SPECIAL ARTICLE

Bradycardia and Prolonged Ventricular Pauses in Competing Athletes

Bradicardia y pausas ventriculares prolongadas en el deportista

ROBERTO M. PEIDRO[MTSAC, 1], ANTONIO PELLICCIA[2]

Cardiovascular modifications generated by high performance sports conform the athlete's hear syndrome. These changes are manifested in the physical exam, electrocardiographic (at rest and with stress exercise) and echocardiographic studies and even in magnetic resonance imaging. (1, 2)

Occasionally, the differentiation from diverse cardiac diseases is difficult and the decision to discharge or nor the athlete from sports activity can be complicated. (3)

Although ending sports activities in a patient with certain cardiac diseases may protect him from a fatal event, unnecessary athlete disqualification is usually associated with psychosocial and economic problems which significantly alter quality of life.

Severe sinus bradycardia (SB) and presence of prolonged ventricular pauses in Holter monitoring is one of the situations generating doubts in the conduct to follow in the asymptomatic athlete. When present, the athletes are occasionally discouraged to continue sports training, and even treatments, such as pacemaker implantation, may be suggested.

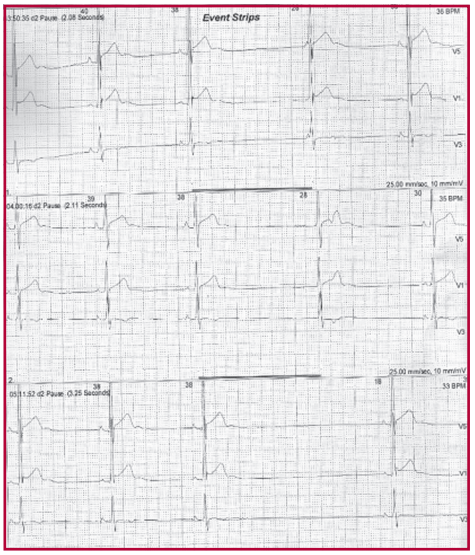
The purpose of this presentation is to pose different cases with comments on the adopted decision.

Four cases of asymptomatic high-performance athletes with prolonged ventricular pauses on Holter monitoring will be presented. Athletes had no structural heart disease (normal Doppler echocardiography and magnetic resonance imaging with and without gadolinium) and with adequate chronotropic response to exercise. A brief review and discussion will follow this presentation.

CASES

Case 1

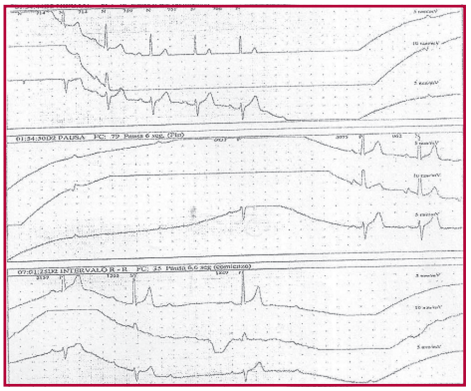
Twenty-three year-old professional football player with up to 3,250 ms pauses on Holter monitoring. At that time, his international transfer to a European sports institution was discouraged.



Progression: Nine-year follow-up. Asymptomatic. Continues working as a football player in a first division team.

Case 2

Asymptomatic 32-year-old competitive male volleyball player in a first division team. Electrocardiogram (ECG) with SB. Up to 6,600 ms pauses on Holter monitoring.



REV ARGENT CARDIOL 2017;85:133-136. http://dx.doi.org/10.7775/rac.v85.i2.9463

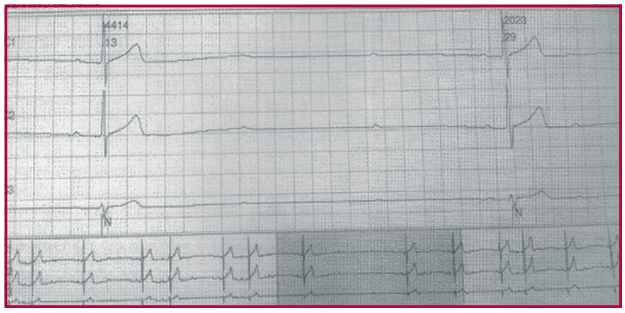
Address for reprints: Roberto M. Peidro - Futbolistas Argentinos Agremiados - Salta 1144 - CABA - e-mail: rpeidro009@hotmail.com

MTSAC Full Member of the Argentine Society of Cardiology
[1] Department of Cardiology, Sports Medicine Institute - Futbolistas Argentinos Agremiados
Chair of Sports Medicine - Universidad Favaloro
[2] Institute of Sports Medicine and Science. Roma, Italy

