

# ATHLETE'S HEART

## WHAT IS IT?

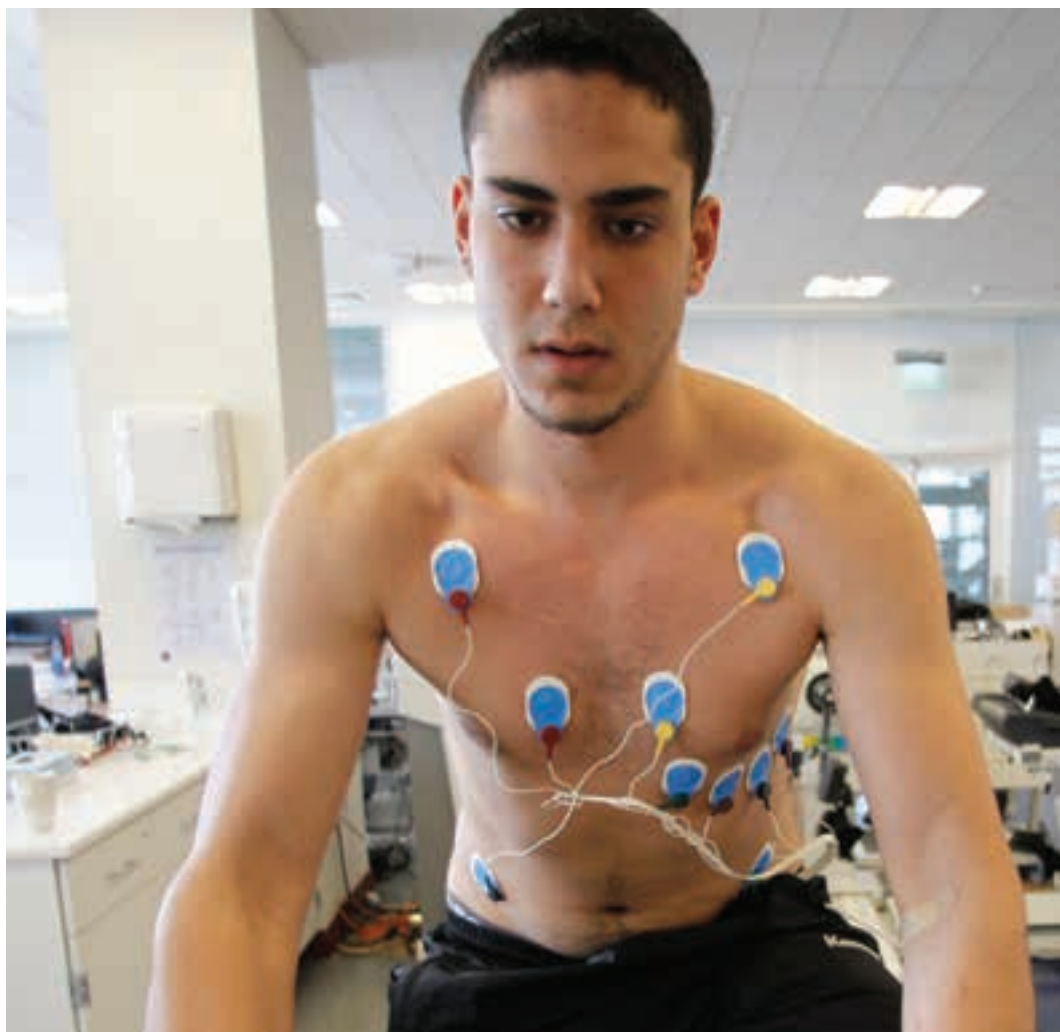
– *Written by Francois Carré, France*

The body's homeostasis is dramatically altered during physical exercise because of neuro-hormonal and haemodynamic constraints. Thus, the cardiovascular system must adapt itself to these acute disturbances. One goal of regular exercise training is to promote the development of effective cardiovascular adaptations to best meet these constraints. Although modest, the beneficial effects are often observed as soon as physical activity is performed. This may occur with only 3 weekly sessions of 30 to 45 minutes duration and between 60 to 80% of maximal oxygen consumption ( $VO_2$  max), with improvements in diastolic filling and systolic emptying with resting bradycardia.

