Sports-Specific Features of Athlete's Heart and their Relation to Echocardiographic Parameters

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Abstract

Chronic physical training may induce morphological and useful functional adaptations which affect all cardiac chambers. Morphological modifications are mainly modest and far from pathologic ones. All these adaptations seem helpful for sport's performance. Hemodynamic and neurohumoral stresses depend on the muscular exercise type performed, static or dynamic. However, sports-specific adaptive cardiac structural changes are yet debated. Actually, it appears that highly trained athletes develop a left ventricular fair combination of cavity dilatation and increased wall thickness. Thus, it is not possible to clearly separate a strength-trained from an endurance-trained athlete's heart. However, this review shows that some specific cardiac adaptations mainly linked to the specific training stimulus may be observed. Dilatation slightly predominates in dynamic endurance-trained athletes whereas increased wall thickness slightly predominates in dynamic resistance- and static-trained athletes. Thus, assessment of athletes' echocardiographic parameters should take into account both sport and training specificities practiced, in terms of quantity and contents.

Key Words:
Echocardiography · Athlete's heart · Specific training stimuli

Herz 2006;31:531–43

DOI 10.1007/s00059-006-2862-2

Sportspezifische Merkmale des Sportherzens und ihr Zusammenhang mit echokardiographischen Parametern

Zusammenfassung

Körperliches Training kann morphologische und nützliche funktionelle Anpassungen bewirken, die alle Herzkammern betreffen. Die morphologischen Anpassungen sind moderat und unterscheiden sich klar von pathologischen Veränderungen. Alle diese Adaptationen scheinen für die sportliche Leistungsfähigkeit hilfreich. Hämodynamischer sowie neurohumoraler Stress hängen von der Art des Muskeltrainings – statisch oder dynamisch – ab. Sportspezifische adaptive Veränderungen der Herzstruktur werden jedoch noch diskutiert. Tatsächlich scheinen hochtrainierte Athleten eine typische linksventrikuläre Kombination aus kavitärer Erweiterung und erhöhter Wanddicke zu entwickeln. Die klare Unterscheidung zwischen einem kraft- und einem ausdauertrainierten Herzen ist daher nicht möglich. Die vorliegende Übersichtsarbeit zeigt, dass einige spezifische kardiale Adaptationen, vorwiegend in Verbindung mit dem jeweiligen Trainingsstimulus, beobachtet werden können. Bei dynamisch ausdauertrainierten Sportlern überwiegt die Herzerweiterung geringfügig, während sich die Wanddickenzunahme etwas häufiger bei statisch und dynamisch krafttrainierten Athleten findet. Bei der Bewertung echokardiographischer Parameter sollten daher die Sportart und deren trainingsspezifische Besonderheiten berücksichtigt werden.

Schlüsselwörter:
Echokardiographie · Sportherz · Spezifische Trainingsstimuli

Introduction

Echocardiography is a noninvasive and repeatable imaging tool largely used in sports cardiology. It is classically used in order to differentiate training-induced adaptations from pathologic changes such as hypertrophic or dilated cardiomyopathy [42].

Echocardiography allows also an evaluation of the heart's functional and structural adaptations in response to physical training [18]. A high level of physical training may be associated with morphological and functional cardiac alterations, the so-called athlete's heart [67].

Morphological adaptations affect the four cardiac chambers' size with a dilatation associated with an increase in both wall thickness and calculated cardiac mass [22, 57, 65].

Concerning the functional adaptations, despite the increased left ventricular (LV) mass, resting myo-

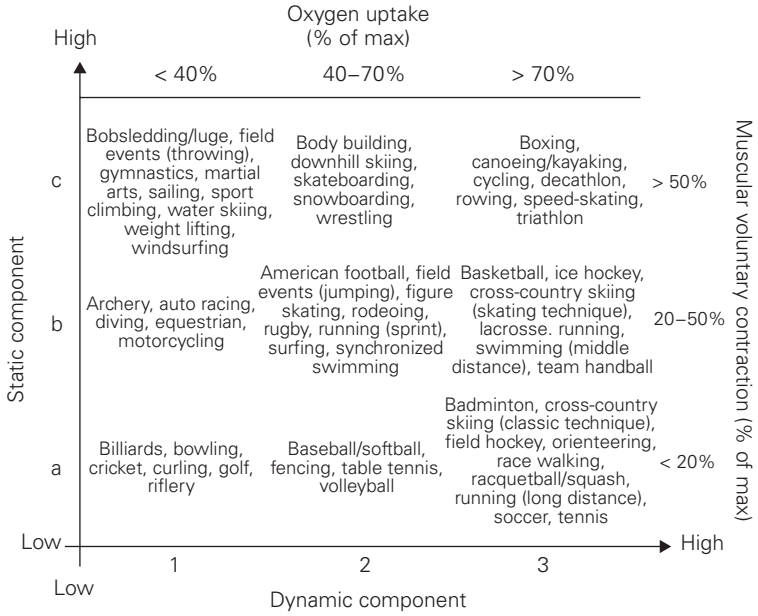


Figure 1. Sports classification according to dynamic and static components (adapted from [47]).

Abbildung 1. Klassifikation der Sportarten in dynamische und statische Komponenten (modifiziert nach [47]).

cardial functions are not significantly altered. Thus, this adaptive cardiac hypertrophy is classically presented as a physiological one [83].

Morganroth et al. were the first to postulate that two different morphological forms, strength-trained and endurance-trained, of athlete's heart can be distinguished [49]. However, the sports-specific adaptive cardiac structural changes are actually somewhat controversial [25, 60, 65, 80, 87].

The aim of this brief review is to resume actual data upon morphological and functional athlete's heart adaptations and particularly their sports-specific features.

Cardiovascular Effects of Sports

Classification of Sports

From the cardiovascular point of view, sports are classified according to their type, dynamic (isotonic) or static (isometric), and to their intensity, low, moderate or high [47]. Briefly, dynamic exercise involves changes in muscle length and joint movement with rhythmic contractions that develop a relatively small intramuscular force, and static exercise induces development of a large intramuscular force with little or no change in muscle length or joint movement [47]. These two types of exercises should be thought of as the two opposite poles of a continuum, with most physical activities involving both static and dynamic components. For example, distance running has low static and high dynamic demands; body building has principally high static and low dynamic demands, and rowing or canoeing have both high static and dynamic demands (Figure 1).

