Review

Second-Degree Atrioventricular Block: A Reappraisal

S. Serge Barold, MD, and David L. Hayes, MD

In this review, we discuss the various forms and causes of second-degree atrioventricular (AV) block and the reasons they remain poorly understood. Both type I and type II block characterize block of a single sinus P wave. Type I block describes visible, differing, and generally decremental AV conduction. Type II block describes what appears to be an all-or-none conduction without visible changes in the AV conduction time before and after the blocked impulse. Although the diagnosis of type II block is possible with an increasing sinus rate, absence of sinus slowing is an important criterion of type II block because a vagal surge (generally a benign condition) can cause simultaneous sinus slowing and AV nodal block, which can superficially resemble type II block. The diagnosis of type II block cannot be established if the first postblock P wave is followed by a shortened PR interval or is not discernible. A pattern resembling a narrow QRS type II block in association with an obvious type I structure in the same recording (eg, Holter) effectively rules out type II block because the coexistence of both types of narrow QRS block is exceedingly rare. Concealed His bundle or ventricular extrasystoles confined to the specialized conduction system without myocardial penetration and depolarization can produce electrocardiographic patterns that mimic type I and/or type II block (pseudo-AV block). All correctly defined type II blocks are infranodal. A narrow QRS type I block is almost always AV nodal, whereas a type I block with bundle branch block barring acute myocardial infarction is infranodal in 60% to 70% of cases. A 2:1 AV block cannot be classified in terms of type I or type II block, but it can be nodal or infranodal. Infranodal blocks require pacing regardless of form or symptoms. The widespread use of numerous disparate definitions of type II block appears primarily responsible for many of the problems surrounding second-degree AV block. Adherence to the correct definitions provides a logical and simple framework for clinical evaluation.

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ACC = American College of Cardiology; AHA = American Heart Association; AV = atrioventricular; ECG = electrocardiogram; WHO = World Health Organization

Second-degree atrioventricular (AV) block remains poorly understood despite major advances in cardiac electrophysiology in the past 3 decades. The literature has numerous differing definitions of second-degree AV block, especially Mobitz type II block.[1] Thus, during formal testing, physicians' scores in detecting second-degree AV block on an electrocardiogram (ECG) are poorer than their scores in detecting other arrhythmias.[2] Indeed, a recently published book correctly stated that "Mobitz II block is misunderstood more than any other abnormality of rhythm or conduction."[3] In this review, we discuss the reasons for these difficulties and outline the pitfalls associated with the diagnosis of second-degree AV block, with emphasis on use of correct definitions.

HISTORICAL BACKGROUND

In 1906, Hay[4] from Liverpool, England, described a new form of second-degree AV block now considered type II second-degree AV block. In 1924 Mobitz,[5] using an ECG, classified the well-known Wenckebach form of second-degree AV block as type I and characterized type II second-degree AV block as "the occasional block of one or more P waves with no change in the PR interval before and after the nonconducted P waves" (Figure 1). Mobitz also classified 2:1, 3:1, etc, AV block as type II only when the PR interval after the blocked impulse (or impulses) remained unchanged. Mobitz believed that conduction in type II block was an all-or-none phenomenon. The definitions of type I and II second-degree AV block still remain descriptive. Type I block describes ECG visible differing, generally decremental AV conduction with large or small increments in conduction time. Type II block describes what appears as an all-or-none conduction without visible changes in AV conduction time, as it appeared to Mobitz.

CHICAGO SCHOOL OF ECG: A NEW DEFINITION OF TYPE II SECOND-DEGREE AV BLOCK

In 1956, Katz and Pick,[6] renowned Chicago electrocardiographers, offered their definition of type II second-degree AV block, which differed substantially from Mobitz's original description. The wide acceptance of their new definition, even at present, appears primarily respon-

From the Electrophysiology Institute, Broward General Hospital, Ft Lauderdale, Fla (S.S.B.); and Division of Cardiology and Internal Medicine, Mayo Clinic, Rochester, Minn (D.L.H.).

Address reprint requests and correspondence to S. Serge Barold, MD, 6237 NW 21st Ct, Boca Raton, FL 33496 (e-mail: ssbarold@aol.com).

sible for many of the problems surrounding the understanding of second-degree AV block. In their 1956 classic book, Katz and Pick described second-degree AV block as follows:

> There are more P waves than QRS complexes. Each QRS can be related to a preceding P wave, but intermittently a single P wave is not followed by a QRS. Type II: In type II, dropping out of ventricular beats takes place without warning; that is, it occurs after a series of beats with a constant either normal or prolonged PR duration. Variation of PR duration is limited to the first beat after the pause in which the PR may be shorter than the otherwise constant PR interval in the record.

The degree of an acceptable PR abbreviation after a blocked beat for the diagnosis of type II block was not specified until 1972, when Langendorf et al[7] indicated that the PR shortening should be 0.02 second or less (Figure 2).[8] As a result, some contemporary definitions of type II block still contain vague statements that the PR interval may shorten or shorten slightly, but *slightly* is not defined.[9-11] Indeed, in various contemporary books, it is common to see tracings of "type II block" in which the first postblock PR interval is shorter than the others. Yet, in a later discussion of AV block, the authors do not specify shortening of the PR interval after the blocked beat as a permissible feature of type II block.[12] Possibly, they realized the diagnostic importance of constancy of all the conducted PR intervals.

Katz, Pick, and others also advocated searching for circumstances suggesting "type I (decremental) and type II (all-or-none) physiology" when typical type I and type II patterns were not recordable in second-degree AV block, but this approach may be misleading and is mostly of academic interest.[6,7,12]

CONTEMPORARY DEFINITIONS

Type I block and type II block are ECG patterns that describe the behavior of the PR intervals (in sinus rhythm) in sequences (with at least 2 consecutively conducted PR intervals) in which a *single* P wave fails to conduct to the ventricles. The anatomical site of block should not be characterized as either type I or type II because type I and type II designations refer only to ECG patterns.

Type II Block

The most appropriate and accepted definitions are those codified in 1978 by the World Health Organization (WHO)[13] and the American College of Cardiology (ACC)[14]; type II second-degree AV block is defined as the occurrence of a single nonconducted P wave associated with constant PR intervals before and *after* a single blocked impulse, as long as the sinus rate or the P-P interval is constant and there are at least 2 consecutively conducted P waves (ie, 3:2 AV block) to determine the behavior of the PR interval. The pause encompassing the blocked P wave must be equal to 2 (P-P) cycles[15-18] (Figures 2 and 3). Stability of the sinus rate is an important criterion because a vagal surge (generally a benign condition) can cause simultaneous sinus slowing and AV nodal block; this can superficially resemble type II second-degree AV block, which is always infranodal.[17,20-22] Although debate continues on whether type II block can occur in the AV node, careful analysis reveals that the cases claiming to show exceptions to the rule are invalid based on a strict definition of type II block.[23,24] Finally, in type II block, the PR interval can be normal or prolonged, and the QRS complex can be narrow or wide. Typically, the PR interval is normal, and the QRS complex is wide because the block is in the His-Purkinje system.

