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Exercise-Induced Distal Atrioventricular Block

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Three patients with 1:1 atrioventricular (AV) conduction at rest developed fixed 2:1 or 3:1 AV block during treadmill exercise testing. Electrophysiologic study documented block distal to the AV node in all three patients, and suggested that the exercise-induced block occurred because of increased atrial rate and abnormal refractoriness of the His-Purkinje conduction system.

The findings in these three patients suggest that high grade AV block appearing during exercise reflects conduction disease of the His-Purkinje system rather than of the AV node, even in the absence of bundle branch block. Patients with this diagnosis should be considered for permanent cardiac pacing.

High degree atrioventricular (AV) block occurring during exercise in patients with intact conduction at rest is rare (1–3). The few published reports (4–7) have speculated on the site and pathophysiology of the block, but have not included confirmation by electrophysiologic study. This report describes three such patients, each with a documented site of block distal to the AV node.

Case Reports

Case 1

A 54 year old man gave a 3 year history of palpitation during exercise. He had no history of dizziness or syncope, and his physical examination, blood chemistry values and chest radiograph were normal. A 12 lead electrocardiogram showed a normal PR interval of 160 ms and normal QRS configuration and duration.

Before exercise testing, when the patient was supine at rest, his heart rate was 105 beats/min and AV conduction was normal (Fig. 1, upper tracing). When he stepped onto the treadmill, his atrial rate increased to 125 beats/min, 3:2 AV block developed and he experienced his usual episodes of palpitation (Fig. 1, middle tracing). The PR interval remained constant at 160 ms. Although block persisted throughout exercise, the patient was able to complete stage III of the standard Bruce protocol. At this time, the atrial rate was 180 beats/min and 2:1 AV block was present (Fig. 1, lower tracing). AV conduction returned to normal by 8 minutes after exercise when the atrial rate was 105 beats/min. The test produced no other symptoms or ST segment change.

After exercise testing, continuous electrocardiographic monitoring by telemetry demonstrated spontaneous AV block and a consistent relation between atrial rate and AV conduction. As long as the atrial rate was less than 110 beats/min, AV conduction was 1:1. However, when the atrial rate exceeded 110 beats/min, 3:2 or 2:1 AV block occurred without lengthening of the PR interval. This relation was consistent with that observed for atrial rate and AV conduction on the exercise test.

During electrophysiologic study, the AH interval was 100 ms and the HV interval was 70 ms. During atrial pacing, 1:1 conduction was maintained at rates less than 110 beats/min, but at rates of 110 beats/min or higher, 3:2 or 2:1 AV block occurred distal to the His bundle potential (Fig. 2). Because of the normal QRS axis, block within the His bundle was suspected but could not be confirmed by demonstration of split His potentials. The paced rate at which block appeared was identical to the sinus rate at which spontaneous AV block occurred.

The patient received a permanent ventricular demand pacemaker and has had no symptoms other than palpitation due to AV block at rapid sinus rates.

Case 2

A 69 year old man presented with a 4 month history of lightheadedness during exertion. He had no history of chest pain or prior heart disease, and his physical examination,

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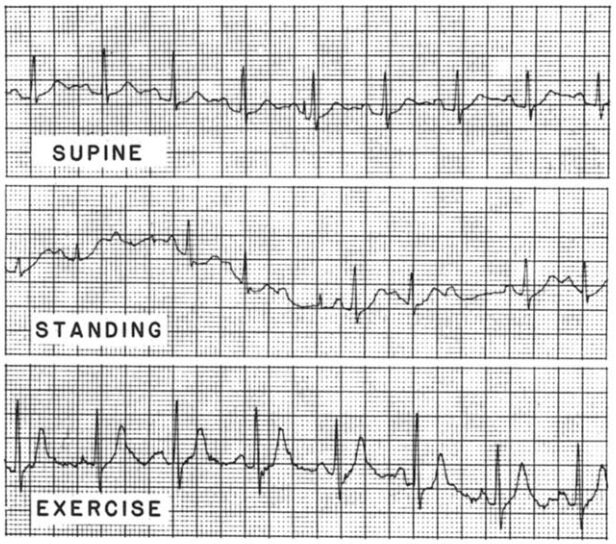

Figure 1. Case 1. Exercise electrocardiograms (lead II). **Top**: patient supine, atrial rate 105 beats/min, 1:1 atrioventricular (AV) conduction; **middle**: patient standing, atrial rate 125 beats/min, 3:2 AV conduction with PR interval constant at 160 ms; and **bottom**: after 9 minutes of exercise, atrial rate 180 beats/min, 2:1 AV conduction.

blood chemistry values and chest radiograph were normal. The electrocardiogram was unchanged from 2 years previously and demonstrated right bundle branch block with a normal QRS axis and PR interval.

