A comparison between the heart of young athletes and of young healthy sedentary subjects: a morphometric and morphofunctional

A comparison between the heart of young athletes and of young healthy sedentary subjects: a morphometric and morpho-functional study by echo-color-doppler method

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SUMMARY

Morphologic and morpho-functional heart differences between healthy young athletes and healthy young subjects who do not practice agonistic sport have been studied using Color Doppler Echography (CDE).

Overall, 68 subjects were enrolled in the study (age range: 19-26 yrs). Of them, 34 subjects (17 men and 17 women) were practicing sport agonistically; the 34 controls (17 men and 17 women) did not practice any sport on a regular basis. In each subject, age, height, weight, body mass index, practiced sport, systolic and diastolic blood pressure were recorded. CDE measures included telediastolic left and right ventricular diameters (LVDd and RVDd, respectively), interventricular septum thickness (IVSd), posterior wall thickness of the left ventricle (PLVWd), left and right atrium diameters during ventricular systole (LAD and RAD respectively), and continence of each heart valve (mitral; tricuspid; aortic; pulmonic).

In women, LADd was significantly higher in the athletes than in the controls (35.04±4.13 vs 31.81±3.34; $p<0.02$). Physiological regurgitation in at least one heart valve was observed in 15 out of 17 (88.2%) of the athletes; in 12 cases only one valve was involved: the mitral valve presented physiological regurgitation in 8 women, the tricuspid in 4, the aortic in 2 and the pulmonic in 6. In the control female population (17 persons), only 2 women showed evidence of regurgitation.

In men, except for RVDd, CDE measurements were all significantly higher in the athletes than in the controls: LVDd (49.4±3.13 vs 46.02±4.46; $p<0.02$); IVSd (9.79±1.24 vs 8.59±0.91; $p<0.003$); PLVWd (8.63±1.29 vs 7.48±0.66; $p<0.002$).

Physiological regurgitation through one or more heart valves was demonstrated in all the 17 male athletes studied; in 9 cases (52.9%) only one valve was involved. Mitral regurgitation was ob-

served in 8 cases (47%); tricuspid in 6 (35.3%). No physiological regurgitation through the aortic valve was found, while 15 cases (88.2%) presented a pulmonic regurgitation.

Among male controls, physiological regurgitation was demonstrated only in 2 persons out of 17 (11.8%), both involving the pulmonic and the aortic valve.

In the total population of athletes compared to controls, analyzing men and women jointly, we found that LAD ($p<0.001$),RAD ($p<0.001$), LIVD ($p<0.01$) were significantly larger in cases than in controls, while for RVD, IVSd and PLVWd such a difference did not reach statistical significance. No relationship was found betweeen CDE data and either age, height, weight or blood pressure.

INTRODUCTION

It is well known that when persons practice a sport agonistically, their heart must face both the increase of metabolism of muscles used, particularly the increase oxygen requirements and the emotional stress, which varies according to the different type of sport but is generally intense. (Bevegard and Shepard, 1967; Astrand and Rodhal, 1970; Raskoff *et al.*, 1976; Frenkl, 1977; Douglas, 1989; Oury *et al.*, 1998; Dickerman *et al.*, 1999; Zandrino *et al.*, 2000). Sport training, reproducing the physical challenges of the competition, prepares the heart to face the increase of metabolic requirements, but no training can reproduce the emotional stress experienced by the athlete during a competition; stress levels may vary in the same person practicing the same sport from a competition to another. Physiologists have shown that, compared to persons who do not practice agonistic sport, athletes present a lower heart rate, a higher heart weight and volume, and a higher telesystolic ventricular volume (Vasconcelos *et al.*, 1982; Parmley, 1985; Ganong, 1987; Penco *et al.*, 1989; Di Bello *et al.*, 1996; Hood and Northcote, 1999). The purpose of this study was to identify any morphologic and morpho-functional heart difference between healthy young athletes and healthy young persons who do not practice agonistic sport which can be related to the heart modifications in athletes described by physiologists and listed above. To address this issue, Color Doppler Ecography (CDE), a non-invasive technique which provides high-definition morphologic as well as functional imaging (Shapiro and Smith, 1983; Fedel *et al.*, 1988; Pelliccia *et al.*, 1996) has been used in this research.