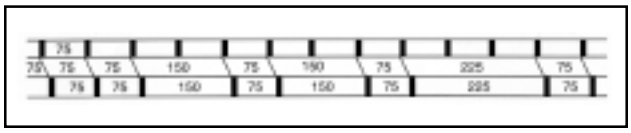

Figure 1. Type II atrioventricular (AV) block, showing atrial, AV, and ventricular intervals in hundredths of a second. Note the constant AV or PR intervals before and after the blocked impulse or impulses. From Mobitz.[5]

Behavior of the First Postblock PR Interval.—The diagnosis of type II block cannot be established if the first P wave after a blocked beat is absent or if the PR interval is shorter than all the other PR intervals of conducted P waves.[8,13,14,17] After a blocked impulse, a shorter PR can be due to improved conduction (type I block), AV dissociation linked to an escape beat, or preempted ventricular activation by an implanted pacemaker (Figure 2). In questionable cases, recording long tracings is important to establish with reasonable certainty that a shorter PR interval represents either a supraventricular conduction or a nonconducted P wave associated with a junctional escape QRS complex. Consequently, the diagnosis of type II block necessitates a statement about an unchanged PR interval of the first *conducted* beat after the blocked impulse. Type II block cannot be diagnosed when a shortened AV interval occurs after a blocked single P wave, regardless of the number of constant PR intervals before the block. This situation represents a type I pattern or is unclassifiable.[8] The sequence remains nondiagnostic because the site of block can be AV nodal or infranodal.

Behavior of the PR Interval After Block of 2 or More Consecutive P Waves.—When all PR intervals remain constant before and after AV block of 2 or more consecutive impulses in the setting of a stable sinus rhythm, the purist will insist on calling this pattern *type II AV block* based on the original description by Mobitz.[5] Some investi-

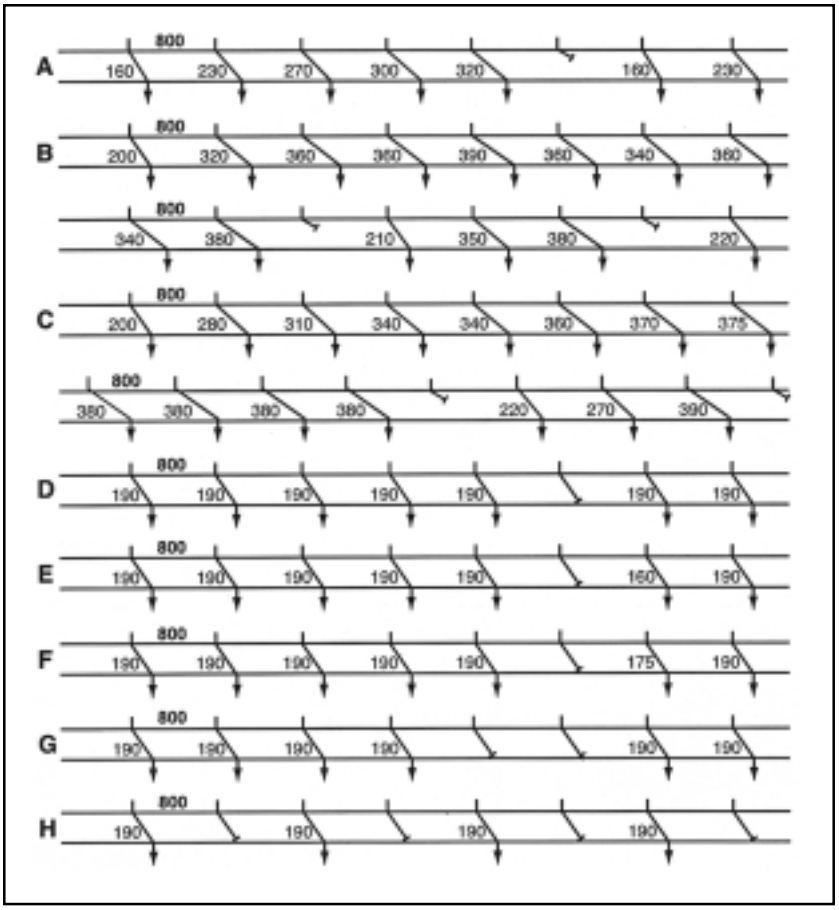

Figure 2. Various forms of second-degree atrioventricular (AV) block. The 3 levels represent activation of the atria, AV junction (PR intervals are shown between the lines), and ventricles, respectively. All values are in milliseconds. A, Classic type I AV block. B, Relatively long and atypical type I sequence. Note the irregular fluctuations of the PR intervals before the dropped beat. C, Relatively long and atypical type I sequence with several constant PR intervals before a dropped beat. Note the *shorter* PR interval after the blocked P wave. This pattern should not be called type II AV block. All the PR intervals in long rhythm strips must be examined, not merely several PR intervals preceding a blocked impulse. D, True type II AV block. Every atrial impulse successfully traverses the AV node, which is not afforded a long recovery time as occurs in type I AV block. Note that the PR interval *after* the blocked beat is unchanged. E, Dropped beat followed by a 30-ms shortening of the PR interval. This pattern should not be called type II AV block. It may be a type I AV block or unclassifiable if shortening of the PR interval is due to an AV junctional escape beat. F, Type II AV block according to the old definition of the Chicago School of Electrocardiography. This is now labeled *type I* with very small increments in conduction. Some investigators still call this arrangement *type II AV block*. The diagnosis of type II block cannot be made if the PR interval *after* the blocked beat is not equal to all the other PR intervals. G, Advanced second-degree AV block (failure of conduction of 2 consecutive P waves without warning). All the PR intervals are constant, including the first one after the block. This suggests infranodal AV block. Some investigators still use the original Mobitz definition and call this sequence *type II block*. H, Fixed 2:1 AV block. This cannot be classified as type I or type II block. Reproduced with permission from Barold and Friedberg.[8]

gators still respect the original definition.[25] According to the codified definitions,[13,14] such an observation should not be labeled *type II AV block* (but advanced second-degree block). Admittedly, the constant PR intervals strongly suggest infranodal block and the potential need for a pacemaker. When the first PR interval after the blocked P waves is not equal to previous PR intervals, the block can be either in the AV node or in the His-Purkinje system.

