Biology of Sport, Vol. 22 N°2, 2005

A FOLLOW-UP STUDY OF THE CHANGES IN LEFT VENTRICLE AND RUNNING PERFORMANCE IN HIGHLY TRAINED RUNNERS

A.Legaz[1], M.González[2], I.Lacambra[2]
[1]Section of Physical Education and Sports, University of Zaragoza, Zaragoza, Spain; [2]Division of Cardiology, Echocardiography Laboratory, Clinical University Hospital, Zaragoza, Spain

Abstract. Long-term studies have not previously been reported concerning the association among left ventricle (LV) adaptation and running performance. This association can be important for differentiation between pathological and physiological cardiac adaptation. The echocardiograms of 11 male and 9 female elite endurance-trained runners were evaluated over at least four times. The best running performance and echocardiography measurements achieved by each athlete in each year were selected in order to observe the individual association among LV and performance. Extreme LV enlargement (i.e., >60 mm) was observed in a 64% of the male runners. In a 75% of athletes, the higher value of the left ventricular internal diameters at end-diastole (LVIDd) was observed during the season whereon they obtained his/her better running performance. The mean value of range went from 5.8 and 4.4 mm for the LVIDd, and of 2.2 and 1.7 for the wall thickness; respectively for male and female runners. Individual analysis showed that LVIDd was associated with running performance in a 50% of runners ($p<0.05$). Wall thickness was negatively associated with performance in a 30% of runners, this relation was linked to LVIDd enlargement. These results represent an important criteria in considering extreme LV enlargement in elite runners to be a physiological adaptation and it leads on to thinking that the echocardiographic would be able to be utilized to determine the fitness of the athlete. However, this study's design and different difficult variables to control do that these conclusions should be taken like the start of a new field of study.

(Biol.Sport 22:135-149, 2005)

Key words: Exercise - Echocardiography - Running performance - Elite runners - Cardiac enlargement

Reprint request to: Dr. Alejandro Legaz Arrese, Dept. de Fisiatría y Enfermería, Universidad de Zaragoza. C/ Domingo Miral S/N, 50009 Zaragoza, España
E-mail: alegaz@posta.unizar.es

Introduction

A considerable body of echocardiographic studies have shown how athletic training induces morphological adaptation of the left ventricle (LV) [1,19,24,26]. Nevertheless, previous studies have involved a cross-sectional analysis and such an approach cannot provide conclusive evidence with respect to LV adaptations. Direct evidence of changes in LV after training programs has only been assessed in short longitudinal studies involving non-athletes or heterogeneous groups of performers [2,5,7,10,11].

The differentiation between physiologic and pathologic LV can be difficult, but it is important in determining the existence of cardiac disease in athletes in order to prevent exercise-related sudden cardiac death. The relationship between heart dimensions and metabolic exercise testing or performance represents an important criterion for differentiating between physiological and pathological cardiac enlargement [22,25]. Only a long-term study on LV morphology is available with elite athletes [1], they described the serial cardiac changes in a group of 37 professional cyclists but could not make the association between these morphologic and functional changes and performance.

In an effort to address these important questions, we set out to establish the serial changes in left ventricular structure and performance caused by intense athletic conditioning by a follow-up study for each athlete of group of elite male and female endurance-trained runners.

Material and Methods

Subject selection: A total of 11 male and 9 female elite runners engaged in intense athletic conditioning underwent, at least 4 times, a screening echocardiogram at the National Centre of Sports Medicine in Spain. The measurements were recorded using a Toshiba SSH-140A Sonolayer ultrasound system (Toshiba Medical System S.A., Spain) equipped with a 2.5–3.75 MHz probe. All examinations were performed each year during the competitive season within the five months of the best performance.

The criteria applied to determine the best performances of those athletes involved in several events was established by means of the corresponding performance equivalence according to the Score established by the International Amateur Athletic Federation (IAAF) [23]. The IAAF, using a database of performances obtained at world level, assigns a definite score to each performance,

enabling them to compare the different performance in different events for the same athlete.

All runners have been training for more than ten years and achieved national or international levels of competition. The mean velocity achieved during their best season performance had to be included among the best fifty ever in the Spanish ranking. The performances were obtained after consulting the official rankings published by the Statistics Department of the Real Federación Española de Atletismo.

