

Haemodynamic effects of paroxysmal supraventricular tachycardia in an endurance athlete during exercise testing

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DESCRIPTION

Atrial fibrillation (AF) is a commonly known heart rhythm abnormality which occurs in the general population as well as in endurance athletes. Various studies have provided evidence of increased prevalence of this condition in such athletes compared to the general population; various potential trigger mechanisms have been discussed previously.^{1,2} We present the case of a 60-year-old male endurance athlete who participated in several triathlons of 'ironman distance' during his sports career. He was 188 cm tall, weighed 92 kg (body mass index (BMI) 26) and his training level averaged 10-15 hours/week (swimming, cycling and running). The 60-year-old patient presented in our department with unknown symptoms, including intermittent shortness of breath during his training sessions and heart palpitations. His medical history showed an isthmus ablation caused by typical atrial flutter and a clinically stable arterial hypertension. The echocardiographic data showed a normal size of the left ventricle (42 mm), no hypertrophy of the left ventricular walls (IVSd 11 mm, PWTs 11 mm), minimal enlargement of the left atrium (LA 21 qcm) and a normal ejection fraction (55%).

In order to examine the patient more extensively, we decided to perform a cardiopulmonary exercise testing (CPEX) on an ergometer bike. During the test, the patient developed an episode of paroxysmal supraventricular tachycardia (SVT, maximum

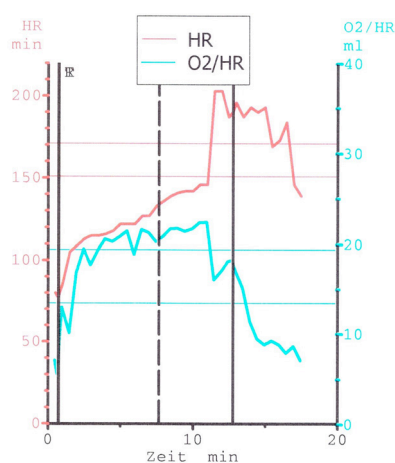


Figure 1 Impairment of the oxygen pulse during the CPEX caused by atrial fibrillation (starting within minute 11) and showing the gap between the tachycardia (red) and oxygen pulse (blue). CPEX, cardiopulmonary exercise testing.

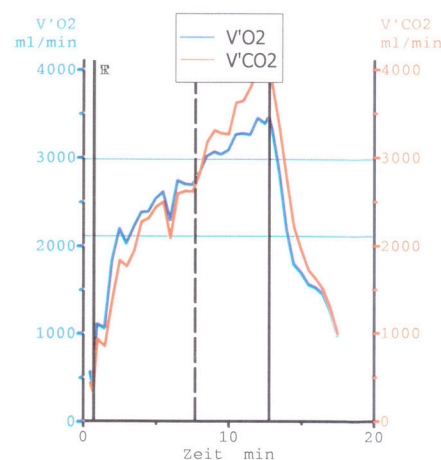


Figure 2 Graphical record of the athlete's impairment referring his maximum VO_2 uptake caused by atrial fibrillation, Missing the typical levelling off phenomenon by endurance athletes at the end of the CPEX testing. CPEX, cardiopulmonary exercise testing.

effort 250 W). We were able to graphically record the degree of impairment of the athlete's performance (figures 1 and 2). The patient's oxygen pulse decreased pathologically and his VO_2 oxygen uptake during the SVT was limited (figures 1 and 2). After he completed ergometer testing, the patient recovered very fast and his heart rhythm returned to a stable sinus rhythm, similar to pretesting conditions. Our case graphically shows the haemodynamic influence and reduced performance of a patient with a paroxysmal AF during CPEX and therefore highlights the importance of a stable sinus rhythm for optimal performance in endurance athletes. Various aspects including the pathological mechanisms and the relation between high endurance training and the prevalence of AF are widely discussed. Changes in the left atrial substrate, that is, an increased size of the LA and histological fibrosis, might also play an important role. Additionally, the amplitude of the autonomic nervous system may increase the risk of developing AF in intensive endurance athletes.^{1,3} These presumptions can be confirmed by an animal model in rats in which chronic endurance exercise increased the AF susceptibility with atrial dilatation, autonomic changes and fibrosis. Even augmented baroreflex responsiveness and increased cardiomyocyte sensitivity to cholinergic stimulation are presumed to cause higher prevalence of AF.⁴ Monitoring the training of endurance athletes shows some evidence that an accumulation of lifetime



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training hours and participation in competitions substantially increases the risk of AF in aging endurance athletes.⁵ The risk of AF in young competitive athletes is estimated to be low⁵ due to a U-shaped dose dependency in relation to the athlete's activity.¹ The development of AF in our endurance athletes collective with performed ablation of atrial flutter is a commonly observed occurrence.⁶ In long-term follow-up, paroxysmal AF remained stable in half of the athletes, only developing into permanent AF in a minority of this population.³ The previously mentioned strenuous endurance exercise (SEE) related AF or paroxysmal AF in young and middle-aged athletes syndrome⁷ is quite a common finding in (young) endurance athletes, and even our athlete/patient could fulfil its criteria. A relatively common finding in highly trained endurance athletes is an atrial cardiomyopathy which is characterised by right and left atria enlargement and fibrosis (often detected in an MRI).

Learning points

- ▶ The presented case graphically shows an impressive haemodynamic influence of heart rhythm abnormality on the athlete's performance during a cardiopulmonary exercise testing.
- ▶ While different pathological mechanisms for underlying etiological reasons are discussed, maximum effort should be exerted to establish a stable sinus rhythm in endurance athletes.
- ▶ Therapeutic approaches should be individually tailored according to the athletes' demands.

Finally, although some evidence regarding atrial anatomic adaptations was reported by Müssigbrodt *et al*¹ and by Fragakis *et al*⁸, alterations in autonomic nervous system, chronic systematic inflammation and fibrosis have been proposed as potential mechanisms for paroxysmal SVT induced by endurance exercise.^{2 5 8} However, this hypothesis is speculative.

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