JACC Vol 7, No 1
January 1986 190–203

Structural Features of the Athlete Heart as Defined by Echocardiography

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The morphologic concepts of the "athlete heart" have been enhanced and clarified over the last 10 years by virtue of M-mode echocardiographic studies performed on more than 1,000 competitive athletes. Long-term athletic training produces relatively mild but predictable alterations in cardiac structure that result in an increase in calculated left ventricular mass. This increase in mass observed in highly trained athletes is due to a mild increase in either transverse end-diastolic dimension of the left ventricle or left ventricular wall thickness, or both. Cardiac dimensions in athletes compared with matched control subjects show increases of about 10% for left ventricular end-diastolic dimension, about 15 to 20% for wall thickness and about 45% for calculated left ventricular mass.

Furthermore, there is evidence that the modest degree of "physiologic" left ventricular hypertrophy (both the cavity dilation and wall thickening) observed in athletes is dynamic in nature, that is, it may develop rapidly within weeks or months after the initiation of vigorous conditioning and may be reversed in a similar time period after the cessation of training. Several echocardiographic studies also suggest that the precise alterations in cardiac structure associated with training may differ depending on the type of athletic activity undertaken (that is, whether training is primarily dynamic [isotonic] or static [isometric]).

Although the ventricular septal to free wall thickness ratio (on M-mode echocardiogram) is almost always within normal limits (<1.3), occasionally an athlete will show mild asymmetric thickening of the anterior basal septum (usually 13 to 15 mm). This circumstance may mimic certain pathologic conditions characterized by primary left ventricular hypertrophy such as nonobstructive hypertrophic cardiomyopathy.

The long-term significance of increased left ventricular mass in trained athletes has not been conclusively defined. However, there is no evidence at this time suggesting that this form of hypertrophy is itself deleterious to the athlete or predisposes to (or prevents) the natural occurrence of cardiovascular disease later in life.

(J Am Coll Cardiol 1986;7:190–203)

The "athlete heart" is a term that has been used for many years by physicians and laymen to describe the cardiovascular effects of long-term conditioning observed in highly trained competitive athletes (1–4). Physiologic responses to prolonged training include increased stroke volume and decreased heart rate under resting conditions; with long-term training there is enhanced extraction of oxygen by peripheral skeletal muscle and reduced blood lactate levels associated with increased maximal arteriovenous oxygen difference and increased maximal oxygen consumption (5–11). These cardiovascular changes are probably produced by a complex interaction of central and peripheral mechanisms operating at structural, as well as biochemical, metabolic and neural levels. The clinical manifestations of the "athlete heart syndrome," as originally described, include sinus bradycardia at rest, a soft systolic murmur, audible third and fourth heart sounds, cardiomegaly on chest X-ray film and a variety of alterations in the scalar electrocardiogram (1).

Initially, heart enlargement in athletes was identified largely by physical examination and chest radiography, or inferred from electrocardiographic patterns suggestive of left ventricular enlargement or hypertrophy (1–19). The emergence of M-mode echocardiography for cardiac diagnosis in the early 1970s permitted a more precise definition of the alterations in cardiac dimensions and function induced by chronic conditioning. Echocardiography is ideal for this purpose because it allows a noninvasive quantitative assessment of cardiac dimensions to be made in individuals who are in good health and have no clinical evidence of cardiac disease and therefore are not candidates for invasive testing.

For the past 10 years a number of echocardiographic

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Manuscript received July 30, 1985; revised manuscript received September 18, 1985, accepted September 23, 1985

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0735-1097/86/$3.50

studies (2,3,10,20–59) have been performed in a variety of athletic populations. Therefore, there now exists a large body of echocardiographic data encompassing studies in more than 1,000 competitive athletes. The purpose of this report is to review and cohesively summarize these data and characterize what is known of the morphologic features of the "athlete heart."

General Comments

Since 1975, 28 echocardiographic studies describing cardiac dimensions in trained athletes have been reported (20–49) (Table 2). Most are investigations of relatively small groups of competitive athletes (19 of the 28 are studies of <25 athletes). Most athletes studied are young adults (usually 18 to 30 years of age, average 23) and primarily male (85%). These investigations are largely cross-sectional in design; that is, performed at one point in time, usually at peak level of conditioning.

The majority of echocardiographic studies were carried out in endurance athletes (usually runners), although a variety of diverse athletic activities were investigated. Some studies compared cardiac dimensions in athletes participating in primarily isotonic or dynamic forms of conditioning (such as running or swimming) with those in athletes performing isometric or static training (such as weight-lifting, shot-putting, wrestling or gymnastics). However, only rarely is athletic conditioning purely isotonic or isometric; most physical activities have both static and dynamic components, although one or the other may be dominant.

It is difficult to standardize and compare precisely the cardiac dimensional data in available published studies in athletes. This is due to differences in the experience and expertise of the echocardiographic laboratories, the variety of sports participated in, the size of the study populations, the age, sex and genetic or racial characteristics of the athletes and the frequency, intensity and duration of the conditioning regimen. Because of this diversity, the response of individual athletes to training is not uniform and there may be considerable variability in cardiac dimensions among subjects. Nevertheless, even with these inherent limitations, the findings of the large number of published echocardiographic studies in athletes are remarkably similar in many respects and provide a reasonably homogeneous perspective regarding the structural and functional consequences of athletic conditioning to the heart.