Immediately before a treadmill exercise test, the rest heart rate was 60 beats/min with 1:1 AV conduction. After 2 minutes of stage I of the standard Bruce protocol, the atrial rate had increased to 140 beats/min, but 2:1 AV block limited the ventricular response to 70 beats/min. At 2½ minutes, the atrial rate was 150 beats/min with alternating 2:1 and 3:1 AV block producing an average ventricular response of 62 beats/min. The patient had no chest pain, but became lightheaded and the test was terminated. As the atrial rate slowed, 2:1 and then 1:1 AV conduction returned. The PR interval remained constant throughout the test.

During diagnostic cardiac catheterization, spontaneous increases in the atrial rate were observed to produce AV block. At atrial rates of 85 beats/min or less, AV conduction was 1:1; however, at faster atrial rates, 2:1 AV block occurred. This pattern continued throughout the procedure, and was unaffected by catheter placement or by left ventricular or coronary angiography. A His bundle recording during 2:1 block demonstrated block distal to the His potential, but no atrial pacing was performed.

Cardiac catheterization revealed 90% stenosis of the proximal right coronary artery and 60% stenosis of the left anterior descending coronary artery. However, normal wall motion and left ventricular function were maintained during two gated nuclear blood pool scans, one performed during exercise and the other during rapid ventricular pacing at a rate of 160 beats/min. After implantation of a permanent ventricular demand pacemaker, the patient was able to complete stage II of the standard Bruce treadmill protocol without lightheadedness or angina.

Case 3

A 55 year old man was referred for evaluation of progressive distal conduction disease. In 1968, his electrocardiogram was normal. In 1974 and 1979, right bundle branch block with a normal QRS axis was noted. In 1981, the appearance of new left posterior hemiblock (rightward QRS axis of $+110°$) prompted his referral for electrophysiologic study. He was asymptomatic and physical examination was remarkable only for a widely split second heart sound. Blood chemistry values and a chest radiograph were normal. A 24 hour Holter monitor recording showed no AV block and no change in the baseline QRS configuration, although the heart rate did not exceed 120 beats/min.

Electrophysiologic study demonstrated an AH interval of 70 ms and an HV interval of 65 ms during sinus rhythm. During atrial pacing, 1:1 AV conduction was maintained at rates of 160 beats/min or less, and AV nodal Wenckebach conduction occurred at rates of 180 beats/min or higher; however, block distal to the His bundle potential occurred at a paced rate of 170 beats/min (Fig. 3B). The first paced beat conducted normally, but the second paced beat conducted with a leftward axis of $-30°$ and an HV interval of 150 ms. We interpreted this pattern to represent block of

