

Clinical and Electrophysiologic Findings in Patients with Paroxysmal Slowing of the Sinus Rate and Apparent Mobitz Type II Atrioventricular Block

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SUMMARY Over five years, 13 patients with episodic apparent type II atrioventricular (AV) block associated with sinus slowing were seen. This phenomenon occurred only transiently during an acute illness in eight patients (group I) but recurred chronically in five (group II). For the group as a whole, the mean spontaneous cycle length was 42% longer during the period of AV block compared with periods of 1:1 AV conduction (800 ± 116 msec to 1138 ± 489 msec) ($P < 0.05$). Electrophysiologic studies in four group I patients showed no abnormalities, whereas abnormalities in AV nodal conduction and refractoriness or provocation of intranodal Mobitz type II AV block (during carotid massage) were observed in three patients in group II and were totally abolished by atropine. In group I patients, apparent type II AV block was self-limited. In the chronic group, recurrent symptoms required insertion of permanent pacemakers in two patients. Simultaneous type II block and sinus slowing appeared to be related to the effects of increased vagal tone on both nodal structures. Intracardiac pacing is not indicated for patients with transient episodes associated with an acute illness, but may be required for symptomatic patients with recurrent episodes.

MOBITZ TYPE II atrioventricular (AV) block (type II block) is diagnosed by the presence of blocked sinoatrial depolarizations without preceding prolongation of the PR interval.¹ A wealth of anatomic and physiologic data confirm the localization of this type of block to the His-Purkinje system.²⁻⁷ Some authorities, in fact, believe that type II block is always localized below the AV node.⁷ In addition, it is well appreciated that patients with type II block have a more guarded prognosis and are at increased risk for development of Stokes-Adams attacks.^{1, 8-11} Therefore, intracardiac pacemaker therapy must be considered in patients with this conduction disturbance.

Thirteen patients who manifested apparent type II AV block simultaneously with abrupt slowing of the sinus rate are reported in this study, and the clinical setting, localization of AV block and natural history of this rhythm disorder are described. The recognition and the prognostic and therapeutic implications of this type of block are reviewed.

Methods

Patient Population

Over the past five years, 13 of 55 patients with Mobitz type II AV block admitted to San Francisco General Hospital Medical Center had associated

simultaneous slowing of the sinus rate. In the 13 patients, apparent type II AV block and prolonged sinus rate were documented during continuous electrocardiographic monitoring: in nine patients while in the coronary care unit, by ambulatory monitoring in two patients, and by both methods in two patients. Four of the patients were monitored during strenuous exercise: during treadmill exercise testing in one patient and during Holter monitoring in three. Continuous electrocardiographic recordings before, during and after carotid massage were available in 11 subjects.

Electrophysiologic Studies

Seven patients underwent further electrophysiologic studies within four days of a documented episode of apparent Mobitz type II AV block. His bundle electrograms were obtained using standard techniques.¹² In brief, a multipolar electrode catheter was inserted into the right femoral vein and positioned across the tricuspid valve. The His bundle electrogram and surface leads X, Y and Z of the Frank orthogonal lead system were simultaneously displayed on the oscilloscope and recorded using an Electronics for Medicine (DR-12) recorder. The AV nodal conduction time was measured from the initial high frequency deflection of the low right atrial electrogram to the first high frequency deflection (A-H), and infranodal conduction was measured from the initial high frequency deflection recorded from the His bundle to the onset of ventricular activation determined from the surface leads (H-Q). Upper limits of normal for these measurements in our laboratory are 120 msec and 55 msec, respectively. Atrial overdrive pacing, using previously described methods,¹³ was performed in all seven patients. Atrioventricular nodal refractory periods were measured in five patients using the atrial extra stimulus technique.¹⁴ Three patients showed ab-

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TABLE 1. Clinical Data for 13 Patients with Mobitz Type II Atrioventricular Block with Simultaneous Prolongation of the Sinus Rate

Patient no.	Age (yr)	Sex	Cardiac diagnosis	Associated symptoms	Cardiac medication	Length of follow-up (months)	Outcome
<i>Group I</i>							
1	54	M	Inferior MI	None	None	9	Abnormalities resolved
2	49	M	Inferior MI	None	None	54	Abnormalities resolved
3	50	M	Inferior MI	None	None	24	Abnormalities resolved
4	65	F	Subendocardial MI	None	Digoxin	44	Abnormalities resolved
5	64	M	Unstable angina	None	Digoxin	6	Abnormalities resolved
6	76	F	CHF	Vomiting	None	6	Abnormalities resolved
7	73	F	CHF	Vomiting	None	24	Abnormalities resolved
8	80	F	ASD, CHF	Vomiting	Digoxin	3	Abnormalities resolved
<i>Group II</i>							
9	31	M	None	Syncope	None	11	Abnormal conduction persists; asymptomatic with pacemaker
10	57	M	CHF	Near syncope	Digoxin	14	Abnormal conduction persists; asymptomatic with pacemaker
11	67	M	Angina	Syncope	None	59	Abnormal conduction persists; symptomatic; refused pacemaker
12	21	M	None	None	None	11	Abnormal conduction persists; asymptomatic
13	30	M	None	None	None	3	Abnormal conduction persists; asymptomatic

Abbreviations: ASD = atrial septal defect; CHF = congestive heart failure; F = female; M = male; MI = myocardial infarction.

normalities during control observations and were retested after intravenous administration of atropine, 0.04 mg/kg body weight.