Echocardiographic studies of athletes are designed to compare cardiac dimensions (under basal conditions) in a highly trained athletic group during a period of active conditioning with those of an age- and sex-matched group of sedentary (nonathletic) subjects. Values for cardiac dimensions in groups of athletes and their matched control subjects usually show substantial overlap, and the two groups fail to demonstrate a bimodal distribution. Values for dimensions in trained athletes are usually only slightly increased compared with those in control subjects and rarely exceed the normal range (60,61). For example, the average difference in the measurement of wall thickness between athletes and control subjects is less than 2 mm and therefore within the methodologic error for M-mode echocardiography. Nevertheless, differences between athlete and control dimensions usually achieve statistical significance; the consistency with which a large number of studies report these differences strongly suggest that they are valid and not due to chance alone.

Finally, it should also be pointed out that M-mode echocardiography allows assessment of only a small portion of the heart and makes the implicit assumption that the limited area visualized with the M-mode beam is representative of overall cardiac structure and function. Although this may be a reasonable assumption to make in studying a young athletic population without heart disease, there have been

Table 1. Ventricular Dimensions in Athletes and Nonathletes as Assessed by Echocardiography in Published Studies*

Echocardiographic Variable	Nonathlete Controls		Athletes		
	Mean Value	No. of Subjects	Mean Value	No. of Subjects	Percent Difference§
Ventricular septal thickness (mm)†	9.1	313	10.4	461	+14.3
Posterior free wall thickness (mm)†	9.0	439	10.7	740	+18.9
LV end-diastolic dimension (mm)‡	49.1	394	53.9	701	+ 9.8
Estimated LV mass (g)‡	175	252	256	381	+46.3
RV internal transverse dimension (mm)†	17.7	146	22.0	147	+24.3

*From 25 studies cited as references (20–26, 28–49); data given only for male athletes because relatively few female athletes have been reported (27,28,37,42). †Measurements obtained at end-diastole ‡An echocardiographic estimate of left ventricular mass derived from measured values of ventricular wall thickness and internal transverse cavity dimension, utilizing the formula of Troy et al. (62) or of Devereux and Reichek (63) §Percent change of the dimension in athletes, as compared with nonathlete control subjects. Controls = control subjects; LV = left ventricular; RV = right ventricular.

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Table 2. Summary of Previous M-Mode Echocardiographic Studies of Cardiac Dimensions in Competitive Athletes‡

Ref. No.	First Author	Year	No. of Controls Studied	No. of Athletes Studied	Athletes		Sports Participated in	Echocardiographic Dimensions in Athletes	
					Age(yr)	Sex		$LVID_d$ (mm)	$LVID_d$ (↑ over controls)
(20)	Morganroth	1975	16	56	(19)	M	ER, swimming, wrestling, shot putting	50 to 61(55)-IT 43 to 52(47)-IM	+ 0
(21)	Roeske	1976	10	10	21 to 31(24)	M	Basketball (elite)	(53)	+
(22)	Zoneraich	1977	20	12	(38)	—	ER, marathon running	(55)	+
(23)	Gilbert	1977	26	20	20 to 39(28)	M	ER	42 to 59(50)	0
(24)	Laurenceau	1977	44	166	(23)	—	Wide variety (elite)	(53)	+
(25)	Underwood	1977	18	20	—	—	ER (including elite)	42 to 56(51)*	+
(26)	Parker	1978	12	11	19 to 40	M	ER	50 to 63 (57)	+
(27)	Zeldis	1978	25	10	18 to 24(20)	F	Field hockey	38 to 53(47)	+
(28)	Ehsani	1978	0	14	17 to 22	13M,1F	ER, swimming	(52)	+
(29)	Ikaheimo	1979	13	22	18 to 36(27)	M	ER, sprinting†	45 to 64(54)	+
(30)	Blair	1980	20	20	20 to 33(27)	M	ER, rowing (elite), bicycling	43 to 63(54)	+
(31)	Nishimura	1980	35	60	20 to 49	M	Bicycling	48 to 62(54)§	+
(32)	Heath	1981	8	15	50 to 72(59)	M	ER (masters)	(54)[b]	+
				11	18 to 27(22)	M	ER (young athletes)	(50)	+
(33) [(34)]	Keul [Dickhuth]	1981	—	90	—	—	ER, bicycling, skiing rowing, weight lifting, shotputting	(53)-IT (50)-IM	− −
(35)	Paulsen	1981	10	8	21 to 44(29)	M	ER, marathon running	(56)	+
(36)	Bekaert	1981	11	14	21 to 29(25)	—	Bicycling	(59)	+
(37)	Mumford	1981	19	19	18 to 38(23)	10M,9F	ER	—	+
(38)	Wieling	1981	17	23‖	(21)	M	Rowing	(57)	+
(39)	Longhurst	1981	24	29	(26)	M	ER, weight lifting	(56)-ER	+
(40)	Longhurst	1980						(54)-Weight lifting	0
(41)	Snoeckx	1982	17	45	(26)	M	ER, bicycling, weight lifting	(56)-IT (51)-IM	+ 0
(42)	Rost	1982	23	155	—	M	ER, weight lifting, rowing, swimming	(59)	+
			0	120	—	F	ER, rowing, swimming	(51)	−
(43)	Fagard	1983	12	12	17 to 35	M	Bicycling	(55)	+
(44)	Brown	1983	9	18	(26)	M	Weight lifting	(55)	0
(45)	Sugishita	1983	25	48	18 to 21(20)	M	ER, judo	(57)-ER (54)-Judo	+ +
(46)	Spirito	1983	48	75	18 to 39(21)	M	Football, weight lifting (elite), wrestling	39 to 63(55)	+
(47)	Fagard	1984	24	24	18 to 40	M	ER, bicycling	(55)-Bicycling (52)-ER	+ +
(48)	Granger	1985	12	11	22 to 40	M	ER, marathon running	(53)	0
(49)	Colan	1985	22	22	17 to 30(22)	—	Swimming, power-lifting	(54)-Swimming (54)-Weight lifting	+ +