MATERIALS AND METHODS

The study population included 68 healthy subjects (34 men and 34 women, age range 19-26). Of them, 34 persons, 17 men and 17 women were practicing sport agonistically; control subjects did not practice any sport on a regular basis. A list of the disciplines practiced included: swimming, long distance walking, cycling, body building, playing tennis. Most of the participants were students of the Superior Institute of Physical Education of Florence, Italy. In each participant, sex, age, height weight, body mass index, practiced sport, systolic and diastolic omeral blood pressure were recorded. CDE measures included telediastolic left and right ventricular diameters (LVDd and RVDd, respectively), interventricular septum thickness (IVSd), posterior wall thickness of the left ventricle (PLVWd), left and right atrium diameters during ventricular systole (LAD and RAD respectively), and continence of each heart valve (mitral; tricuspid; aortic; pulmonary).

An Acuson 128XP Echo-Color-Doppler, equipped by 5 and 7 MHz probes, was used for CDE.

Radiologic, ultrasonographic and hemodynamic data were correlated with age, sex, and body surface using Student's "T" test or correlation coefficient when appropriate.

RESULTS

In Table 1 the mean values (± standard deviation) of each measurement are presented: in the first section, results are presented separately for cases and controls; in the second section, they have been grouped by sex; age, height, weight, body index, systolic and diastolic blood pressure and CDE measurements expressed in mm are shown for each proband.

The presence or absence of physiological regurgitation in the mitral (1), tricuspid (2), aortic (3), and pulmonic (4) valves in each sex are presented in Table 2.

In women, the mean values of age, height, weight and body mass index were very similar in the athletes and in the controls and the slight differences were not significant; also CDE measurements were similar except for LADd, which was significantly higher in the athletes than in the controls (35.04±4.13 vs 31.81±3.34; $p<0.02$).

Physiological regurgitation in at least one heart valve was observed in 15 out of 17 (88.2%) of women athletes; in most cases (12 out of 15), only one valve was involved: the mitral valve presented physiological regurgitation in 8 women, the tricuspid in 4, the aortic in 2 and the pulmonic in 6 (Tab. 3). In the control female population (17 persons), only 2 women showed evidence of a physiological regurgitation (11.8%). The mitral valve was involved in both women; one of them presented also a physiological regurgitation through the pulmonic valve.

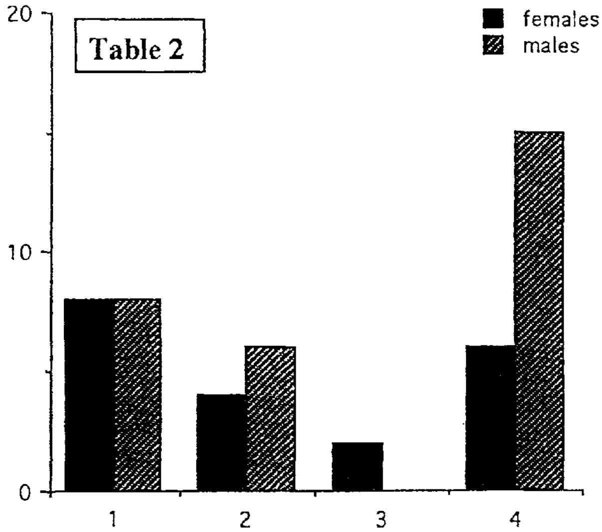
In men, the mean values of age, height,weight and body mass index were also remarkably similar in cases and in controls; except for RVDd, CDE measurements were significantly higher in the athletes than in the controls: LVDd (49.4±3.13 vs 46.02±4.46; $p<0.02$); IVSd (9.79±1.24 vs 8.59±0.91; $p<0.003$); PLVWd (8.63±1.29 vs 7.48±0.66; $p<0.002$).

