Natural History of Chronic Second-degree Atrioventricular Nodal Block

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SUMMARY This report details our experience with documented chronic second-degree atrioventricular (AV) nodal block (proximal to His [H]) in 56 patients. Forty-six men (82%) and 10 women (18%), ages 18–87 years, were studied. Nineteen of the patients (34%) had no organic heart disease (including seven trained athletes) and 37 (66%) had organic heart disease. ECGs in all patients demonstrated episodes of type I second-degree block; five patients also had periods of 2:1 block. Prospective follow-up of patients with no organic heart disease (157–2280 days, mean 1395 ± 636 days) revealed one patient with clear indication for permanent pacing because of bradycardiac symptoms (permanently placed on day 220 of follow-up). Two patients died nonsuddenly.

In patients with organic heart disease (prospective follow-up of 60–2950 days, mean 1347 ± 825 days), pacemakers were implanted in 10 patients, primarily for treatment of congestive heart failure in eight and syncope in two. Sixteen patients died — three suddenly, seven with congestive heart failure, two of an acute myocardial infarction and four of causes unrelated to cardiac disease.

In summary, chronic second-degree AV nodal block has a relatively benign course in patients without organic heart disease. In patients with organic heart disease, prognosis is poor and related to the severity of underlying heart disease.

SECOND-DEGREE atrioventricular (AV) nodal block is most often recognized in the acute clinical setting, complicating inferior wall myocardial infarction, digitalis intoxication, acute myocarditis or recovery after open heart surgery. In such circumstances, second-degree AV nodal block is usually reversible with time, and usually plays no major role in determining clinical outcome.

Chronic second-degree AV nodal block is also seen in a variety of circumstances. Although generally it is considered a benign conduction defect, few systematic data have been reported. In this report, we describe our experience in 56 consecutive patients with chronic second-degree AV nodal block and report clinical, electrocardiographic and electrophysiologic findings. We also report the follow-up data and summarize the clinical significance of chronic second-degree AV nodal block.

Materials and Methods

Definitions

Second-degree AV block was defined as incomplete AV block with dropped ventricular beats. Type I block (Wenckebach) was defined as an episode of second-degree block characterized by a measurable increase in PR or AH intervals from the first conducted beat of a sequence to that of the last conducted beat before the dropped beat. Type II block was defined as an episode of second-degree block with no measurable increase in PR from the first to the last conducted beat of a sequence. Two-to-one (2:1) block was not classified by type. Patterns of left and right bundle branch block were diagnosed using standard electrocardiographic criteria. The duration of the QRS complex was considered narrow when less than 0.12 second and was considered wide when at least 0.12 second.

Patient Selection

Fifty-six consecutive patients with chronic second-degree AV nodal block were detected, studied and followed between January 1970 and March 1980. Approximately 50% of patients were detected within the inpatient and outpatient services of our medical center and 50% were referred because of the electrocardiographic diagnosis of second-degree AV block.

Criteria for inclusion in the present study were (1) electrocardiographic documentation of second-degree AV block; (2) documentation of chronicity on multiple ECGs; (3) availability of prospective life history, obtained through follow-up in our conduction clinics, or through direct correspondence with referring physicians; and (4) electrophysiologic diagnosis of second-degree AV nodal block. This diagnosis was based on either direct demonstration of second-degree AV nodal block during sinus rhythm (block proximal to the His bundle recording site) or the finding of significantly depressed AV nodal function during electrophysiologic study as manifest by at least two of the following: prolonged AH intervals (greater than 130 msec), AV nodal Wenckebach periodicity developing.
at atrial paced rates less than 101 beats/min, or pro-
longed AV nodal refractory periods (AV nodal effec-
tive refractory period greater than 365 msec and/or
AV nodal functional refractory period greater than
495 msec).

Patients with acute second-degree AV nodal block
complicating acute myocardial infarction and digitalis
intoxication were excluded from the study.