*Values given are for the elite athletes, but no significant differences were identified between the elite and the nonelite competitive athletes. †Endurance runners significantly exceeded sprinters with regard to ventricular septal and posterior wall thickness, the derived estimate of left ventricular mass and left atrial size. ‡Limited to analyses of adult competitive athletes studied with echocardiography during periods of active training; does not include values obtained after detraining, or those published reports of nonathletes undergoing standardized training programs, studies in which left ventricular cavity dimensions were reported only as estimates of left ventricular volumes, or when values are presented only as corrected for body surface area. §Values given are for 20 to 29 year old bicyclists; left ventricular internal dimension in diastole in these subjects was similar to that in both 30 to 39 and 40 to 49 year old bicyclists; however, septal and posterior wall thickness, derived left ventricular mass and left atrial size were increased in the 40 to 49 year olds. ‖Values given are only for the 14 senior oarsmen. ¶Posterior wall thickness and left ventricular mass were significantly increased over control subjects only when those values were corrected for body surface area. **Values for left ventricular mass are estimates derived from measurements of wall thickness and transverse cavity dimension, utilizing the formula of Troy et al. (62) or of Devereux and Reichek (63). ††Values for left ventricular

Table 2. *(continued)*

Echocardiographic Dimensions in Athletes

Ref. No.	VS Thickness(mm)	VS (↑ over controls)	PW Thickness(mm)	PW (↑ over controls)	RVID ↑ (mm)	RVID (↑ over controls)	LV Mass[b] (↑ over controls)	LA in mm (↑ over controls)	EF
(20)	10 to 11(10)-IT	0	10 to 12(11)-IT	0	—	–	+	0	—
	10 to 15(13)-IM	+	11 to 16(13)-IM	+	—	–	+	0	—
(21)	(13)	+	(11)	+	(20)	+	+	–	NL
(22)	—	–	(10)	+	(20)	+	+	+	NL
(23)	8 to 13(10)	0	8 to 14(10)	+	15 to 26(21)	+	+	0	NL
(24)	—	–	(10)	+	—	–	+	–	—
(25)	9 to 16(11)*	0	7 to 11(10)*	0	12 to 26(18)	+	+	+	NL
(26)	10 to 13(11)	+	10 to 12(11)	+	—	–	+	–	NL
(27)	(8)	0	(10)	0	(20)	0	+	+	NL
(28)	—	–	(10)	+	—	–	+	–	NL
(29)	8 to 14(11)	+	8 to 13(11)	+	—	–	+	+ (Only ER)	NL
(30)	—	–	8 to 12(11)	–	11 to 33(26)	+	–††	+	↓9 of 20
(31)	(9)§	0	(8)§	0	—	–	+	0	NL‡‡
(32)	(10)	0	(9)	0	—	–	+[b]	–	NL
	(11)	0	(9)	0	—	–	+	–	NL
(33)	(11)-IT	–	(9)-IT	–	—	–	–	–	NL
[(34)]	(12)-IM	–	(10)-IM	–	—	–	–	–	NL
(35)	—	–	(10)	+	—	–	+	–	NL
(36)	(12)	+	(11)	+	—	–	–††	+	NL
(37)	—	–	10 to 15(11)	+	11 to 28(20)	0	+	–	NL
(38)	(11)	+	10	+	(27)	+	–††	+	NL
(39)	(9)-ER	0	(9)-ER	0	—	–	+	–	—
(40)	(9)-Weight lifting	0	(9)-Weight lifting	+	—	–	+	–	—
(41)	(11)-IT	+	(11)-IT	+	—	–	+	0	—
	(9)-IM	0	(9)-IM	+	—	–	0	0	—
(42)	—	+	(11)	+	—	–	–	–	—
	—	–	(9)	–	—	–	–	–	—
(43)	(12)	+	(13)	+	—	–	–††	–	NL
(44)	(9)	0	(9)	+¶	—	–	+¶	–	—
(45)	—	–	(11)-ER	+	—	–	–††	–	—
	—	–	(14)-Judo	+	—	–	–	–	
(46)	8 to 15(11)	+	8 to 14(11)	+	—	–	+	–	—
(47)	(12)-Bicycling	+	(12)-Bicycling[a]	+	—	–	–	–	NL(%FS)
	(11)-ER	+	(10)-ER	0	—	–	–	–	NL(%FS)
(48)	—	–	(10)	+	—	–	+	–	NL
(49)	—	–	(10)-Swimming	0	—	–	+	–	↑ (%FS)
	—	–	(13)-Weight lifting	+	—	–	+	–	↑ (%FS)

