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Does mild symptomatic bradycardia in a young active female need pacemaker?

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Case report

46 years old women presented suddenly as fainting attack during her work shortly admitted to the emergency department and the initial work-up, a baseline electrocardiogram revealed irregular rhythm at 41 beats per minute (bpm). The patient denied ever having symptoms of presyncope, syncope, or generalized weakness. A Junior doctor in the emergency department treated patient with Amiodaron infusion to overcome atrial fibrillation.

The patient was monitored on telemetry during his 3-day admission and received Amiodaron orally 200mg×3, in addition, had a 48-hour Holter monitoring performed. He was found to have episodes of atrial fibrillation. A thorough history of the patient's dietary supplements was obtained, none of which were known to cause bradycardia.

During this period, transthoracic echocardiogram was performed, which did not reveal any evidence of structural heart disease. Specifically, left ventricular chamber size and wall thickness were normal. Electrophysiology consultation was obtained; after reviewing the tracings, the impression was that the patient exhibited the effects of increased vagal tone during sleep. Because the patient refused any further testing, the electrophysiologist referred to the phenomena of sinus node slowing.

The patient discontinues of Amiodaron after 3 months, she has regular rhythm 41bpm and return back to her normal work, the patient has another sudden fainting attack after 5 months of first one, but without AF. The heart rate 38 bpm regular rhythm her doctor recommends pacemaker.

Keywords: Bradycardia; Electrophysiology; Pacemaker

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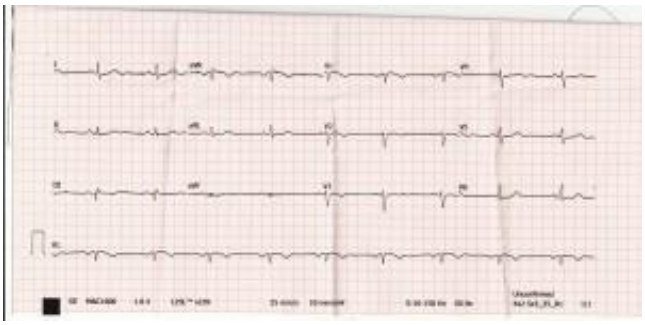

Discussion

Sinus bradycardia is one of the two major causes bradycardia, or slow heart rate. In sinus bradycardia, the slowing occurs because the heart's sinus node has reduced the rate at which it generates the electrical impulses that control the heart rhythm. In many circumstances, sinus bradycardia is entirely normal.

In other circumstances, sinus bradycardia causes the heart rate to become inappropriately low, which can lead to symptoms and may require a pacemaker. It has been well documented that bradyarrhythmias are a common finding in highly trained athletes, resulting in resting heart rates as low as 25 bpm [1], First, second, and third-degree heart block as well as sinus pauses have been recognized to occur in athletes [2].

Several medical conditions can cause abnormal sinus bradycardia that is sinus rates are inappropriately reduced. The most common is the intrinsic sinus node disease. Usually, sinus node disease is caused by an age-related fibrosis of the sinus node; it is a disorder of aging. The intrinsic sinus node disease is most commonly seen in people who are 70 years old or older. Sinus pauses lasting more than 2 seconds occurred in greater than one third of athletes evaluated in 1 study [3].

In the absence of organic heart disease, these changes appear to be reversible with the cessation of high-performance sports and are thought to be due to the elevated vagal tone common in athletes [4]. Less has been written about sustained ventricular pauses in a patient in sinus rhythm. While prolonged ventricular pauses as long as 16 seconds have been documented during episodes of OSA [5], they are of uncertain significance, because they occur in a patient population which is typically obese with significant comorbidity.

Less has been studied about the significance of ventricular pauses in seemingly healthy, athletic individuals.

One prospective study found that highly trained athletes with ventricular pauses as long as 3 seconds failed to show evidence of increased clinical risk compared with age-matched controls concerning syncope, near syncope, or death at long-term follow-up, despite continued training [6].

Our patient in the above case is unique in that the length of ventricular pauses was significantly longer than those found in the literature with regard to athletes. Our patient did not have sinus

pauses, as he maintained sinus rhythm throughout each event. His sinus node activity becomes relatively slowed during these ventricular pauses.

Vagal inhibition of AV nodal activity and slowing of ventricular rhythm have been demonstrated [7-9], which would explain the lack of junctional or ventricular escape rhythm in our patient. Interestingly, bradycardia and a systole were observed during direct vagal nerve stimulation in the treatment of epilepsy, further demonstrating the susceptibility of the AV node to vagal stimulation [10-12].

The prognostic significance of this patient's clinical presentation is unknown, although the literature suggests that sinus bradycardia in athletes is usually not concerning unless it is symptomatic or produces ventricular pauses exceeding 4 seconds.

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