Measurements: Conventional two-dimensional-guided M-mode traces of the LV were recorded at a paper speed of 50 mm/s via the parasternal approach after obtaining long-axis cross-sectional echocardiograms. All recordings were performed in the manner recommended by Devereux *et al.* [8] and by the American Society of Echocardiography [20]. These recordings enabled left ventricular internal diameters at end-diastole (LVIDd), end-diastolic interventricular septal wall thickness (IVSTd), and posterior wall thickness (PWTd) to be measured. The Devereux-modified American Society of Echocardiography cube formula was used to calculate LV mass [17]. Relative wall thickness (RWT) was calculated with a distinction between eccentric (RWT <0.44) and concentric (RWT ≥0.44) patterns [1].

DCM was defined in clinical terms as LVIDd >60 mm [15] and HCM as IVSTd and/or PWTd >13 mm for male and >11 mm for female subjects [26].

Statistical analysis: Pearson analysis was performed to examine in each athlete the association between left ventricle and running performance. A p value <0.05 was considered indicative of statistical significance.

Results

Tables 1 and 2 show, respectively for athletes engaged in middle- and long-distance events, the descriptive analysis out of every one of the athletes. This analysis is showed in Tables 3 and 4 for the female sample.

Table 1. Serial left ventricle and performance changes in elite male middle-distance runners engaged in systematic exercise conditioning.

Table 2. Serial left ventricle and performance changes in elite male long-distance runners engaged in systematic exercise conditioning.

Table 3. Serial left ventricle and performance changes in elite female middle-distance runners engaged in systematic exercise conditioning.

Table 4. Serial left ventricle and performance changes in elite female long-distance runners engaged in systematic exercise conditioning.

Table 1
Serial left ventricle and performance changes in elite male middle-distance runners engaged in systematic exercise conditioning

		1er year	2º year	3er year	4º year	5º year	6º year	7º year	8º year	9º year
"Runner 1" 800m	IAAF Score	1034	1092	1125	1139	1127	1176	1124	1091	1121
	LVIDd, mm	49.0	52.0	58.0	56.3	54.1	57.1	56.3	55.5	56.6
	PWT, mm	11	11	10	10	9	8	10	10	9
	LVM, g	204	225	239	213	186	190	227	222	201
"Runner 2" 800m	IAAF Score	1029	1054	1084			1095	1109	1116	1084
	LVIDd, mm	51.0	52.0			45.8	46.9	45.1	51.6	48.7
	PWT, mm	8	8			12	11	8	8	8
	LVM, g	156	150			206	202	126	159	144
"Runner 3" 800m	IAAF Score	894		950	1071	1145	1172			
	LVIDd, mm	53.9		57.0	58.8	60.9	60.6			
	PWT, mm	10		9	9	8	8			
	LVM, g	198		203	215	198	182			
"Runner 4" 800m	IAAF Score	946	1062	1096	1111	1085				
	LVIDd, mm	50.1	54.5	51.6	51.6	51.6				
	PWT, mm	8	8	9	9	8				
	LVM, g	140	163	171	159	148				

"Runner 5" 1500 m	IAAF Score	998	1048	1076	1108	1086	1118	1136	1145	1120
	LVIDd, mm	62.0	60.0	57.6	62.4	60.3	60.6	62.1	64.9	61.3
	PWT, mm	10	9	10	8	9	9	9	8	8
	LVM, g	269	223	237	207	225	227	237	222	201
"Runner 6" 1500 m	IAAF Score	978	1019	1029	1048	1055	1103	1088	1119	1089
	LVIDd, mm	55.0	55.0	54.5	55.9	54.8	55.9	57.0	55.5	57.7
	PWT, mm	9	9	11	11	10	9	9	9	8
	LVM, g	191	191	229	254	231	210	203	194	180

IAAF Score = the score established for race time by the International Amateur Athletic Federation; LVIDd = left ventricular internal diameter at end-diastole; LVM = left ventricular mass; PWT = end-diastolic posterior wall thickness

Table 2
Serial left ventricle and performance changes in elite male long-distance runners engaged in systematic exercise conditioning

		1er year	2º year	3er year	4º year	5º year	6º year	7º year	8º year	9º year	10º year
"Runner 7" 3000 m	IAAF Score	1145	1135	1137	1135	1093	1125	1165	1114	1234	
	LVIDd, mm	58.1	59.0				58.1		57.3	60.6	
	PWT, mm	10	9				10		8	9	
	LVM, g	240	231				240		178	227	
"Runner 8" 5000 m	IAAF Score	1100		1107	1111	1134	1108	1162	1139	1180	1197
	LVIDd, mm	56.1			58.4		57.5		60.0	59.5	62.7