mass are not presented in this report, but mass was likely to be increased judging from the reported wall thicknesses and transverse cavity dimensions. ‡‡Except in 40 to 49 year old bicyclists in whom ejection fraction was decreased. [a]Differences between bicyclists and endurance runners achieved statistical significance only for posterior wall thickness. [b]Masters athletes had significantly larger left ventricular end-diastolic volume index than young athletes; all other comparisons of echocardiographic dimensions between the two groups did not achieve statistical significance. Controls = control subjects; EF = ejection fraction; ER = endurance running; F = female; %FS = percent fractional shortening; IM = isometric exercise; IT = isotonic exercise; LA = left atrium; LV = left ventricle, $LVID_d$ = left ventricular internal (transverse) dimension in diastole; M = male; No. = number; NL = normal; PW = posterior left ventricular free wall; ref. = reference; RVID = right ventricular internal (transverse) dimension in diastole; VS = ventricular septum, + = present; 0 = absent; – = data not available; ( ) = mean value; ↑ = increased; ↓ = decreased.

few correlative two-dimensional and M-mode echocardiographic investigations in athletes (50) to substantiate this premise.

Cardiac Effects of Chronic Conditioning in Athletes: Cardiac Structure and Dimensions

Left ventricular mass. Echocardiographic studies have shown that most highly trained athletes competing in a variety of sports show an increase in calculated left ventricular mass (Tables 1 and 2). Left ventricular mass was significantly greater (average increase 46%) in athletes than in control subjects in each of the 20 studies in which this calculation was reported (Tables 1 and 2). Such echocardiographic estimates of mass are usually derived employing the formula of Troy et al. (62), which incorporates end-diastolic left ventricular transverse dimension and posterior free wall thickness, or that of Devereaux and Reichek (63). The increased left ventricular mass identified in most athletes is not related solely to body size; that is, the "athlete heart" is not larger than normal because the athlete may have a larger than normal body size; cardiac dimensions still differ significantly between athletes and control subjects when normalized for body surface area or weight (9,20,27,36,39,40,46–48). Maximal oxygen consumption, probably the single most objective indicator of a training effect, is also increased in athletes (average 38%) (23,27, 28,30,31,35,36,38,43,47,48).

Left ventricular cavity. The increased left ventricular mass observed in athletes is due in large part to an increased transverse end-diastolic dimension of the left ventricle (Fig. 1); however, this increased dimension (and the inferred increase in volume) does not often exceed the accepted normal limits for adults without heart disease and is relatively mild compared with values commonly encountered in patients with cardiac diseases producing left ventricular cavity dilation. Echocardiographic data obtained from previous studies (Tables 1 and 2) show that the left ventricular end-diastolic transverse cavity dimension in trained athletes is increased by approximately 10% over that in matched sedentary control subjects; this is equivalent to a 33% difference in left ventricular volume. The average reported left ventricular end-diastolic dimension in athletes is almost 54 mm and the maximal dimension is 64 mm; however, it is unusual for the normal "athlete heart" to show a left ventricular