The patients were either followed in the Pacemaker and Arrhythmia Clinic at San Francisco General Hospital Medical Center or pertinent follow-up data were obtained from their private physicians. The data were analyzed using paired and unpaired Student's *t*-tests.

Results

Clinical Setting

The pertinent clinical and electrocardiographic data are summarized in tables 1 and 2. Patients were subdivided into two groups on the basis of their clinical

condition. Group I consisted of eight acutely ill patients in whom apparent type II AV block occurred transiently during the course of acute myocardial infarction (inferior wall in three patients, subendocardial in one patient), during prolonged retching (three patients), or during an episode of angina (one patient). Figure 1 shows an episode of Mobitz type II block with abrupt sinus slowing in a patient (number 2) with an acute myocardial infarction. Group II consisted of five patients who were not acutely ill who had recurrent episodes of apparent type II AV block. Four patients (three patients from group I, one patient from group II) were taking digoxin without evidence of toxicity; serum digoxin levels ranged from 0.3 to 1.6 ng/ml at the time of AV block.

Only two patients in group I had more than one

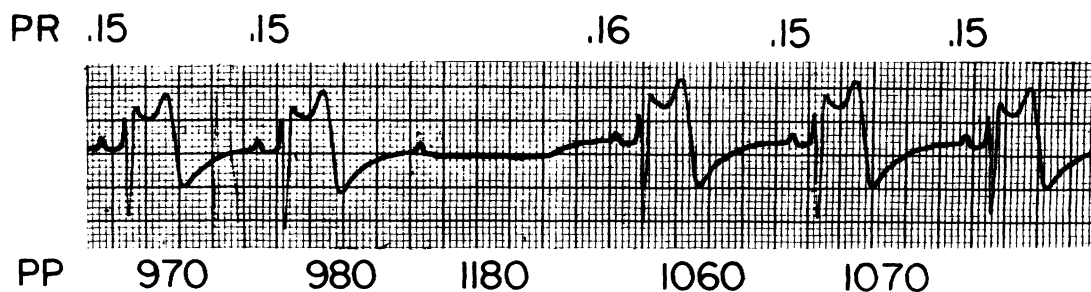


FIGURE 1. A, Mobitz type II AV block associated with sinus slowing in patient 2 during the course of an acute inferior myocardial infarction. The PR intervals remained essentially constant while sinus cycle lengths (P-P) were prolonged.

TABLE 2. *Electrocardiographic Findings in 13 Patients with Mobitz Type II Atrioventricular Block with Simultaneous Prolongation of the Sinus rate**

Patient no.	Electrocardiogram	Monitoring results		Response to carotid sinus massage
		Episodes of type II AVB	Associated rhythm and conduction disturbances	
Group I				
1	Acute inferior MI	Multiple	None	Prolonged sinus rate
2	Acute inferior MI	Single	None	Prolonged sinus rate
3	Acute inferior MI	Single	None	Not performed
4	Subendocardial MI	Single	None	Prolonged sinus rate, type II AVB
5	LVH, old anterior MI	Multiple	1° AVB, type I AVB	Prolonged sinus rate, type I AVB
6	LVH	Single	1° AVB	Prolonged sinus rate, type I AVB
7	LBBB	Single	None	Not performed
8	RVH	Single	None	Prolonged sinus rate
Group II				
9	Normal	Multiple	SAB, type I AVB	Prolonged sinus rate, SAB
10	Old inferior MI; RBBB, LAFB	Frequent	Sinus arrest, type I AVB	Prolonged sinus rate, sinus arrest; type II AVB
11	Normal	Multiple	1° AVB, type I AVB	Prolonged sinus rate, type I AVB
12	Normal	Multiple	1° AVB, type I AVB	Prolonged sinus rate, type I AVB
13	Normal	Frequent	SAB, 1° AVB, type I AVB	Prolonged sinus rate, types I and II AVB

*Rhythm abnormalities included under monitoring results.
 Abbreviations: AVB = atrioventricular block; LBBB = left bundle branch block; LAFB = left anterior fascicular block; LVH = left ventricular hypertrophy; MI = myocardial infarction; RVH = right ventricular hypertrophy; RBBB = right bundle branch block; SAB = sinoatrial exit block.

episode of Mobitz type II AV block with sinus slowing, whereas all group II patients had multiple episodes. Two patients had chronic intraventricular conduction delay: left bundle branch block was present in one (in group I) and right bundle branch block and left anterior fascicular hemiblock in the other (in group II).

Sinus Rate and PR Interval

The mean PR intervals and sinus cycle lengths during the period before the first recorded occurrence of type II block and those of the immediately preceding, blocked, and subsequent beats are shown in table 3. Sinus cycle length increased, usually abruptly, simultaneously with the appearance of AV block in each patient. Maximum prolongation occurred either in the cycle terminated by the blocked P wave or in the subsequent cycle. For the group as a whole, mean sinus cycle length was 42% longer during the period of AV block compared with the cycle length immediately preceding the block, from a mean of 800 ± 116 msec (mean \pm SD) before the appearance of block to a maximum of 1138 ± 489 msec ($P < 0.05$). Although the spontaneous sinus cycle lengths were similar in both groups (803 ± 123 msec and 796 ± 116 msec in groups I and II, respectively), the mean increment in P-P interval was significantly greater in group II than in group I (696 ± 707 msec versus 115 ± 73 msec, $P < 0.05$). The changes in P-P cycle in length in all patients are shown in figure 2. The PR interval preceding the blocked beat was identical to the mean

PR during 1:1 AV conduction. However, a variable relationship existed between the PR preceding and following the dropped beat. The PR intervals were essentially unchanged in seven patients (± 10 msec), decreased by 20 msec in two, decreased by more than 20 msec in three, and increased in one (table 3). The variable responses in PR are illustrated in figures 1, 3 and 4.