	PWT, mm	9			9		9		9	8	7
	LVM, g	184			198		207		208	190	179
"Runner 9" Marathon	IAAF Score	987		1163	1139	1168	1156	1208	1176	1138	
	LVIDd, mm	56.0			57.1		59.3	60.4		57.7	
	PWT, mm	8			9		9	8		9	
	LVM, g	171			190		218	195		194	
"Runner 10" Marathon	IAAF Score	1095	1130	1201	1196	1180	1158				
	LVIDd, mm	53.9	53.4	61.9	59.5	58.1	57.9				
	PWT, mm	10	9	8	8	9	10				
	LVM, g	240	195	250	232	210	219				
"Runner 11" Marathon	IAAF Score	1173	1177	1167	1191	1166					
	LVIDd, mm	56.6		56.5	60.0	56.6					
	PWT, mm	10		9	8	9					
	LVM, g	215		214	207	201					

IAAF Score = the score established for race time by the International Amateur Athletic Federation; LVIDd = left ventricular internal diameter at end-diastole; LVM = left ventricular mass; PWT = end-diastolic posterior wall thickness

In male runners, LVIDd was found to be above the upper clinical limit of 55 mm [12] in 71% of the measurements, with more substantial dilatation (i.e., >60 mm) in 23%, a 64% of the male runners. In female runners, only a runner exceeded 55 mm. The maximal wall thickness values did not exceed 11 mm in males and 9 mm in females. The LV geometry was eccentric in all runners.

Table 3
Serial left ventricle and performance changes in elite female middle-distance runners engaged in systematic exercise conditioning

		1er year	2º year	3er year	4º year	5º year	6º year	7º year	8º year	9º year
"Runner 12" 800m	IAAF Score	893	960	1013	982					
	LVIDd, mm	49.0	51.0	53.0	52.6					
	PWT, mm	9	8	8	8					
	LVM, g	157	145	143	141					
"Runner 13" 800m	IAAF Score	1003	1040	1050	1082	1084				
	LVIDd, mm	49.8	50.8	56.3	55.5	56.3				
	PWT, mm	7	9	8	8	7				
	LVM, g	118	167	172	155	147				
"Runner 14" 1500m	IAAF Score	1107	1035		1206	1151	1142	1153	1132	1176
	LVIDd, mm	52.0	55.0		52.7	52.6	52.5	51.9	50.1	53.4
	PWT, mm	8	8		7	7	7	7	7	6
	LVM, g	150	165		130	130	129	127	119	111
"Runner 15" 1500m	IAAF Score	1015	1061	1028	1026					
	LVIDd, mm	45.0	49.6	45.0	46.2					
	PWT, mm	8	6	8	8					
	LVM, g	126	107	126	122					

IAAF Score = the score established for race time by the International Amateur Athletic Federation; LVIDd = left ventricular internal diameter at end-diastole; LVM = left ventricular mass; PWT = end-diastolic posterior wall thickness

Table 4
Serial left ventricle and performance changes in elite female long-distance runners engaged in systematic exercise conditioning

		1er year	2º year	3er year	4º year	5º year	6º year	7º year
"Runner 16" Marathon	IAAF Score	1043		1088	1114	1065	1095	1073
	LVIDd, mm	48.5	49.8	48.3		51.2	51.0	49.8
	PWT, mm	8	8	8		7	8	8
	LVM, g	133	139	132		135	145	139
"Runner 17" Marathon	IAAF Score	1138		1122	1193	1157		
	LVIDd, mm	48.9		49.0	52.8	49.8		
	PWT, mm	8		8	7	9		
	LVM, g	145		146	142	161		
"Runner 18" 5000 m	IAAF Score	1143	1162	1152	1181	1092		
	LVIDd, mm	45.2		48.5	51.6	49.0		
	PWT, mm	8		6	8	9		
	LVM, g	117		94	148	146		
"Runner 19" Marathon	IAAF Score	1147	1095	1108	1169	1050		
	LVIDd, mm	51.6		52.1	54.8	52.8		