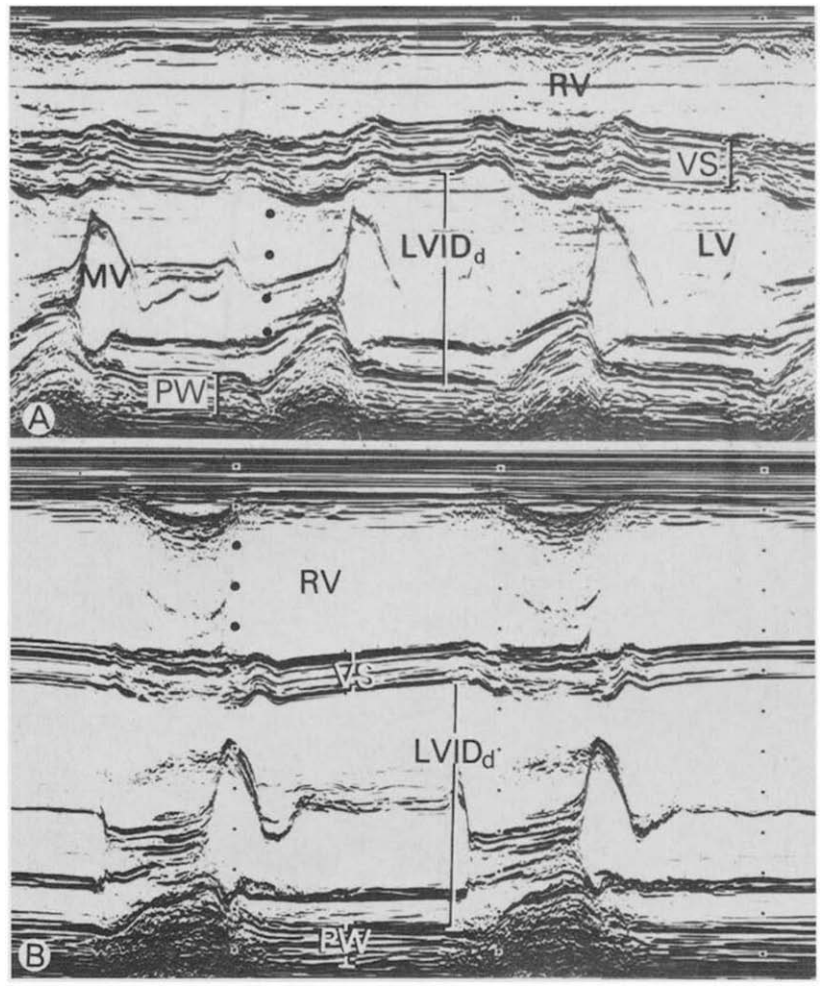

Figure 1. M-mode echocardiograms recorded at the level of the mitral valve (MV) showing cardiac dimensions in two trained athletes. **A**, From a 19 year old male football player showing findings typical of an endurance athlete. The ventricular septum (VS) and posterior left ventricular free wall (PW) are both mildly thickened (12 mm each) and the left ventricular cavity is slightly increased in transverse dimension ($LVID_d$) (56 mm); the right ventricular (RV) cavity is of normal size. LV = left ventricle; MV = mitral valve. **B**, From an 18 year old lacrosse player showing more substantial enlargement of ventricular cavities; the left ventricular transverse end-diastolic dimension ($LVID_d$) is 60 mm and the right ventricular (RV) dimension is 32 mm. In contrast to the athlete shown in **A**, ventricular septal (VS) and posterior (PW) thicknesses are normal (10 mm each). **Calibration dots** are 10 mm apart in both panels.

dimension of more than 60 mm. The most impressive increases in cavity dimension are most consistently identified in endurance athletes; an increase in left ventricular end-diastolic transverse dimension compared with that in control subjects has been reported in 24 to 26 studies of such athletes (Table 2). The only exceptions to this principle are the studies of Gilbert (23) and Granger (48) and their coworkers, in which long-distance runners showed normal left ventricular diastolic dimension. There are few data currently available on cardiac dimensions in older (>40 years of age) nonelite athletes, including marathon runners, although it is our impression that such individuals do not achieve the same degree of cavity enlargement as younger runners.

Left ventricular end-systolic dimension is usually increased in athletes compared with control subjects (22,25–27,29,31,36,38,43,47), although this difference is usually mild and has achieved statistical significance in only some investigations (27,29,31,36,43,47).

Left ventricular wall thickness. Ventricular septal and posterior left ventricular free wall thicknesses are often increased in athletes compared with values in sedentary control subjects (in 11 of 18 investigations for the septum and in 22 of 26 for the posterior wall) (Tables 1 and 2). In absolute terms, these reported wall thicknesses are not substantially increased and are rarely greater than 14 mm (the maximal reported thickness is 16 mm). Average values for ventricular septal and posterior free wall thickesses in previous studies of athletes are only 10.4 and 10.6 mm, respectively. These thicknesses are well within the accepted normal range, but are 14% (for the septum) and 19% (for the free wall) greater than values reported for matched sedentary control subjects (Table 1).

The majority of athletes studied by M-mode echocardiography show symmetric ventricular septal and posterior free wall thicknesses (normal septal/free wall ratio of <1.3). Some reports (21,25,50) have described a small number of athletes with an increased septal/free wall ratio suggesting an asymmetric pattern of left ventricular hypertrophy. However, these athletes showed only minimal absolute thickening of the septum and, therefore, such "abnormal" ratios cannot be considered diagnostic of hypertrophic cardiomyopathy. Only Shapiro (50) reported a significant proportion (28%) of athletes with a septal/free wall ratio greater than 1.3. However, his measurements of left ventricular wall thicknesses were apparently made from the two-dimensional echocardiogram, a method that is much less desirable than M-mode echocardiography for quantitative measurement of wall thickness. Our experience with two-dimensional echocardiography in athletes has shown that their mild left ventricular wall thickening is almost always symmetrically distributed.

Right ventricular cavity. The increase in ventricular mass induced by long-term conditioning may often include the right ventricle. Transverse right ventricular cavity dimension is commonly (although not invariably) increased in athletes. In the studies shown in Tables 1 and 2, right ventricular dimension averaged 22 mm in athletes (maximum 33) compared with 17 mm for control subjects, a difference of 24%; right ventricular size was greater in athletes than in control subjects in six of the eight studies in which this variable was measured.

Left atrial size. Many athletes also have enlargement of the left atrium. Left atrial transverse dimension was greater in athletes than in control subjects in 7 out of the 13 studies in which this variable was reported (Tables 1 and 2). The cause of left atrial enlargement in athletes is unknown. It has been suggested that the increased atrial size is a reflection of impaired left ventricular compliance; however, as will be discussed later, several recent studies have shown that indexes of diastolic function are normal in athletes.