TABLE 3. *P-P and PR Intervals During Episodes of Mobitz Type II Atrioventricular Block in 13 Patients*

Patient no.	Basic intervals (mean)		Preceding beat		Dropped beat	Following beat	
	P-P	PR	P-P	PR	P-P	P-P	PR
Group I							
1	930	0.20	970	0.20	1010	960	0.18
2	970	0.15	970	0.15	980	1180	0.16
3	850	0.19	990	0.19	1000	860	0.18
4	650	0.16	710	0.16	740	740	0.16
5	840	0.27	860	0.27	900	840	0.22
6	820	0.26	820	0.26	860	840	0.26
7	730	0.17	700	0.17	790	780	0.17
8	630	0.23	750	0.23	840	860	0.26
Group II							
9	740	0.18	1040	0.18	1500	1340	0.18
10	720	0.16	720	0.16	2620	1460	0.16
11	980	0.42	1120	0.42	1150	1200	0.36
12	840	0.20	900	0.20	1100	980	0.18
13	700	0.38	860	0.38	960	1000	0.24

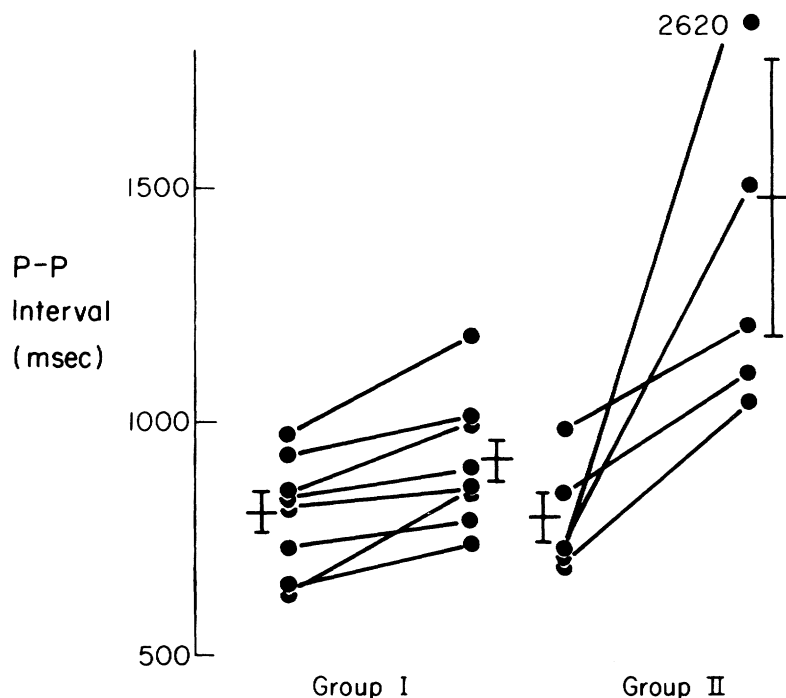


FIGURE 2. Individual (•) and mean (±) spontaneous cycle length (P-P interval) before and during spontaneous episodes of type II AV block are illustrated for groups I and II. Increases in P-P interval are significant ($P < 0.05$) for both groups.

Associated Rhythm Disturbances

Frequent episodes of type II AV block were present in all group II patients, and associated abnormalities in sinus node automaticity, sinoatrial conduction and AV conduction were more commonly observed in these patients than in group I patients. All patients in group II, for example, also manifested periods of type I AV block. An example (patient number 13) of both types of AV conduction disturbance is shown in figure 3. Three patients, all in group II, also manifested sinus node dysfunction in addition to AV conduction abnormalities. Patients 9 and 13 had frequent episodes of 3:2 and 2:1 sinoatrial exit block, which on several oc-

casions occurred simultaneously with type II AV block. Patient 10 had several episodes of sinus arrest, lasting up to 4100 msec, associated with AV block; one of these episodes is shown in figure 4. In contrast, only one instance of type I AV block and no instance of disturbances of the sinoatrial conduction were recorded in group I patients.