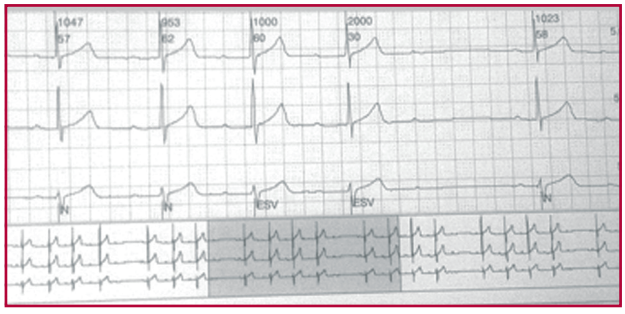
Sinus bradycardia. Mobitz I second degree A-V block and blocked P waves in the context of SB with no prior PR prolongation. Up to 6,600 ms nocturnal pauses. The patient refused pacemaker indication. He continued with sports activity to the age of 35 years. 20-year follow-up: He remains asymptomatic and without events, leading a sedentary life.

Case 3

Nineteen-year-old rugby player with SB in the ECG and up to 4,414 ms pauses on 24-hour Holter monitoring. Two blocked P waves are also observed in the ECG.

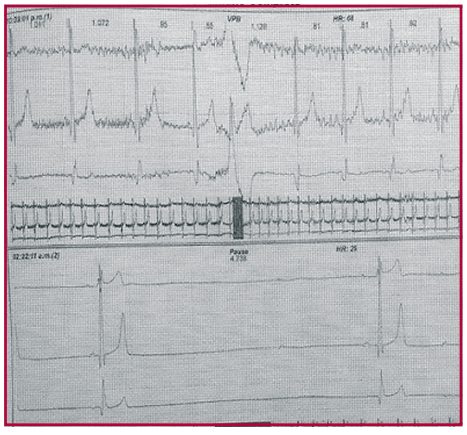


Progression: New assessments performed with transient training suspension showed significant reduction in the number of prolonged ventricular pauses >3,000 ms.

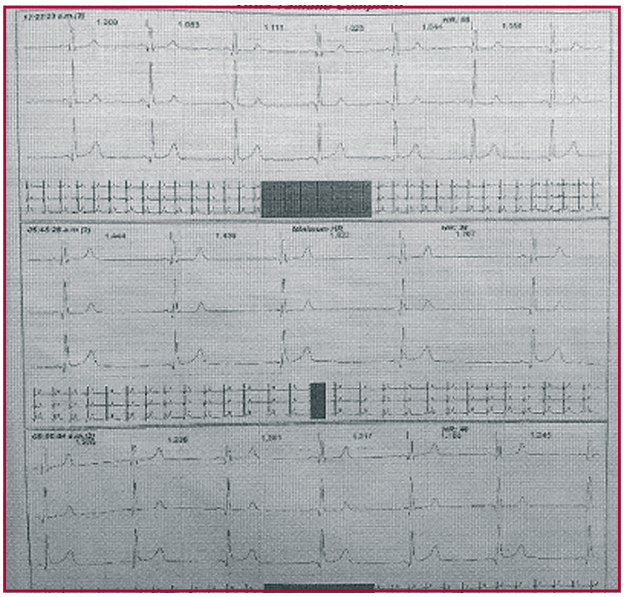


Case 4

Nineteen-year old rugby player with up to 4,730 ms pauses on 24-hour Holter monitoring and blocked P waves. More prolonged posterior PR than prior to the blocked P wave.



Progression: Sixty-day training interruption: no pauses >1,700 ms were recorded.



FINDINGS AND MECHANISMS

Sinus bradycardia is considered the most frequent finding in the athlete ECG. Although its prognosis is generally benign in young athletes, its limits and long-term progression have not been sufficiently studied. (4-7)

Sinus bradycardia may be attributed to increased vagal tone and receptor density reduction in the whole organ or, specially, in the inferior left ventricular wall. (8-10)

Additional mechanisms have been described, such as reduced sensitivity to catecholamines, altered neural input to the sinus node and carotid bulb and left ventricular baroreceptor stimulation due to greater contractile force and increased afferent vagal reflex with acetylcholine release and blockade of adrenaline release. (11, 12)

However, electrophysiological studies performed in athletes and sedentary subjects with sympathetic and parasympathetic blockade showed the existence of non-autonomic influences in the athlete SB at rest. (13). Ion channel remodeling is another mechanism proposed for the athlete SB, and even more, for abnormal A-V conduction, more prevalent in athletes than in the general population. (14) The elevated presence of SB explains the greater prevalence of A-V junctional escape rhythms in athletes than in sedentary subjects.

Electrocardiographic and Holter monitoring studies showed that PR interval prolongation >200 ms occurs in 10% to 37% of trained individuals compared with only 0.65% of normal sedentary subjects. (15) Its frequency of occurrence, as in SB; depends on the circumstances in which the electrocardiogram was recorded, the subject's anxiety level and state of relaxation, as slight increases in the sympathetic tone normalize conduction. The PR interval shortens with

exercise while the delay in A-V conduction elicited by exertion may be considered pathologic.