Other sports classifications mainly proposed by sports medicine physicians are based upon the energetic, i.e., aerobic and anaerobic, demand of the sport concerned. The terms dynamic and static exercise characterize physical activity on the basis of the mechanical action of skeletal muscles involved and differ from the terms aerobic and anaerobic exercise. For example, high-intensity static exercise is performed with anaerobic metabolism, whereas high-intensity dynamic exercise lasting for more than several minutes is performed mainly with aerobic metabolism. However, some dynamic exercises, such as sprinting or jumping, are performed primarily with anaerobic metabolism.

Acute Cardiovascular Adaptations to Physical Exercise

During a progressive dynamic exercise, oxygen uptake (VO_2) increases from the resting value (3.5 ml O_2/min/kg = 1 MET) until maximal VO_2 (VO_2 max) is achieved. Thus, well-adapted transport and uptake of O_2 from atmosphere to mitochondria are required in order to adapt to the large increase of oxygen flux induced by maximal aerobic exercise. VO_2 max, which is an objective measure of aerobic fitness, is also a classic index of the integrity of cardiovascular function. Indeed, according to the Fick principle, VO_2 is the product of cardiac output (CO) multiplied by the arteriovenous O_2 difference [(a–v)O_2D] which represents the extraction of oxygen from the blood by the active tissue. Thus, central factors, CO, as well as peripheral factors, (a–v)O_2D, determine systemic O_2 transport. During progressive dynamic exercise both CO components, heart rate (HR) and stroke volume (SV), increase. SV increase is achieved by both an increase in end-diastolic volume (Frank-Starling mechanism) and a decrease in end-systolic volume (increased contractile state). Total peripheral resistances deeply decrease. Thus, systolic and mean blood pressure increase moderately and diastolic blood pressure is maintained or decreases slightly [39].

By contrast, static exercise induces a small increase in VO_2, HR and CO and no change in SV. Total peripheral resistances do not decrease. Associated systolic, mean and diastolic blood pressure increases are linked to the muscle mass involved, the percent of maximal voluntary contraction (MVC) used and the contraction duration [41].

Finally, dynamic exercise induces mainly a volume load on cardiac cavities, whereas static exercise causes mainly a pressure load [39].

Exercise Cardiovascular Adaptations in Trained Subjects

Maximal CO reported in endurance athletes (35–40 l/min) is greatly increased in comparison with sedentary people (20–25 l/min). Even if some authors described a small decrease of the individual maximal HR (HR max) in top-level endurance-trained athletes, classically physical training does not alter HR max which is mainly linked to age [5]. Thus, the CO max increase observed in trained subjects is linked to an increase in maximal SV (SV max). For example, in endurance-trained subjects, because of bradycardia, resting SV is approximately 130–150 ml/beat (vs. 70–80 ml/beat in sedentary people) and can attain 200–220 ml/beat (vs. 120–140 ml/beat in sedentary people) at maximal aerobic power. Thus, this increased SV associated with a larger maximal $(a–v)O_2D$ explains the high level of VO_2 max classically described in endurance-trained subjects [39, 47]. This large SV improvement is greatly due to the association of endurance training-induced hypervolemia [26] and to the morphological and functional adaptations of athlete's heart.

In contrast to dynamic exercise training, static exercise training results in a small or no increase in VO_2 max. The most striking adaptation concerns a lowered increase in blood pressure for the same absolute work level developed [41].

Determinants of Physical Training Cardiac Hypertrophy

Physiological Determinants

Three main physiological determinants, genetic, hemodynamic and neurohumoral, are classically proposed to explain the cardiac hypertrophy induced by chronic physical training [7, 74]. The associated effects of these determinants induce quantitative cellular growth response but also many specific perturbations in the expression of the cellular constituents [74]. It must be noted that separable signaling pathways regulate cardiac hypertrophy due to a pathologic basis and cardiac hypertrophy, such as athlete's heart one, observed in response to physiological cause [7].

Genetic determinants present two different aspects. First, it has been shown that individual genotypes, for example those associated with angiotensin-converting enzyme (ACE), angiotensinogen or endothelin-1, may influence cardiac hypertrophy development as vascular adaptations to chronic exercise [7]. For example, the ACE genotype DD is both positively correlated to performance in resistance sports and to hypertrophy of myocardial wall thickness. Second, physical training alters many genes' expression, for example the natriuretic factor or the nitric oxide ones, which explain some beneficial adaptations of physical training [7].

Multifactorial hemodynamic determinants' effects, such as diastolic load and resting bradycardia-exercise tachycardia alternation, are proposed in the physical training cardiac hypertrophy development [74].

The effects of several neurohumoral factors, for example autonomic nervous system [17], plasma catecholamines and angiotensin II, are also proposed [74]. The anabolic effects of some hormones are illustrated both by the lack of myocardial wall thickness increase in boys before puberty [51, 62] and by the low level of myocardial wall hypertrophy observed in trained women [58, 92].

Influence of Doping on Cardiac Hypertrophy in Athletes

Because of controversial and scarce validated data, it is actually not possible to affirm the effects of doping on myocardium. However, some clinical and experimental studies argue for the development of a concentric cardiac hypertrophy with altered diastolic function in case of anabolic steroid or growth hormone use [9, 31, 87] and of an eccentric cardiac hypertrophy in case of erythropoietin use [1]. In the latter study, echocardiography was performed twice, in 1995 and 1998, by the same operator, in some professional cyclists. They showed a marked increase in LV dilatation without any increase in wall thickness [1].

Echocardiographic Findings in Well-Trained Athletes

Chronic physical training may induce morphological and functional myocardial alterations which affect all cardiac chambers and both resting systolic and diastolic functions whatever the age, sex, and race. In accordance with its genetic determinant, the degree of cardiac hypertrophy may vary between subjects involved in the same training program. However, usual cardiac dimensions in trained people are mildly increased in comparison with sedentary subjects (cavity diameter 3–6 mm and wall thickness 2–3 mm) and generally remain within or slightly above the classic normal range (Tables 1 to 3).

Cardiac Chambers' Diameters

Left ventricular end-diastolic diameter (LVID) is increased in athletes, and is greater in male than in female athletes [56]. LVID is usually < 60 mm in an endurance athlete with normal body size and seldom exceeds 62 mm (see Table 1). In the study by Pellicia et al., LVID exceeded 60 mm in 14% of the athletes

Table 1. Review of echocardiographic data concerning endurance athletes and matched untrained controls. A: Doppler peak velocity of LV filling after atrial contraction; E: Doppler peak velocity of early LV filling wave; EF: ejection fraction; HR: heart rate; h/R: relative wall thickness = LV wall thickness/LV end-diastolic radius; IVST: interventricular septal thickness; LVID: left ventricular internal diameter; LVM: left ventricular mass; LVM index: left ventricular mass related to body surface area; LVV: left ventricular volume in diastole; NS: not significant; PW: left ventricular posterior wall thickness; SF: shortening fraction; SP: soccer players; TT: total trained people; VO_2 max (ml · min^{-1} · kg^{-1}): weight-indexed maximal O_2 uptake.