Initial Evaluation

All patients were hospitalized. A complete history,
physical examination, routine laboratory studies,
serial ECGs and chest roentgenograms were obtained.
Thirty-nine patients had echocardiograms, 10 had
Holter monitoring and 34 had graded treadmill exer-
cise tests. Diagnostic cardiac catheterization was
performed in 12 selected cases. The diagnosis of specific
organic heart diseases was established by the follow-
ing criteria. Arteriosclerotic heart disease was diag-
osed if the patient had a previous diagnosis of
definite myocardial infarction or a typical history of
exertional angina or both (with coronary arterio-
diagnostic documentation in several cases). Hyperten-
sion was diagnosed if three or more determinations
of blood pressure were 160 mm Hg systolic or 95 mm Hg
diastolic or higher. Aortic valvular disease was
diagnosed by physical examination and by echo-
cardiographic and cardiac catheterization studies.
Mitral valve prolapse was diagnosed by physical ex-
amination and echocardiography. Primary conduction
disease (Lev’s or Lenegre’s disease) was diagnosed
when patients had bundle branch block with no evi-
dence of additional cardiovascular disease (other
than chronic second-degree AV nodal block).

Electrophysiologic Studies

Each patient gave informed written consent. His
bundle electrograms were recorded using standard
catheter techniques. In localizing the site of block,
special attention was paid to ruling out block within
the His bundle by careful scrutiny of potentials
recorded from the AV junction, looking for split H
potentials. Refractory periods were determined using
the extrastimulus technique. Intracardiac electro-
grams from the high right atrium and His bundle, with
simultaneous electrocardiographic leads 1, 2, 3 and
V_{1}, were recorded on a multichannel photographic
recorder (Electronics for Medicine DR-20) at paper
speeds of 100 and 200 mm/sec. The atrial and ventric-
ular rates and the AH (AV nodal conduction time)
and HV (His-Purkinje conduction time) intervals were
measured. In our laboratory, normal values are
54–130 msec for the AH interval and 31–55 msec for
the HV interval. Sinus node recovery times (normal
<1620 msec) and sinoatrial conduction time (normal
32–152 msec) were calculated as previously
described.

Patient Follow-up

The follow-up period was defined from the time of
the initial electrophysiologic study. After the initial
evaluation, most patients were followed in conduc-
tion disease clinics to define the natural history of
chronic second-degree AV nodal block. Some of the
patients were followed by communication with the
referring private physician. Syncope was defined as an
episode of transient loss of consciousness (usually
witnessed). The development of recurrent syncope, or
congestive heart failure with slow ventricular rates (or
requiring digitalization), were considered cause for
rehospitalization and insertion of permanent pac-
makers. Follow-up, as defined in this study (in regard
to development of syncope or mortality), was con-
tinued even when pacemakers were inserted.

Results

Clinical Findings

Detailed clinical data are presented in table 1. Forty-six men (82%) and 10 women (18%) were
studied, ages 18–87 years (57 ± 21 years, mean ± sp).
Patients without organic heart disease were younger
(39 ± 4 years, mean ± sem) than those with organic
heart disease (66 ± 3 years) (p < 0.001).

Nineteen patients (34%) had no evidence of organic
heart disease, and second-degree AV nodal block was
considered to be idiopathic. Of these 19, seven were
trained athletes (five were engaged in physical training
at the time of evaluation and two had stopped athletic
training 4–5 years before evaluation). All had been
trained in long-distance running or swimming.

Thirty-seven patients (66%) had clinically diag-
nosable organic heart disease. Nineteen patients (34%)
had arteriosclerotic heart disease, six of them had old
inferior wall myocardial infarctions, 10 (18%) had
hypertensive cardiovascular disease, three (5%) had
aortic valvular disease, three (5%) had primary con-
duction disease (Lev’s and Lenegre’s disease), one
(2%) had mitral valve prolapse and one (2%) had sar-
coidosis.

Chest roentgenogram revealed cardiomegaly in 27
patients (48%), with all these patients having organic
heart disease, except for a single trained athlete with
mild cardiomegaly. Thirty patients (53%) were in New
York Heart Association functional class I, 15 (27%)
were in class II, nine (16%) were in class III, and two
(4%) were in class IV at the time of evaluation.