Physiological regurgitation through one or more heart valves was demonstrated in all the 17 male athletes studied (100%); in 9 cases (52.9%) only one valve was involved (Tab. 5). Mitral regurgitation was observed in 8 cases (47%); tricuspid in 6 (35.3%). No physiologic regurgitation through the aortic valve was found, while 15 cases (88.2%) presented a pulmonic regurgitation.

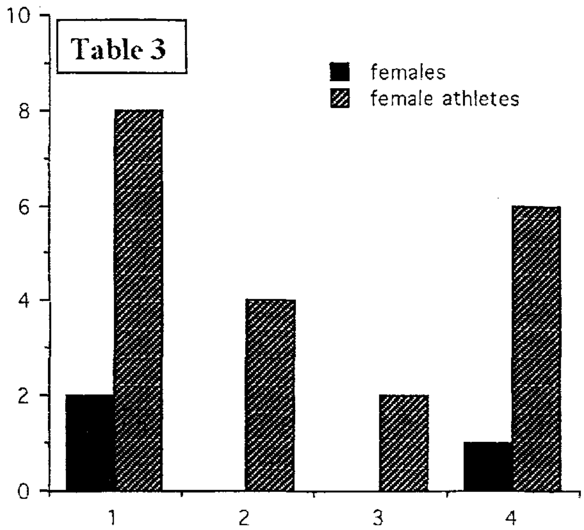
TABLE 1. MEAN AND STANDARD DEVIATION OF ALL VALUES (WEIG.=WEIGHT; BODY IND.=BODY INDEX; SYST.P.=SYSTOLIC PRESSURE; DIAS. P.=DIASTOLIC PRESSURE; LEFT V.=LEFT VENTRICLE DIAMETER ; RIG.V. = RIGHT VENTRICLE DIAMETER ; SEPT. = SEPTUM DIAMETER ; POST.WALL = POSTERIOR WALL THICKNESS; L.A.=LEFT ATRIUM DIAMETER; R.A.=RIGHT ATRIUM DIAMETER).

	AGE	HEIGHT	WEIG.	BODY IND..	SYST.P.	DIAS.P.	LEFT.V.	RIG.V.	SEPT.	POST.WALL	L.A.	R.A.
	ATHLETHES											
MEAN	23	172,7	67,7	22,6	122,4	79,8	49,4	32,1	9,1	8,2	36,6	33,8
ST.DEV.	2,27	6,82	10,59	2,36	8,76	3,91	3,98	4,45	1,55	1,30	4,27	6,99
	SEDENTARY SUBJECTS											
MEAN	22	166,6	63,2	22,7	118,4	77,9	46	29,6	8,4	7,6	33,4	29,8
ST.DEV.	2,35	8,20	10,77	3,05	10,12	5,72	4,13	3,20	1,00	0,89	3,34	3,83

	AGE	HEIGHT	WEIG.	BODY IND.	SYST.P.	DIAS.P.	LEFT.V.	RIG.V.	SEPT.	POST.WALL	L.A..	R.A.
	MALES											
MEAN	22,3	175,8	73,7	23,8	124,9	80,4	48,9	31,7	9,3	8,3	36,4	33,4
ST.DEV.	1,90	5,26	6,85	1,74	6,51	2,45	4,56	4,63	1,32	1,25	3,78	6,91
	FEMALES											
MEAN	22,8	163,5	57,2	21,4	115,9	77,3	46,5	30,1	8,2	7,6	33,3	30,2
ST.DEV.	2,72	5,31	7,20	2,98	10,19	6,24	3,89	3,24	1,13	0,94	4,03	4,30