Tabelle 1. Literaturübersicht zu echokardiographischen Daten bei Ausdauerathleten und normalen untrainierten Kontrollen. A: Doppler-Spitzengeschwindigkeit des atrialen Einstroms; E: Doppler-Spitzengeschwindigkeit der frühen linksventrikulären Füllung; EF: Ejektionsfraktion; HR: Herzfrequenz; h/R: relative Wanddicke = LV-Wanddicke/LV-enddiastolischer Radius; IVST: Dicke des interventrikulären Septums; LVID: linksventrikulärer enddiastolischer Durchmesser; LVM: linksventrikuläre Muskelmasse; LVM index: linksventrikuläre Muskelmasse, bezogen auf die Körperoberfläche; LVV: linksventrikuläres enddiastolisches Volumen; NS: nicht signifikant; PW: linksventrikuläre Hinterwanddicke; SF: Verkürzungsfraktion; SP: Fußballspieler; TT: alle Trainierten; VO_2 max (ml · min^{-1} · kg^{-1}): maximale Sauerstoffaufnahme.

Authors	Subjects	Methods	VO_2 max	Correlations	LVM	LVM index	LVID	PWT	IVST	h/R	LVV	E	A	E/A	EF	SF
Zeidis et al. 1978 [94]	10 female hockey players	M-mode echo	51.70**	VO_2 max vs. LVID			2.93**	NS	NS							
	10 controls		41.20				2.63									
Cohen & Segal 1985 [10a]	10 distance runners	M-mode echo			311.80*		5.58*									
	10 wrestlers				325.90*			1.29*	1.35*							
	10 controls				215.90		4.97	1.01	1.91							
Csanady et al. 1986 [13]	15 boys canoeists	M-mode, 2-D,			261.00**		4.61*	0.75	0.83**					NS	NS	NS
	17 controls	Doppler echo			202.00		4.43	0.65	0.76							
Maron 1986 [41a]	20 athletes	M-mode echo				256.00*	5.39*	1.07*	1.04*							
	20 controls					175.00	4.91	0.90	0.91							
Miki et al. 1987 [45]	117 professional cyclists	Doppler echo										NS	NS	NS	NS	NS
	40 controls															
Milliken et al. 1988 [46]	Cross-country skiers	MRI	77.00	VO_2 max vs. LVM	239.00***											
	Endurance cyclists		72.00		244.00***											
	Long-distance runners		75.00		258.00***											
	Controls				189.00											
Cohen et al. 1989 [10]	99 professional cyclists	M-mode, 2-D,			305.00***					0.40***			36.00***	1.81***		
	26 controls	Doppler echo			174.00					0.35		NS	41.00	1.61	NS	NS
Osborne et al. 1992 [52]	15 endurance athletes	M-mode echo		VO_2 max vs. LVID	214.00*	113.00*	5.53*	0.92	0.84							
	26 controls			and LVM	178.00	91.00	5.20	0.87	0.78							
Riley-Hagan et al. 1992	36 endurance athletes	MRI	63.00*		159.00*			1.15*			122.00*					
[66]	11 controls		35.00		115.00			0.98			93.00					
Bjornstad et al. 1993 [3]	30 athletic students	2-D, Doppler echo				147.00**										
	30 controls					100.00						NS	NS	NS	NS	
Di Bello et al. 1993 [15a]	12 elite isotonic athletes,	M-mode, 2-D,			315.00***		5.07	1.25	2.22						75.50	
	11 controls	Doppler echo			225.00		4.81	1.31	2.24						71.80	
Levy et al. 1993 [40]	13 endurance athletes	Radionuclide	68.00**		210.00**							94.10**				
	13 controls		47.00		190.00							85.00				
Fagard 1996 [24]	138 cyclist	M-mode, 2-D,				262.00	5.51*	1.16*	1.17*	0.42*						
	84 controls	Doppler echo				159.00	5.05	0.89	0.91	0.35						
Fagard 1996 [24]	119 long-distance runners	M-mode, 2-D,				216.00*	5.30	1.05*	1.08*	0.40*						
	81 controls	Doppler echo				149.00	4.80*	0.88	0.93	0.37						
Pellicia et al. 1996 [58]	600 elite female athletes	M-mode,2-D			133.00**	80.00**	4.89**	0.78**	0.81**	0.34**						
	65 controls	Doppler echo			98.00	60.00	4.60	0.67	0.70	0.31						
Karjalainen et al. 1997	32 endurance athletes	M-mode, 2-D,			263.00***	141.00***	5.48***	1.12***	1.21***	0.53		72.00**	31.00***	2.42***		
[36]	15 controls	Doppler echo			167.00	84.00	5.13	0.91	0.88	0.53		65.00	40.00	1.66		
Iglesias-Cubero et al.	37 cyclists	M-mode, 2-D,	57.60*	VO_2 max vs. LVM		123.00*	NS	1.10*	1.10*							
2000 [34]	15 soccer players	Doppler echo	48.50	for TT		92.00	NS	1.00	0.90							
	12 canoeists		53.30	VO_2 max vs. LVM for SP		113.00	NS	1.10	1.00							
Jungblut et al. 2000 [35]	12 endurance trained	M-mode, 2-D,	44.00***			61.00*										
	12 controls	Doppler echo	27.00			51.00	NS	NS	NS		NS	NS	NS	NS		
Sozen et al. 2000 [79]	83 soccer players	M-mode, 2-D,				142.00***	5.24***	1.08***	1.14***		133.00***			2.08***		
	52 controls	Doppler echo				103.00	4.88	0.91	0.99		113.00			1.65		
Schannwell et al. 2002	49 endurance athletes	M-mode, 2-D,			225.00**	99.00**	5.00	1.40**	1.50**			65.00	51.00	1.27		44.00
[72]	25 controls	Doppler echo			119.00	52.00	4.80	0.80	0.90			67.00	52.00	1.28		43.00
Scharhag et al. 2002 [73]	21 endurance athletes	MRI	68.00**	VO_2 max vs. LVM	200.00***		5.60***	1.12***	1.14***		167.00***				59.00	35.00
	21 controls		42.00		148.00		5.00	0.95	1.02		125.00				59.00	37.00
Abergel et al. 2004 [1]	285 cyclists	M-mode				141.00***	6.01***	1.00***	1.11***						66.10**	33.90**
	52 controls					73.00	4.90	0.80	0.86						63.90	36.10
Gates et al. 2004 [28]	10 trained kayak canoeists				240.00**		NS	1.12*	1.14*			NS	NS	NS	NS	NS
	10 moderately active men				169.00*			0.92	0.94							
	10 controls				124.00			0.91	0.93							
Saito et al. 2004 [70]	22 female rowers	M-mode, 2-D,	47.00	VO_2 max vs. LVID	158.00	95.00	31.00	0.80	0.80			91.00	38.00	2.50	75.00	
		Doppler echo		and LVM												
Barbier et al. [2]	16 cyclists	M-mode, 2-D,	70.70***	VO_2 max vs. LVM,	281.00***	152.60***	5.65	1.00	1.00		166.00	77.30	42.60	1.85	57.40	34.40
	12 canoeists	Doppler echo	51.50	LVV, E, A(–),	213.00*	126.50*	4.95	1.01*	1.04*		144.90*	84.10	46.80	1.89	53.90	36.50
	12 tumblers		50.7	LVID, HR (–)	195.00	111.20	5.01	0.81	0.84		139.70	90.20	46.10	2.01	52.10	36.60
	19 controls			for TT	180.00	96.20	4.77	0.89	0.87		131.40	86.80	48.80	1.82	59.90	37.90