In the case of high level training ( $\geq 6$  to 8 hours per week with intensity greater than 60% of  $VO_2$  max for longer than 6 months) and only in this case, marked functional and morphological cardiovascular adaptations can be observed. These adaptations are not considered pathological.

Instead, they play a major role in the physical performance of these athletes. These clinical, electrocardiographic and morphological cardiovascular adaptations are combined under the so-called 'athletic heart syndrome'. The signs of athlete's heart are important for sports physicians and cardiologists to know. While in the vast majority of cases these adaptations are moderate and asymptomatic, in 3 to 5% of cases they can cause diagnostic problems with certain inherited cardiac pathologies associated with exercise-related sudden cardiac death. The etiological diagnosis is, however, essential. Indeed, giving medical clearance for sports participation to a patient with heart disease exposes that athlete to an elevated risk of an adverse cardiac event. On the other hand, it is not acceptable to unreasonably prohibit intense sports competition to someone whose livelihood depends on their sport. This brief review will describe the main patterns of the athlete's heart.





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#### ATHLETE'S HEART DETERMINANTS

The athlete's heart pathophysiology is multifactorial<sup>1</sup>. As with skeletal muscle, exercise modulates expression of the genome part which encodes for structural and metabolic proteins of the cardiovascular system. The acute haemodynamical, mechanical and neuro-hormonal disturbances due to the exercise have a synergistic action on the genome. These determinants not only alter myocardial vessels but the autonomic nervous system as well. The characteristics of these determinants vary with the exercise performed (isotonic, static or mixed). Thus, it is easy to understand why different exercise patterns can influence the athlete's heart characteristics differently (see below).

#### ATHLETE'S HEART FEATURES VARIABILITY

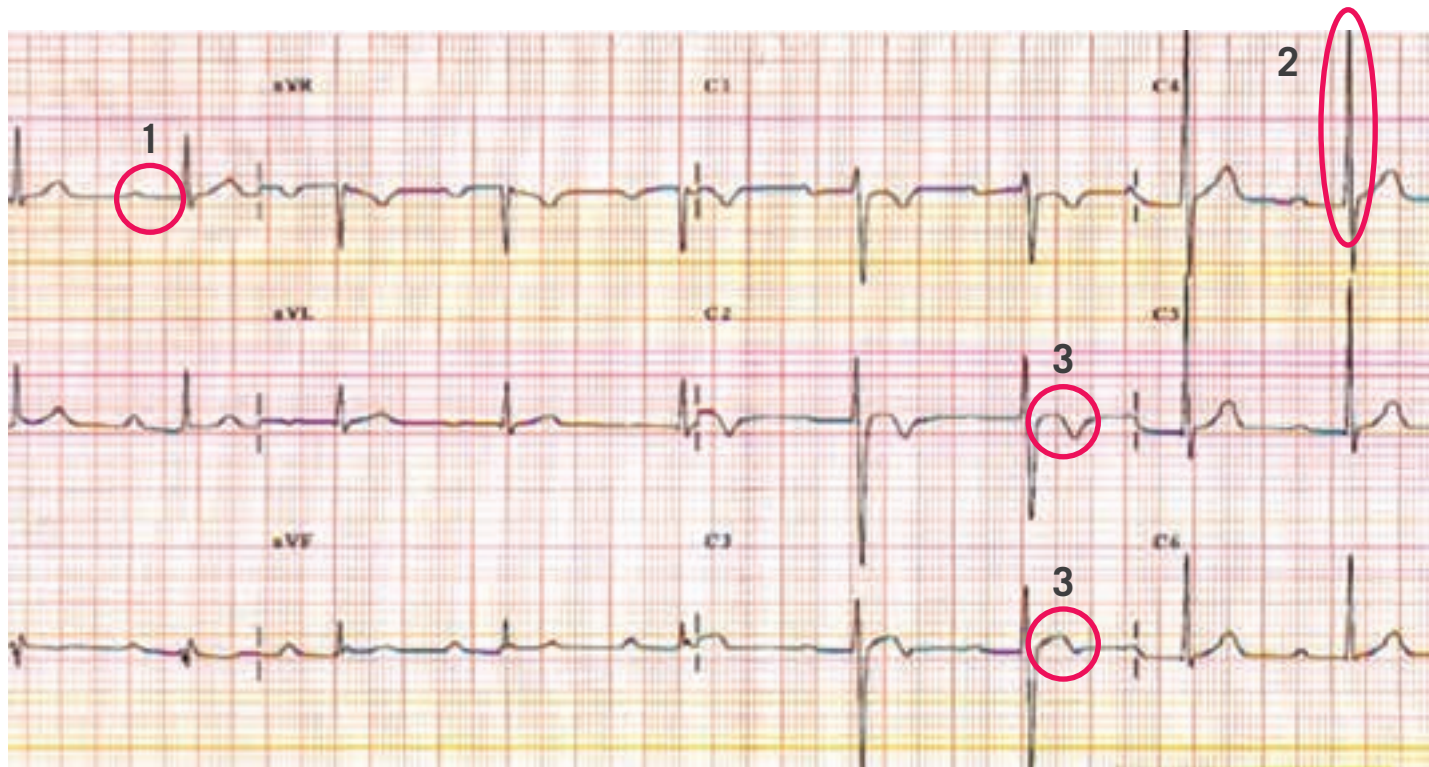
The athlete's heart patterns present with great individual variability<sup>1</sup>. It largely depends on the individual genetic background but also on the type of sport,