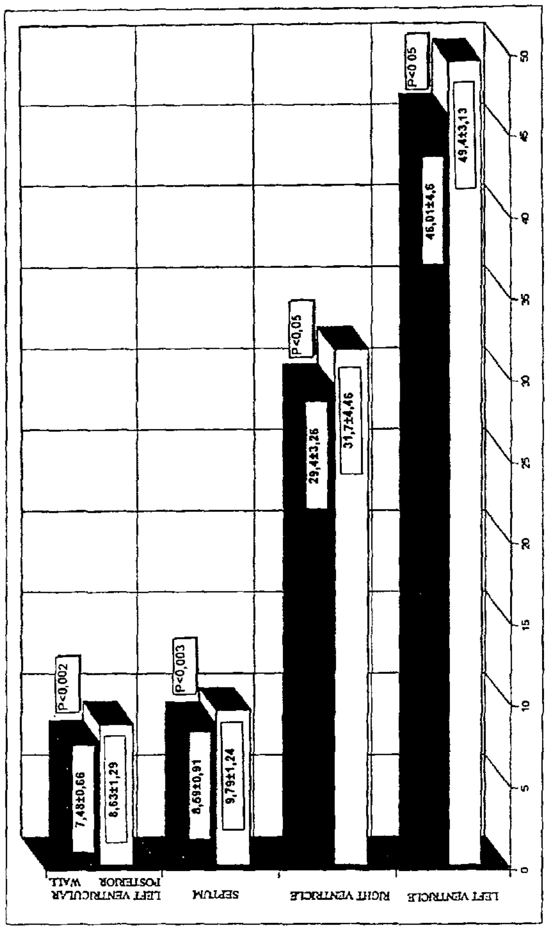


Incidence of physiological valvular regurgitation.
1)mitral r. 2)tricuspid r. 3)aortic r. 4) pulmonic r.



Incidence of physiological valvular regurgitation.
1)mitral r. 2)tricuspid r. 3)aortic r. 4) pulmonic r.

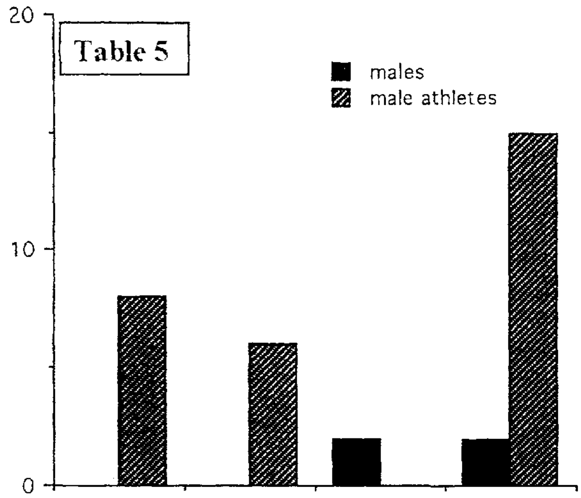
Table 4 MALES : comparison between athlethes (white) and sedentary subjects (black).



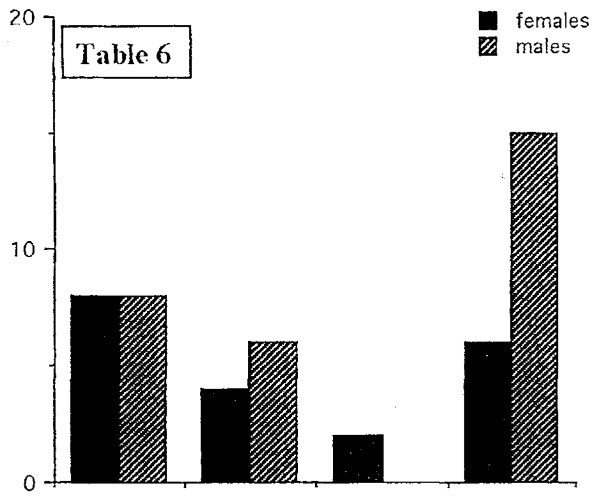
Among male controls, physiological regurgitation was demonstrated only in 2 persons out of 17 (11.8%), both involving the pulmonary and the aortic valve.

In Tables 6-7, the distribution of the physiological regurgitations in the population of the athletes is shown.