	PWT, mm	8		8	8	9
	LVM, g	148		150	164	166
"Runner 20" Marathon	IAAF Score	1141	1169	1176	1149	1134
	LVIDd, mm	49.8		53.1	53.5	49.4
	PWT, mm	9		8	9	9
	LVM, g	161		168	182	159

IAAF Score = the score established for race time by the International Amateur Athletic Federation; LVIDd = left ventricular internal diameter at end-diastole; LVM = left ventricular mass; PWT = end-diastolic posterior wall thickness

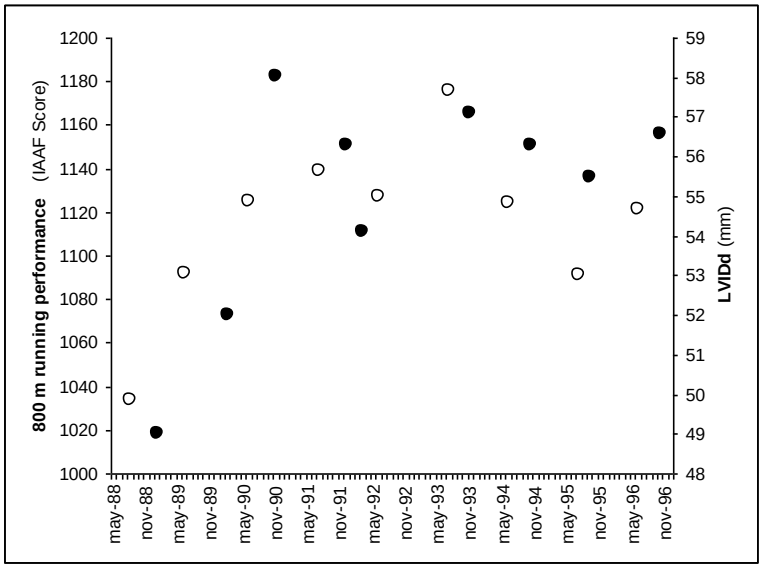

Fig. 1
Plot of the changes in left ventricular internal diameter at end-diastole (LVIDd) against changes in 800 meters running performance caused by intense athletic conditioning in the "Runner 1" (open circles = Running performance, IAAF score; solid circles = LVIDd, mm)

In 9 of the 11 male (Table 1 and 2) and in 6 of the 9 female runners (Table 3 and 4), the higher value of the LVIDd was observed during the season whereon they obtained his/her better running performance.

The mean value of range went from 5.8 and 4.4 mm for the LVIDd, and of 2.2 and 1.7 for the PWT; respectively for male and female runners.

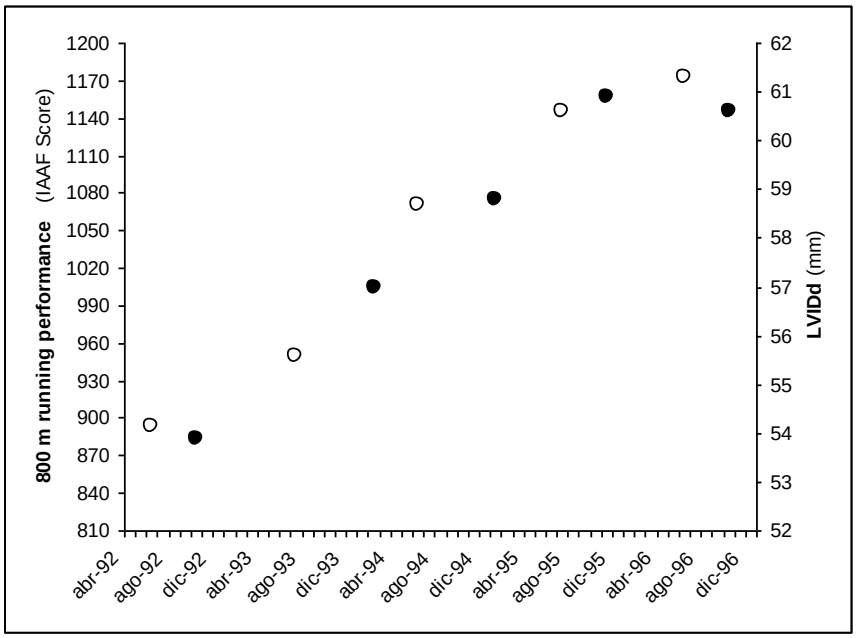

Fig. 2
Plot of the changes in left ventricular internal diameter at end-diastole (LVIDd) against changes in 800 meters running performance caused by intense athletic conditioning in the "Runner 3" (open circles = Running performance, IAAF score; solid circles = LVIDd, mm)

