Exercise-Induced Second-Degree Atrioventricular Block

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In this report we describe 2 patients with exercise-induced, second-degree atrioventricular (AV) block. Case 1 was a 49-year-old man with normal AV conduction at rest but who developed dyspnea on exertion. Treadmill testing showed an exercise-induced 2:1 AV block. Electrophysiologic study (EPS) demonstrated rate-dependent, presumably intrahissian, AV block. Case 2 was a 31-year-old woman with first-degree AV block and complete right bundle branch block with dyspnea on exertion and occasional syncope. She had twice undergone surgical patch closure of an ostium primum atrial septal defect. Exercise testing induced type II second-degree AV block. Atrial pacing during EPS did not disclose rate-dependent type II AV block, but disopyramide induced second-degree AV block.

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Second-degree atrioventricular (AV) block induced by exercise is uncommon and has been found to be sinus rate dependent. Here we describe 2 cases of exercise-induced second-degree AV block. One (case 2) showed an apparent lack of dependency on sinus rate during EPS.

Case 1

Case 1 was a 49-year-old man who was hospitalized because of a 6-month history of palpitation and dyspnea on exertion. He had no history of dizziness, syncope, or chest pain. Chest radiography and routine laboratory test, including 2-dimensional echocardiography, were normal. A standard 12-lead electrocardiogram (ECG) revealed normal sinus rhythm at a rate of 68/min with normal 1:1 AV conduction (PR interval=180 msec; Fig 1A).

During treadmill exercise, 2:1 AV block occurred without prolongation of the PR interval at an atrial rate >85 beats/min (Fig 1B, upper trace), and the patient became dyspneic. There was no ischemic ST change. After exercise, AV conduction returned to normal at an atrial rate <85 beats/min. Twenty-four-hour Holter monitoring disclosed type II second-degree AV block at atrial rates above 84 beats/min (Fig 1B, lower trace).

After written consent was obtained, an electrophysiologic study (EPS) was performed with the patient in a non-sedated condition. His basic sinus cycle length was 780 msec, right atrial-His bundle deflection (AH) interval 80 msec and His bundle-right ventricle deflection (HV) interval 40 msec (Fig 2A). On atrial pacing at cycle lengths <710 msec, type II second-degree AV block occurred, and at a cycle length of 670 msec progressed to 2:1 AV block (Fig 2B). At <430 msec, the AV block advanced to a higher grade. Upon withdrawal of atrial pacing, 1:1 AV conduction was promptly restored.

Intravenous atropine- or isoproterenol-induced sinus tachycardia also provoked second-degree AV block at sinus cycle lengths <710 msec (Fig 2C). Coronary angiograms were normal. The patient underwent implantation of a permanent dual chamber (DDD) pacemaker and is now symptom free.

Case 2

Case 2 was a 31-year-old woman who underwent surgical patch closure of an ostium primum atrial septal defect (ASD) at the age of 8. She had been well until 23, when she underwent a second closure operation because of ASD patch failure. After this operation, 24-h Holter ECG monitoring disclosed...
Fig 1. Case 1. (A) Standard 12-lead ECG at rest. (B) Exercise ECG (lead II) during (top) and after treadmill exercise (bottom). P-P intervals are shown below the traces (in msec).

Type I second-degree AV block (Wenckebach), although the patient remained asymptomatic. For the past 5 years, she has experienced dyspnea and faintness on effort, and occasional syncope. Her physical examination revealed a regular pulse of 59 beats/min and blood pressure of 122/70 mmHg. Chest radiography showed a cardiothoracic ratio of 57% without signs of heart failure.

Her resting ECG exhibited a normal sinus rhythm at a rate of 60/min with 1:1 AV conduction, first-degree AV block (PR interval=220 msec) and complete right bundle branch block (QRS interval=180 msec) with a leftward deviation of QRS axis (−31°, Fig 3A).

During an exercise tolerance test, type II second-degree AV block was induced at an atrial rate of 107 beats/min, and the patient complained of dyspnea (Fig 3B). Immediately after the exercise, AV conduction returned to 1:1. Twenty-four-hour Holter ECG monitoring failed to detect any episodes of second- or advanced-degree AV block. Two-dimensional echocardiography showed slight dilation of the left atrium (41 mm).

EPS revealed 1:1 AV conduction associated with prolongation of the HV interval (75 msec) at sinus rhythm (Fig 4A). Atrial pacing induced type I second-degree AV block at cycle lengths <750 msec and 2:1 AV block at <600 msec. The block was intranodal.

Fig 2. Case 1. Intracardiac electrograms during sinus rhythm (A), during atrial pacing at a cycle length of 670 msec (B), and after intravenous injection of 0.04 mg/kg atropine (C). The sinus cycle length decreased to 660 msec after atropine injection. Abbreviations: HRA, high right atrial electrogram; HBE, His bundle electrogram; RV, right ventricular electrogram; S, stimulation of right atrial pacing; A, right atrial deflection; H, His bundle deflection; V, right ventricular deflection.
Fig 3. Case 2. (A) Resting standard 12-lead ECG. (B) Exercise ECG (lead II) during an exercise tolerance test.

(AH block, Fig 4B), and the HV interval remained identical. After intravenous administration of atropine (0.04 mg/kg), the longest pacing cycle length at which type I second-degree AH block occurred decreased to 375 msec. Atropine did not disclose latent HV block, but subsequently intravenous disopyramide (1 mg/kg) induced type II second-degree HV block at an atrial pacing length of 857 msec (Fig 4C).

Discussion

Approximately 20 cases of exercise-induced second-degree AV block in patients with 1:1 AV conduction at rest have been reported, 10 of whom have been investigated by EPS.

All patients showed provocation of second-degree AV block by atrial pacing in a rate-dependent manner, and the sites of block were distal to the AV node. Our case 1 also demonstrated a rate-dependent induction of type II second-degree AV block. The location of the AV block in this case was probably intranodal, because neither β-adrenergic stimulation nor vagolyis by atropine improved AV conduction, although the splitting of His potential could not be recorded. Exercise-induced increase in the atrial rate provoked AV block distal to the AV node because the effective refractory period of the His-Purkinje system was unaffected by autonomic modulation.

Case 2 appeared to develop second-degree AV block in a manner independent of the heart rate. Low-grade exercise such as talking induced syncope, whereas second-degree AV block with dyspnea developed at
an atrial rate of 107 beats/min during exercise testing. Although atrial pacing alone could produce only type I second-degree AV block at pacing rates up to 200/min, intravenous application of disopyramide disclosed the presence of infrahisian type II second-degree AV block. This latent infrahisian AV block (revealed by disopyramide) may be exacerbated by exercise-induced factors other than increases in the sinus rate. Ischemia-induced change in conductivity may be one such factor. However, this was unlikely because the patient showed no changes in the ST segment during exercise and AV block. Post-operative AV block may be another cause, and progressive fibrosis at the site of surgery may gradually affect the conduction system, although delayed-onset post-operative AV block is considered uncommon^{10}

References