duration, resting potential, conduction velocity, maximum rate of rise of the upstroke (dV/dt), overshoot, safety factor for conduction and threshold of activation, compared with cells in the His-Purkinje system.

Recovery of excitability in AV nodal cells is delayed beyond the time for restoration of the membrane potential to its full diastolic potential (time dependence), while refactoriness is voltage-dependent in normal cells of the His-Purkinje system. Summation is another feature of AV nodal cells.

Based on studies in the rabbit AV node, the middle or N portion does not exhibit automaticity, while normal cells of the His-Purkinje system commonly undergo spontaneous diastolic depolarization.

The ionic currents responsible for the depolarization and repolarization of cardiac cells appear to be different for AV nodal cells and cells in the His-Purkinje system. In the His-Purkinje system, the fast, inward, sodium current, which is responsible for the rapid upstroke of the cardiac action potential, triggers a second, slower current carried by calcium (and possibly sodium) ions, which is responsible primarily for the plateau phase of the cardiac action potential.

These two inward currents, interdependent and more complex than simply a rapid current followed by a slow component, are present in specialized conducting tissues and in atrial and ventricular muscle cells. In the N region of the AV node, it is possible that only the second, slow current may be operative, and propagation through this area may occur normally without engaging the fast component.

Cells on either side of the AV node may demonstrate a continuum: Depending upon their distance from the N region, these cells show progressively less dependence on the slow-current contribution to depolarization.

Slow-channel blockers, such as manganese and verapamil, strikingly depress AV nodal propagation without significantly affecting conduction in the His-Purkinje system or in ventricular or atrial muscle. Tetrodotoxin, the poison obtained from the puffer fish, blocks the fast channel, making His-Purkinje cells inexcitable, and fails to inhibit AV nodal activity.

These distinctive electrophysiologic differences between normal cells in the AV node and normal cells in the His-Purkinje system account for differences in
the nature of conduction and block. Subjecting cells in the His-Purkinje system to a variety of interventions, such as ischemia, elevated potassium concentrations or other ionic perturbations, depolarizing currents, or superfusion with ionic blockers such as tetrodotoxin, may depress the normal fast response and, under appropriate circumstances, may evoke slow conduction that can be a typical slow response, a depressed fast response, or possibly a mixture of the two (for review, see Cranefield). Such depressed His-Purkinje conduction may respond more like the normal AV node and show large increments in conduction time before a blocked impulse.

Mechanisms of Block

Various mechanisms have been proposed to explain the nature of type I block, including fatigue, accumulation of extracellular potassium ions and encroachment of succeeding impulses on incompletely recovered tissue, possibly due to prolongation of the absolute and relative refractory periods. Slow conduction can result in lengthening of the action potential duration and the refractory period, leading to a positive feedback interplay of RP and PR intervals that progresses to the point of block. The block then shortens the action potential duration and refractoriness in fibers that were activated proximal to the site of block, and the sequence restarts.

Decremental conduction, in which the properties of the fiber change along its length so that the action potential loses its efficacy as a stimulus for the unexcited fiber ahead of it, may be responsible for type I block. Alternatively, electrotonus may play an important role. Decreased stimulating efficacy of a transmitted electrotonic potential, rather than a regenerative, propagating impulse, has been suggested as the cause of Wenckebach periodicity.

Recently, it was demonstrated during 4:3 AV nodal Wenckebach cycles in the dog, that the effective refractory period after the fourth (blocked) beat was shorter than after any other beat. The effective refractory period cumulatively and progressively increased in succeeding cycles, so that the effective refractory period of the third beat exceeded the duration of the basic cycle length, and caused the fourth beat to block. The functional refractory period, on the other hand, decreased in succeeding Wenckebach cycles, primarily because the control conduction time increased in successive beats of the cycle.

Other phenomena implicated in the genesis of block include automaticity, geometric configuration of the tissue, the role of intercellular connections, uniformity of the propagating wavefront, alterations in excitability, and concealed re-entry. All of these factors may be modulated in vivo by potential hemodynamic alterations and the influence of the autonomic nervous system.

Despite multiple mechanisms that may be responsible for causing type I AV block, relatively few hypotheses have been advanced to explain the electrophysiologic basis of type II AV block. Recently, it has been suggested that the two types of block may be different manifestations of the same electrophysiologic mechanism.