Problematic Definitions.—In 1956, Katz and Pick[6] described type II block as a condition that "develops without

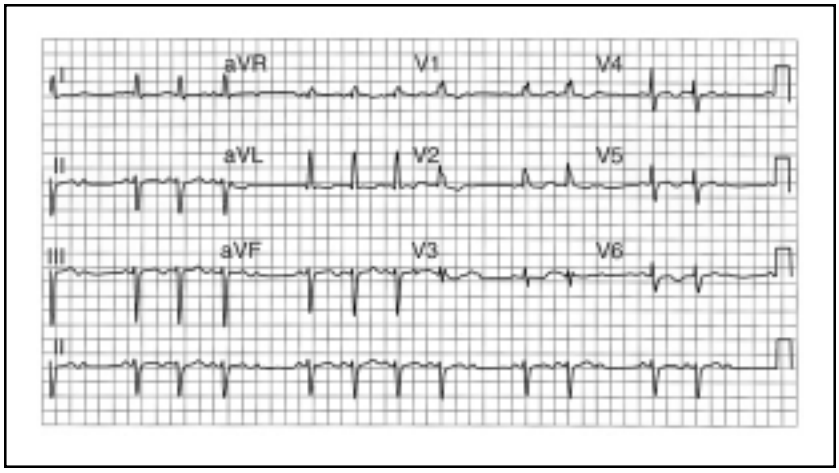

Figure 3. Sinus rhythm with second-degree type II atrioventricular (AV) block in the presence of right bundle branch block and left anterior hemiblock. The tiny q waves in V_2 and V_3 probably relate to left anterior hemiblock rather than old anterior myocardial infarction. Note that the sinus rate is constant and that the PR interval after the blocked beat remains unchanged. Reproduced with permission from Barold.[19]

warning, that is it occurs after a series of beats with a constant PR prolongation." Of note, this statement does not mention the number of constant PR intervals required before the blocked beat. The Katz and Pick description of a type II block created the still-popular definition of a type II block found in most textbooks, an "electrocardiographic pattern characterized by failure of a single impulse to conduct to the ventricles in the absence of antecedent lengthening of the PR interval (normal or prolonged)."[26-38] In fact, this definition of type II block also describes type I block when the terminal portion of a long atypical type I sequence shows PR intervals with no discernible or measurable change before the blocked impulse[1,8,39] (Figure 2). Consequently, by citing the Chicago definition of type II block, some investigators have diagnosed type II block with only 2 or 3 constant PR intervals before block of a single P wave (ignoring the first postblock PR interval).[23]

Definitions of type II block with statements such as "all conducted PR intervals are constant or all the conducted P waves have constant PR intervals"[12] also contain a loophole. Such definitions could be interpreted to mean that an absent or nonconducted P wave after the blocked impulse has no diagnostic value and can be ignored because it is *nonconducted*.

Type I Block

Based on the 1978 WHO and ACC statements, type I second-degree AV block can be defined as the occurrence of a single nonconducted P wave associated with *inconstant* PR intervals before and after the blocked impulse as long as there are at least 2 consecutive conducted P waves (ie, 3:2 AV block) to determine the behavior of the PR intervals.[13,14] The PR interval after the blocked impulse always shortens when the P wave is conducted to the ventricle. Variations in the sinus rate do not usually interfere with the diagnosis of type I block (Figure 2). Friedman[40] believes that the aforementioned definition is appropriate because the traditional concept that type I block is synonymous with the classic Wenckebach phenomenon is no longer tenable in view of the common occurrence of an atypical type I sequence.[41,42]

Atypical Sequences Are Typical of Type I Block.— The term *inconstant PR* or *AV intervals* is important because many type I sequences are atypical and do not conform to the traditional mathematical structure of the Wenckebach phenomenon associated with progressive prolongation of the PR intervals.[6,8,41,42] For example, the second (conducted) PR interval (after a blocked impulse) often fails to show the greatest increment. A PR interval may actually shorten and then lengthen in the middle of a type I sequence. In the middle or for a few beats just before termination of a sequence, the duration of PR intervals may show no discernible change[39] (Figures 2 and 4). The 1978 WHO document on the terminology of cardiac arrhythmias emphasized that most cases of type I block involve atypical varieties, and it promulgated a useful but generally ignored definition of type I block: "intermittent failure of impulse conduction in which the blocked impulse is preceded by prolongation of conduction time relative to the first conducted impulse."[13] The term *progressive* in the popular definitions of type I block should be replaced by *generally*

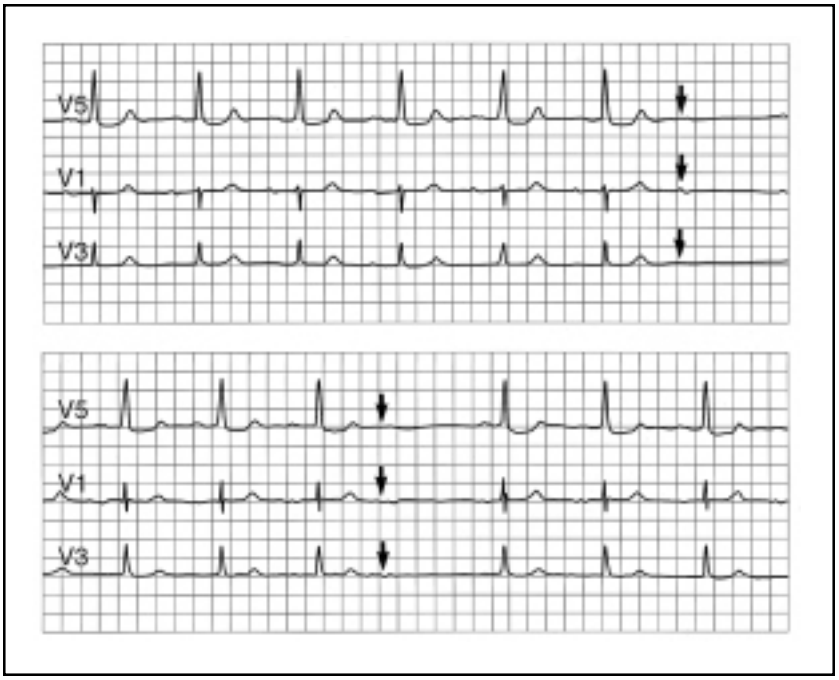

Figure 4. Two examples of narrow QRS complex atypical type I second-degree atrioventricular (AV) block registered in 3-lead Holter recordings. Top, Sinus arrhythmia. The last 3 PR intervals before the nonconducted P wave (arrows) are constant. Diagnosis of type II block is untenable because the first PR interval on the left is shorter than the ones immediately before the block. Actually, the first postblock P wave was conducted (not shown) with a shorter PR interval compared with the one before the block. The pattern is consistent with type I second-degree AV block. Bottom, The 3 PR intervals before the blocked P wave (arrows) are constant. The postblock PR interval is slightly shorter. The second postblock PR interval is longer than the first postblock PR interval, and the last PR interval is very slightly longer than the second postblock PR interval. The pause is slightly shorter than 2 P-P intervals. This establishes the diagnosis of type I block. Applying the strict definition of type II block to these 2 situations ensures that the correct diagnosis is made.

progressive to describe the changing PR intervals before a blocked impulse.

All Type II Blocks Are Really Type I Blocks.—Type II blocks are type I blocks with increments in AV conduction so miniscule that they cannot be recorded or measured with standard equipment.[8] Although a type II block is an artifact of the standard ECG because it cannot record ultrashort changes in AV conduction, the resultant all-or-none pattern remains immensely useful to localize the site of block.