gender and age. Indeed, for the same level of training, the type and extent of observed adaptations can vary widely. This paper describes the most typical characteristics of athletes' hearts as seen in endurance athletes. They are always more marked in adult male athletes than in females, with some other specific aspects described in relation to the athlete's ethnicity. While not part of this review, it should be noted that the use of some banned performance enhancing and recreational drugs can have some detrimental effects.

#### CLINICAL ASPECT OF ATHLETE'S HEART

First, it must be emphasised that the healthy athlete must be asymptomatic; confirmed by family and personal histories and by physical examination. Personal history must also confirm a good correlation between the characteristics of training and the athlete's level of performance.

Physical exam can show some peculiarities<sup>1</sup>. One involves a thin subject



**Figure 1:** Resting ECG of a black professional soccer player with common training-related changes.  
**1:** First degree atrio-ventricular block. **2:** Isolated left ventricle hypertrophy criteria.  
**3:** Early repolarization pattern with diphasic T waves.

with a slow heart rate and a marked apex beat and arterial pulsatility. A non-pathological murmur can be heard from the cardiac area. It presents a low intensity which varies with chest position and with inspiration/expiration. The venous system, mainly legs in cyclists or dominant arm in asymmetrical sports such as tennis, can be marked. Blood pressure is rather low with a tendency to orthostatic hypotension.

#### ELECTROCARDIOGRAM OF THE ATHLETE

The resting 12-lead electrocardiogram (ECG) has no role in checking the training impact or guiding it. This exam is a part of the cardiovascular evaluation recommended for competitive sport pre-participation eligibility<sup>2</sup>. In the case of cardiovascular symptoms, resting ECG must be recorded in the first line investigation. Both ECG recording and analysis in the athlete must be classical. Its interpretation should always be performed in accordance with history and physical exam. It should especially consider the type of training, quality and quantity, and the period of the sports season. The widespread belief that all athletes have specific ECG features, which can pose diagnostic difficulties with

cardiac diseases, is false. It must be kept in mind that ECG alterations linked to high levels of training are mainly modest. Thus, resting ECG is normal in 50 to 60% of male and in 75 to 80% of female athletes aged 12 to 40 years. Minor peculiarities which do not evoke cardiovascular symptoms are observed in 25 to 40% of cases and mainly in endurance athletes. Lastly, very marked peculiarities are observed in less than 5% of ECGs. These patterns are described in Table 1. ECG alterations are explained by

intrinsic cellular and autonomic nervous system adaptations (both marked decrease of sympathetic and slight increase of parasympathetic drives).

