CASE REPORT

Prolonged Ventricular Pauses in an Asymptomatic Athlete with "Apparent Mobitz Type II Second-Degree Atrioventricular Block"

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We report the case of a 30-year-old basketball player with asymptomatic, nocturnal ventricular pauses of >3,000 ms, the longest being ~12,000 ms, who was misdiagnosed with Mobitz type II second-degree atrioventricular (AV) block. Conversely, the tracings were characteristic of a vagally mediated AV block, a phenomenon first described by Massie and called "apparent Mobitz type II AV block."

Although the patient was asymptomatic with ventricular pauses occurring only at night, it was decided to implant a permanent pacemaker to prevent neurological damage or life-threatening ventricular arrhythmias resulting from repeated, abnormally prolonged ventricular pauses. The persistence of AV block after a 3-month detraining period led us to believe that our decision was reasonable. (PACE 2011; 1–4)

electrocardiogram, imaging, pacing

A 30-year-old basketball player attending regular training sessions (~6 hours per week) was referred to our institution because of prolonged ventricular pauses during Holter monitoring, requested by the sports physician for an occasional diagnosis of first-degree atrioventricular (AV) block on the rest electrocardiogram (ECG). The patient was asymptomatic with a history of vasovagal syncope after a blood draw, which occurred several years previously.

Holter monitoring documented 11 asymptomatic, nocturnal ventricular pauses of >3,000 ms (Fig. 1), labeled as Mobitz type II second-degree AV block. Several pauses were significantly prolonged, the longest being 11,976 ms (Fig. 1A). During 24-hour monitoring, average heart rate (HR) was 70 beats per minute (bpm), maximum HR was 138 bpm (four intervals), and minimum HR was 34 bpm (four intervals). The circadian variations of HR were normal. Three ventricular and no supraventricular premature beats were observed.

On admission, the patient was completely asymptomatic. The clinical examination did not reveal any abnormalities. A 12-lead ECG showed sinus rhythm at 55 bpm with PR interval of 206 ms. Laboratory tests, chest radiography, and transthoracic echocardiography were completely normal.

During the first day of recovery, a high number of prolonged nocturnal pauses were recorded by ECG telemetry, the longest being 8,800 ms (Fig. 2). The electrophysiological study did not reveal any conduction abnormalities of the sinoatrial and AV nodes, nor in the His bundle and bundle branches.

During the pauses recorded either with ambulatory Holter monitoring or by in-hospital ECG telemetry, there were one or more blocked P waves with a conduction ratio of 2:1, 3:1, and 4:1. The longest ventricular pauses were associated with profound sinus slowing and irregular PP intervals. The first PR intervals after the last dropped beat were equal, or shorter, or longer than the PR interval preceding the block. The QRS complex was always narrow. Several sequences were consistent with classic Mobitz type I second-degree AV block (Fig. 1B).

Although the patient was asymptomatic with ventricular pauses occurring only at night, it was decided to implant a VVI-mode single chamber permanent pacemaker because of the extremely long duration of pauses. The patient was discharged 2 days later.

After a 3-month detraining period postimplantation, Holter recording showed the persistence of AV block with appropriate pacemaker intervention (Fig. 3).

Discussion

In our patient, AV block was initially misdiagnosed as a Mobitz type II second-degree