*p < 0.05, **p < 0.01, ***p < 0.001: compared with controls

Table 2. Review of echocardiographic data concerning mixed and strength athletes and matched untrained controls. For abbreviations see Table 1.
Tabelle 2. Darstellung der echokardiographischen Daten bei gemischten und Kraftathleten sowie normalen untrainierten Kontrollen. Abkürzungen s. Tabelle 1.

Authors	Subjects	Methods	VO_2 max	Correlations	LVM	LVM index	LVID	PWT	IVST	h/R	LVV	E	A	E/A	EF	SF
Morganroth et al. 1975 [49]	4 shot-putters	M-mode, 2-D echo				138.00	5.00	1.35	1.38							
Dickhuth et al. 1979 [20]	9 weight lifters	M-mode, 2-D echo					5.10	1.26	1.03							
	15 athletic putters, throwers						5.20	1.26	1.09*							
Menapace et al. 1982 [44]	13 weight lifters (top level)	M-mode, 2-D echo					5.20	1.39	0.97							
Rost 1982 [66a]	8 weight lifters	M-mode, 2-D echo					5.60	1.21	1.04							
Snoeckx et al. 1982 [78]	14 weight lifters	M-mode,2-D echo					5.10	0.90*	0.90*							
	17 controls						5.00	0.85	0.82							
Brown et al. 1983 [6]	18 weight lifters	M-mode, 2-D echo				134.00*	5.50	0.88*	0.94*							
	9 controls					116.00	5.50	0.86	0.89							
Shapiro 1984 [76]	34 athletic throwers and weight lifters	M-mode 2-D echo					5.40	1.25	1.05							
Salke et al. 1985 [71]	15 steroid-using body builders	M-mode, 2-D echo					5.30	1.37*	0.94							
	15 steroid-free body builders						5.20	1.24	0.94							
Pearson et al. 1986 [54]	16 weight lifters	M-mode, 2-D echo					5.60	1.00	0.90							
Colan et al. 1987 [11]	11 power lifters	M-mode, 2-D echo					54.0		1.32							
Roy et al. 1988 [68]	46 body builders	M-mode, 2-D echo				114.00	5.50	1.01	0.96							
Van den Broeke &	10 athletic throwers	M-mode, 2-D echo				165.00	5.30	1.07	1.05							
Fagard 1988 [88]	10 controls	M-mode, 2-D echo				131.00	5.10		1.05							
Urhausen et al. 1989 [84]	14 steroid-using body builders	M-mode, 2-D echo				111.00	5.50	1.26*	1.25*							
	7 steroid-free body builders					102.00	5.70	1.16	1.03							
Longhurst et al. 1980	17 competitive weight lifters	M-mode, 2-D echo				91.00*	5.40	0.93	0.95*							
[40a]	7 recreational weight lifters					86.00*	5.20	0.97*	0.85							
	10 controls					72.00	5.40	0.83	0.82							
Pellicia et al. 1991 [60]	9 weight lifters	M-mode, 2-D echo				91.00	5.50*	1.00	1.00							
	7 controls					100.00	5.30	1.04	1.04							
Fagard 1996 [24]	100 strength athletes	M-mode, 2-D,				198.00*	5.30	0.95*	1.03*	0.37*						
	80 controls	Doppler echo				159.00	5.20	0.84	0.89	0.33						
Pluim et al. 2000 [65]	413 endurance athletes	M-mode, 2-D,			249.00**		5.37**	1.03*	1.05*					2.20	68.80	34.40
	494 combined endurance and strength athletes	Dopplerecho			288.00***		5.62***	1.10*	1.13**					1.89	66.10	34.70
	544 strength athletes				267.00**		5.21**	1.10*	1.18**					2.11	66.30	35.70
	813 controls				174.00		4.96	0.88	0.88					1.84	67.20	34.40
Wermstedt et al. 2002 [19]	10 endurance male athletes	M-mode, 2-D,	66.00**	VO_2 max vs.	210.00**	208.00**	0.57*	1.03	1.06*		191.00*					
	8 strength male athletes	Doppler echo	46.00	LVM, LVID, SV	163.00	161.00	0.53	1.03	1.04		147.00					
	9 controls		46.00		144.00	161.00	0.53	0.94	1.00		150.00					
	10 endurance female athletes		58.00**		120.00	155.00*	0.52*	0.91*	0.94*		138.00					
	10 strength female athletes		42.00		110.00	129.00	0.49	0.79	0.86		120.00					
	10 controls		40.00		107.00	132.99	0.49	0.80	0.84		117.00					

*p < 0.05, **p < 0.01, ***p < 0.001: compared with controls

Table 3. Review of echocardiography Doppler tissue imaging (DTI) studies of athletes and matched untrained controls.
Am: DTI peak velocity of atrial diastolic wave; Em: DTI peak velocity of early diastolic wave in DT; Sm: DTI peak velocity of systolic wave; other abbreviations see Table 1.

Tabelle 3. Literaturübersicht zu echokardiographischen Gewebe-Doppler-Analysen (DTI) bei Athleten und normalen untrainierten Kontrollen.
Am: Gewebe-Doppler-Spitzengeschwindigkeit zum Zeitpunkt der atrialen Füllungswelle; Em = Gewebe-Doppler-Spitzengeschwindigkeit zum Zeitpunkt der frühen Diastole; Sm: Gewebe-Doppler-Spitzengeschwindigkeit in der Systole; übrige Abkürzungen s. Tabelle 1.