Electrocardiographic Findings

ECGs available at the time of initial evaluation, as
well as serial ECGs, revealed episodes of second-
degree AV block type I (Wenckebach periodicity) in
all patients. Six patients also had persistent episodes
of 2:1 AV block with prolongation of the PR interval
of conducted beats in four. One patient had a single
recorded episode of repetitive block (two successive
blocked P waves during a Wenckebach period). No
patient had type II block.

The morphology of the QRS was narrow in 43
patients (77%), including all patients without organic
heart disease. Eighteen patients (32%) had intra-
ventricular conduction defects, 15 with organic disease
and three with primary conduction disease. One had
Table 1. Clinical, Electrocardiographic, and Follow-up Data

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Abbreviations: A = alive; ASHD = arteriosclerotic heart disease; ASMI = anteroapical myocardial infarction; AVD = aortic valve disease; Ca = neoplasia; CHF = congestive heart failure; CRF = chronic renal failure; D = death; HCVD = hypertensive cardiovascular disease; I = idiopathic; IRRBBB = incomplete right bundle branch block; IWMI = inferior wall myocardial infarction; LAHB = left anterior hemiblock; LBBB = left bundle branch block; LVH = left ventricular hypertrophy; MI = myocardial infarction; MVP = mitral valve prolapse; N = normal; NYHA = New York Heart Association; P = pneumonia; PCD = primary conduction disease; RBBB = complete right bundle branch block; TA = trained athlete; ? = reason unclear.
incomplete right bundle branch block, two had complete right bundle branch block, eight had right bundle branch block and left anterior hemiblock, one had right bundle branch block and left posterior hemiblock, four had left anterior hemiblock, and two had complete left bundle branch block.

Atrial rates ranged from 54–105 beats/min (73 ± 11 beats/min, mean ± SD). Seven patients had persistent sinus bradycardia and two had sinus rates consistently above 100 beats/min. Mean observed ventricular rates ranged from 32–102 beats/min (mean 62 ± 15 beats/min).

Electrophysiologic Data

Table 2 presents the electrophysiologic findings. Thirty-two patients (57%) were in spontaneous type I second-degree AV nodal block (block proximal to H) at the time of study and two patients were in 2:1 AV block, with AH intervals of conducted beats of 306 and 260 msec. The remaining 22 patients had 1:1 AV conduction at the time of study. These 22 had evidence of AV nodal dysfunction as manifest by at least two of the following: prolonged AH intervals (131–196 msec, mean 160 ± 20 msec) (14 patients); development of Wenckebach periodicity at atrial pacing rates less than 101 beats/min (70–100, 94 ± 10 beats/min) (20 patients); and prolonged functional and effective AV nodal refractory periods (all patients).

The HV interval was measured in all patients and ranged from 30–70 msec (mean 47 ± 9 msec). In patients without organic heart disease, the HV was normal (mean 44 ± 7 msec), but in patients with organic heart disease, it ranged from 33–70 msec (mean 49 ± 10 msec) and was prolonged in seven (19%).

The sinus node recovery time was measured in 42 patients, and ranged from 680–1490 msec (mean 1093 ± 174 msec). Sinoatrial conduction time was measured in 31 patients, and ranged from 15–144 msec (mean 89 ± 32 msec). Both measurements were within normal limits in all patients.

Clinical Course

The clinical course differed between the patients with and without organic heart disease, so that these groups are presented separately (table 1).

Patients with No Organic Heart Disease

Before electrophysiologic study, six of the 19 patients (32%) without organic heart disease had a history of syncope. Three of these patients had single syncopal episodes and three had multiple episodes (two, three and five episodes per patient). One of the patients with single syncopal episodes and all of the patients with multiple syncopal episodes were trained athletes.

None of the patients (with or without syncope) were treated with pacemakers at the time of electrophysiologic study because of the prospective nature of this study. Follow-up after electrophysiologic study ranged from 157–2280 days (mean 1395 ± 636 days). Five of the six previously syncopal patients did not have further syncope. One of the patients (a trained athlete) with recurrent syncope continued to have syncope, necessitating permanent pacemaker insertion on day 220. Pacing alleviated his syncopal episodes. Another patient received a pacemaker in another hospital for reasons that are unclear.