SITE OF BLOCK

The anatomical site of a block should not be described as either type I or type II because these designations refer only to ECG patterns.[43] Any form of second-degree infranodal AV block should not be automatically called *type II block* unless it fits the aforementioned definition. Thus, all type II blocks are infranodal (discussed subsequently), but not all infranodal blocks are type II blocks. Similarly, labeling all forms of second-degree AV nodal block as type I blocks is inappropriate.

Type II Second-Degree AV Block

According to the strict definition, a type II block occurs in the His-Purkinje system[44-50] and rarely above the site of recording of the His bundle potential in the proximal His bundle or nodo-Hisian junction.[51,52] Type II block has not yet been convincingly demonstrated in the body of the AV node or the N zone. Most of the purported exceptions involve reports in which type I blocks (a shorter PR interval after the blocked beat) are claimed to be type II blocks by using loopholes in the definitions of second-degree AV block.[23,24] About 70% of cases of type II block are associated with bundle branch block, and 30% are associated with a narrow QRS complex and are therefore within the His bundle.[44-48]

A revision of the 1998 ACC/American Heart Association (AHA) guidelines for pacemaker implantation should

Table 1. **Indications for Permanent Pacing in Acquired Second-Degree Atrioventricular Block in Adults***

Permanent or intermittent second-degree AV block regardless of the type or the site of block, with correlated symptomatic bradycardia
Permanent or intermittent asymptomatic type II second-degree AV block
Asymptomatic type I or advanced second-degree AV block at intra-His or infra-His levels
Exercise-induced second-degree AV block (absent at rest) regardless of symptoms but in the absence of reversible ischemia
Infranodal second-degree AV block induced by atrial pacing, provided functional block is ruled out
Infranodal second-degree AV block induced by a pharmacological challenge with a type I antiarrhythmic agent in patients with undiagnosed syncope, bundle branch block, and negative results on an electrophysiologic study
Infranodal second-degree AV block induced by bradycardia (phase 4 block)
Second-degree AV block of any type in asymptomatic patients with neuromuscular diseases, eg, myotonic muscular dystrophy, Kearns-Sayre syndrome, limb-girdle dystrophy, and peroneal muscular atrophy

*AV = atrioventricular.

eliminate confusing recommendations for type II second-degree AV block.[53] In the section on acquired AV block, the guidelines state that "asymptomatic type II second-degree AV block, is class IIa indication" (width of the QRS complex is not stated). In the same clinical situation in the section on bifascicular and trifascicular block, the guidelines state that "bifascicular and trifascicular with type II second-degree AV block, symptoms not specified," constitutes a class I indication for pacing. A type II block should be a class I indication for permanent pacing, even in asymptomatic patients, because of its poor prognosis[54] (Table 1).

Type I Second-Degree AV Block

Decremental conduction is not diagnostic of AV nodal block. A type I block with a narrow QRS complex is almost always due to a lesion in the AV node because a type I block in the His bundle is rare. In a type I block with a wide QRS complex (≥0.12 second), except for acute myocardial infarction, the block is AV nodal in 30% to 40% of cases and is in the His-Purkinje system in 60% to 70% of cases[44-48] (Figure 5). Type I infranodal block indicates diffuse disease of the His-Purkinje system, and its prognosis is believed to be the same as for type II block; in most patients, even asymptomatic patients, a permanent pacemaker should be considered.[54] The diagnosis of type I infranodal block requires invasive His bundle recordings, and the increments in AV conduction occur in the HV interval. Type I infranodal block is commonly associated with tiny but measurable increments of AV conduction,[55] but this finding is not diagnostic because the increments of AV nodal conduction delay can also be miniscule.[56] The relative frequency of such patterns at these sites is unknown. Conversely, large increments in AV conduction strongly favor a site in the AV node, but large increments and atypical type I sequences can also occur with His-Purkinje disease.[39] Thus, the incremental pattern of type I

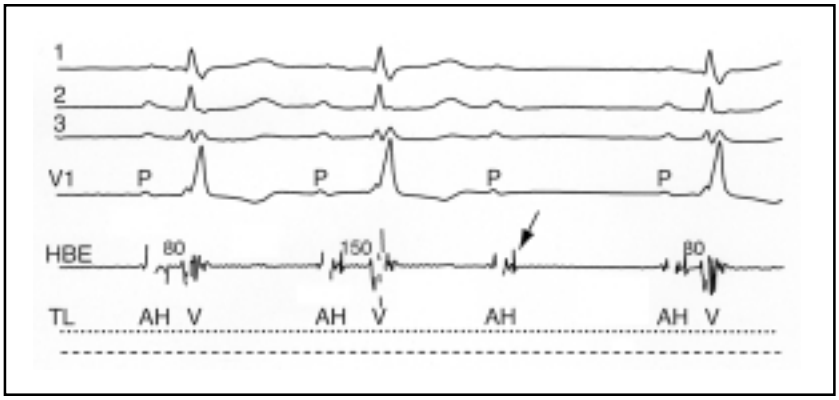

Figure 5. Sinus rhythm with second-degree type I 3:2 infranodal atrioventricular (AV) block and right bundle branch block. Note that the AH interval (reflecting conduction through the AV node) remains constant. The HV interval (reflecting conduction through the His-Purkinje system) increases from 80 milliseconds (following first P wave [P]) to 150 milliseconds (following second P wave). The third P wave is followed by a His bundle deflection (H) but no QRS complex. An AV block occurs in the His-Purkinje system below the site of recording of the His bundle potential (arrow). Note the shorter PR interval after the nonconducted P wave, a feature typical of type I second-degree AV block. A = atrial deflection; HBE = His bundle electrogram; TL = time lines, 50 milliseconds; V = ventricular deflection. Reproduced with permission from Barold.[19]

Table 2. **Indications for Invasive Study of Second-Degree Atrioventricular Block***

Asymptomatic type I second-degree AV block with bundle branch block
Asymptomatic advanced second-degree AV block with bundle branch block
Questionable diagnosis of type II block with a narrow QRS complex
Suspicion of concealed AV junctional or ventricular extrasystoles
Confirmation of bradycardia-dependent (phase 4) infranodal block in selected cases
Transient second-degree AV block with bundle branch block in patients with inferior myocardial infarction where the site of block is suspected to be in the His-Purkinje system rather than the AV node

*AV = atrioventricular.

block cannot provide an absolute diagnosis of the site of block, AV node vs His-Purkinje system.