Responses to Carotid Sinus Massage and Exercise

Carotid sinus massage was performed in 11 patients within 48 hours of an observed episode of type II AV block; the results are shown in table 2. In patients 4 and 10, type II AV block with accompanying sinus

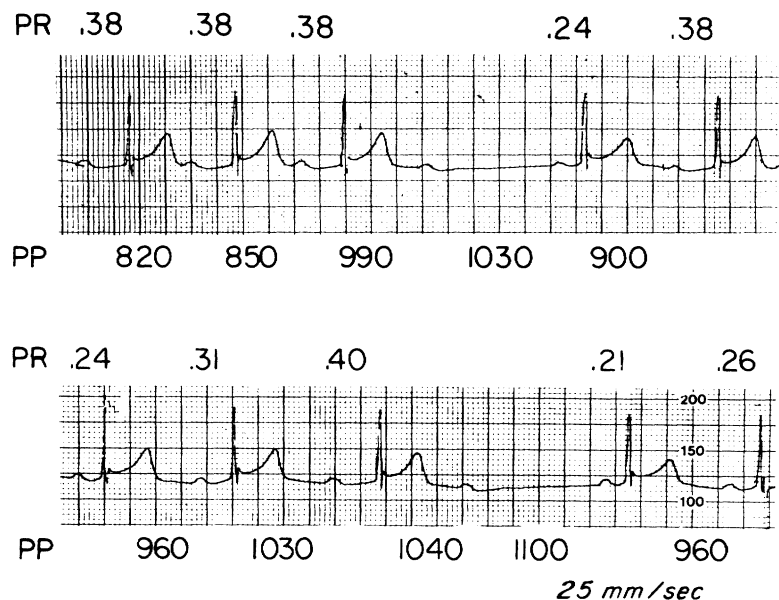


FIGURE 3. The PR and P-P intervals before and after the blocked P wave are shown for patient 13. The upper panel shows type II AV block (constant PR interval proximate to the blocked beat), while panel B shows an atypical Wenckebach sequence.

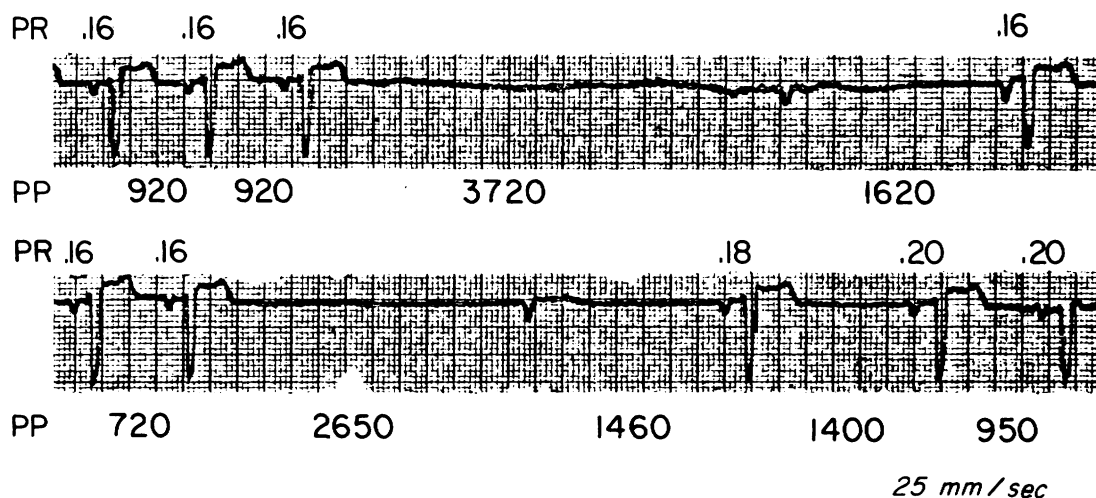


FIGURE 4. Illustrative tracings from patient 10 show episodic sinus arrest associated with type II AV block.

slowing could be induced (fig. 5). A third patient (number 13) displayed type II block on the standard electrocardiogram, but during electrophysiologic studies utilizing high speed recordings a slight prolongation of the A-H interval was observed before the blocked sinus beat (fig. 6). The responses to carotid massage of the other patients are shown in table 1.

Four of the five group II patients (numbers 9, 11, 12 and 13) were monitored during exercise. Atrio-ventricular conduction normalized completely in three and the PR interval shortened but remained abnormal in one (number 11).

Electrophysiologic Studies

Electrophysiologic data from the seven patients studied are summarized in table 4. Four of the seven patients were in group I. Although all were studied within four days (two within 16 hours) of an episode of type II AV block, none manifested disturbances in AV conduction either spontaneously or during carotid

massage at the time of study. One of the group I patients had left bundle branch block, but had a normal infranodal conduction time.

In contrast, each group II patient manifested abnormalities of AV conduction. In patient 10, who had bifascicular block and a slightly prolonged H-Q interval (62 msec), episodes of type II block associated with sinus slowing were always localized to the AV node and could be induced repeatedly by carotid sinus massage (fig. 5); however, after atropine, no abnormalities could be induced by carotid sinus massage. Patients numbers 12 and 13 had prolonged A-H intervals and normal H-Q intervals and manifested numerous spontaneous episodes of AV nodal Wenckebach conduction (no type II AV block was present) during baseline electrophysiologic studies. Both also displayed markedly abnormal responses in AV conduction to atrial pacing and prolonged AV nodal effective refractory periods. Abnormalities of AV nodal conduction and refractory periods in these patients were also abolished by atropine. Figure 6 shows the change in response to carotid massage after