Determinants of increased cardiac dimensions in athletes. From the vast amount of echocardiographic data now available on athletes, it would appear that the increased cardiac dimensions (particularly the left ventricular wall thickness and cavity) of these individuals are truly a direct response to training. However, it is also possible that such cardiac alterations are not produced solely by intense training, but are due, in part, to a genetic predisposition that exists before training (10). Although this issue has not been resolved, it would seem unlikely that increased left ventricular mass is importantly determined by genetic factors in most athletes because of the facility and rapidity with which change in the activity level can alter the magnitude of left ventricular mass (28,38,43) (as discussed in detail later). It is also necessary to consider the possibility that the increase in left ventricular transverse dimension commonly present in athletes is partially due to training-induced bradycardia and the concomitant prolongation of the diastolic filling period (10). However, Hirshleifer et al. (64) found that in normal subjects very small changes in left ventricular dimension accompanied marked changes in heart rate (induced by atropine). Hence, reduced heart rate is probably not the sole determinant of the increase in left ventricular cavity size observed in trained athletes.

Relation of Cardiac Structural Changes to the Nature of Athletic Conditioning

The findings of several investigations (20,33,34, 39,40,45,46,49,52), particularly those of Morganroth et al. (20) and Longhurst et al. (39,40), suggest that the precise alterations in cardiac structure in athletes may differ depending on the type of training activity undertaken (Fig. 2). For example, athletes participating in isotonic (dynamic) endurance sports are exposed primarily to conditions producing a volume load and therefore show increased left ventricular cavity dimension without a significant increase in wall thickness. In contrast, athletes participating in sports

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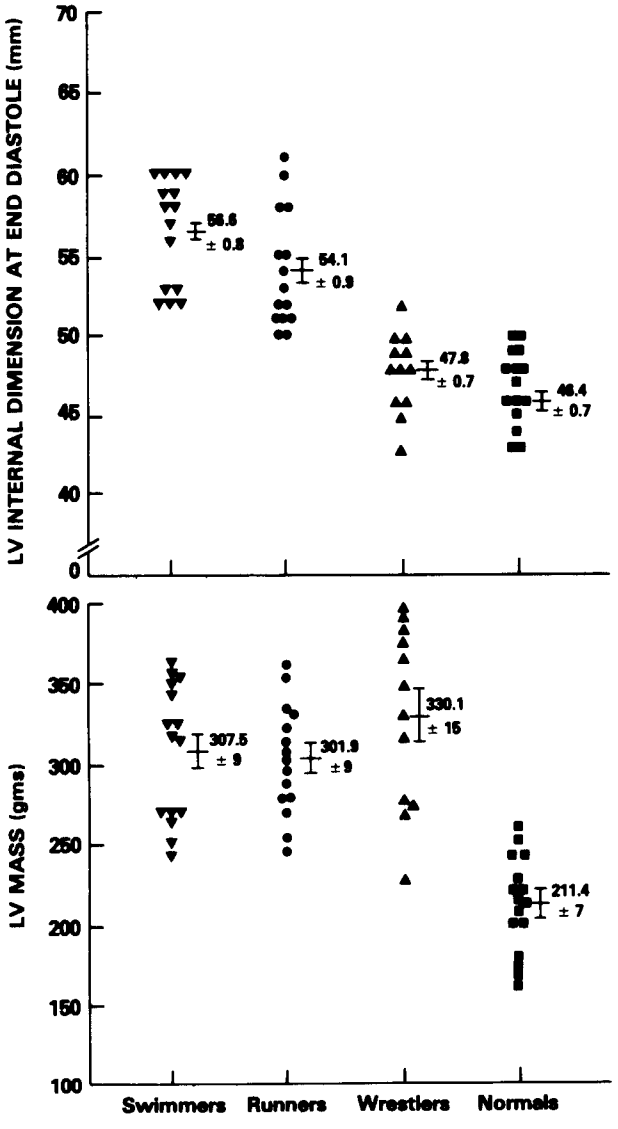


Figure 2. Left ventricular (LV) internal dimension at end-diastole **(upper panel)** and estimated left ventricular mass **(lower panel)** measured with M-mode echocardiography in college athletes participating in a variety of sports. The numbers represent mean values ± SEM. For *left ventricular end-diastolic dimension,* data of swimmers and runners are statistically different from those of wrestlers and normal subjects ($p < 0.001$), although values in wrestlers and normal subjects are similar. For *left ventricular mass,* data of swimmers, runners and wrestlers are all statistically different from those of normal subjects ($p < 0.001$). (Reproduced with permission from Morganroth et al. [20].)

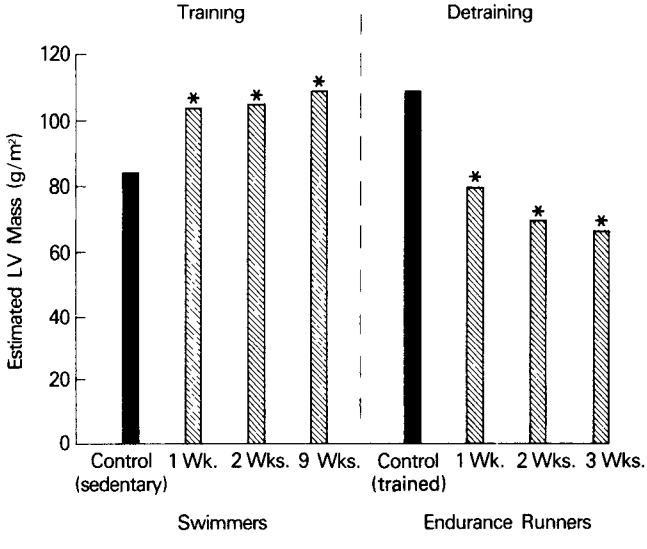
involving isometric (static) exertion are exposed primarily to a pressure load and often show increased left ventricular wall thickness with little or no increase in the cavity dimension.