The 1998 ACC/AHA guidelines for pacemaker indications state that "type I second-degree AV block at intra- or infra-His levels found incidentally at electrophysiological study performed for other indications is a class II a for pacing."[53] All asymptomatic patients with type I block with bundle branch block should be considered for an electrophysiologic study to determine the site of block (Table 2). An invasive study is usually not recommended or necessary in asymptomatic patients with type I block and a narrow QRS complex.[54]

INFLUENCE OF HEART RATE ON DIAGNOSIS

A modest increase or decrease in the sinus rate indicates the diagnosis of type I block. Classically, the sinus rate must be constant when PR intervals are measured to determine the presence of type II block. However, a block in the His-Purkinje system can be tachycardia dependent; thus, a type II block can be diagnosed in the setting of an increasing sinus rate. Second-degree (or third-degree) AV block absent at rest but precipitated with exercise is almost always due to His-Purkinje disease and represents a tachycardia-dependent block.[57,58]

The diagnosis of type II block has not been carefully studied during sinus arrhythmia. Type II block can be diagnosed when the sinus rate increases (and a block occurs in the shorter cycle) because a vagal effect can be ruled out based on the rate increase and provided there are no associated episodes of type I block. An AV block in a long cycle during sinus slowing is problematic because a vagal effect cannot be ruled out. However, the absence of an associated type I block, the presence of a wide QRS, and a repetitive pattern showing absolutely constant PR intervals before and after the single blocked impulse raise the suspicion of an infranodal lesion in a rare bradycardia-dependent or phase 4 block in the His-Purkinje system[59] (Table 1). This phenomenon should not be termed *type II block.* One should not diagnose a type II block with a narrow QRS if the P-P interval lengthens before the block, even if the PR intervals are constant before and after the block. This situation is almost always a type I variant with AV nodal block. A phase 4 block in this setting is extremely rare. The presence of phase 4 block can be suspected by observing prolonged ventricular asystole and the absence of type I block in long tracings. A definitive diagnosis of phase 4 AV block requires His bundle recordings (Table 2).

2:1 AV BLOCK

Second-degree AV block in the form of a 2:1 AV block or involving 2 or more consecutively blocked P waves is usually characterized as advanced second-degree AV block.[13,14] A 2:1 AV block can be in the AV node or the His-Purkinje system. Much of the confusion surrounding a 2:1 AV block can be traced to previous publications[6,7,12,60-64] and the unfortunate practice of investigators using the term *type I 2:1 AV block* when the lesion is in the AV node or there is evidence of decremental conduction and using the term *type II 2:1 AV block* when it is infranodal or there is evidence of all-or-none conduction. This practice violates the well-accepted traditional definitions of type I block and type II block based on ECG patterns, not on the anatomical site of block. Perhaps investigators who continue to use these designations still incorrectly believe that all type I blocks are confined to the AV node.[65-67] (Conversely, the belief that all type II blocks are infranodal is correct.) Consequently, they freely interchange descriptive patterns with anatomical site of disease.

In a 2:1 AV block, there is only 1 conducted P wave for each P wave blocked; thus, only 1 PR interval is available for examination before the block. A 2:1 AV block cannot be classified as type I or type II, a concept emphasized in the ACC/AHA guidelines for clinical intracardiac electrophysiologic studies.[68,69] Unfortunately, the 1998 ACC/AHA guidelines for pacemaker implantation do not mention a 2:1 AV block, and an advanced second-degree AV block is defined as a block of 2 or more consecutive P waves.[53]

Authors of a recent review stated that a 2:1 AV block and bundle branch block indicate block infranodal block and therefore indicate pacing, even in an asymptomatic patient.[27] This recommendation ignores the fact that, in this situation, the block is in the AV node in 15% to 20% of patients with a better prognosis and is not necessarily an indication for a pacemaker in an asymptomatic patient[44-48] (Table 2).

Both type I and type II blocks can progress to a 2:1 AV block, and a 2:1 AV block can regress to a type I or type II block. Consequently, the importance of the lesion in a 2:1 block can often be determined by the company it keeps. If the conduction ratio changes to or previous ECGs show a 3:2, 4:3, etc, AV block with at least 2 consecutively conducted P waves, the abnormalities may evolve into a type I, type II, or an unclassifiable situation in circumstances with a shorter PR interval after the blocked impulse. In a persistent 2:1 AV block, His bundle recordings are needed to localize the site of the block (Table 2).

3:1 AND 4:1 AV BLOCK

The sudden appearance of a 3:1 block or higher is sometimes mislabeled a *type II block*.[70] Although a 3:1 block occurs more commonly in the His-Purkinje system than in the AV node, it is illogical to call a block type II simply because it involves many consecutive sinus P waves.

ACUTE MYOCARDIAL INFARCTION

During vomiting or other strong vagal stimulation, a narrow QRS AV block is AV nodal and transient and does not require specific therapy. In acute myocardial infarction, a wide QRS type II block occurs only in an anterior infarction.[71] A narrow QRS type II second-degree AV block has not been reported as a complication of acute anterior myocardial infarction. Several investigators have reported the occurrence of a narrow QRS type II block in acute inferior myocardial infarction by using incorrect criteria for the diagnosis. Critical review of a large sample of study patients with inferior myocardial infarction failed to reveal a single case of type II block in this setting.[72-76]

Transient or persistent second-degree AV block of any form and associated bundle branch block in acute *anterior* wall myocardial infarction are almost certainly infranodal, and a permanent pacemaker is indicated if a vagal block is excluded. In contrast, in transient second-degree AV block (nodal) with bundle branch block in acute *inferior* wall myocardial infarction, no permanent pacemaker is needed (Table 3).

AUTONOMIC CHALLENGE FOR DIAGNOSIS

Carotid sinus massage, exercise, or administration of atropine has been advocated to distinguish between type I and type II block. The use of autonomic maneuvers may be misleading, and they are seldom needed in clinical practice.[77] The assumption that vagal stimulation and atropine will decrease the block in type I block is based on the effect on the AV node. If the effect is greater on the sinus rate than on the AV node, atropine will increase the degree of block, and conversely vagal stimulation may reduce it.

Table 3. **Indications for Permanent Pacing After Acute Myocardial Infarction***

Persistent or transient second-degree AV block in the His-Purkinje system associated with bundle branch block (anterior infarction). Transient second-degree AV block with acute inferior myocardial infarction is almost always AV nodal and not an indication for permanent pacing
Persistent advanced second-degree AV nodal block after 16 days

*AV = atrioventricular.

VAGALLY MEDIATED AV BLOCK

In 1978, Massie et al[20] reported that a vagal surge can cause simultaneous sinus slowing and AV nodal block, an often benign condition that can superficially resemble type II block. This phenomenon has been termed *apparent type II block*, but the best term is *type I variant*. A vagally mediated AV block occurs in the AV node, is generally benign, and must be carefully distinguished from a true type II block.[21,22,57,78] It is associated with a narrow QRS complex, and results of an electrophysiologic investigation are usually normal.

A vagally induced AV block can occur in otherwise normal patients, in those with cough or hiccups, and during swallowing or micturition when vagal discharge is enhanced. A vagally mediated AV block is characteristically paroxysmal and is often associated with clearly visible sinus slowing on an ECG. As a rule, AV nodal block is associated with obvious irregular and longer P-P intervals and is associated with bradycardia (not bradycardia dependent), ie, both AV block and sinus slowing result from vagal effects. Vagal AV block differs from neurally mediated (malignant vasovagal) syncope in which head-up tilt testing causes sinus arrest and rarely predominant AV block.