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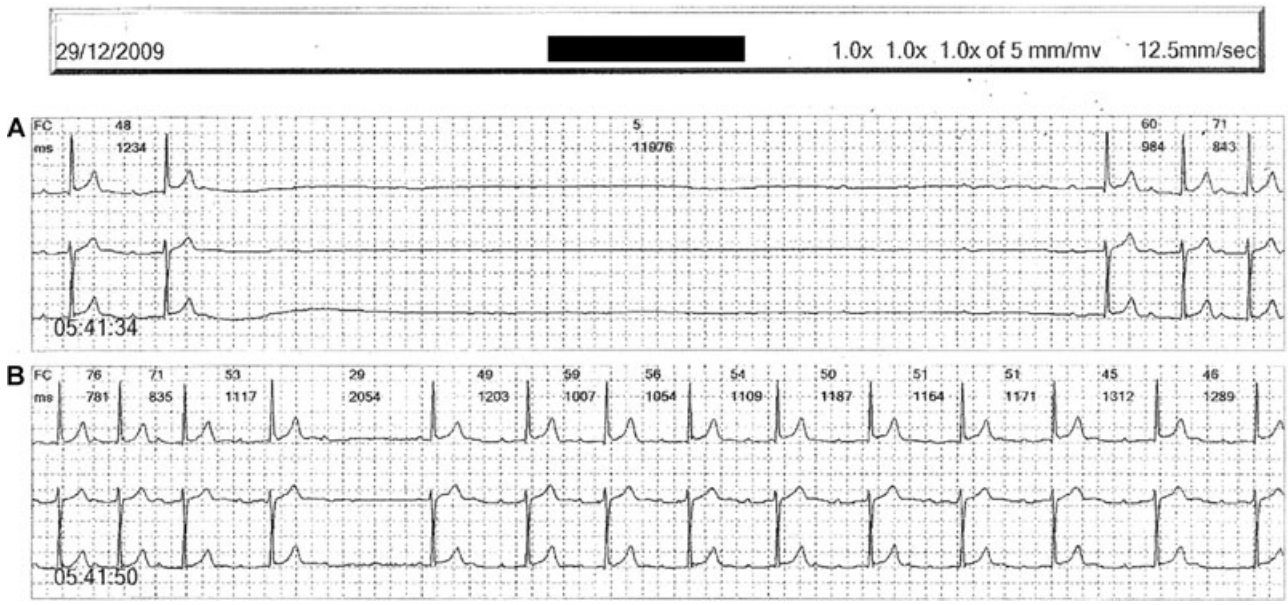

Figure 1. *Holter monitoring showing very long ventricular pauses. (A) 3:1 atrioventricular block with ventricular pauses of up to 11,976 ms. Severe sinus slowing with irregular PP interval. Late onset of the first P wave after the last QRS complex. The first PR interval after the last dropped P wave is equal to the PR interval preceding the block. (B) Type I atrioventricular block.*

AV block rather than a typical vagally mediated paroxysmal AV block, as described by Massie et al. in 1978.[1] Massie et al. called this phenomenon "apparent Mobitz type II AV block" due to the fact that, when observed in a superficial manner, this condition may mimic Mobitz type II second-degree AV block.[2]

The difference between these two diagnoses is not purely academic. Indeed, in Mobitz type II block, the anatomical location of the block is almost always below the AV node resulting in a poor prognosis, whereas in vagally mediated AV block, the site is within the AV node and the prognosis is generally good.[3]

A vagally mediated AV block is characterized by the simultaneous occurrence of abrupt sinus slowing and AV block. This type of AV block is considered a "type I variant" block[3] and is bradycardia-associated (not bradycardia-dependent). PP intervals are longer and irregular, the first dropped P wave occurs long after the QRS complex; the block is not always preceded by typical progressive PR prolongation or followed by PR shortening after the last dropped beat (the

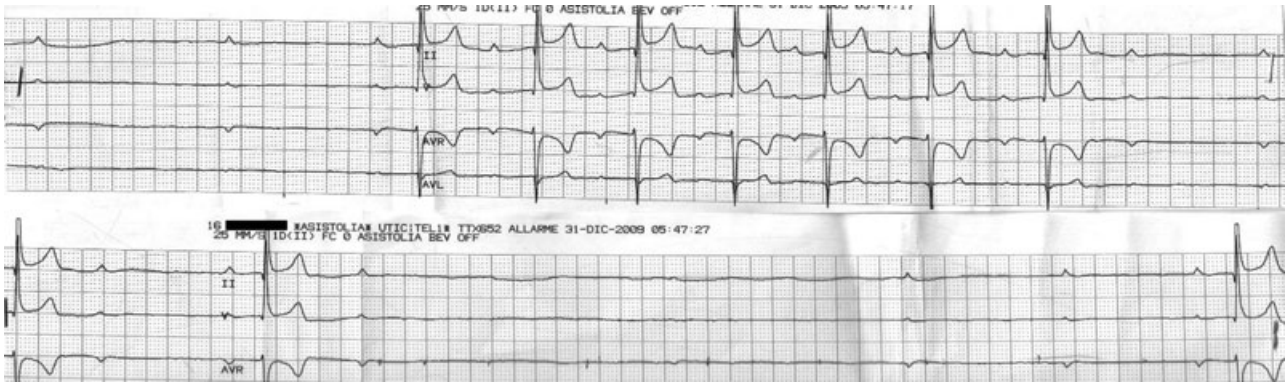

Figure 2. *4:1 atrioventricular block with ventricular pauses of up to 8,800 ms. The first PR interval after the last dropped beat is longer than the PR interval preceding the block.*