Mobitz 1 second degree A-V block (with Wenckebach periods) has been described in 1% to 10% of athletes practicing different disciplines, and different publications consider it a common finding in the athlete ECG. (16) Also, 2:1 A-V and even high grade blocks are described, with ventricular pauses including various non-propagated P waves. This presentation, associated with SB (though not bradycardia–dependent), is not preceded by a progressive prolongation of the A-V interval or followed by a shorter PR after the blocked P. Even the appearance of the first blocked P wave may be delayed after the QRS complex. This type of blockade is caused by an acute increase of the vagal tone acting on the sinus and A-V nodes and has been described as "apparent Mobitz II A-V block". It is characterized by the simultaneous occurrence of SB and A-V block and could be considered a type I A-V block variant. (17-18)

CONDUCTS AND PROGNOSIS

Some athletes present with ventricular pauses > 3000 ms at rest. These findings are more common in aerobic resistance athletes, though they can also be registered in other sports. According to several authors, in the absence of symptoms and evidence of structural cardiac disease and adequate chronotropic response to exercise, these athletes should not be excluded from their usual sporting activities. (19)

However, the tolerable limit of pause duration to define sports interruption, or even pacemaker implantation, is not well established.

Pacemaker therapy guidelines do not recommend its implant in asymptomatic trained subjects with prolonged ventricular pauses, though they do not mention the maximum extent of these pauses. Neither do they recommend pacemaker implantation in Mobitz 1 second degree A-V block nor when it is not possible to know whether the blockade is supra or infrahisian, or when it responds to non-permanent situations as the increase in vagal tone. (20)

The natural history of asymptomatic athletes with prolonged ventricular pauses detected by Holter monitoring is not clearly known. Few investigations have assessed the prognosis of these athletes. In some cases, pacemaker implantation has been indicated due to very prolonged pauses (over 10 ms in Holter monitoring), though not based on randomized controlled trials. (18)

A study with more than six thousand consecutive patients evaluated with Holter monitoring due to bradycardia symptoms or findings in the resting ECG showed a prevalence of pauses above 3 seconds in 0.8% of cases. These pauses were not an indication of poor prognosis at an average follow-up of 3 years. (21) Asymptomatic nocturnal pauses above 3 seconds, either by SB or atrial fibrillation, in elderly non-athletic individuals did not produce greater mortality compared with control groups. (22) Ogawa et al. at Sapporo Hospital in Japan studied 30 high-performance skiers with Holter monitoring and compared them with 24 sedentary controls. (23) Ventricular pauses were more frequent in the athletes (2,200±600 vs. 1,600±300 ms), with 66.7% of athletes having pauses >2,000 ms and 16.5% >3,000 ms. No athlete died or had symptoms during the 3-year follow-up in which they continued with their high-performance sports activity, which means that the prognosis of athletes with pauses above 3 ms does not differ from those with normal rhythm.

Among 16 medium and long-distance runners with 48-hour Holter monitoring, 12 presented with pauses between 2,000 and 3006 ms and Mobitz I and II second-degree A-V block was verified. (24). Compared with sedentary subjects, runners had in average 14 beats per minute less than non-active persons in the nocturnal recording. These findings were associated with greater heart rate variability in the whole spectral band, suggesting an increased parasympathetic tone that might explain the pronounced SB in athletes.

A recent review of the literature including studies of athletes with pauses above 3 seconds concluded that no adverse effects could be demonstrated at follow-up or that these pauses were predictors of symptoms. (25) Furthermore, the same publication presented the case of an athlete with multiple nocturnal pauses between 7 and 13 seconds without alterations or interruption of his sports activities at a follow-up of 3.5 years.

The American recommendations to disqualify or select athletes with cardiac disease claim that sinus bradycardia or block does not require treatment in athletes nor do they inhibit the practice of sports. However, they do not mention the length of ventricular pauses. (26)

Is there scientific support to discourage the practice of sports in an asymptomatic athlete with pauses at rest >3,000 ms? And, even more, to indicate a pacemaker? We believe there is not enough evidence or randomized controlled studies demonstrating this position.

Transient interruption of training could, in many cases, banish longer pauses or decrease them in number and/or time between RR intervals. In these cases, should sports practice be interrupted in order not to return to previous findings? It does not seem to be the most reasonable approach without structural heart disease and with adequate heart rate response to exercise, where no secondary effects have been demonstrated due to the presence of prolonged ventricular pauses. Presence of a "threshold pause" should be established in studies with extended follow-up, since 3 seconds do not seem to discriminate athletes with or without future abnormalities.

Conflicts of interest
None declared.

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