Depending on its timing and duration, vagal discharge can produce AV block without preceding prolongation of the PR intervals, but this does not constitute type II block (Figure 6). Moreover, the first PR interval after the blocked beat can be equal to the PR intervals before the vagal effect either from a fortuitous relationship of a nonconducted P wave and an escape QRS complex or from an actual conduction of the P wave with an unchanged PR interval. The latter occurs when a slower sinus rate facilitates AV conduction and overcomes the depressant effect of a residual vagal effect on AV conduction. Thus, when all the PR intervals remain constant, type II block will be diagnosed erroneously if sinus slowing is ignored. An increase of the P-P interval by as little as 0.04 second may occasionally be clinically important.[78-80]

The large variety of ECG patterns seen with vagal AV block depends on the interplay of several factors influenc-

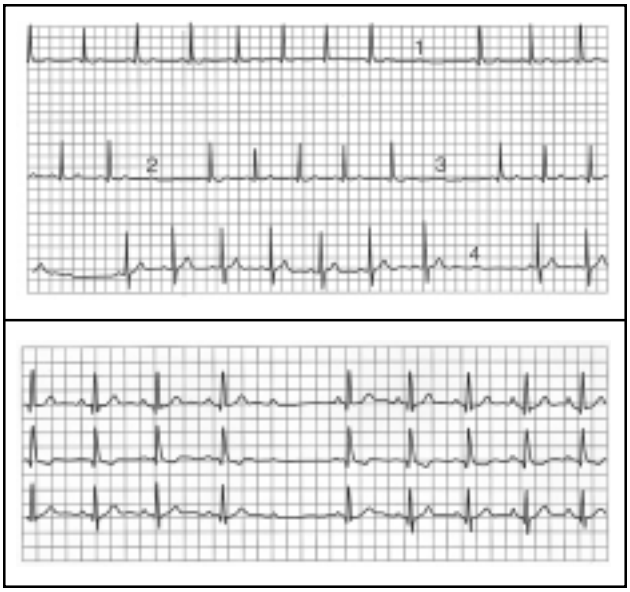

Figure 6. Top, Representative electrocardiographic (ECG) strips recorded during continuous monitoring in a 27-year-old man with a history of syncope. The patient was referred for implantation of a permanent pacemaker with a diagnosis of Mobitz type II second-degree atrioventricular (AV) block. Note that the nonconducted P waves labeled 1, 2, 3, and 4 occur coincidentally with sinus slowing secondary to increased vagal tone affecting both the sinus node and the AV node, with resultant AV nodal block. The PR interval after the blocked P waves (2, 3, and 4) shows subtle shortening compared with the PR interval before the nonconducted beats compatible with a diagnosis of type I second-degree AV block. Bottom, Holter recording shows a more typical pattern of type I second-degree AV block preceded by slight slowing of the sinus rate (left side of tracing). The occurrence of AV block with sinus slowing suggests a vagal phenomenon and AV nodal block (phase 4 infranodal block is rare, especially in the presence of narrow QRS complex). Type I and type II second-degree AV blocks can occur together in the same patient, but it is a rare combination seen almost exclusively in patients with bundle branch block. The upper strip shows "apparent type II AV block" or a variant of type I block. A tilt table confirmed the diagnosis of neurocardiogenic syncope; the patient has remained asymptomatic for 3 years while taking a small dose of β-blocker therapy. Reproduced with permission from Barold.[19]

ing the net vagal effect: (1) the moment in the cycle when the vagal effect occurs, (2) the intensity (and speed) of the vagal surge, (3) the atrial rate, (4) the sensitivity of the AV node, and (5) the background sympathetic activity. Although vagal stimulation usually causes simultaneous sinus bradycardia and depression of AV nodal conduction, it may occasionally be restricted primarily to 1 site. When the effect on the sinus node predominates, sinus bradycardia may occur without an obvious effect on the AV node. Sinus bradycardia itself may mask the depressing vagal effect on the AV node.

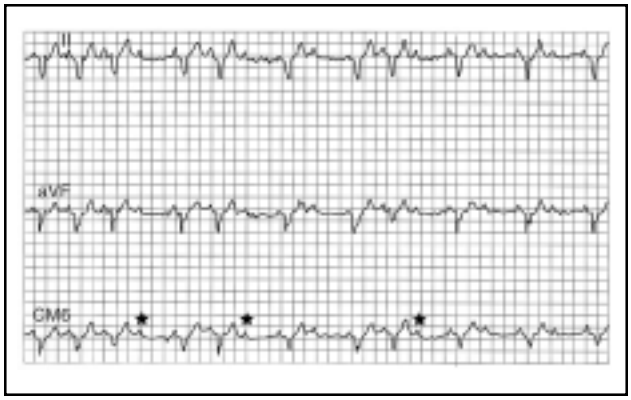

Figure 7. Type II atrioventricular (AV) block induced by exercise shown in 3-lead electrocardiogram (ECG) of a 68-year-old patient who had near-syncope while playing tennis. Holter recordings were unremarkable. The ECG showed sinus rhythm at 60 beats/min, complete right bundle branch block, and left anterior hemiblock. During treadmill testing, the patient developed type II block (stars), followed by 2:1 AV block (right side of tracing). The development of type II block is diagnostic of infranodal block.

AV BLOCK IN ATHLETES

Atrioventricular block in athletes is typically type I second-degree block and probably an expression of hypervagotonia related to physical training; it resolves after physical deconditioning.[81] This form of AV block may be associated with sinus bradycardia because the relative effects of sympathetic and parasympathetic systems on the AV and sinus node may differ. An AV block in athletes responds to exercise or atropine. Several investigators have indicated that type II second-degree AV block can occur in young athletes. The diagnosis of a type II AV block immediately suggests a permanent pacemaker. We believe that type II second-degree AV block (always infranodal) occurring in apparently healthy athletes should be diagnosed by using intracardiac recordings for confirmation (Table 2). In some articles, the reported occurrence of type II AV block appears related to failure to apply the correct definition of type II second-degree AV block.

The development of second-degree AV block with exercise is often infranodal[57,58] (Figures 7 and 8). This form of AV block is often reproducible in the electrophysiologic laboratory by rapid atrial pacing because it is tachycardia dependent (Figure 8). Exercise-induced second-degree AV block is rarely due to AV nodal disease or cardiac ischemia.

PERPLEXING SITUATIONS

The problems associated with the diagnosis of second-degree AV block are listed in Table 4. When confronted with a pattern that appears to be a type II block with a narrow QRS complex, one must consider the possibility of

a type I block without discernible or measurable increments in the PR intervals. Sinus slowing with AV block essentially rules out type II block. Although intense vagal tone, as during malignant vasovagal syncope, can be associated with block of multiple consecutive P waves, vagally mediated AV block rarely involves more than block of 2 consecutive P waves. Difficulty arises when the sinus rate is stable. When a narrow QRS type II-like pattern occurs with type I sequences (as in Holter recordings), a true type II block can be safely excluded because narrow QRS type I and type II blocks virtually never coexist within the His bundle. A true narrow QRS type II block is relatively rare and occurs without sinus slowing. It is typically associated with AV conduction ratios greater than 2 (3:1, 4:1, which are rare in vagal block) without associated type I structures.[17] Sustained advanced second-degree AV block is far more common in association with true type II block than with type I block or its variant. Based on these facts, the diagnosis of a true narrow QRS type II block almost never requires invasive His bundle recordings.