In addition, Longhurst et al. (39,40) demonstrated that although both isotonic and isometric training increased left ventricular mass, they did so in different ways relative to lean body mass. Isotonic training produced an increase in left ventricular mass that was disproportionately greater than the increase in the athlete's skeletal muscle mass. In contrast, isometric training increased left ventricular mass only in proportion to the increase in skeletal muscle mass. The different effects of isotonic and isometric exercise on cardiac dimensions are not always clear-cut; some investigators have not found significant differences in cardiac morphology between athletes participating in each of these two types of activity (41).

Changes in Left Ventricular Dimensions and Mass Associated With Conditioning or Deconditioning

Conditioning. Several studies show that the modest degree of left ventricular hypertrophy present in some athletes can develop quite rapidly (within weeks or months) in response to the initiation of a vigorous physical conditioning program. Ehsani et al. (28) used M-mode echocardiography to study eight competitive swimmers at the conclusion of a period of deconditioning (2 to 7 months) and then serially for a subsequent 9 week period of intense training (Fig. 3). Left ventricular end-diastolic dimension increased significantly (by about 10% compared with the deconditioned state) after only 1 week of training and then remained relatively constant for the next 8 weeks. Posterior wall thickness increased more gradually with training; significant increases (of about 7% compared with the deconditioned state) were not evident for 5 weeks and subsequently posterior wall thickness remained relatively constant. Similarly, Wieling

Figure 3. Effect of training and detraining on the hearts of competitive athletes. Echocardiographic data for estimated left ventricular (LV) mass (corrected for body surface area) were obtained in eight swimmers who trained for 9 weeks after a sedentary period (during which they had become deconditioned) and in six endurance runners who trained for at least 3 months, but subsequently stopped their conditioning program for 3 weeks. The **asterisk** indicates mean values obtained during training or detraining that were significantly different compared with the respective control values **(solid bars)** ($p < 0.005$). (Reproduced with permission from Ehsani et al. [28].)



et al. (38), in studies on collegiate oarsmen, found relatively rapid increases in left ventricular mass shortly after initiation of training. Statistically significant increases in left ventricular end-diastolic dimension and ventricular septal thickness were evident after 4 months of training and posterior wall thickness increased after 7 months of training.

Deconditioning. Observations on the structural cardiac changes after a period of deconditioning also emphasize the dynamic nature of the increased left ventricular mass present in trained athletes. Ehsani et al. (28) showed that within 1 week of total cessation of training, a significant decrease in left ventricular diastolic dimension (8%), posterior wall thickness (15%) and calculated left ventricular mass (27%) occurred (Fig. 3); these changes became even more marked over the next 2 weeks (overall change of 9, 25 and 38%, respectively). Fagard et al. (43) studied 12 competitive bicyclists and also found a decrease in left ventricular mass associated with detraining, manifested by a reduction in ventricular septal and posterior left ventricular wall thicknesses, but without significant change in left ventricular end-diastolic dimension. Shapiro and Smith (59) reported regression in left ventricular mass after a 6 week period of detraining in 10 nonathletes who had previously participated in a standardized 6 week exercise program. Shapiro (50) also observed normal cardiac dimensions in a group of deconditioned ex-athletes 5 or more years after they had ceased competitive activities (although serial echocardiographic studies were not available in these subjects).

Such changes in left ventricular mass with training and detraining have been shown (23,27,28,30,31,35,36, 38,43,47,48) to occur in parallel with alterations in maximal oxygen consumption. Furthermore, maximal oxygen consumption has been shown (30,36) to have a relation to the magnitude of left ventricular cavity enlargement.

Left Ventricular Function

Systolic function. M-mode echocardiographic assessments of left ventricular systolic function in athletes have shown that the percent fractional shortening (or the derived ejection fraction or velocity of circumferential fiber shortening) is within normal limits in most athletes (Table 2). Such assessments of systolic function are segmental in nature and do not constitute a measure of global function. Nevertheless, relatively mild deviations from normal left ventricular contractility have been reported (31,43) in only two studies, both involving competitive bicyclists.

Nishimura et al. (31) found indexes of left ventricular contractility to be mildly decreased in competitive bicyclists in the 40 to 49 year old age group who had trained for most of their adult lives (an average of 27 years). Fagard et al. (43) found a significant decrease in percent fractional shortening associated with regression in left ventricular mass after 8 weeks of detraining. Percent fractional shortening (and ejection fraction) is usually normal in athletes because long-term conditioning induces increases of similar magnitude in left ventricular end-diastolic and end-systolic dimensions. Rerych et al. (65) assessed global cardiac function in 18 competitive swimmers with radionuclide angiography before and after 6 months of training. These investigators found that ejection fraction at rest decreased from a mean value of 73% before training to 67% after training, although left ventricular end-diastolic volume was increased.