Overall, compared to the sedentary individual, the ECG of an athlete reveals a marked respiratory arrhythmia with a sinus bradycardia, mainly between 45 to 55 beats per minute. QRS complex presents a slightly increased duration with marked amplitude and a left-deviation of its frontal axis. Tall T waves are frequent. They must be positive,

TRAINING-RELATED ECG CHANGES	TRAINING-UNRELATED ECG CHANGES	
<i>Bradycardia (&lt;50 bpm)</i>	<i>Arrhythmias</i>	<i>T waves inversion</i>
<i>Isolated QRS voltage criteria for left ventricular hypertrophy</i>	<i>Complete left or right bundle branch block</i>	<i>Brugada like early repolarization</i>
<i>Incomplete right bundle-branch block</i>	<i>Marked QRS axis deviation</i>	<i>Long or short QT interval</i>
<i>First degree atrio-ventricular block</i>	<i>Right ventricular hypertrophy</i>	<i>Ventricular pre-excitation</i>
<i>Early repolarization</i>	<i>Left atrial enlargement</i>	<i>ST segment depression</i>
		<i>Marked Q waves</i>

**Table 1:** Athlete's ECG training effects or not? Adapted from Corrado D et al<sup>2</sup>. ECG = electrocardiogram, bpm = beats per minute.





**Above:** Detail shot of echocardiogram, which shows how effectively the heart is pumping.

except in aVR where it is always negative, and this is also often the case in Lead III and V1. In the master's athlete, supraventricular arrhythmias are more common. Ethnicity also plays a role and some features such as biphasic (20 to 25%) or negative (4 to 6%) T waves are more common in black male and female athletes<sup>3</sup> (Figure 1).

#### ECHOCARDIOGRAM OF THE ATHLETE

Echocardiography plays a central role in the cardiovascular examination of the heart of an athlete. This exam has no value as a guide for training. However, it is highly valued as a diagnostic procedure. The functional myocardial adaptations that occur rapidly after 1 to 3 weeks of training are mainly due to lower heart rate and to hypervolaemia induced by hormonal adaptations. They concern firstly the ventricular filling (diastolic function).