Finally, when evaluating second-degree AV block, one must remember that (1) a sudden increase in the atrial pacing rate during invasive testing may cause a functional AV block in the His-Purkinje system in a normal person[83] (Figure 9) and (2) concealed His bundle or ventricular extrasystoles (confined to the specialized conduction system and not propagated to the myocardium) can produce

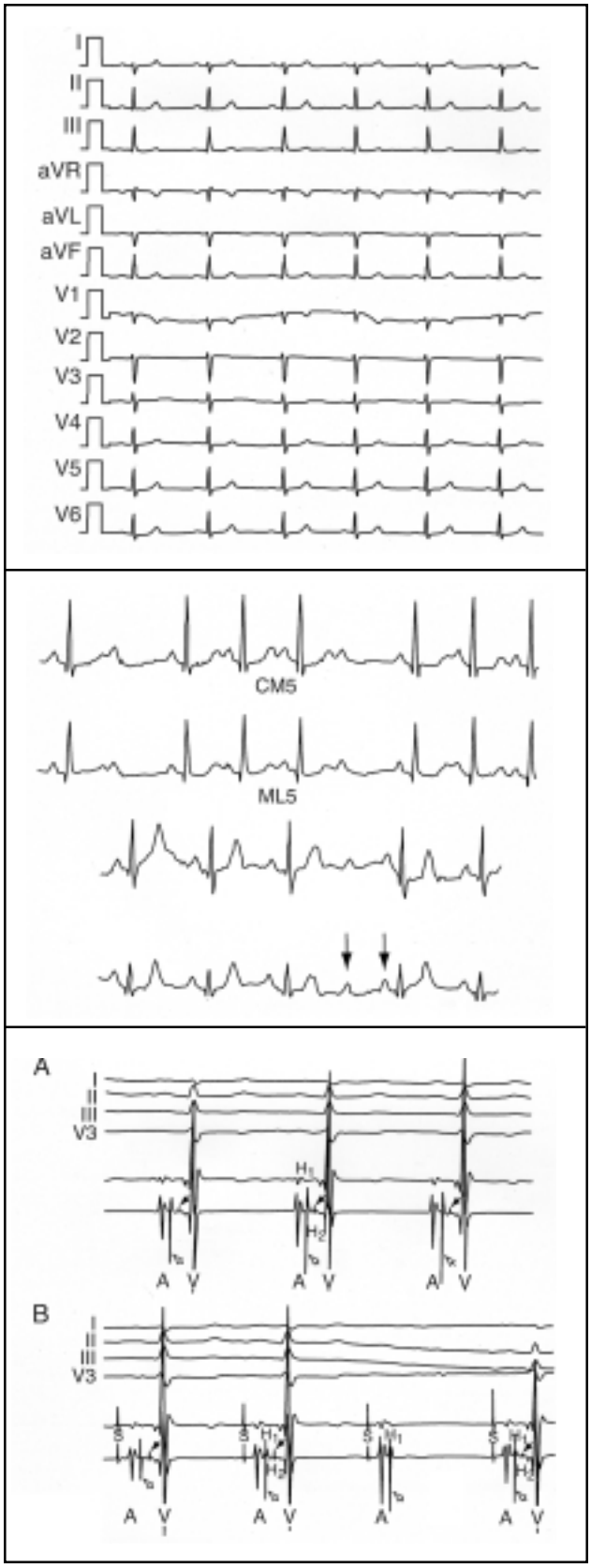

Figure 8. Exercise-induced narrow QRS atrioventricular (AV) block. All tracings were recorded from an electrocardiogram of a 47-year-old woman with dyspnea on effort. Top, The tracing is normal except for right axis deviation. There was no evidence of AV block at rest or on Holter recordings. Middle, Development of narrow QRS type I block during treadmill testing, followed by 2:1 and advanced second-degree AV block (arrows). Exercise-induced type I block is almost always infranodal, and the narrow QRS complex suggested an intra-Hisian block. Bottom, Panel A, Type II intra-Hisian block induced by atrial pacing. There are 4 electrocardiographic leads at the top and 2 intracardiac recordings at the bottom. The A (atrial) wave is followed by split His bundle potentials (H1 and H2), indicating conduction delay within the His bundle. The open arrows point to the large proximal His deflections (H1), and the solid arrows point to the smaller distal His deflections (H2). The AH interval (reflecting AV nodal conduction with H being the first His deflection) is normal. The His-Purkinje conduction time (from the first His deflection to the ventricle V or H1-V interval) is 80 milliseconds and abnormal (normal upper limit, 55 milliseconds). The H1-H2 interval is 50 milliseconds. Panel B, Atrial pacing induces intra-Hisian block between the 2 His deflections (third beat). All the PR intervals and intracardiac intervals are constant before and after the blocked impulse. The heart rate is just less than 90 beats/min and constant during atrial pacing. The abnormal sequence represents type II second-degree AV block induced by atrial pacing. S = stimuli. Reproduced with permission from Barold et al.[82]

Table 4. **Pitfalls in the Diagnosis and Treatment of Second-Degree Atrioventricular Block***

Electrocardiographic pitfalls	*Clinical pitfalls*
Nonconducted atrial premature beats masquerading as AV block	Type I AV block can be physiological in athletes, resulting from heavy physical training, and occasionally in young people
An apparent narrow QRS type II block may be a type I variant. Failure to suspect type I block in the presence of miniscule increments of the PR interval	Type I AV block can be physiological during sleep in patients with high vagal tone
Atypical type I sequence mistaken for type II block	During invasive testing, a sudden increase in the atrial pacing rate may cause functional infranodal block
Diagnosing type II block without seeing a truly conducted first postblock P wave (“shortage” of PR intervals)	Failure to recognize reversible causes of AV block, ie, Lyme disease, electrolyte abnormalities, inferior myocardial infarction, sleep apnea
Concealed extrasystoles causing pseudo-AV block. (Look for associated unexpected sudden PR prolongation, combination of what appears to be type I and type II and isolated retrograde P waves from retrograde conduction of the concealed extrasystole)	Failure to suspect vagally induced AV block, ie, vomiting
	Poor correlation between narrow QRS type I block and symptoms
	Belief that all type I blocks are AV nodal
	Failure to realize that type I and type II blocks almost never occur in the same electrocardiogram or Holter recording

*AV = atrioventricular.

ECG patterns mimicking a 2:1, type I, and/or type II AV block[84,85] (Figure 10, Table 2).