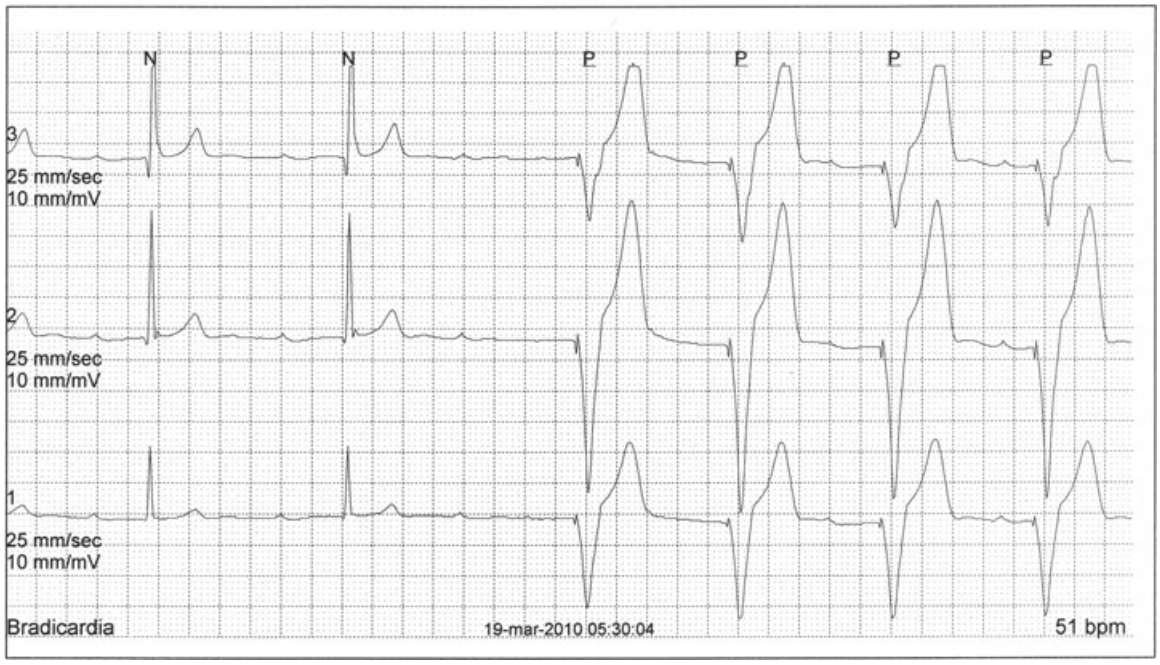

Figure 3. *After a 3-month detraining period, Holter recording showed the persistence of atrioventricular block with appropriate pacemaker intervention.*

first PR interval after the AV block may be longer than the PR interval immediately preceding the block). This type of AV block is caused by an acute increase in vagal tone that acts both on the AV and sinus nodes.

The fact that a 3:1 or higher AV block is often misdiagnosed as Mobitz type II block, in which the level of the block is more commonly infranodal rather than within the AV node,[3] may account for the initial erroneous diagnosis. According to current definitions, the correct diagnosis should have been "advanced second-degree block."[4] Another source of confusion derives from the fact that some authors cling to the original Mobitz definition,[5] whereby when all PR intervals remain constant before and after AV block of $\geq$2 consecutive beats in the setting of a stable sinus rhythm, such an observation should be labeled type II block.

In our athlete, the diagnosis of vagally mediated paroxysmal AV block was confirmed by (1) the simultaneous occurrence of sudden and serious sinus bradyarrhythmia (irregular PP intervals); (2) the late onset of the first blocked P wave after the last QRS complex; (3) the variable behavior of the first PR intervals after the last dropped P wave; and (4) the presence of concomitant Mobitz type I second-degree AV block.

Although sinus bradycardia, sinus arrhythmias, AV blocks, and ventricular pauses are common in athletes of all ages with a generally favorable prognosis, ventricular pauses of $\geq$3,000 ms are quite rare[6] but pauses of $>$10,000 ms are even rarer.

The clinical significance of ventricular pauses remains controversial, especially in asymptomatic and apparently healthy young athletes.[7,8] The length of pauses that appears critical to symptom onset can hardly be determined, as brain damage may be caused by either ventricular asystole or inadequate cardiac output and cerebral circulation.

Although not recommended by current guidelines,[4] we deemed it appropriate to implant a pacemaker to prevent neurological damage or life-threatening ventricular arrhythmias resulting from repeated, abnormally prolonged pauses. A 3-month detraining period can be considered long enough to abolish the effects of vagal hyperactivity induced by regular aerobic exercise training.[9] The persistence of AV block at 3 months postimplantation led us to believe that our decision was reasonable.

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