Two patients, ages 83 and 73 years, died nonsuddenly on days 341 and 1640 of the study, one of pneumonia and the second of acute myocardial infarction. The rest of the patients are all alive and well. None of these patients has shown progression of AV block during prospective observation.

Patients with Organic Heart Disease

In contrast to the patients without organic heart disease, 29 of the 37 patients (78%) with organic heart disease were symptomatic before study. Twenty-four (65% of organic heart disease patients) had clinically manifest congestive failure. Seven (19%) had a history of syncope before electrophysiologic study (single syncope in five and recurrent syncope in two).

Follow-up in the patients with organic heart disease ranged from 60–2950 days (mean 1347 ± 825 days). Permanent pacemakers were implanted in 10 of these 37 patients. In two patients, pacemakers were implanted for recurrent syncope (on days 2 and 77). Both had recurrent syncope before electrophysiologic study. In six, pacemakers were inserted to alleviate the bradycarrhythmic component of congestive heart failure or to facilitate management with cardiac glycosides. Two patients received permanent pacemakers at other institutions, one because of the development of advanced AV block with digoxin and one for reasons unknown to us. In two other patients, pacemakers were recommended to facilitate management of congestive failure, but were refused by the patients. No patient developed established complete AV block during follow-up.

Sixteen patients died, three suddenly (none had bifascicular block or permanent pacemakers), seven with congestive failure (including both patients who refused pacemakers), two with acute myocardial infarction, and four with non-cardiac-related causes (neoplasia in two and uremia in two). The remaining 21 patients are alive and symptomatically controlled. One of the patients with recurrent syncope who received a pacemaker was cured of syncopal episodes (the patient received a pacemaker on day 2), and another who received a pacemaker died with congestive failure but without recurrence of syncope.

Discussion

The incidence of second-degree AV block in healthy populations is considered low. Johnson et al.20 and Manning and Sears21 detected second-degree AV block in 12-lead ECGs in three of 67,375 healthy air force personnel and one of 19,000 healthy soldiers. More recently, Brodsky et al.22 using a 24-hour elec-
TABLE 2.  

Electrophysiologic Findings

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Abbreviations: ERP = effective refractory period; F = fast pathway; FRP = functional refractory period; PCL WP = paced cycle length producing Wenckebach periodicity; S = slow pathway; SACT = sinoatrial conduction time; SNRT = sinus node recovery time; W = spontaneous Wenckebach periods with block proximal to II.
trocardiographic monitoring, found nocturnal second-de гree AV block in three of 50 healthy male medical students. Although electrophysiologic studies were not performed in these studies, second-degree block in healthy persons was presumably AV nodal.

Second-degree AV nodal block appears in a variety of clinical situations. Most frequently, second-degree AV nodal block is seen in acute clinical settings, as in acute inferior wall myocardial infarction, digitalis intoxication, myocarditis (including rheumatic fever), and after cardiac surgery.1

In contrast to acute second-degree AV nodal block, chronic AV nodal block is recognized less frequently. It has been seen with chronic ischemic heart disease,2 mesothelioma of the AV node,3 atrial septal defect,4 aortic valvular disease,5 amyloidosis,6 Reiter's syndrome,7 mitral valve prolapse,8 in healthy populations,9 10 and in trained athletes.11

The electrocardiographic diagnosis of second-degree AV nodal block is traditionally based on the observation of type I block (Wenckebach periodicity), narrow QRS and facilitation of conduction with atrupine. Electrophysiologic studies performed in patients with second-degree AV block (acute and chronic) demonstrate an AV nodal site of block (block proximal to the His bundle recording site) in most patients with type I block.23 24 The results of the present study are consistent with these observations. We noted type I block on surface ECGs in all 56 patients with documented chronic second-degree AV nodal block. We also noted 2:1 AV block in six of the patients. One patient had a single episode of two consecutive blocked P waves,25 while no patient had type II block. Forty-three had narrow QRS complexes during second-degree block, and in 13, the QRS was wide (0.12 second or more) due to preexisting bundle branch block. (The accuracy of the surface ECG in predicting site of second-degree block is not addressed in this study.)