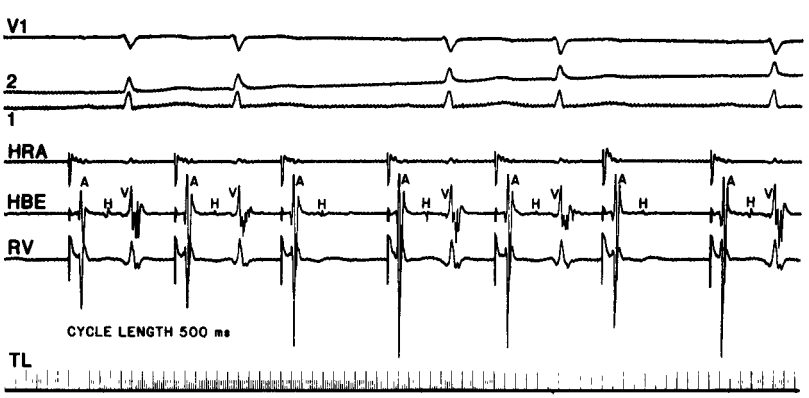

Figure 2. Case 1. Standard electrocardiographic leads and intracardiac electrographic tracings during atrial pacing, demonstrating 3:2 AV block distal to the His bundle potential. The AH interval is constant at 190 ms. A = right atrial deflection; H = His bundle deflection; HBE = His bundle electrogram; HRA = high right atrial electrogram; RV = right ventricular electrogram tracing; TL = time lines (inscribed at 10 and 50 ms intervals); V = right ventricular deflection; V_1, 2, 1 denote standard electrocardiographic leads V_1, II and I.

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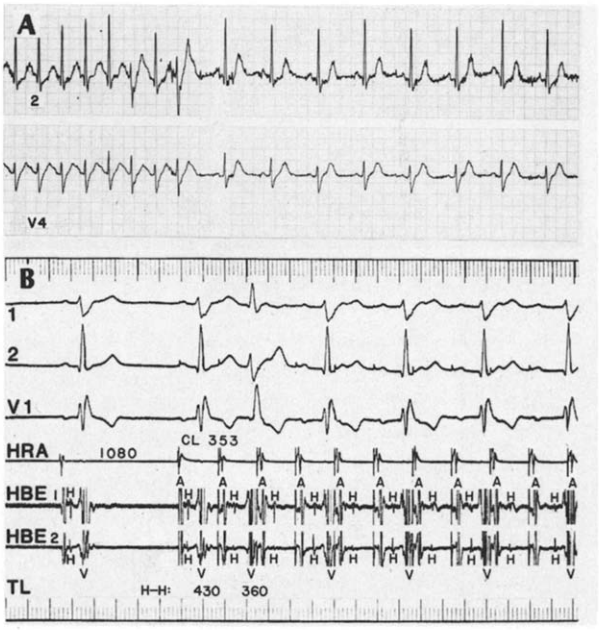

Figure 3. Case 3. **A**, Exercise electrocardiogram (standard leads II and V_4) showing an abrupt leftward shift in the QRS axis followed by 2:1 AV block. **B**, Standard electrocardiographic leads (I, II and V_1) and intracardiac electrograms during atrial pacing, demonstrating axis shift toward the left and prolonged HV interval of the second paced beat, followed by 2:1 AV block distal to the His bundle (H) potential. Paced cycle length is 353 ms. HBE_1, HBE_2 = His bundle electrograms from two different electrode pairs; other abbreviations as in Figure 2.

the impulse in the left anterior fascicle and slow conduction in the left posterior fascicle. The next impulse blocked distal to the His bundle potential, and a fixed pattern of 2:1 distal AV block followed.

Exercise testing the next day yielded a similar result. After 8 minutes of the standard Bruce protocol, at a heart rate of 140 beats/min, the QRS complex alternated for three beats between the baseline right bundle branch block with right axis configuration and the right bundle branch block with left axis configuration seen prior to the block during atrial pacing. This was followed by 2:1 AV block and a ventricular response of 70 beats/min (Fig. 3A). The patient immediately became lightheaded and dyspneic and the test was terminated. As the atrial rate slowed, AV conduction improved to 3:2 and finally to 1:1. The PR interval did not change during the test.

Because of the AV block during atrial pacing and exercise testing, a permanent ventricular demand pacemaker was implanted. The patient remains asymptomatic.

Discussion

AV nodal versus His-Purkinje block during exercise. Several authors (4–7) suggest that exercise testing may reliably differentiate between AV nodal and infranodal heart block. AV nodal block has been shown to improve or resolve during exercise (7–9), because the enhancement of AV nodal conduction by increased sympathetic and decreased parasympathetic tone during exercise more than compensates for the increase in atrial rate. In contrast, because the His-Purkinje system is relatively insensitive to autonomic modulation (10), comparable enhancement of distal conduction during exercise should not occur. Therefore, one would expect AV block that is induced by exercise to be located distal to the AV node. This hypothesis has not previously been confirmed. Reports (4,5,7) of high degree AV block developing with exercise in patients with 1:1 conduction at rest are rare and do not include documentation of the site of block.