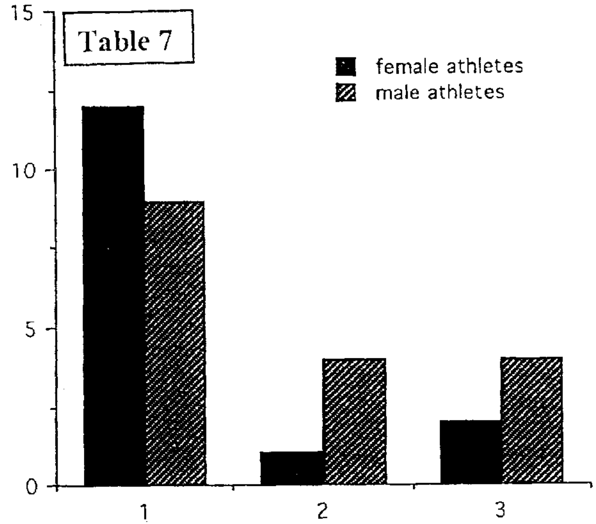
Finally, we compared the total population of athletes to the controls, analyzing men and women jointly. We found that LAD (36.1±4.09 vs 33.1±2.49; $p<0.001$), RAD (34.1±5.43 vs 29.4±2.97; $p<0.001$), LIVD (48.4±3.5 vs 46.1±4.2; $p<0.01$) were significantly larger in cases than in controls, while for RVD, IVSd and PLVWd such a difference did not reach statistical significance (Tab. 8). No relationship was found betweeen enlargement of the cardiac chambers and either age, height, weight or blood pressure.



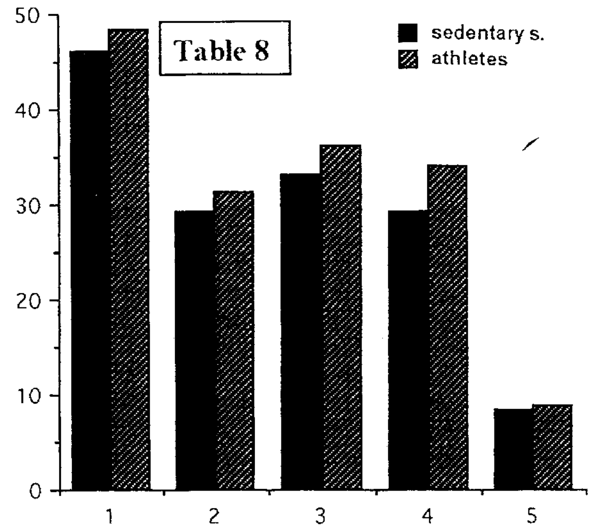
Incidence of physiological valvular regurgitation.
1)mitral r. 2)tricuspid r. 3)aortic r. 4) pulmonic r.



Incidence of physiological valvular regurgitation.
1)mitral v. 2)tricuspid v.3)aortic v. 4)pulmonic v.



Physiological Valvular regurgitations : 1) in single valve;2) in two valves; 3) in three valves.



Diastolic thickness of : 1) left ventricle; 2) right ventricle; 3) left atrium;4) right atrium;5)septum.

DISCUSSION AND CONCLUSIONS

These results have shown that the heart of young athletes develops early morphological as well as morpho-functional changes that, although still within the limits of physiology, must be promptly detected and monitored, so that practicing sports may help psychologic and physical development without being an hazard for the health of some predisposed subjects.

Altogether, our results may be synthetized in three major points. First, we have shown that young athletes (men and women considered together) present an higher diameter for all the four heart chambers than controls, with differences that reach significance for all except the right ventricular chamber. In agreement with most of the literature (Ikaheimo *et al.*, 1979; Nishimura *et al.*, 1980; Shapiro and Smith, 1983; Fedele *et al.*, 1988), these findings are not associated with heart hypertrophy, expressed as significant increase in the thickness of the IVS or of the PLVW.

Second, we have demonstrated a physiological regurgitation in 32 of the 34 athletes studied; furthermore, 11 of them presented a regurgitation through more than one valve. As opposed to these findings, only 4 of the 34 controls (11.8%) had a physiological regurgitation, involving a single valve in one case, and two in the other three. In our population of young athletes, the distribution of physiological regurgitation through any of the four valves did not appear to follow a recognizable pattern (for instance right heart vs left heart).