The relationship of AV nodal dysfunction to dysfunction elsewhere in the conduction system is unclear. Among our 56 patients with chronic AV nodal block, 18 had an associated intraventricular conduction defect. All these patients had organic heart disease and most presented with either congestive heart failure or syncope. This is consistent with our report of an association of prolonged AH intervals and myocardial dysfunction in patients with chronic bundle branch block.26 Chronic sinus node dysfunction (sick sinus syndrome) has been associated with concomitant AV nodal dysfunction.27 In the present series, only seven patients had associated sinus bradycardia, with atrial rates ranging between 54-58 beats/min. Sinus node recovery time and sinoatrial conduction time were normal in all patients, including the six patients with sinus bradycardia. None of the patients developed symptomatic sinus node dysfunction during follow-up. Our results suggest that clinically significant sinus node dysfunction is uncommon in patients with chronic AV nodal block.

Although chronic second-degree AV nodal block is traditionally considered benign, few data (other than anecdotal) support this premise. Young et al.28 reported 16 otherwise healthy children and adolescents with type I second-degree AV block. They noted progression to complete AV block in seven patients. They suggested that the prognosis of AV nodal block was not as benign as expected, and suggested that second-degree AV nodal block might be a step in the evolution of idiopathic AV block.

In the present series, 19 patients had no organic heart disease, and their course was relatively benign. Only one patient developed clear bradyarrhythmias necessitating permanent pacing (one patient received a pacemaker elsewhere for reasons that were not clear), and no patient developed established complete AV block. Two patients died nonsuddenly at ages 83 and 73 years. The remaining patients are alive and well. We do not know why our patients did better than those of Young et al.28 Perhaps natural history is worse when AV nodal block is detected in a predominantly pediatric population.

Our 37 patients with organic heart disease and AV nodal block had relatively advanced heart disease. Their prognoses were considerably worse compared with those without organic heart disease. Ten patients required permanent pacing, in most to facilitate management of heart failure. Sixteen patients died, 12 of related cardiac causes (including three sudden deaths) and four of nonrelated cardiac causes. The three sudden deaths were in nonpaced patients, suggesting that untreated bradyarrhythmias might have been responsible. However, all three patients were older than 60 years and had congestive failure and arteriosclerotic heart disease. We believe (although we do not have direct evidence) that these three sudden deaths were probably tachyarrhythmic and related to ischemic heart disease and could not have been prevented with permanent pacing.

Clinical Implications

Our study suggests that chronic second-degree AV nodal block occurs both with and without organic heart disease. More males than females have it, whether or not they have organic heart disease (in contrast to our previously reported series with chronic His bundle block, in which females predominated).29 Chronic second-degree AV nodal block is almost always characterized by episodes of type I block. The natural history is related to the presence or absence of organic disease; it is usually benign in the former and more malignant in the latter.

Without complicating organic heart disease, chronic second-degree AV nodal block is usually benign. Most patients do not need permanent pacing, because of lack of progressive bradyarrhythmia or bradyarrhythmic symptoms. These patients can be reassured without need for prophylactic pacing and followed periodically.

When chronic second-degree AV nodal block complicates organic heart disease, the clinical course tends to be more malignant (congestive failure and both nonsudden and sudden death). However, the clinical
course probably reflects the extent and severity of organic heart disease and not usually the presence of AV nodal block. We believe that permanent pacing in the patients with organic heart disease was of some value in management of congestive failure by correcting bradyarrhythmias and simplifying the use of cardiac glycosides. We cannot determine whether permanent pacing in some of the patients with organic heart disease made a significant positive impact on life history. We do not feel that routine prophylactic pacing is justified in the patients with organic heart disease, although we recommend pacing in this group when recurrent syncope occurs or when pacing might facilitate management of heart failure.

References
Natural history of chronic second-degree atrioventricular nodal block.
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