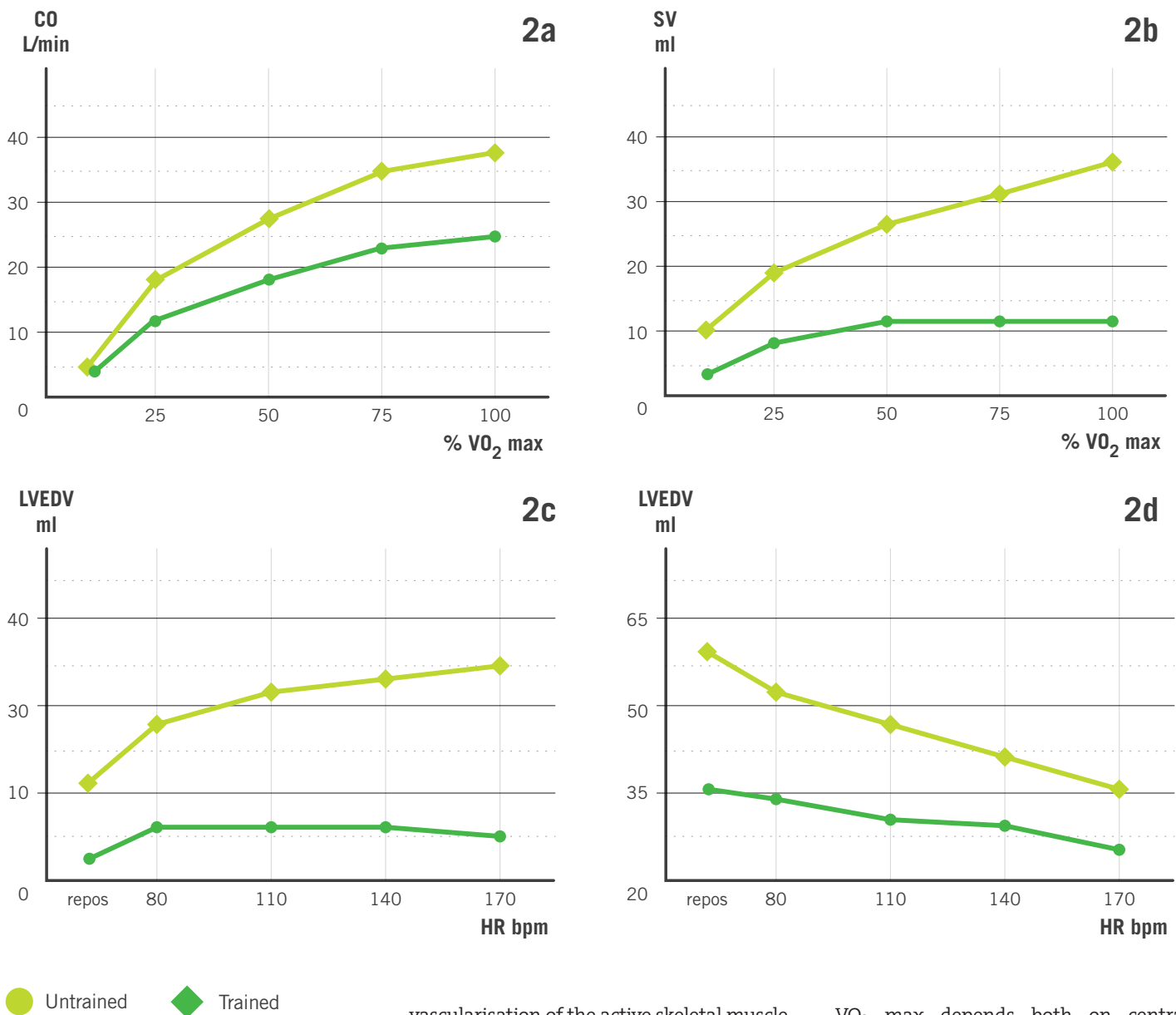
Morphological adaptations are delayed, but are significant after 2 to 6 months of training<sup>4</sup>. They are characterised by a cardiac hypertrophy with a predominant dilation which concerns both ventricles and atria, and left as well as right heart. Wall thicknesses, mainly of left ventricle, are slightly increased in order to decrease the

parietal tension due to the cavity dilation. This chamber enlargement is associated with normal or supra-normal ventricular functions of filling and emptying. These morphological modifications are always slight in comparison with cardiac diseases that involve more marked individual variations. The classical upper limits proposed concern the left ventricle with an end-diastolic diameter less than 60 and 56 mm and a parietal wall less than 13 and 11 mm in male and female athletes, respectively. Here again, some ethnic specific patterns are reported in (male and female) black athletes with a less marked left ventricle dilatation and a more marked wall hypertrophy.

#### VASCULAR ADAPTATIONS IN ATHLETES

Heart and blood vessels are closely linked, especially on the functional level. Athlete's heart is also characterised by vascular adaptations, even if they are less-pronounced than myocardial changes<sup>5</sup>. Functional and morphological vascular adaptations are observed in athletes. The ability for endothelial dilation is increased. On the morphological level, angiogenesis, as well as arteriogenesis, is increased. Thus,

**In the master's athlete, supraventricular arrhythmias are more common.**



**Figure 2:** Comparison of the exercise's adaptations of the CO (2A) and of the left ventricle SV (2B) and of the SV components LVEDV (2C) and LVESV (2D). CO = cardiac output, SV = stroke volume, LVEDV = left ventricle end diastolic volume, LVESV = left ventricle end systolic volume.

vascularisation of the active skeletal muscle is increased. These adaptations, besides the fact that they optimise ventriculo-arterial coupling, will participate in the performance improvement reported in the athlete.