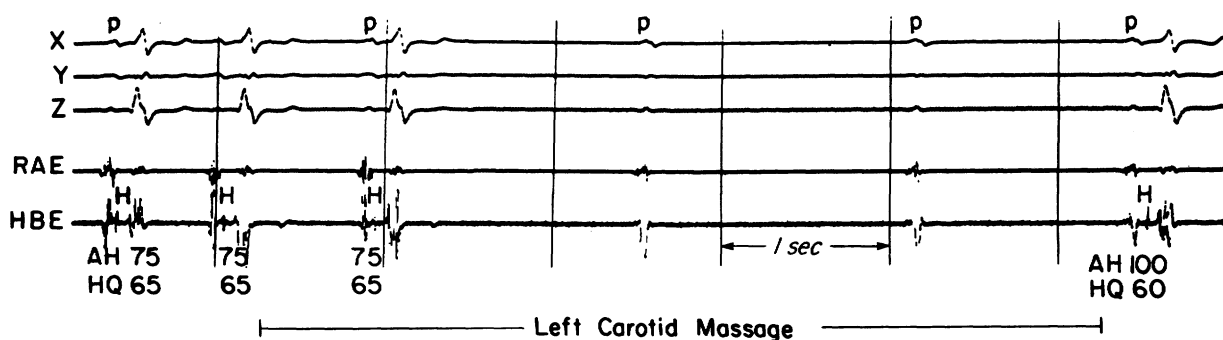


FIGURE 5. Simultaneous recordings of surface leads X, Y and Z (of the Frank orthogonal lead system), right atrial electrogram (RAE), and the His bundle electrogram (HBE) for patient 10. Carotid massage results in marked sinus slowing and two blocked P waves. The AV conduction disturbance is localized to the level of the AV node because no His depolarizations are recorded during AV block. The absent His spikes are unlikely due to technical problems because well defined His potentials are recorded both before and after block.

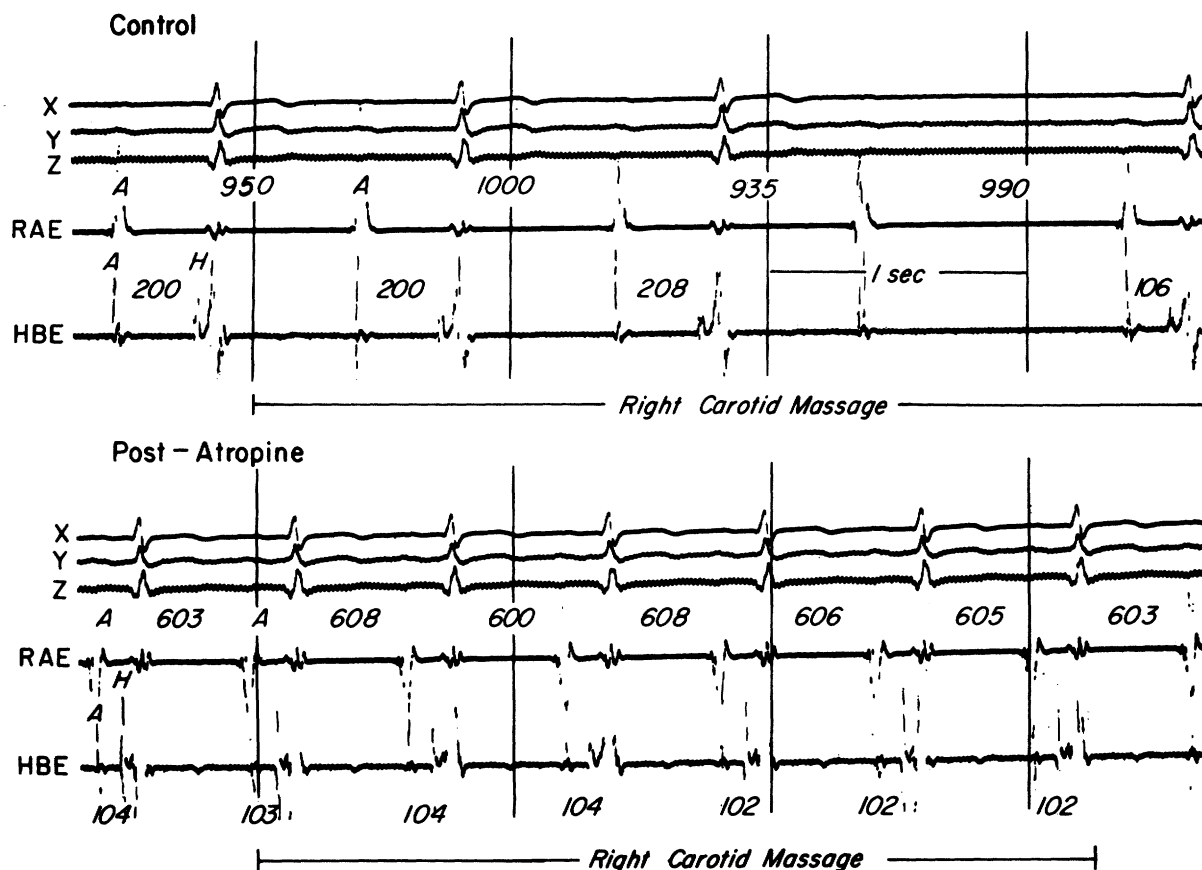


FIGURE 6. Sinus cycle length and A-H interval are inscribed for patient 13 during carotid sinus massage before and after intravenous administration of atropine. The top panel reveals minimal (8 msec) increase in the A-H interval proximate to the blocked P wave. This phenomenon could not be reproduced (bottom panel) after atropine. RAE = right atrial electrogram; HBE = His bundle electrogram.

the administration of atropine in patient 13; normalization of AV conduction and refractoriness after atropine in this patient is shown in figure 7.