Individual analysis showed that LVIDd was associated with running performance in 7 of the 11 male runners: "Runner 1" (Fig. 1), r=0.83, p=0.006; "Runner 3" (Fig. 2), r=0.97, p=0.007; "Runner 7" (Fig. 3), r=0.92, p=0.026; "Runner 8", r=0.89, p=0.019; "Runner 9", r=0.87, p=0.047; "Runner 10", r=0.92, p=0.01 and "Runner 11", r=0.96, p= 0.036; and in 3 female runners: "Runner 12", r=0.98, p=0.02; "Runner 15", r=0.95, p=0.047 and "Runner 17", r=0.95, p=0.049.

PWT was negatively associated with IAAF Score in 6 athletes, "Runners 1, 3, 8, 12, 14, 15". LVM was not significantly associated to running performance.

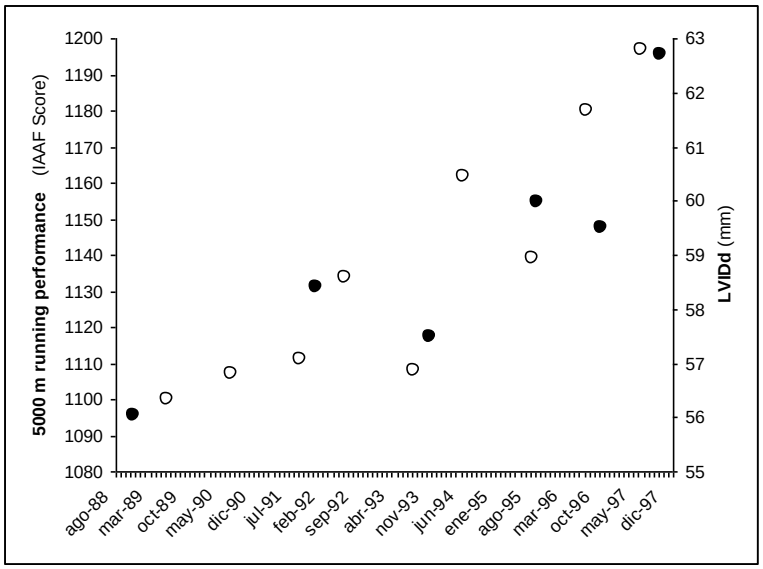

Fig. 3
Plot of the changes in left ventricular internal diameter at end-diastole (LVIDd) against changes in 5000 meters running performance caused by intense athletic conditioning in the "Runner 7" (open circles = Running performance, IAAF score; solid circles = LVIDd, mm)

Discussion

This study examined the implications of the changes in LV and performance caused by athletic conditioning for disease screening in high-levels runners and provides unique information from two perspectives. First, only one follow-up study [1] described the serial cardiac changes in elite athletes. Second, the relationship between the changes in LV morphology and function and competition performance is not available. These analyses represent important criteria for differentiating between physiological and pathological cardiac enlargement.

Individual analysis evidenced important changes in LVIDd during intense endurance athletic conditioning, mean value of range was 5.8 and 4.4 mm for male and female, respectively. Increase in LVIDd was accomplished with wall thinning

over time. The LV geometry was therefore eccentric (<0.44) in all runners. In the only long-term study available on LV morphology with elite athletes, Abergel *et al.* [1] showed the same pattern of LV adaptation in professional cyclists.

In both studies, the unexpected decrease in LV wall thickness with the increase in LVIDd is inexplicable and we can therefore only speculate on its significance. It's possible that the mechanism be similar to the effect produced by blowing up a balloon [18].

LV enlargement exceeding 60 mm, in the dimensional range of DCM, was observed in 67% of male athletes, all runners engaged in distance higher than 1500 m showed LVIDd >60 mm. Exist different theories to be able to explain the extreme enlargement and the great variation observed in the LVIDd of these athletes. Compared with the subjects of previous reports [1,15,17], the BSA of our athletes was significantly smaller, meaning that a larger cardiac load per unit body size may be related to other factors; besides all runners had over 20 years at the beginning of study, consequently, the variations in BSA were insignificant. It seems unlikely that these changes were due to the reproducibility of measure [9] and/or to the changes in the basal heart rate or the plasma volume expansion [6]. The association shown among the LVIDd and running performance can represent an important criterion in considering LV enlargement in our athletes to be a physiological adaptation to systematic exercise conditioning. Previously, the relationship between cardiac morphology and human performance, only was presented for the VO_{2max} [22] and habitually in very heterogeneous groups. VO_{2max} was found not to be a good predictor of performance in homogeneous groups of runners (i.e Billat *et al.*) [4] and the plateauing of maximal oxygen uptake in elite endurance athletes has been documented [13,21].