Authors	Subjects	Methods	LVM index	LVID	PWT	IVST	h/R	LVV	E	A	E/A	Sm	Em	Am	Em/Am	EF	SF
Caso et al. 2000 [8]	20 highly competitive water polo players	DTI	120,40***	5.05*	0.99*	1.04**			77.00	46.00	1.98**	8.00	13.00	6.00	2.32*		38.20*
	20 controls	2-D, Doppler echo	82.60	4.73	0.84	0.89			77.00	50.00	1.54	8.00	13.00	7.00	1.84		34.70
Vinereanu et al. 2001 [90]	30 endurance athletes	DTI	193.00*	5.50*	1.20*	1.30*			81.00	58.00	1.39	11.20	14.50				
	20 controls	2-D, Doppler echo	177.00	5.00	0.90	1.00			79.00	58.00	1.36	10.70	13.70				
D'Andrea et al. 2003 [14]	32 competitive endurance athletes	DTI	93.10***	5.64	0.94*	0.98**	0.38***		98.00**	46.00	2.20***	17.00	26.00**	9.00	2.30***		45.70
	26 strength-trained athletes	2-D, Doppler echo,	158.60	4.82	1.18	1.23	0.46		77.00	48.00	1.50	16.00	17.00	10.00	1.70		46.10
Nottin et al. 2004 [51]	12 endurance-trained adult men	DTI	118.10*	6.07*	1.40*	1.42*			88.40*	43.40*	2.08*		16.10**	8.10**	2.20**	65.00	
	13 age-matched sedentary controls	2-D, Doppler echo	89.10	5.50	1.22	1.22			81.40	52.80	1.56		14.30	6.30	1.70	63.20	
	12 endurance-trained boys		86.40	5.79	1.14	1.28			101.50*	49.60	2.12*		17.40**	6.70	2.60**	66.80	
	11 controls		75.60	5.40	1.14	1.22			90.50	54.70	1.70		15.20	6.60	2.10	66.00	
Pelà et al. 2004 [55]	20 athletes	DTI	150.00***	5.50***	1.07***	1.14***	0.41**				1.96*	12.20				65.00	37.00
	15 controls	2-D, Doppler echo	94.00	4.90	0.82	0.86	0.37				1.75	10.30				65.00	37.00
King et al. 2005 [38]	17 international rowers	DTI	188.90**	5.60**	1.30**	1.13**			65.50*	36.6	1.70*	19.20**		7.10**		64.00	
	30 controls	2-D, Doppler echo	101.96	4.80	1.00	0.89			72.80	40.01	1.50	17.90		6.60		64.00	

*p < 0.05, **p < 0.01, ***p < 0.001: compared with controls

and the sports more often concerned were cycling (49%), ice hockey (42%), basketball (40%) rugby (39%), canoeing (39%), and rowing (34%) [56]. The largest LVIDs reported in this study were 66 mm for a female and 70 mm for a male athlete. However, the impact of large body area is notable and it may be more useful to index all values by body area or other anthropometric parameters.

Trained children have been scarcely studied, and data show that endurance training induces, first, an enlargement of the left ventricle which can be followed by an increase in wall thickness only in postpubescent boys [51, 62].

Transverse dimension of left atrium (LA) measured with M-mode echocardiography is also increased in athletes (mean increase 16%) as compared to controls [83]. Very recently, it has been shown that LA dimension ≥ 40 mm was observed in 20% of athletes with upper limits of 45 mm and 50 mm in women and men, respectively [59]. Enlarged LA dilatation was mainly observed in dynamic or mixed sports such as cycling, canoeing, and rowing. LA enlargement was explained for a large part by LV dilatation (r^2 = 0.53) and minimally by body area [59].

Because of its complex shape and the effects of position on echocardiographic measurements, right cardiac cavities have been less studied, and mainly in endurance athletes [21, 32, 83]. Both right ventricle (RV) and right atrium are slightly increased in athletes in comparison with controls [30, 32, 75, 82]. LV and RV mass are similarly increased (36% and 37%, respectively) in athletes in comparison with controls.

Myocardial Wall Thickness

As shown in Tables 1 to 3, in trained subjects, both interventricular septum (IVS) and posterior LV free wall (PW) thickness are 15–20% greater than values observed in age- and sex-matched untrained people [24, 50, 52, 65]. However, athletes' values are most often within the normal range [83]. Indeed, IVS and PW thickness exceed the generally accepted upper limits of normal (12 mm) in 1.1% and 0.3%, respectively [56]. LV wall thickness values ranging between 13 and 16 mm may be observed in some athletes without any pathologic findings and particularly in rowing, triathlon, cycling or in older athletes who have trained regularly for many years [60, 76]. In female athletes, LV wall thickness is mostly ≤ 11 mm [60]; exceptionally, a value of 12 mm has been reported [67].

The septal-to-posterior wall ratio is mainly < 1.3, and asymmetric cardiac hypertrophy is not usual in athletes. The wall thickness/LV radius ratio is used to define eccentric or concentric cardiac hypertrophy (Tables 1 to 3). For example in a large group of triath-

letes (n = 235), concentric and eccentric myocardial remodeling have been reported in only 2% and 4%, respectively [83]. Right ventricular free wall thickness is also significantly increased in athletes [32]. Classically, myocardial wall thickness is related to the pressure load imposed by exercise [34, 36]. However, multivariate analysis showed that type of sport, body surface area, age and sex, as well as LV diameter are independently related to wall thickness [83].

Left Ventricular Mass

Because of both cavity dilatation and wall thickness hypertrophy, LV mass is increased in most of highly trained athletes (Tables 1 to 3). Indeed, echocardiographically calculated LV mass with M-mode parameters shows that LV mass is, on average, 45–50% greater than in matched untrained subjects [83]. LV mass normalized by body surface area, height or weight is always higher in athletes than in controls, but exceeds normal limits only in 9% of male and 7% of female athletes [56]. An acceptable upper limit of 3.5 g/kg body weight (normal range 2 g/kg body weight) has been proposed [19].

LV shape alterations can be studied only with the two-dimensional echocardiography method. By this method, physical training cardiac hypertrophy shows marked differences with the pathologic one. Briefly, trained heart is characterized by an LV elongated shape associated with a marked shortening of its longitudinal axis [19]. This shape alteration may explain the fact that classic M-mode methods of LV mass calculations often overestimate the values calculated with cardiac magnetic resonance. Thus, in athletes the use of other LV mass formulas, such as that proposed by Dickhuth et al., is recommended [19, 73].