The corrected sinus node recovery times were within normal limits in all patients studied, including patients 10 and 13, who had spontaneous episodes of sinus arrest and/or sinoatrial exit block.

Clinical Follow-up

The 13 patients were followed for a mean of 21 months (range 3–59 months), and their clinical outcome is shown in table 1. In the eight group I patients, episodes of AV block occurred during the course of acute myocardial infarction, unstable angina or prolonged retching. These episodes appeared to be self-limited and the patients' subsequent course was benign. In contrast, three of the five patients in group II had repeated bouts of syncope or near syncope. These symptoms were alleviated only after insertion of a permanent intracardiac pacemaker in two, and persisted in a third who refused pacemaker insertion.

Discussion

Our study is unique in that we define the clinical profile and present long-term follow-up data for a group of patients with apparent type II AV block

associated with simultaneous sinus slowing. We found that these patients could be segregated into two groups with marked differences in both clinical condition and electrophysiologic findings.

In eight patients (group I), the AV block proved to be transient and related to a well-defined acute illness. None required cardiac pacing and no episodes occurred subsequently. Subsequent carotid massage usually failed to provoke AV block, and electrophysiologic studies showed no abnormalities. These findings suggest that the AV block was functional and probably related to acute increases in vagal tone. In contrast, the five group II patients were not acutely ill and had recurrent episodes of type II AV block with simultaneous slowing of the sinus rate. These abnormalities could be readily provoked by carotid massage and largely abolished by exercise. In addition, abnormalities of sinus pacemaker function and AV conduction were commonly present. Three of the five had recurrent syncope, and symptoms disappeared after insertion of a permanent pacemaker in two; symptoms persist in the other patient who refused pacemaker insertion.

Mechanism of Type II Atrioventricular Block

Electrophysiologic studies after subsidence of acute symptoms in four of the eight patients in group I

TABLE 4. Electrophysiologic Findings in Seven Patients with Mobitz Type II Atrioventricular Block with Simultaneous Slowing of the Sinus Rate

Patient no.	Spontaneous cycle length (msec)	A-H (msec)	H-Q (msec)	AV node ERP (msec)	Pacing cycle length producing AVB (msec)	Corrected SNRT (msec)
Group I						
1	790	95	45	—	350	230
4	680	80	45	—	420	270
5	670	120	40	300	400	350
7	880	95	53	320	380	410
Group II						
10*	Control	725	65	340	400	425
	Atropine†	615	57	280	320	280
12	Control	1100	156	400	800	380
	Atropine†	990	120	240	500	330
13‡	Control	870	240	580	650	340
	Atropine‡	590	100	300	380	350

*Developed type II block during carotid massage; this response was abolished after administration of atropine.

†After intravenous administration of atropine, 0.04 mg/kg body weight.

‡Developed a "type II-like" response with minimal prolongation of A-H (8 msec) just before the blocked beat; this response was abolished after administration of atropine.

Abbreviations: AVB = atrioventricular block; ERP = effective refractory period; SNRT = sinus node recovery time.

revealed no evidence of impaired AV conduction. We hypothesize that in these patients type II AV block was probably related to acute increases in vagal tone. This interpretation is supported by the presence of conditions known to be associated with increased vagal tone in each (myocardial infarction in four, nausea and retching in three and digoxin therapy in two).

Control electrophysiologic studies in three of five group II patients revealed abnormalities in AV conduction and AV nodal refractoriness (two patients) or Mobitz II-like responses to carotid sinus massage

(two patients). These findings were completely abolished after atropine, again emphasizing the role of vagal overactivity in this conduction disorder. The inability to provoke type II AV block after carotid massage in some patients does not exclude vagal stimulation as an initiating event, since carotid sinus massage may not be the appropriate trigger for the intense vagal discharge.

Depression of sinus node automaticity and AV nodal conduction may be produced by vagal stimulation. The simultaneous occurrence of slowing of the sinus rate with AV block in these patients, as well as

