Cardiovascular adaptations to physical exercise

Carlo Manca, Giovanna Pel *, Roberto Bolognesi, Nicola Gaibazzi, Alberico Borghetti*

*Department of Internal Medicine and Biomedical Sciences, *Department of Clinical Medicine, Nephrology and Prevention Sciences, University of Parma, Parma, Italy*

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Address:

Prof. Carlo Manca

Dipartimento di Medicina Interna e Scienze Biomediche Università degli Studi Via Gramsci, 14 43100 Parma E-mail: climed@ ipruniv.cce.unipr.it

Physiological responses to physical exercise lead to specific cardiovascular adaptations depending on the intensity and schedule of training and on other factors such as age and genetic factors. Two forms of conditioning can be distinguished: isotonic exercise also referred to as endurance or aerobic exercise, and isometric exercise or static or anaerobic training[1]. Athletes who participate in isotonic endurance sports, for example runners and swimmers, are primarily exposed to conditions producing a volume load[2]. Athletes who participate in sports involving isometric exertion, such as weightlifters or body-builders, are primarily exposed to a pressure load[3]. However athletic conditioning is seldom purely isotonic or isometric; most physical activities have both static and dynamic components, although one of them may be dominant.

Lower resting heart rates and reduced heart rates at any submaximal exercise are the manifestations of neurohumoral adaptations; parasympathetic tone tends to prevail over sympathetic tone in the trained subject as it is clearly demonstrated by the increased heart rate variability measured in trained versus sedentary subjects.

Many recent papers have focused on the role of regular training in improving endothelial function; this has been demonstrated with a high-resolution brachial artery ultrasound assessment of flow-mediated dilation on older endurance-trained men[4].

The athlete s heart

The athlete s heart is a term that has been used for many years to describe the effects of long-term conditioning observed in trained competitive athletes.

Athletes involved in sports with a high dynamic component develop predominantly increased left ventricular chamber size with a proportional increase in wall thickness caused by volume overload associated with the high cardiac output of the endurance training[1,2,5,6]. The increase in septal and wall thickness neutralizes the increase in wall stress according to the Laplace law and has been described as eccentric hypertrophy with an unchanged ratio of wall thickness to radius. Athletes involved in mainly static or isometric exercise develop predominantly increased left ventricular wall thickness with unchanged left ventricular chamber size; this is caused by pressure overload which accompanies the high systemic arterial pressure found in this type of exercise. Left ventricular hypertrophy, in power athletes, has been described as concentric hypertrophy which is characterized by an increased ratio of wall thickness to radius[1,3]. The shape of this chamber is more elliptical, whilst in endurance athletes it is more spherical. In endurance athletes, the left ventricular enlargement is associated with an increase in the dimensions of both atria and right ventricular cavity; these changes, due to the higher cardiac filling dependent on an increase in venous return and slower heart rate, do not occur in power athletes[7]. The left ventricular systolic function, assessed as ejection fraction, fractional shortening or velocity of circumferential fiber shortening, is normal[1]. The left ventricular diastolic function, evaluated by the pattern of transmitral flow, is normal or slightly enhanced in both groups of athletes[1]. These findings came from echocardiography which is the most common imaging technique in the study of morphological

Table I. Left ventricular adaptations in elite cyclists.

	Athletes	Normals
Diastolic ventricular septum thickness (mm)	11.1 – 1.4	10.5 – 1.3
Diastolic posterior wall thickness (mm)	10.1 – 1.1	9.8 – 1.3
Diastolic short dimension (cm)	5.3 – 0.3	4.9 – 0.4**
Systolic short dimension (cm)	3.2 – 0.2	2.9 – 0.3*
Diastolic long dimension (cm)	9.1 – 0.7	8.2 – 0.6**
Systolic long dimension (cm)	6.8 – 0.6	6.2 – 0.5**
Diastolic volume (ml)	137 – 19	84 – 23***
Systolic volume (ml)	42 – 11	29 – 11**
Ejection fraction (%)	69 – 7	66 – 6
Short-axis fractional shortening (%)	40.1 – 4.3	39.5 – 6.3
Long-axis fractional shortening (%)	24.9 – 7.3	24.0 – 6.2

Data are expressed as mean – SD. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ normals vs athletes.

and functional changes induced by training in different types of sport and it can be used both at rest and during exercise[2,3,5-7]. By using this technique we observed an increase of left ventricular septal and posterior wall thickness in trained cyclists; this group had higher diastolic short and long dimensions, increased end-diastolic and end-systolic volumes and normal short-axis, long-axis fractional shortening and ejection fraction[5] (Table I). The E/A ratio from mitral flow also increased suggesting an improved diastolic function[5].

A meta-analysis regarding all published echocardiographic data on the athlete s heart has recently confirmed the hypothesis of divergent cardiac adaptations in dynamic and static sports[8]. Conventional echocardiography may also help to distinguish physiological hypertrophy from pathological hypertrophy as found in hypertrophic or hypertensive cardiomyopathy. Highly trained athletes usually show a mild increase in septum and posterior wall thickness that does not exceed 13 mm and an increase in transversal left ventricular diameter which remains within the upper normal limits (5.5 cm)[9]. Conversely, in patients with hypertrophic cardiomyopathy the increase in septum thickness is more evident (15 mm), the left ventricular cavity is normal or reduced, the pattern of left ventricular filling is abnormal and evidence of dynamic subaortic obstruction can be observed[9].

Myocardial tissue characterization, based on videodensitometric analysis or on integrated backscatter, has also been used to differentiate physiological from pathological hypertrophy, like hypertrophic cardiomyopathy or hypertensive heart disease[10].

Doppler tissue echocardiography, which is able to analyze regional systolic and diastolic wall motion[11], has been applied in the study of cardiac adaptations to exercise[5,6,12]. Using this technique an increase in left ventricular systolic function and an improved relaxation in trained athletes have been shown; this technique has also proved helpful to differentiate cardiac responses to different training programs[6] and is useful to distinguish physiological from pathological hypertrophy[5].

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