Our observations in these three patients support this hypothesis. All three patients had 1:1 AV conduction at rest and developed 2:1 or 3:1 AV block during exercise. Conduction disease evident on pre-exercise electrocardiograms was varied: one patient (Case 3) had bifascicular block, one (Case 2) had right bundle branch block only and one (Case 1) had a normal QRS pattern. In each patient, AV block occurred without antecedent PR prolongation, suggesting block in the distal conduction system. Electrophysiologic study confirmed a distal site of block during spontaneous increases in sinus rate in one patient and during atrial pacing in the remaining two.

Rate-related AV block: mechanism. The pattern of block in our patients resembles that seen in patients with rate-dependent aberrancy. These patients reproducibly develop bundle branch block with a critical increase in sinus rate, and the block resolves when the sinus rate slows (11). This phenomenon has also been reported to occur during the tachycardia induced by exercise testing (12). Exercise-induced block in our patients was also related to increases in atrial rate. In Patients 1 and 2, an atrial rate was identified above which AV block consistently occurred, whether the increase in rate was spontaneous, paced or induced by exercise. When the atrial rate decreased below this critical level, 1:1 AV conduction returned.

The precise mechanism of rate-related block remains obscure (11). However, the pattern of AV block in these patients may be explained by a relatively fixed effective refractory period of the His-Purkinje system, which fails to decrease sufficiently with decreasing atrial cycle length to permit continued 1:1 AV conduction. If this mechanism is correct, the critical atrial rate above which distal block occurs is the atrial rate at which the HH intervals become less than the effective refractory period of the His-Purkinje system.

Block during exercise versus atrial pacing. In Patient 3, AV block also occurred with increases in atrial rate, but the rates at which block developed during exercise and atrial pacing differed by 30 beats/min. In both instances, 2:1 block was preceded by a change in the QRS complex from a left posterior to a left anterior fascicular block configuration. During atrial pacing (Fig. 3B), this change was likely ini-

tiated by a long-short cycle relation occurring at the onset of pacing, that is, a relatively long HH interval of the preceding sinus beat is followed by a relatively short HH interval which is less than the effective refractory period of the left anterior fascicle determined by the long preceding cycle length. However, during exercise (Fig. 3A), left anterior fascicular block occurred when the heart rate was stable, and 1:1 conduction had been maintained at this same rate during atrial pacing. The reason for block at this rate during exercise is not clear, but variability in the heart rate at which rate-dependent aberrancy appears on serial exercise tests has been reported (12). It is also possible that the block was initiated by a slightly premature atrial beat not evident from the exercise tracing. Despite this uncertainty, the similarity of the exercise-induced block to the block during pacing suggests a common site of block for both in the His-Purkinje system.

Role of ischemia during exercise. It is unlikely that exercise-induced ischemia was responsible for the development of heart block in our patients. Reversible fascicular block (13,14) and Mobitz type II block (5) may occur during exercise-induced ischemia in patients with coronary artery disease, and transient complete block may occur in the AV node during ischemia from coronary artery spasm (15). However, none of our patients had chest pain or electrocardiographic evidence of myocardial ischemia during exercise. In addition, AV block was reproduced at rest during electrophysiologic study when ischemia was unlikely. Although one patient (Case 2) had coronary artery disease, myocardial ischemia was not detected and pacemaker implantation alone relieved his symptoms.

Therapeutic implications. Rate-related AV block may be detected noninvasively by exercise testing, and should be considered in the differential diagnosis of dizziness or syncope. Because the natural history of patients with exercise-induced AV block is unknown, guidelines concerning pacing in these patients are speculative. In symptomatic patients, implantation of a permanent pacemaker seems justified. In asymptomatic patients the decision is more difficult, but the site of block is an important consideration. Asymptomatic patients with second degree or higher grade AV block distal to the AV node, either spontaneous (16) or induced by atrial pacing (17), have been shown to have a high incidence of subsequent symptomatic block. Permanent pacing for these patients is recommended (16–18). Because patients with exercise-induced AV block are likely to have a site of block distal to the AV node, permanent pacing may be appropriate in these patients as well. However, until the natural history of exercise-induced AV block is more clearly defined, evaluation for spontaneous or pacing-induced distal block should probably precede decisions concerning permanent pacing in these patients.

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