The LV/RV mass ratio studied with magnetic resonance imaging is not altered in athletes in comparison with untrained people [27, 73]. Cardiac hypertrophy induced by physical training can vary during the competitive season as it has been shown in cyclists [23], and a decrease in wall thickness without marked decrease in LV dilatation has been reported after 3 months of physical deconditioning [60].

Sports-Specific Athlete's Heart Echocardiographic Adaptations

In case of physiological cardiac hypertrophy, according to Laplace's law and in order to reduce the increased wall tension induced by LV dilatation, LV wall thickness must increase. Thus, in accordance with this theory, the greatest increase in LV wall thickness must occur in the largest dilated LV. However, Spirito et al. have shown that if most endurance sports are associated with a large LV diastolic diameter, they are not always associated with the same increase in wall thickness [80]. Thus, other factors and mainly training specificity may also be involved in these adaptations.

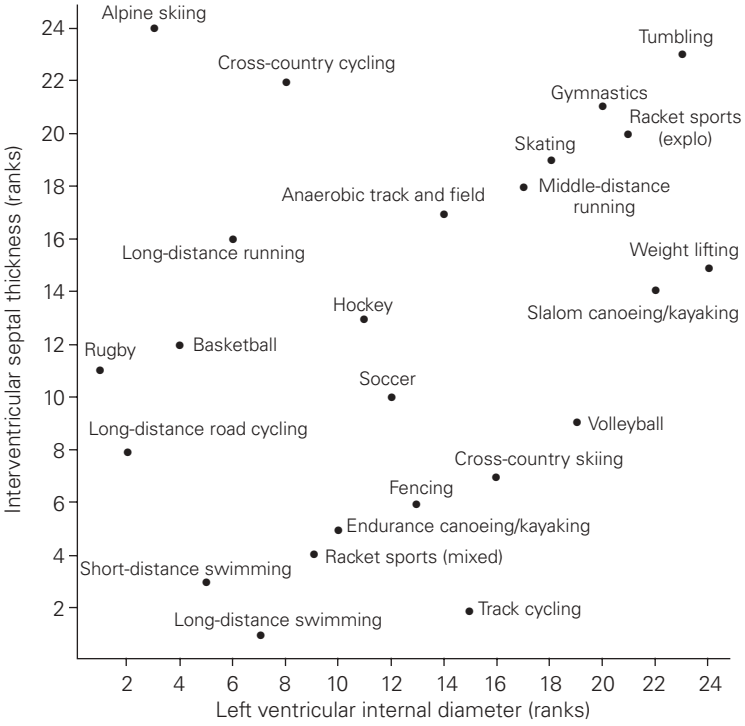
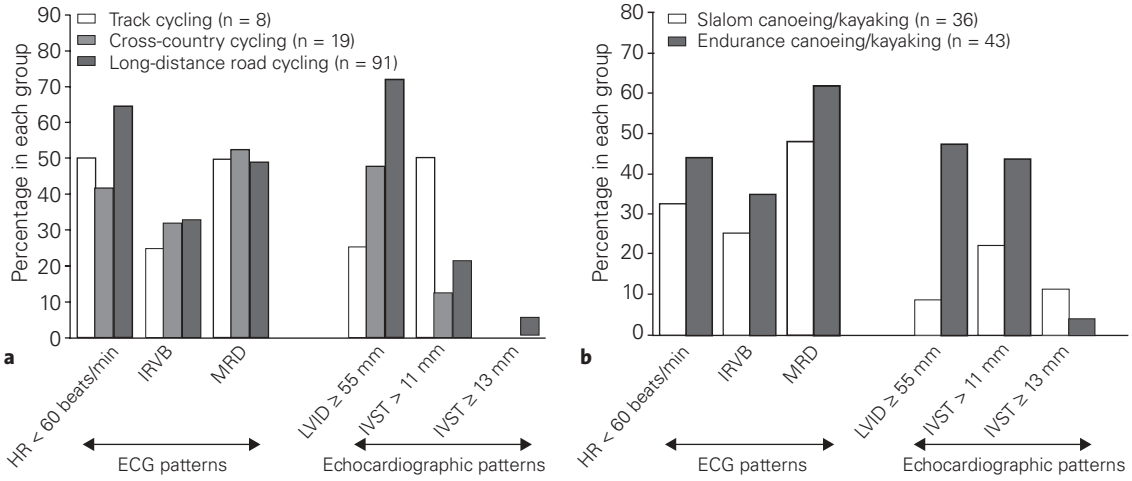


Figure 2. Interventricular septal thickness plotted against left ventricular internal diameter in 24 sports of 916 French elite athletes. Data is expressed by its rank (lower rank corresponds to larger cardiac dimension). Anaerobic track and field: short-distance running, jumping, and throwing. Racket sports (explosive): squash, table tennis, badminton. Racket sports (mixed): tennis.

Abbildung 2. Darstellung der Beziehung zwischen linksventrikulärem enddiastolischem Durchmesser und Dicke des interventrikulären Septums bei 916 französischen Spitzenathleten. Aufgeführt sind 24 Sportarten; die Arten sind ausgedrückt in Bezug auf die Rangfolge, d.h., niedriger Rang bedeutet großen Herzdurchmesser. Anaerobes Feld: Kurzstreckenlauf, Springen und Werfen. Explosiver Sport: Squash, Tischtennis, Badminton. Gemischte Sportart: Tennis.

Sports-specific adaptive cardiac structural changes are somewhat controversial. The two different classic morphological forms of athlete's heart, strength-trained and endurance-trained, previously proposed [49] are not really confirmed [24, 31, 65, 87]. Briefly, and according to the theory by Morganroth et al., athletes involved in sports with a high dynamic component are presumed to demonstrate eccentric LV hypertrophy, with a great LVID and a proportional increase in wall thickness, in response to volume overload, whereas strength-trained athletes are presumed to develop concentric LV hypertrophy, with unchanged LVID and increased wall thickness, in response to pressure overload.

In accordance with recent data (reviews in [24, 31, 65, 87]), it actually appears that most of highly trained



Figures 3a and 3b. Electrocardiographic and echocardiographic patterns in a) cycling and b) canoeing/kayaking: distinction between fields. Data is expressed by its percentage observed in each group. HR: heart rate; IRVD: incomplete right ventricular block; MRD: minor repolarization disturbances (negative or diphasic T-wave in V_1); LVID: left ventricular internal diameter; IVST: interventricular septal thickness. Concerning LVID $p < 0.01$ between subgroups in cycling and in canoeing/kayaking.