Evaluation of Electrophysiologic Evidence for One Type of Second-degree AV Block

El-Sherif et al. showed that dogs subjected to ligation of the anterior septal artery developed second-degree AV block in the proximal His-Purkinje system. After coronary artery occlusion, the block was initially preceded by no perceptible or only 1-2-msec increments in conduction time. After 1-2 hours, the increment in conduction time before the block increased 100-200 msec. Upon recovery of the excised tissue during superfusion in vitro, the large increments that occurred before the blocked impulse once again decreased, but not consistently in each preparation. These observations were offered as support for the concept that second-degree AV block encompassed a spectrum that ranged from no perceptible or small increments preceding the blocked impulse “at an early stage of departure from normal” to increments of several hundred milliseconds as the abnormality increased. The authors suggested that type I and type II AV block “represent various degrees of the same electrophysiologic mechanism,” differing only quantitatively, in the size of the increments.

While these studies demonstrated that ischemia can alter conduction and block in the His-Purkinje system, the data do not support the conclusion that the mechanism giving rise to the block preceded by small increments was the same as when the block was preceded by large increments. In these studies, El-Sherif and colleagues compared the response of cells in the His-Purkinje system exposed to short periods of ischemia with that of cells exposed to long periods of ischemia. If these cells were electrophysiologically different — and it is likely that they were after exposure to different durations of ischemia — the cause of the initial block may have differed from the mechanism that caused the later block. Also, the site of early and late block may have changed. These investigators observed that paroxysmal AV block (that is, block of multiple, successive P waves) only occurred early, when the increments were still small, and could not be produced in their preparation when the increments were large. This finding also supports the concept that the two types of block are different.

From these studies and other studies of block in the His-Purkinje system, several facts emerge: 1) Most, but possibly not all, examples of apparent type II AV block in the His-Purkinje system are preceded by small increments in conduction time that can be found if carefully searched for. 2) The His-Purkinje system, both in animals and in patients who appear to have typical type II AV block, can be perturbed by ischemia or other factors (figs. 1 and 2) to show large increments in conduction before the blocked impulse.

However, increments in conduction time before a blocked impulse, whether large or small or present or absent, are descriptive phenomena and cannot be
equated with the electrophysiologic mechanisms that give rise to the block. Block can result from complex interactions of multiple mechanisms.5, 21-42

The present classification of only two types of second-degree AV block may be too simplistic, as it is based on a description of only input and output data: A certain number of atrial impulses enter a "black box," i.e., the AV node and His-Purkinje system, and

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**Figure 1.** Type II second-degree AV block in a patient with left-axis deviation and right bundle branch block. During right atrial pacing at a constant cycle length (510 msec), P waves intermittently blocked in the His-Purkinje system, distal to the His bundle recording site. No change occurred in the duration of the PR interval (188 msec), AH interval (125 msec) or HV interval (42 msec) before or after the blocked P wave. 

**Figure 2.** Increase in the HV interval before block in the same patient as in figure 1. Continuous recording: The stimulus and part of the last paced atrial complex (unlabeled) in the top recording are repeated as the first stimulus and first paced atrial complex in the bottom recording. Right atrial pacing at a shorter cycle length (450 msec) than in figure 1 resulted in 3:2 conduction. The HV interval increased from 44 to 81 msec before the blocked P wave and shortened to 44 msec after the block. The PR interval only increased from 197 to 226 msec before the block, because of shortening of the AH interval from 133 to 125 msec. Note a slight change in the contour of the second QRS complex, when the HV interval prolonged. A = atrial deflection; H = His deflection; V = ventricular deflection; RA = right atrial electrogram; S = stimulus. Paper speed was 250 mm/sec.
a certain number of His-Purkinje or ventricular impulses exit in a certain pattern. From an analysis of these patterns, one attempts to deduce the mechanisms that cause the block. Probably, different electrophysiologic mechanisms within the "black box" can cause similar patterns to emerge, and, conversely, similar electrophysiologic mechanisms can produce different emerging patterns. Even if the "black box" is partially opened, and the cells are studied by the microelectrode technique, depressed transmembrane potentials may appear remarkably similar, but be ionically and therefore, "mechanistically" distinct.

Differentiating Type I and Type II
Second-degree AV Block

Although the classification is descriptive, clinically separating second-degree AV block into type I and type II serves a useful function, and, in most instances, the differentiation can be made easily and reliably from the surface ECG.

Clinically, type II AV block often antedates the development of Adam-Stokes syncope and complete AV block, while type I AV block with a normal QRS complex is generally more benign, and does not progress to more advanced forms of AV conduction disturbance. In the patient with an acute myocardial infarction, type I AV block usually accompanies an inferior myocardial infarction, is transient and does not require temporary pacing, while type II AV block results in the setting of an acute anterior myocardial infarction, may require temporary or permanent pacing, and is associated with a high mortality.