CONCLUSION

Thirty years after the advent of invasive electrophysiology, several controversies still surround second-degree AV block. The literature is replete with diagnostic errors, mainly because standard definitions are not being used universally. Knowledge of the strict but logical definitions will facilitate understanding of second-degree AV block and its management. When applied appropriately, the simple ECG patterns of type I and type II block have stood the test of time and remain clinically useful. On this basis, invasive electrophysiologic studies are rarely indicated (Table 2), and the indications for permanent pacing are better understood (Tables 1 and 3).

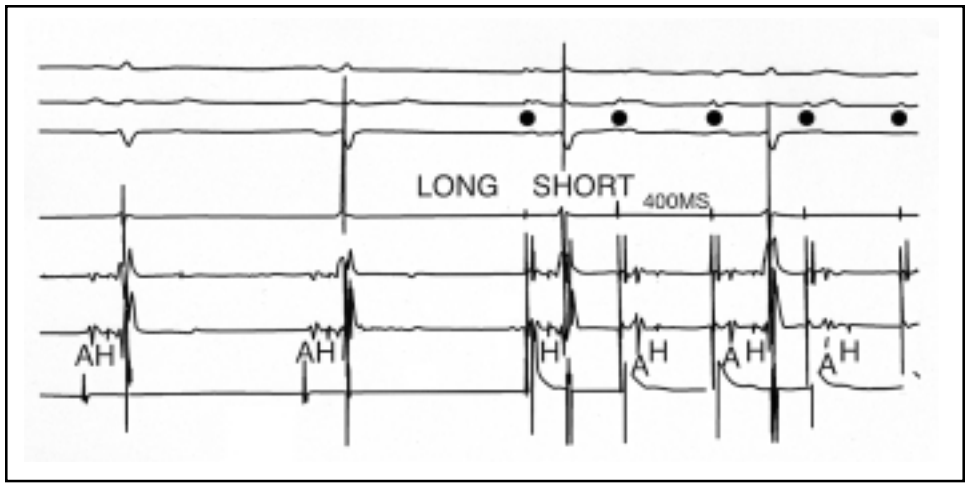

Figure 9. Functional block in the His-Purkinje system by sudden initiation of atrial pacing at a cycle length of 400 milliseconds (MS) or rate of 150/min. There are 3 electrocardiographic leads at the top and 3 intracardiac recordings at the bottom. The atrial stimuli are depicted by the solid black circles. A LONG interval followed by a SHORT interval causes block below the site of recording of the His bundle (H). The block due to Ashman phenomenon continues in a 2:1 fashion. This is a normal response to the sudden introduction of rapid atrial pacing. A = depolarization of the low atrium, and H = depolarization of the His bundle.

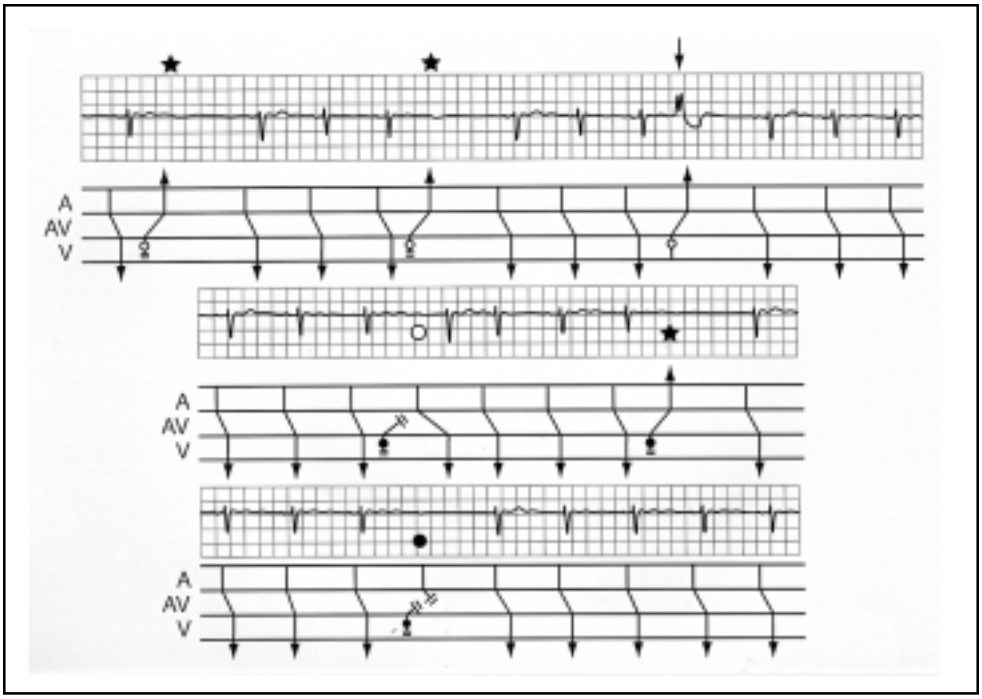

Figure 10. Electrocardiographic (ECG) recordings of lead V_1 with standard ladder diagrams. The conducted QRS complex is narrow, and the PR interval is normal. The lower strip shows all the criteria for the diagnosis of type II second-degree atrioventricular (AV) block, an indication for a permanent pacemaker. However, true type II block can be ruled out by scrutiny of the upper 2 strips. Upper strip, there is an aberrantly conducted beat (arrow) consistent with a ventricular extrasystole (VE) based on the configuration though a junctional extrasystole with aberrant conduction cannot be ruled out. This VE induces retrograde atrial depolarization (arrow) judged by P-wave morphology and resetting of the sinus cycle with delay of the succeeding sinus P wave. By the same criteria, the stars (upper and middle strips) depict retrograde activation from a concealed VE that does not depolarize the ventricular myocardium (anterograde block). The ECG diagnosis of concealed conduction is often made by observing conduction delay or block of the beat immediately after "concealed'' penetration into the specialized conduction system. The fourth beat in the middle strip (open circle) is a good example of this phenomenon. Bidirectional block of the concealed VE causes a sudden marked prolongation of the PR interval (without a change in the timing of sinus node discharge) because the sinus impulse that follows the concealed VE arrives during the relative refractory period of the AV junction engendered by retrograde conduction of the concealed VE. The bottom tracing shows the typical pattern of type II second-degree AV block with a constant sinus rate at the time of block, constant PR intervals, and importantly unchanged PR intervals before and after the blocked impulse so that the pause is equal to 2 P-P intervals. The blocked P wave is sinus and is not retrograde in origin judged by its timing and configuration (solid circle). The concealed VE penetrated the AV junction and not the atrium (bidirectional block). In this instance, the normal sinus impulse (solid circle) encountered a totally refractory AV junction and was blocked, a phenomenon simulating type II second-degree AV block. The tracings are interesting because the various manifestations of a concealed VE causing so-called pseudo-AV block allowed the confident diagnosis without invasive confirmation with His bundle recordings. Patients with concealed junctional or VEs commonly have disease of the His-Purkinje system and may require permanent pacing. However, some patients can be treated conservatively. In this patient, the cause of the phenomenon could not be determined. Because the 12-lead ECG was otherwise normal and the conducted QRS complex was narrow, the patient was successfully treated without a pacemaker.

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