Finally, the observed heart modifications were more consistent in men than in women; indeed, although chamber diameters and thickness of IVS and PLVW were always higher in female athletes than in controls, this difference reached significance only for LAD diameter. Furthermore, while all the male athletes presented a physiological regurgitation, two females (11.8%) did not.

It is necessary to bear in mind that all the modifications observed in the hearts of these young athletes fall strictly within the limits of normality. Indeed, standard echocardiographic criteria are ≤40 mm for atrial diameter, ≤56mm for ventricular diameter and ≤11 mm for the thickness of the IVS and of the PLVW (Feigenbaum, 1986). However, the significant increase of these parameters may evolve in time to the triad defined by physiologists as the "athletes heart": decreased heart frequency, elevation of cardiac output and cardiac hypertrophy (Hanne-Pararo *et al.*, 1976; Bettini *et al.*, 1985; Caru' and Mauri, 1987; Mockel and Stork, 1996), which in turn may lead few, selected cases to develop pathologic heart function. For this reason, adequate detection and monitorage of the heart adaptive modifications is recommended, so that sport practice may be corrected before an irreversible damage may occur.

The association between agonistic sport practice and presence of physiologic heart valve regurgitation was very strong in our population. Phyisiological regurgitation occurs in structurally normal valves as a small regurgitant flow through the valvulary leaflets which do not perfectly appose; the use of CDE applied to the dynamic study of intra cardiac blood flow is allowing to identify physiological regurgitation as an highly frequent finding, to be distinguished from the pathologic regurgitation which occurs through an anatomically abnormal valve (Perry and Nanda, 1987; Takao *et al.*, 1988; Macchi *et al.*, 1994). From our data, it is not possible to establish whether the observed significant increase in heart chambers' diameters is the cause or the consequence of the regurgitation. Previous studies (Takao *et al.*, 1988; Pollack *et al.*, 1988; Douglas *et al.*, 1989; Zeppilli, 1990) reported a higher prevalence of physiological regurgitation through the right heart valves; in their hypotheses, this finding could be explained by an enlargement of right heart chambers due to adaptation to a countinuous physical effort, which would have led to valve incontinence through enlargement of the annulus and/or relative shortening of the chordae tendineae. However, our results do not support these hypotheses, both because we could not demonstrate an higher prevalence of physiologic regurgitations involving the right heart valves and because we have found that in female athletes, where the heart chambers' enlargement was not significant, physiological regurgitation was highly frequent, as if the latter would preceed the former in time.

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REFERENCES

Astrand PO, Rodhal K. 1970.Textbook of work physiology.Mc Graw Hill Inc., New York.

Bettini R, Visonà L, Antolini R, Irler WJ, Gramegna L, Cozzi F, Debiasi M, Resina A, Bevegard B, Shepard J. 1967. Regulation of the circulation during exercise in man. Physiol Rev 47: 178-185.

Carù B, Mauri L. 1986. Bradyarrhythmias in sport activity and cardiovascular diseases. Rulli V, Vajole SF (Eds), Ed. Pozzi, pp. 85-91, Roma.

Di Bello V, Santoro G, Talarico L, Di Muro C, Caputo MT, Giorgi D, Bertini A, Bianchi M, Giusti C. 1996. Left ventricular function during exercise in athletes and in sedentary men. Med Sci Sports Exerc, 28:190-6.

Dickerman RD, Schaller F, Mc Guirre S. 1999. Does aortic valve calcium correlate with coronary artery calcium in young power athletes?. Am J Cardiol Apr, 15; 83:1300.

Douglas PS. 1989. Cardiac considerations in the triathlete. Med Sci Sports Exerc, 21: S214-8.

Douglas PS, Berman GO., O'Toole ML, Hiller WD, Reichek N. 1989. Prevalence in multivalvular regurgitation in athletes. Am J Cardiol, 64: 209-212.

Fedele F, Agati L, Penco M, Sciomer S, Gagliardi MG, Di Renzi P. 1988. Exercise echocardiography in athletes. A study of the mechanism responsible for the increasing in the ejection fraction. Int. J Sport Cardiol, 2: 1-12.