While type I conduction disturbance is ubiquitous and may occur in any tissue in the in situ heart, as well as in vitro, the site of block for the usual forms of second-degree AV block can be judged with sufficient reliability from the surface ECG to permit clinical decisions without requiring invasive electrophysiologic studies in most instances (table 1). Type I AV block with a normal QRS complex almost always takes place at the level of the AV node (proximal to the His bundle). An exception is the uncommon patient with type I intrahisian block. Type II AV block, particularly when it occurs with a bundle branch block, is localized in the His-Purkinje system. Type I AV block in a patient with a bundle branch block may represent block in the AV node or in the His-Purkinje system. Type II AV block in a patient with a normal QRS complex may be due to intrahisian AV block, but the block is likely to be type I AV nodal block, which exhibits small increments in AV conduction time.

The above generalizations encompass the vast majority of patients who present with second-degree AV block. However, certain caveats must be heeded to avoid misdiagnosis because of subtle ECG changes or exceptions:

1) Two:one AV block may be a form of type I or type II AV block (fig. 3). If the QRS complex is normal, the block is more likely to be type I, located in the AV node, and one should search for a transition of the 2:1 block to 3:2 block, during which the PR interval lengthens in the second cardiac cycle. If a bundle branch block is present, the block may be located either in the AV node or in the His-Purkinje system.

2) AV block may occur simultaneously at two or more levels, and render the distinction between type I and type II difficult.

3) If the atrial rate varies, it may alter conduction times and cause type I AV block to simulate type II or change type II AV block into type I. For example, if the shortest atrial cycle length that just achieved 1:1 AV nodal conduction at a constant PR interval is decreased by as little as 10 or 20 msec, the P wave of the shortened cycle may block at the level of the AV node without an increase in the antecedent PR interval. Apparent type II AV block in the His-Purkinje system may be converted to type I in the His-Purkinje system in some patients by increasing the atrial rate.

4) Concealed premature His depolarizations may create electrocardiographic patterns that simulate type I or type II AV block (fig. 4).

5) Abrupt, transient alterations in autonomic tone may cause sudden block of one or more P waves without altering the PR interval of the conducted P wave before or after the block. Thus, apparent type II AV block would be produced at the A-V node. In the absence of atrial pacing, a burst of vagal tone would probably lengthen the PP interval as well as produce AV block.

6) The response of the AV block to autonomic changes, either spontaneous or induced, to distinguish type I from type II AV block, may be misleading. Though it is considered that vagal stimulation generally increases and atropine decreases the extent of type I AV block, such conclusions are based on the assumption that the intervention acts primarily on AV conduction and fail to consider rate changes. For example, an injection of atropine that minimally improves conduction in the AV node, but markedly increases the heart rate, can increase AV conduction time and the degree of block as a result of the faster atrial rate. Conversely, if an increase in vagal tone,
Figure 3. Two: one second-degree AV block distal (top panel) and proximal (bottom panel) to the His bundle recording site in two different patients. Conventions as in figure 1. BAE = bipolar esophageal recording; BHE = bipolar atrial electrogram; BEE = bipolar His electrogram. (Reproduced with permission from Zipes DP, Watanabe AW, Besch HR: Clinical electrophysiology and electrocardiography, In The Science and Practice of Clinical Medicine. Clinical Cardiology, edited by Wilkerson JT, Sanders CA. New York, Grune and Stratton, 1977, pp 235–248.)

produced pharmacologically or by carotid sinus massage, minimally prolongs AV conduction time, but greatly slows the heart rate, the net effect on type I AV block may be an improvement in conduction. Analogous examples can be proposed for changes effected by alterations in adrenergic tone due to infusion of isoproterenol or after exercise.

7) During type I AV block with high ratios of conducted beats, the increment in the PR interval may be quite small and simulate type II AV block if only the last few PR intervals before the blocked P wave are measured. By comparing the PR interval of the first beat in the long Wenckebach cycle with that of those beats immediately preceding the blocked P wave, the increment in AV conduction becomes readily apparent.62

8) Finally, the classic AV Wenckebach structure depends on a stable atrial rate and a maximum increment in AV conduction time for the second PR interval of the Wenckebach cycle, with progressive decrease in subsequent beats. Unstable or unusual alterations in the increment or atrial rate, often seen with long Wenckebach cycles, result in atypical forms of type I AV block and are quite common.63

PR Shortening after the Blocked P Wave

The duration of the PR interval of the first P wave that conducts to the ventricle after AV block is a source of disagreement. After discounting AV junctional escape complexes that follow a nonconducted sinus P wave at a short interval, the question is: In type II AV block, can the PR interval after the blocked P wave shorten and if so by how much? Does “marked” shortening (i.e., >20 msec)64 eliminate the possibility of type II AV block?