Abbildungen 3a und 3b. Elektrokardiographische und echokardiographische Parameter bei a) Radfahrern und b) Kanuten/Kajakfahrern. Die Daten drücken die Prozentveränderung in jeder Gruppe aus. HR: Herzfrequenz; IRVB: inkompletter Rechtsschenkelblock; MRD: geringe Repolarisationsstörungen (negative oder diphasische T-Welle in V_1); LVID: linksventrikulärer enddiastolischer Durchmesser; IVST: Dicke des interventrikulären Septums. LVID: $p < ,01$ zwischen den Untergruppen der Radfahrer und Kanuten/Kajakfahrer.

athletes develop an LV fair combination of cavity dilatation and increased wall thickness. However, ventricular dilatation slightly predominates in endurance athletes, whereas the increased wall thickness slightly predominates in static ones [83]. The association of dynamic and static training sessions, whatever the sport practiced, may partly explain these results.

The first echocardiographic studies performed in strength-trained athletes reported an increased LV wall thickness similar to the concentric hypertrophy observed in chronic pressure overload [71, 81]. However, data from more recent studies does not confirm this hypothesis. The results of the meta-analysis by Pluim et al. [65] show that the strength-trained weight lifters, power lifters, body builders, throwers and wrestlers, who are considered to develop pure concentric LV hypertrophy, demonstrated in fact an increase in both absolute and relative wall thickness and a significant enlargement in LVID. Moreover, no significant difference in LV diameter has been reported between strength athletes and heavy controls [24, 40a, 78]. Thus, it is important to keep in mind that LVID is not decreased in resistance-trained athletes, in contrast to findings in pathologic pressure load and various forms of cardiomyopathy [31, 84]. Concerning LV wall thickness, the clearly increased absolute values reported in comparison with untrained subjects [40a, 71, 78] disappeared after indexation by body surface area [24, 65, 87].

Thus, correction for body dimensions and/or for lean body mass appears as a major point in the assessment of echocardiographic measurements, particularly in strength-trained athletes [16a, 40a, 86, 87, 91]. The relatively minor differences observed in indexed LV dimensions between strength-trained athletes and matched controls might be due to a better aerobic capacity in the former.

However, besides this general description, it is possible to observe some sports-specific echocardiographic adaptations. Spirito et al. [80] have studied echocardiography parameters of athletes from 27 different sports. They classify sports according to their impact on LVID dimensions and LV wall thickness. Rowing was ranked first according to the calculated effect in LV wall thickness and seventh according to the calculated effect on LVID. For comparison, using the same method cycling was ranked second and first, respectively. Endurance cycling and rowing are mixed sports (Figure 1) which have a great impact on LV cavity dimensions due to dynamic exercise performed by lower body, and also on wall thickness due to the part of static exercise performed by upper body [23, 78, 80, 83].

We have observed nearly the same results in French top-level athletes (Figure 2, from unpublished data). From this data we have more specifically studied cycling and canoeing which are classified as both high-intensity static and dynamic sports (Figure 1). First, we have noted that sports-induced cardiac hypertrophy was quite different in these two sports [2].

Eccentric cardiac hypertrophy was observed in cyclists and predominantly concentric cardiac hypertrophy was noted in canoeists. The same results have previously been reported in canoeists [28]. Despite the same classification (Figure 1), cycling and canoeing present hemodynamic specificities. Cycling is mainly a lower-body dynamic exercise with a smaller static work part of the arms. Canoeists are kneeling in their boat and use only a single paddle to progress. Thus, venous return in canoeists during exercise cannot be deeply increased and they quite exclusively use the upper body to perform their sport. Upper-body exercise is well known to induce a higher relative blood pressure than lower-body exercise [5, 47] and a relationship has been reported between exercise blood pressure and LV mass and geometry [34, 36].

Second, we observed that in the same sport, i.e., cycling, cardiac hypertrophy patterns seem quite different according to the metabolic request (Figure 2). Indeed, endurance cyclists (aerobic training) show larger LVID than track cyclists (anaerobic training) who exhibit larger parietal wall thickness. Cross-country cyclists show quite large LVID associated with low parietal wall thickness (Figure 3a). The same differences may be noted between endurance and slalom canoe/kayak practitioners (Figures 2 and 3b). An electrical pattern of athlete's heart has also been well described [67]. Few of these aspects have been studied in relation with cardiac hypertrophy. QT dispersion seems not to be altered in cardiac hypertrophy induced by physical training [95]. Figure 3 (unpublished data) shows both specific electrocardiographic and echocardiographic patterns observed in cycling and canoeing/kayaking athletes in relation with the specificity of training.

Thus, sports-specific cardiac morphological patterns may be observed, even if it is not possible to oppose a strength-trained to an endurance-trained athlete's heart.

Table 4. Correlations between resting echocardiographic parameters and VO_2 max in a group of elite athletes (n = 40) from three different sports (adapted from [2]). A: Doppler peak velocity of LV filling after atrial contraction; AOd: aortic diameter; E: Doppler peak velocity of early LV filling wave; LAd: left atrial diameter; LVd vol: left ventricular volume in diastole; LVIDd: left ventricular internal diameter in diastole; LVIDs: left ventricular internal diameter in systole; LVM: left ventricular mass; LVM/BSA: left ventricular mass related to body surface area; LVs vol: left ventricular volume in systole.

Tabelle 4. Korrelationen zwischen echokardiographischen Parametern und der Sauerstoffaufnahme bei Hochleistungsathleten (n = 40) für drei verschiedene Sportarten (nach [2]).
A: Doppler-Spitzengeschwindigkeit des atrialen Einstroms; AOd: Aortendurchmesser; E: Doppler-Spitzengeschwindigkeit der frühen linksventrikulären Füllung; LAd: linksatrialer Durchmesser; LVd vol: linksventrikuläres diastolisches Volumen; LVIDd: linksventrikulärer enddiastolischer Durchmesser; LVIDs: linksventrikulärer endsystolischer Durchmesser; LVM: linksventrikuläre Muskelmasse; LVM/BSA: linksventrikuläre Muskelmasse, bezogen auf die Körperoberfläche; LVs vol: linksventrikuläres systolisches Volumen.

Parameter	Correlation with VO_2 max	
	r	p
LVIDd (mm)	0.92	0.001
LVIDs (mm)	0.57	0.01
LVd vol (ml)	0.63	0.001
LVs vol (ml)	0.39	0.05
LVM (g)	0.60	0.001
LVM/BSA (g/m²)	0.53	0.001
AOd (mm)	0.39	0.01
LAd (mm)	0.36	0.05
E (cm/s)	0.38	0.05
A (cm/s)	−0.33	0.05

Myocardial Functions

Functional adaptations, which seem to precede structural myocardial adaptations, have been described in trained subjects [18]. However, the question of whether athlete's cardiac hypertrophy is a purely physiological phenomenon or should be considered a risk factor like cardiac hypertrophy induced by pathologic, i.e., hypertrophic cardiomyopathy, hypertension or aortic stenosis, is still under debate [64, 83].