However, another factors would be able to have influence in the changes of the LVIDd as performance enhancing drugs and the significance of genetics in athletes heart.

The most significant finding in the current study was the association between LVIDd and running performance. These results highlight the relation found among the LVIDd and running performance in a recent cross-sectional study presented by our fact-finding group [14].

The causes of this relationship between LVIDd and running performance are not clear. In spite of not having relative data to the VO_{2max}, for the data of studies presented above the increment of the LVIDd does not seem to be associated to an increment of VO_{2max}. Then, an important reflection was that the LVIDd would be able to explain part of variability of performance due to physiological factors that they are not associated to the VO_{2max}. It is possible that in middle distance events,

higher LVIDd could enable a more rapid onset of oxygen transport mechanisms. The result of this phenomenon would be a higher rate of energy obtained from the aerobic metabolism in the initial seconds of competition as well as an easing of the acidity level and the disposal of the products derived from metabolism. Muscle glycogen is the preferential fuel for endurance running and it isn't until glycogen is significantly depleted that the runner experiences fatigue. Centrally, an efficient oxygen transport system thus seems to be vital for success in endurance events. Higher LVIDd increased, in submaximal exercise, the capacity of the athlete to pump unusually large volumes of blood and oxygen to the muscles with a lower HR. This allows the muscles to achieve higher work rates before they outstrip the available oxygen supply, developing skeletal muscle anaerobiosis. Besides, this will come accompanied of reduction in the rate of expiratory ratio and consequently in a lower utilization of the carbohydrates. In addition, these changes increase the capacity of the muscles to use fat as a fue during exercise, there-by enhancing endurance performance. Furthermore, a higher LVIDd would be able to intervene in processes such as the removal of products derived from metabolism, functional economy and others environmental factors and the body fluid losses in order to prevent hyperthermia, especially when the race is run under warm conditions.

Limitations

The results of this study are not based in a solid statistical analysis and in a great sample of subjects. Nevertheless, the lack of longitudinal studies with elite athletes not only limits our understanding of the degree to which physiological changes continue to evolve over years of training but also how and when specific changes interact with performance improvement [3]. Consequently, and in front of the difficulty to make a solid longitudinal study with international athletes, we thought than the conclusions presented in this work, although they must be taken with care, can be useful to many trainers and scientists.

Conclusions

Serial echocardiograms evidenced that both male and female elite endurance-trained runners engaged in systematic exercise conditioning showed important changes in LVIDd with wall thinning over time. The association between LVIDd and running performance represent important criteria in considering extreme LV enlargement in elite runners to be a physiological adaptation and it leads on to

thinking that the echocardiographic would be able to be utilized to determine the fitness of the athlete and for the bigger knowledge of physiology applied to the sports performance. However, this study's design and different difficult variables to control do that these conclusions should be taken simply like the start of a new field of study

References

1. Abergel E., G.Chatellier, A.A.Hagege, A.Oblak, A.Linhart, A.Ducardonnet, J.Menard (2004) Serial left ventricular adaptations in world-class professional cyclists. *J.Am.Coll.Cardiol.* 44:144-149

2. Adams T.D., F.G.Yanowitz, A.G.Fisher, J.D.Ridges, K.Lovell, T.A.Pryor (1981) Non-invasive evaluation of exercise training in college men. *Circulation* 64:958-965

3. Berg K. (2003) Endurance training and performance in runners. Research limitations and unanswered questions. *Sports Med.* 33:59-73

4. Billat V., A.Demarle, J.Slawinski, M.Paiva, P.Koralsztein (2001) Physical and training characteristics of top-class marathon runners. *Med.Sci.Sports Exerc.* 33:2089-2097

5. Cox M.L, J.B.Bennett, G.A.Dudley (1986) Exercise training-induced alterations of cardiac morphology. *J.Appl.Physiol.* 61:926-931