During type I or type II second-degree AV block, the PR interval after the blocked P wave generally is the shortest, because the pause after the blocked P wave provides a period of recovery for conduction distal to the site of block. Increments in AV conduction time that accrued during the sequence of conducted beats before the block are lost in this recovery period, and the PR interval usually shortens by an amount equal to this increment.

During the usual type I AV nodal block, the increments in AV conduction time are fairly large, and the decrease in the PR interval after the blocked P wave will be similarly large. During the usual type II
AV block in the His-Purkinje system, the increments, if any, in His-Purkinje conduction time are small or nonmeasurable. In this instance, the PR interval after the blocked P wave will shorten minimally or not at all. However, if the increments in the His-Purkinje conduction time are large (fig. 2), the reduction in the PR interval will also be large, even exceeding 20 msec. However, the argument is sophistic because, given the latter situation, the block is called type I in the His-Purkinje system, rather than type II, and large increments, as well as marked PR shortening, are expected.

Finally, the PR interval in the scalar ECG is made up of conduction through the atrium, the AV node and the His-Purkinje system. An increment in HV conduction can be masked in the scalar ECG by a reduction in the AH interval, and the resulting PR interval will not reflect the entire increment in His-Purkinje conduction time (fig. 2). The AH interval can shorten due to changes in autonomic tone, multiple AV nodal pathways, concealed conduction or other reasons. Also, a shorter AH interval must shorten the HH interval of the preceding cycle, a change that can be conductive to His-Purkinje conduction delay or block.

These potential variations in the duration of the PR interval make difficult the accurate deduction of the type of block based on a particular pattern of the duration of the PR interval immediately before and after the blocked P wave. Nevertheless, certain generalizations about the PR interval can be made. First, very long PR intervals (>200 msec) are more likely to result from AV nodal conduction delay (and block), with or without concomitant His-Purkinje conduction delay. Second, when second-degree AV block occurs during incomplete AV dissociation, variations in the PR interval that reflect the usual inverse relationship between RP and PR intervals generally are due to changes in AV nodal conduction time and suggest that the block is type I; constant PR intervals during ventricular captures are more consistent with the presence of type II AV block. Finally, although it is clear that large increments in the PR interval before the blocked P wave and a large decrement in the PR interval after the blocked P wave may occur with His-Purkinje block, such PR changes would be more consistent with, though not diagnostic of, type I AV nodal block.

Some patients may require an electrophysiologic (His bundle) study to determine with certainty the nature and site of block. For example, patients with type I AV block and a bundle branch block, those with type II AV block and a normal QRS complex, those with fixed 2:1, or greater, degrees of block and those with a type of block that does not appear to conform to any of the patterns mentioned, may profit from such a study.

Limitation of Measurements

Measurement errors may prevent small changes in conduction time from being detected. At the standard electrocardiographic paper speed of 25 mm/sec, a
finely drawn line or the tip of the commonly used caliper has a width equal to approximately 5 msec; at a paper speed of 100 mm/sec (the usual recording speed for electrophysiologic studies in man) the line or caliper tip has a width of 1 or 2 msec. Recording speeds in the range of 250 mm/sec may be required to detect 1- or 2-msec changes (fig. 2).

Summary

1) While it is possible only one type of second-degree AV block exists electrophysiologically, the available data do not justify such a conclusion and it would seem more appropriate to remain a “splitter,” and advocate separation and definition of multiple mechanisms, than to be a “lumper,” and embrace a unitary concept.

2) The clinical classification of type I and type II AV block, based on present scalar electrocardiographic criteria, for the most part accurately differentiates clinically important categories of patients. Such a classification is descriptive, but serves a useful function and should be preserved, taking into account the caveats mentioned above. The site of block generally determines the clinical course for the patient. For most examples of AV block, the type I and type II classification in present use is based on the site of block. Because block in the His-Purkinje system is preceded by small or nonmeasurable increments, it is called type II AV block; but the very fact that it is preceded by small increments is because it occurs in the His-Purkinje system. Similar logic can be applied to type I AV block in the AV node. Exceptions do occur. If the site of AV block cannot be distinguished with certainty from the scalar ECG, an electrophysiologic study will generally reveal the answer.

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