Systolic LV function (Tables 1 to 3) most often expressed as the fractional shortening, is usually normal in athlete's heart whatever the sports practiced [2, 10, 13, 37, 45, 65, 90]. Myocardial tissue Doppler assessment of systolic function has recently confirmed these results [21, 90]. In professional cyclists, however, a decreased resting systolic function has been noted lately [1]. The potentially deleterious myocardial impact of prohibited drugs has been hypothesized [1]. Some studies have also shown that resting right cardiac function is normal in athlete's heart [21].

In trained subjects, despite the increased LV mass and whatever the sports practiced, cardiac diastolic function is normal [2, 13, 23, 28, 45, 70, 72] or increased [10, 12, 36, 43] in comparison with untrained subjects. Data obtained with Doppler tissue imaging confirms these results [21, 51, 90] and shows an improvement in diastolic passive properties of myocardium in endurance athletes [8]. However, it must be kept in mind that some markers of diastolic function (i.e., mitral E/A ratio) are not only related to LV compliance but are also influenced by other factors such as HR, preload, and afterload, which are all altered in trained subjects [21, 33, 48]. Thus, in comparison with pathologic cardiac hypertrophy [77], the physiological cardiac hypertrophy of athlete's heart is not accompanied by disturbances of resting diastolic parameters [24, 65, 87]. Improvement of LV diastolic

Table 5. Correlations between resting echocardiographic parameters and absolute VO_2 max (l/min) in a group of elite athletes according to sport's specificity (adapted from [2]).
LAd: left atrial diameter; LVIDs: left ventricular internal diameter in systole. LVs vol: left ventricle volume in systole; PWTs: left ventricular posterior wall thickness in systole; SF: shortening fraction.

Tabelle 5. Korrelationen zwischen echokardiographischen Parametern in Ruhe und absoluter Sauerstoffaufnahme in einer Gruppe von Spitzenathleten in Abhängigkeit von der Sportart (nach [2]).
LAd: linksatrialer Durchmesser; LVIDs: linksventrikulärer endsystolischer Durchmesser; LVs vol: linksventrikuläres systolisches Volumen; PWTs: linksventrikuläre Hinterwanddicke in Systole; SF: Verkürzungsfraktion.

Group	Parameter	Correlation with VO_2 max	
		r	p
Tumblers (n = 12)	SF (%)	–0.61	0.05
	LVIDs (mm)	0.65	0.05
	LVs vol (ml)	0.64	0.01
Canoeists (n = 12)	PWTs (mm)	0.71	0.01
	LVs vol (ml)	0.65	0.05
	LAd (mm)	0.66	0.01
Cyclists (n = 16)	LAd (mm)	0.59	0.05

function favors an appropriate ventricular filling during exercise, particularly when diastolic period is reduced, due to increased HR [16, 33, 40].

Concerning athlete's heart perfusion, experimental and humans studies show that the increased LV mass described in athletes is associated with an increased size and with an improved response to nitroglycerin of the proximal coronary arteries [29, 61].

On the whole, we can therefore conclude that when echocardiographic variables used to assess systolic and diastolic performances are load-independent, resting intrinsic myocardial contractility is not altered in athlete's heart. Other imaging methods, such as ultrasonic integrated backscatter tissue characterization [15] and magnetic resonance imaging [63, 64], confirm that LV and RV [73] hypertrophy induced by physical training are physiological cardiac adaptations.

Beneficial Effects of Athlete's Heart Adaptations on Physical Performance

Athlete's heart adaptations may be mainly involved in endurance performance. Thus, potential correlations between myocardial morphological and/or resting functional parameters and aerobic power have been studied. Correlations between resting echocardiographic or magnetic resonance imaging parameters and VO_2 max have been described in endurance-trained male and female athletes [46, 52, 53, 66, 69, 70, 94]. Moreover, we recently showed significant relationships between VO_2 max and echocardiographic morphological and functional parameters in a group of top-level athletes from various sports (Table 4) [2].

First studies show that trained subjects with the largest resting cardiac dimensions determined echocardiographically present higher VO_2 max values [4, 53, 94]. Several relationships between VO_2 max and LV parameters have been described particularly in trained individuals (Tables 2 and 3). They concern both morphological and functional adaptations which may be linked. For example, in sedentary and endurance-trained males and females (see Table 4), VO_2 max is positively correlated with LV diastolic morphological (diameter and volume) parameters, LV mass, and functional parameters such as mitral peak E-wave velocity, and negatively with peak A-wave velocity [2, 40, 52, 69, 70, 89]. These last results confirm the relationship between resting LV filling parameters and maximal aerobic performance [48]. Some studies also show a correlation between systolic parameters, such as LV diameter and SV and VO_2 max [2, 91]. Magnetic resonance imaging data confirm these results in male [46, 91] and female athletes [66, 94].

When we compared cyclists, canoeists and tumblers [2], we noted that the LV/VO_2 max ratio was higher in canoeists than in cyclists and tumblers; however, it was lesser than the upper level of 80 g/l proposed in trained athletes [86]. In addition, we have shown that the echocardiographic parameters concerned (Table 5) depend on the sport practiced [2]. The same results have been reported when studying different athletic populations [34, 66, 69] and comparing sedentary subjects and trained people [89, 93]. This data may be explained, at least partly, by the different cardiac hypertrophy patterns observed in the three trained groups (Figure 2). Another possible explanation is that individual cardiac adaptations are nearly the same in athletes with comparable levels of training and performance [1]. Thus, it is difficult to show a significant correlation between physiological variables and physical performance in a homogeneous group. Lastly, it is well known that cardiovascular adaptations do not explain all training VO_2 max adaptations [5, 17, 18, 39]. This last point is well illustrated by the significant increase of LV diameters and wall thickness observed during intensive training periods in rowers without any significant alteration in maximal aerobic capacity [93].

The main limit of these results is the comparison between resting echocardiography data and exercise parameters. Thus, further research is needed, particularly concerning the relationships between exercise echocardiography and metabolic parameters in well-trained subjects.

Conclusion

High level of chronic physical training may induce the development of the so-called athlete's heart characterized by a moderate and harmonious hypertrophy-dilatation of the four cardiac chambers associated with quite normal or enhanced functions. In endurance athletes these adaptations should play a main role in the physical performance. Globally, it seems impossible to clearly separate a strength-trained from an endurance-trained athlete's heart. However, some slight differences in echocardiographic patterns may be observed according to the training specificities.

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