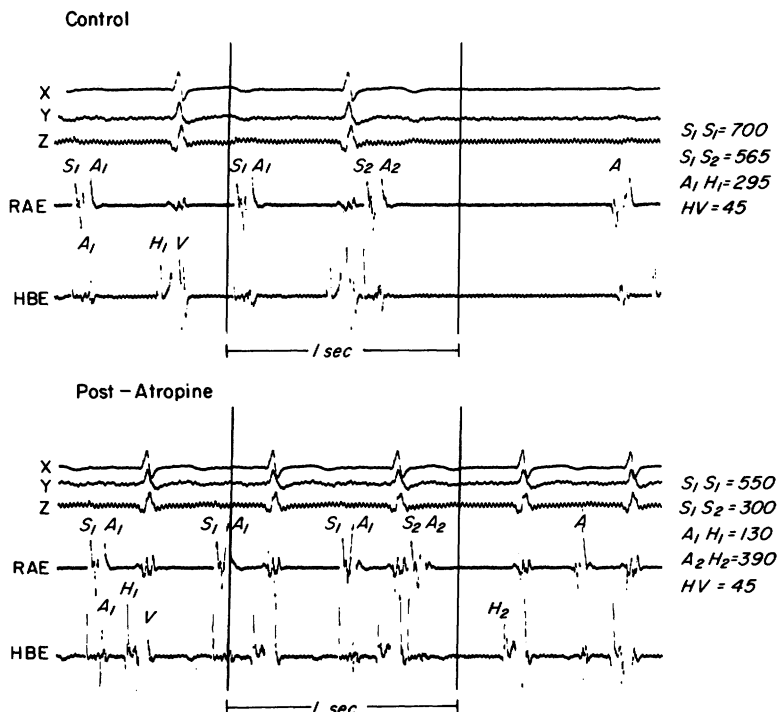


FIGURE 7. Example of marked improvement in AV nodal conduction and refractoriness after atropine administration (patient 13). The upper panel (control observations) shows the shortest driven cycle length ($S_1 S_1 = 700$ msec) at which 1:1 conduction was possible. A late atrial premature depolarization (A_2) (565 msec after the last driven beat [A_1]) is blocked in the AV node. After atropine, 1:1 conduction proceeds at a paced cycle length of 550 msec and an even more premature induced atrial depolarization (300 msec) is conducted to the His bundle.

the response to exercise, provides further circumstantial evidence for the role of increased vagal tone in the production of these disturbances.

Alternatively, Jonas et al.¹⁵ reported infranodal block during carotid massage that they attributed to a phase four depolarization block in the His-Purkinje system. Several findings in our patients make this latter hypothesis unlikely. Spontaneous episodes of even more pronounced spontaneous sinus slowing in all patients failed to produce AV block, the episodes of AV block recorded during electrophysiologic studies were intranodal, and premature ventricular depolarization, which usually terminates phase four block,¹⁶ was recorded in only one of our patients. Furthermore, it is important to emphasize the distinction between vagally mediated AV block described in this report from bradycardia-dependent paroxysmal AV block.^{17, 18} In the latter, a number of P waves are blocked and the AV block is terminated by an escape pacemaker provided the interval from the escape pacemaker and subsequent P wave is appropriate for conduction. The vagally mediated AV block almost always involves block of a single P wave with resumption of 1:1 AV conduction without need for the escape of a subsidiary pacemaker.

Two other previously described mechanisms for the appearance of type II AV block also appear unlikely. Intranodal type II AV block was observed by Spear and Moore¹⁹ in the normal calf heart; they showed that slight (20 msec) decreases in the atrial cycle length can produce typical type II block localized to the AV node and suggested that these findings might be applicable to humans with slight sinus arrhythmia where changes in cycle length may not be detectable on standard electrocardiographic tracings. Our patients, on the other hand, always showed prolongation of sinus rate before the dropped beat. His bundle extrasystoles can result in episodic AV block,²⁰ but these were not observed during electrophysiologic studies of our patients.

Previous Related Observations

Spontaneous Mobitz type II block is rarely localized to the AV node. Rosen et al.²¹ described this phenomenon in several patients, one of whom had associated prolongation of the sinus rate during carotid massage. This finding was attributed to the sudden increase in vagal tone. Similarly, others have reported the occurrence of paroxysmal AV block, which in rare instances has been of the "Mobitz type II" pattern, during carotid sinus massage or other possible hypervagotonic states (e.g., hiccups, micturition, swallowing, anxiety).^{12, 21, 22-32} Our studies support these observations in terms of localization of the block and the role of vagal stimulation.

El-Sherif et al.^{33, 34} suggested a scheme of localization of AV block from surface recordings on the basis of the length of the PR interval preceding and following the blocked beats. Our experience emphasizes the limitations of these criteria for patients with

"vagotonic block." For example, the PR interval after the dropped beat was less than (six patients), equal to (five patients), or greater than (one patient) the PR interval preceding the dropped beats (table 3). Moreover, there was no correlation between the change in PR and its control duration. For patients with AV block due to increased vagal tone, the PR interval after the blocked P wave probably results from the slowing of the sinus rate (which would be expected to facilitate AV nodal conduction) and an intrinsic vagal effect (which slows AV nodal conduction).

Limitations

Certain important limitations of this study require emphasis. Inclusion of three patients who showed pronounced (>20 msec) decreases in the PR following the blocked beat do not conform to either Mobitz's original description¹ or to more recently proposed criteria for diagnosis of type II AV block.³⁵ These patients were included in the present study because aside from the PR immediately following the pause, all subsequent and preceding PR were essentially constant. While shortening of the PR following the blocked P wave may be due to junctional escape beats, in two of the three patients other episodes of "typical" type II block were recorded. These patients were, therefore, included in the present study in order to emphasize the spectrum of AV conduction responses observed in this setting.

Although most patients had the "classic criteria" for Mobitz type II AV block on their surface electrocardiograms, routine electrocardiographic recordings lack sufficient sensitivity to detect slight changes in the PR interval. Indeed, this was the case in patient 13, in whom no prolongation of the PR interval was apparent on the surface electrocardiogram but slight prolongation of the A-H interval (8 msec) was detected using more accurate high speed recordings. Similar slight prolongation in AV nodal conduction time may have been present in other patients.

In our study, two of the five patients with the chronic recurrent type II pattern required permanent pacing. It must be appreciated that our series reflects a biased subgroup in that the five patients in group II were referred to us primarily for evaluation of symptoms and/or frequency of conduction abnormalities. Neither the general incidence of this type of conduction disturbance nor the frequency of associated symptoms is known.