6. De Maria A.N, A.Neumann, P.J.Schubart, G.Lee, D.T.Mason (1979) Systematic correlation of cardiac chamber size and ventricular performance determined with echocardiography and alterations in heart rate in normal persons. *Am.J.Cardiol.* 43:1-9

7. DeMaría A.N., A.Neumann, G.Lee, W.Fowler, D.T.Mason (1978) Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography. *Circulation* 57:237-243

8. Devereux R.B, P.R.Liebson, M.J.Horan (1987) Recommendations concerning the use of echocardiography in hypertension and general population research. *Hypertension* 9 (Suppl. 2):II97-104

9. Feigenbaum H. (1986) Echocardiography.4th Ed. Lea & Febiger, Philadelphia

10. Giada F., E.Bertaglia, B.De Piccoli, M.Franceschi, F.Sartori, A.Raviele, P.Pascotto (1998) Cardiovascular adaptations to endurance training and detraining in young and older athletes. *Int.J.Cardiol.* 65:149-155

11. Haykowsky M.J., D.Humen, K.K.Teo, A.H.Quinney, M.Souster, G.Bell, D.A.Taylor (2000) Effects of 16 weeks of resistance training on left ventricular morphology and systolic function in healthy men >60 years of age. *Am.J.Cardiol.* 85:1002-1006

12. Henry W.L., J.M.Gardin, J.H.Ware (1980) Echocardiographic measurements in normal subjects from infancy to old age. *Circulation* 62:1054-1061

13. Legaz A., E.Serrano, J.A.Casajús, D.Munguía (2004) The changes in running performance and maximal oxygen uptake alter long-term training in elite athletes. *J.Sports Med.Phys. Fitn.* (in press)

14. Legaz A, E.Serrano, M.González, I.Lacambra (2004) The echocardiography to measure the fitnnes of elite runners. *J.Am.Soc.Echocardiog.* (in press)

15. Pelliccia A, F.Culasso, F.M. Di Paolo, B.J.Maron (1999) Physiologic left ventricular cavity dilatation in elite athletes. *Ann.Intern.Med.* 130:23-31

16. Pelliccia A., B.J.Maron, F.Culasso, A.Spataro, G.Caselli (1996) Athlete´s heart in women: echocardiographic characterization of highly trained elite female athletes. *JAMA* 276:211-215

17. Pelliccia A., J.Barry, B.J.Maron, A.Spataro, M.A.Proschan, P.Spirito (1991) The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *N.Engl.J.Med.* 324:295-301

18. Perrault H., R.A.Turcotte (1994) Exercise-induced cardiac hypertrophy. Fact or fallacy? *Sports Med.* 17:288-308

19. Pluim B.M., A.H.Zwinderman, A. van der Laarse, E.E. van der Wall (2000) The athlete´s heart: a meta-analysis of cardiac structure and function. *Circulation* 101:336-344

20. Sahn D.J., A.DeMaria, J.Kisslo, A.Weyman (1978) Recommendations regarding quantification in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation* 58:1072-1083

21. Secher N. (1993) Physiological and biomechanical aspects of rowing. Implications for training. *Sports Med.* 13:24-42

22. Sharma S., P.M.Elliot, G.Whyte, N.Mahon, M.S.Virdee, B.Mist, W.J.McKenna (2000) Utility of metabolic exercise testing in distinguishing hypertrophic cardiomyopathy from physiologic left ventricular hypertrophy in athletes. *J.Am.Coll.Cardiol.* 36:864-870

23. Spiriev B. (1998) IAAF scoring tables of athletics. IAAF

24. Spirito P., A.Pellicia, M.A.Proschan, M.Granata, A.Sparato, P.Bellone, G.Caselli, A.Biffi, C.Vecchio, B.J.Maron (1994) Morphology of the "athlete´s heart" assessed by echocardiography in 947 elite athletes representing 27 sports. *Am.J.Cardiol.* 74:802-806

25. Urhausen A., W.Kindermann (1999) Sports-specific adaptations and differentiation of the athlete´s heart. *Sports Med.* 28:237-244

26. Whyte G.P., K.George, S.Sharma, S.Firoozi, N.Stephens, R.Senior, W.J.McKenna (2004) The upper limit of physiological cardiac hypertrophy in elite male and female athletes: the British experience. *Eur.J.Appl.Physiol.* 92:592-597

Accepted for publication 18.02.2005