Feigembaum H. 1986. Echocardiography, Ed 4, Philadelphia, Lea & Febiger.

Frenkl R. 1977. Athlete's heart: definition and semeiology, clinical and experimental observations. In: "Cuore e Sport" Int Congr, Crippa-Berger(Eds.), pp. 38-47, Milano.

Ganong WF. 1987. Fisiologia medica. Piccin, Padova.

Hanne-Pararo N, Drory Y, Schdenfeld Y. 1976.Common ECG changes in athletes Cardiology 61: 267-273.

Hood S, Northcote RJ. 1999. Cardiac assessment of veteran endurance athletes: a 12 year follow up study. Br J Sports Med. 33: 239-43.

Ikaheimo MJ, Palatsi IJ, Takkunen JT. 1979. Noninvasive evaluation of the athletic heart: sprinters versus endurance runners. Am J Cardiol, 44: 24-30.

Macchi C., S.Zecchi Orlandini and Giovanni E.Orlandini (1994). An anatomical study of the healthy human heart by echocardiography with special reference to physiological valvular regurgitation. Ann Anat, 176: 81-86.

Mockel M, Stork T. 1996. Diastolic function in various forms of left ventricular hypertrophy: contribution of active Doppler stress echo. Int J Sport Med, 17: S184-190. Review.

Nishimura T, Wink K, Kawai C. 1980. Echocardiography evaluation of long -term effects of exercise on left ventricular hypertrophy and function in professional bicyclist. Circulation, 61: 832-841.

Oury JH, Doty DB, Oswalt JD, Knapp JF, Mackey SK, Duran CM. 1998. Cardiopulmonary response to maximal exercise in young athletes following the Ross procedure. Ann Thorac Surg, 66: S153-4.

Parmley WW. 1985. Mechanics of ventricular muscle. In: "The ventricle", 41st Ed, Levine HJ, Gaash WH (Eds), M Nijhoff Pbs, Boston.

Pelliccia A, Maron BJ, Culasso F, Spataro A, Caselli G. 1996. Athlete's heart in women. Echocardiographic characterization of highly trained elite female athletes. JAMA 276: 211-5.

Penco M Agati L, Dagianti A, Fedele F, Pelliccia A, Zeppilli P. 1989. Attuali conoscenze sulla morfologia e funzione del cuore d'atleta. Atti del 4° congresso Nazionale della Società Italiana di Cardiologia dello Sport, pp. 178-189, Venezia.

Perry JL., Nanda NC. 1987. Recent advances in color doppler evaluation of valvular regurgitation, Echocardiography 4: 271.

Pollack SJ, Mclillan SA, Urnip KW. 1988.Cardiac evaluation of women distance runners by echocardiography color doppler flow mapping. J Am Coll Cardiol, 11: 89.

Raskoff WS, Goldman S, Cohn K. 1976. The athletic heart. JAMA 236: 158-163.

Shapiro LM, Smith RG. 1983. Effect of training on left ventricular structure and function. An echocardiographic study. Br Heart J, 50: 534-542.

Takao S, Miyetake K, Izumi S. 1988. Clinical implications of pulmonic regurgitation in health individuals: detection by cross-sectional pulsed doppler echocardiography. Br Heart J, 59: 542.

Vasconcelos DF, Junqueira Junior LF, Sanchez Osella OF. 1993. Doppler echocardiographic comparison of valvular dynamics in bicycling, running and football athletes and sedentary subjects. Arq Bras Cardiol, 61: 161-4.

Vecchiet L, Furlanello F. 1985. Heart rate analysis of top-level football players during training on altitude. New Trends Arrhyt 1: 347-352.

Zandrino F, Molinari G, Smeraldi A, Odaglia G, Masperone MA, Sardanelli F. 2000. Magnetic resonance imaging of athlete's heart: Myocardial mass, left ventricular function and cross-sectional area of coronary arteries. Eur Radiol, 10: 319-25.

Zeppilli P. 1990. Cardiologia nello sport. CESI Editore Roma.

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