Clinical Implications

In this study, we defined two subsets of patients with sudden type II AV block associated with sinus slowing. In group I, the block was associated with acute illness and was self-limited. Although it is generally agreed that patients with acute myocardial infarction (particularly when anterior in location) with associated type II AV block require temporary pacing, our observations suggest that if the block is associated with abrupt prolongation of the sinus rate

(particularly in the setting of an acute inferior myocardial infarction), careful observation or use of an appropriate dose of atropine is a reasonable initial therapeutic approach.

In addition, two patients with bundle branch block were initially diagnosed as showing episodic bilateral bundle branch block. Atropine abolished the AV conduction disturbance induced by carotid massage in one and the AV nodal conduction time normalized in the other. These observations suggest that appearance of Mobitz type II AV block in patients with intraventricular conduction delay does not always indicate localization of the block to the His-Purkinje system.

The elegant experimental observations by El-Sherif et al.^{33, 34} showed that Mobitz types I and II blocks may be localized either in the ischemic AV node or in the His-Purkinje system. They concluded that the different types of block may be a continuum and reflect a difference in degree of damage to the cardiac conduction system rather than a difference in pathophysiologic mechanisms. Our findings are similar in that typical Mobitz I, Mobitz II or Mobitz II-like (minimal increments in A-H just before the blocked beat) were frequently observed (at times in the same patient) either spontaneously or as a result of carotid sinus massage. These observations suggest that enhanced vagal tone can also produce a continuum of AV nodal responses. The clinician must be aware of these possible responses in order to evaluate properly patients with AV conduction disturbances.

The natural history of patients manifesting chronic recurrent episodes of Mobitz type II-like AV block with accompanying sinus slowing is less clear. Some patients have remained without symptoms for a long period of time, whereas others have required pacing. In all of these patients, the type II block was most often associated with both sinus node and AV conduction abnormalities. It is unclear whether the associated disorders are strictly related to increased vagal tone or result from independent sinoatrial and AV nodal disease. Complete normalization after atropine administration favors the former hypothesis.

Young et al.³⁶ recently reported the long-term follow-up of a group of young patients without apparent heart disease who had type I AV block; some of the patients eventually required chronic pacing for alleviation of symptoms. This group may, in fact, be similar to the three younger patients in our series who predominantly displayed type I block. Chronic pacemaker intervention in group II patients appears to be indicated only for those with transient neurologic symptoms.

Finally, this report emphasizes that proper diagnosis of true Mobitz II AV block (and hence appropriate integration of this finding into the total clinical presentation) demands rigorous attention to the sinus rate. Though not explicitly stated in the original or in most subsequent reports of Mobitz II AV block, our observations (together with those of Spear and Moore¹⁹) suggest that this diagnosis can only be made when the sinus rate is stable.

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Dual Echocardiographic Determination of Atrial Contraction Sequence in Atrial Flutter and Other Related Atrial Arrhythmias

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SUMMARY We have applied the new technique of dual echocardiography to determine the sequence of atrial contraction as reflected in the simultaneously recorded movements of the tricuspid and mitral valves. The study group included 29 normal subjects and 23 patients with either atrial flutter, coarse atrial fibrillation or atrial tachycardia with block. In normal individuals, right atrial contraction preceded left atrial contraction, with an average interatrial contraction time of 17 ± 8 msec. In contrast, the atrial contraction sequence was reversed in atrial flutter, with left preceding right atrial contraction and a prolonged interatrial contraction time of 82 ± 20 msec. In two patients with atrial tachycardia with block, atrial contraction was either simultaneous or left preceded right atrial contraction by a brief interval.

The sequence of atrial excitation, as determined by electrode catheter recordings from the right and left atria in one patient with atrial flutter and one patient with normal sinus rhythm, was the same as the contraction sequence. Left atrial pacing reversed both excitation and contraction sequences. After cardioversion of three patients from atrial flutter to normal sinus rhythm, interatrial contraction time was shortened but remained longer than in normal subjects, suggesting an interatrial conduction disturbance in patients with atrial flutter.

In coarse atrial fibrillation, the contraction sequence varied. Significant motion of both mitral and tricuspid valves coincident with fibrillatory waves occurred frequently, especially when the fibrillatory waves were coarse and regular.

Dual echocardiography permits the noninvasive determination of the sequence of atrial contraction and excitation, and may be useful in studying the characteristics of atrial arrhythmias.

DESPITE MANY STUDIES of the electrophysiologic and mechanical characteristics of atrial arrhythmias,¹⁻²⁶ lack of satisfactory techniques has prevented simultaneous examination of the mechanical events in

the two atria in intact human subjects. The new technique of dual echocardiography, recently developed in our laboratory,^{27, 28} makes possible the simultaneous noninvasive recording of tricuspid and mitral valve movements. Because these valvular movements reflect right and left atrial contraction respectively, this technique provides a new method of studying the effects of atrial arrhythmias on the contraction patterns of the two atria. The present study uses high speed dual echocardiographic recording to observe the sequence of atrial contraction on a beat-to-beat basis, as reflected in tricuspid and mitral valve motion. This study was performed on normal subjects and patients with atrial flutter, coarse atrial fibrillation and atrial tachycardia with block. In two patients, right and left

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