#### ATHLETE'S HEART INVOLVEMENT IN AEROBIC PHYSICAL PERFORMANCE

Oxygen uptake ( $VO_2$ ) increases with exercise intensity up to an individual level, called maximal aerobic power, beyond which it levels off ( $VO_2$  max). Cardiovascular adaptations play a major role in the oxygen uptake in accordance with the Fick's equation  $VO_2 = CO \times a-VO_2$  ( $CO$  = cardiac output and  $a-VO_2$  = arteriovenous difference in oxygen). Thus, the cardiovascular system ensures the delivery of oxygen to the active skeletal muscles by increasing blood flow and facilitating its transport by vasodilatation and redistribution<sup>6</sup>.

$VO_2$  max depends both on central (cardiac output) and peripheral (vessels and muscles) factors. With intense physical training, especially aerobic,  $VO_2$  max increases on average by 20 to 30%. This improvement is related to both cardiovascular and muscle adaptations.

Cardiac output is the product of heart rate and stroke volume. The maximal cardiac output is markedly increased by intense training. Its value, which is between 20 to 25 l/minute in sedentary subjects, can reach 35 to 40 l/minute in an athlete (Figure 2).

In athletes, resting heart rate is decreased but maximal heart rate is not significantly modified. It can be slightly decreased but it is never increased. The slope of heart rate increasing during exercise is decreased in athletes in comparison with untrained people because of autonomic nervous system adaptations<sup>7</sup>. Thus we can say that



during sub-maximal exercise, the athlete preferentially uses his/her stroke volume and saves their heart rate for the highest levels of exercise.

Stroke volume plays a major role in the improvement of maximal cardiac output (Figure 2). Physical training alters both myocardial intrinsic and extrinsic factors involved in stroke volume.

The calculated left ventricular mass of the athlete is positively and significantly correlated to  $VO_2$  max. However, myocardial morphological changes are not sufficient to explain the stroke volume adaptations. The functional adaptations also play a large role. During exercise there is a great increase of venous return because of the effects of the 'muscle pump', which by venous compression promotes blood flow to the right heart. Through the improvement of diastolic function due to physical training, the athlete's heart fills smoothly, even at high heart rates. Indeed, the hypertrophy-dilatation of the athlete's heart, unlike hypertrophy secondary to pathological causes, presents a myocardium which is histologically balanced, without fibrosis and with a suitable vascularisation. Again, its ability to relax is also improved. The systolic function is also increased because of a higher sensibility to catecholamine

effects. Thus, the athlete's heart fills and empties better during exercise than when sedentary<sup>1</sup>.

There is also a linear positive relationship between  $VO_2$  max and the maximal vascular conductance. Because of the increased capacity of vasodilatation, the after load is decreased in athletes during exercise. This collapse of peripheral resistance accounts for the major decline in exercise diastolic blood pressure in athletes.

Arteriovenous difference in oxygen adaptation during exercise is increased in athletes. This is due to the decrease in venous  $O_2$  concentration caused by both an increase in mitochondrial oxidative capacity and interface between mitochondria and muscle capillaries. Haemoglobin concentration and arterial  $O_2$  are not increased with physical training<sup>5</sup>.

#### CONCLUSIONS

Regular physical activity induces myocardial and vascular adaptations. These adaptations are essentially functional in the case of moderate training. In intense training, they can also be morphological. In all cases they are beneficial and the 'athlete's heart' adaptations contribute largely to the high level of performance achieved by these athletes.

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