CASE REPORT

Arrhythmias in an athlete: the effect of de-training

O A Obel, C Davidson

A 53 year old athlete with a history of severe palpitations and lightheadedness presented for a second opinion. He was found to exhibit very frequent atrial ectopy, frequent runs of symptomatic atrial tachyarrhythmia, and sinus bradycardia at rest. During exercise testing, his tachyarrhythmias increased in relation to the duration and intensity of exercise. A therapeutic trial of de-training was suggested. As a result, his symptoms completely resolved with a marked reduction in the frequency of atrial arrhythmia. Repeat exercise testing revealed an excellent exercise tolerance with no atrial ectopy. De-training should be considered when athletes present with arrhythmias.

Patients who present with both tachyarrhythmias and bradyarrhythmias pose complex clinical and therapeutic challenges. Adults who are highly trained have an increased incidence of both tachyarrhythmias and bradyarrhythmias. A standard approach to symptomatic tachy-brady syndrome is likely to include long term antiarrhythmic drugs, often in combination with permanent pacing. We describe a middle aged athlete who presented with highly symptomatic arrhythmias in whom a short de-training regimen resulted in total resolution of his symptoms.

CASE REPORT

A 53 year old man presented for a second opinion. He had a one year history of severe palpitations, particularly during exercise, and light headedness occurring at times of rest. His symptoms had been diagnosed as being due to the tachy-brady syndrome and he had been told that this would require antiarrhythmic drug treatment combined with permanent pacemaker implantation. He was a business executive with no prior medical history of note who had been a dedicated athlete throughout adult life. At the time of consultation he was actively training for a triathlon, which would comprise a 20 mile run, followed by a 1.5 mile swim, and finally a 50 mile cycle ride. He had no known risk factors for ischaemic heart disease or cardiomyopathy.

On examination he appeared healthy. The pulse was intermittently irregular, ventricular rate averaged 90 beats per minute (bpm). Resting 12-lead electrocardiography revealed frequent atrial ectopy with no evidence of pre-excitation, axis deviation, QT prolongation, or prolonged atrioventricular conduction. Transthoracic echocardiography revealed normal left ventricular internal dimensions (5.4 cm diastole, 3.2 cm systole), normal septal and posterior wall thickness (0.9 cm and 1 cm respectively), an ejection fraction estimated at 70%, a normal diastolic ventricular filling pattern (E/A ratio 1.25), and normal valvular function.

A 24 hour Holter monitor revealed high degree atrial ectopy (up to 680 ectopic beats per hour), and frequent paroxysms of symptomatic atrial tachycardia and atrial fibrillation. There was sinus bradycardia during rest and sleep with a minimum heart rate of 29 bpm, and frequent sinus pauses, up to 3.4 seconds. During an exercise test he easily reached stage 4 of the Bruce protocol. High degree atrial ectopy was observed, increasing in relation to the duration and intensity of effort such that at peak exercise, the rhythm consisted mostly of rapid runs of atrial tachycardia (fig 1).

As a therapeutic measure, a period of de-training was recommended. After an initial three week period of no physical training whatsoever, he was allowed to exercise to a maximum level of a three mile run, up to twice weekly. At follow up after three months he reported an amelioration of his palpitations and light headedness. Follow up Holter monitor at that time yielded only seven atrial ectopics/24 hours compared with 8510 ectopics/24 hours before de-training therapy, and no atrial fibrillation. There was no further bradycardia. Repeat exercise testing revealed an excellent exercise tolerance with a peak sinus rate of 175 bpm; no atrial ectopy was noted (fig 2). This therapeutic response was sustained at follow up six months later.

DISCUSSION

We describe an athlete who, after presenting with symptomatic exercise related atrial tachyarrhythmias and resting sinus bradycardia, had a complete therapeutic response to de-training. He was able to return to a moderate level of exercise, free of arrhythmia, and without the need for antiarrhythmic drug treatment, or pacemaker implantation.

When presented with an otherwise healthy, middle aged patient with symptomatic tachy-brady syndrome, many physicians and cardiologists alike would consider antiarrhythmic drug treatment combined with pacemaker implantation as a reasonable first option in many cases, although few would suggest that this strategy is not without significant obstacles, and is rarely completely effective. Severe bradycardia occurring in athletes can sometimes respond to de-training. Ector et al reported a complete resolution in eight of 16 patients of severe, symptomatic bradycardia in response to de-training.1 To our knowledge ours is the first case report of pathological tachycardia resolving in response to de-training.

Abbreviations: bpm, beats per minute; PRAP, prolonged rapid atrial pacing
Atrial fibrillation occurs in conditions of enhanced sympathetic activity such as hyperthyroidism, and in the period after cardiac surgery. Atrial fibrillation occurs at an increased frequency in elite athletes, and in a significant percentage of patients, arrhythmias occur during effort. A study which followed up 300 male veteran athletes aged 35–59 years for 11 years found that, despite a lower mortality and a lower incidence of coronary heart disease compared with age matched male controls, the prevalence of atrial fibrillation was increased in the athletic group, with 5.3% of veteran athletes compared with 0.9% of controls developing atrial fibrillation during the follow up period.

The autonomic triggers of atrial fibrillation in athletes are complex. One mechanism by which long periods of intense physical activity can result in a propensity to atrial tachyarrhythmia is apparent from studies using prolonged rapid atrial pacing (PRAP) as a method of inducing sustained atrial fibrillation in animal models. Studies examining the effects of PRAP on the electroanatomic remodelling of the atria have shown that sympathetic hyperactivity occurs, which has a powerful influence on the maintenance of atrial fibrillation in such conditions.

A tendency to bradycardia has been well characterised in athletes. In 37 asymptomatic, highly trained adults aged 17–40 years, nocturnal bradycardia <40 bpm was present in 67%. The symptomatic bradycardia and sinus pauses that occurred in our patient are likely to have arisen, at least in part, due to increased parasympathetic tone which is present in athletes. Chronic sinus node suppression may also occur as a result of long periods of exercise related sinus tachycardia and atrial tachyarrhythmia. This phenomenon was demonstrated in a study in which two to six weeks of atrial fibrillation (induced by PRAP) resulted in prolongation of the sinus node recovery time, and a reduction in the maximal sinus rate, independent of autonomic tone.

In our patient, sympathetically mediated atrial tachyarrhythmia resolved in response to de-training, apparently as a result of down-regulation of atrial sympathetic activity. The bradycardia also responded to detraining, probably as a result of a fall in resting parasympathetic tone and/or from the resolution of sinus node suppression by supraventricular tachyarrhythmia. Thus a period of de-training should be always considered as a therapeutic strategy when highly trained athletes present with
tachyarrhythmias or bradyarrhythmias, once cardiac and other causes have been excluded.

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References

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