Diastolic function. Several recent reports have described diastolic function in trained athletes using a number of noninvasive techniques, including digitized M-mode echocardiography (49), Doppler echocardiography (66) and radionuclide angiography (48). These studies uniformly show various indexes of diastolic function to be within normal limits under basal conditions. Granger et al. (48), using radionuclide angiography, found that despite a 43% increase in left ventricular mass over that of control subjects, athletes showed no alteration in left ventricular filling. Hence, unlike pathologic hypertrophy due to chronic systolic hemodynamic overload (67–71), coronary artery disease (72) or primary myocardial disease (70,71,73,74), "physiologic" left ventricular hypertrophy induced by exercise is apparently not accompanied by impaired left ventricular diastolic function. Recently, Matsuda et al. (75), using digitized M-mode echocardiography reported an augmentation of early diastolic filling with exercise in athletes.

Elite Athletes

An intriguing speculation concerns whether the magnitude to which cardiac dimensions are altered may reflect the potential or achieved level of performance in individual competitive athletes. At present, there are few data defining cardiac dimensions in "elite" athletes who have achieved a world-class or national level of performance and few data comparing these individuals with less accomplished (although highly trained) competitive athletes. Morganroth et al. (20) found no differences in cardiac dimensions between world-class runners and shot-putters and their nonelite counterparts. In addition, Underwood and Schwade (25) found no differences in cardiac morphology between elite endurance runners and other competitive runners. On the other hand, Shapiro (50) reported that national standard athletes had significantly greater left ventricular wall thicknesses and mass compared with those of collegiate and recreational sportsmen. However, absolute wall thicknesses that have been reported in elite athletes still fall largely within the accepted, normal range. Therefore, it is unlikely that "supranormal" cardiac dimensions account for the fact that some athletes achieve significantly higher levels of excellence than do others. Enhanced athletic performance is more likely to be related to genetically determined advantageous body structure and composition, superior psychological mo-

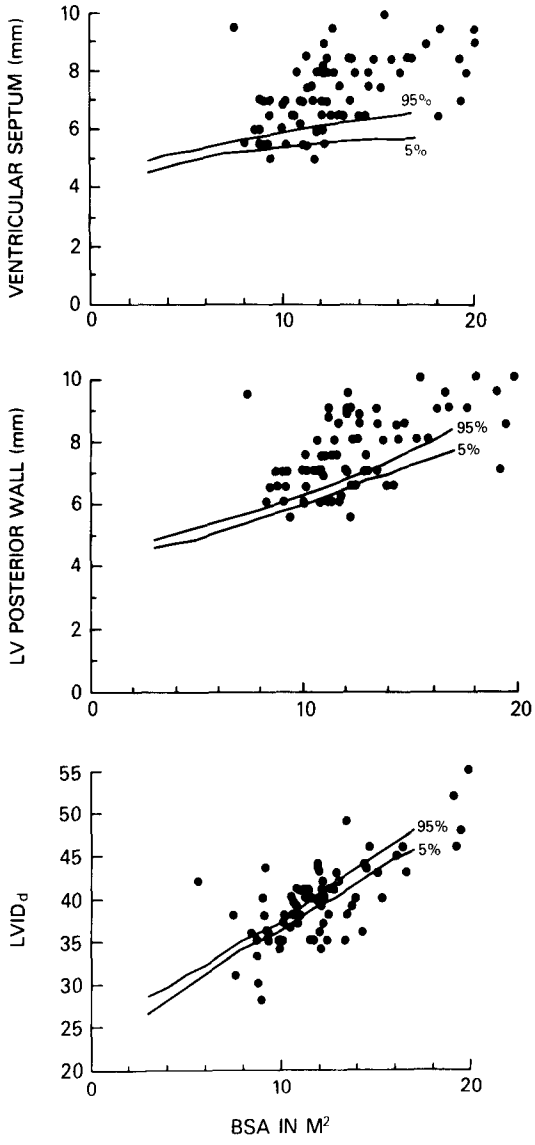


Figure 4. Athlete heart in childhood. Cardiac dimensions in 77 competitive, chronically trained swimmers 5 to 17 years of age, assessed with echocardiography. Measurements of ventricular septal and posterior left ventricular (LV) free wall thickness exceed 95% confidence limits in most athletes. In contrast, left ventricular end-diastolic internal dimensions ($LVID_d$) are more frequently normal. BSA in m^2 = body surface area in square meters. (Reprinted from Allen HD, Goldberg SJ, Sahn DJ, Schy N, Wojcik R. A quantitative echocardiographic study of champion childhood swimmers. Circulation 1977;55:142–5. By permission of the American Heart Association, Inc.)

tivation, proclivity for difficult training, great motivation and instinct for competition as well as the ill-defined attribute of "talent."

Childhood Athletes

Few echocardiographic data are available regarding the cardiovascular effects of training on younger athletes who are still maturing physically and have not achieved their fully developed body habitus. The study of Allen et al. (76) in 77 competitive age-group swimmers (5 to 17 years of age) demonstrated that the morphologic changes of the athlete heart may be identified early in life, presumably because of the training regimen to which these children were exposed. These investigators reported increased thickness of the septum and left and right ventricular walls as well as increased right ventricular cavity size in young swimmers compared with sedentary control subjects of similar age and body size. However, in only 30% of the childhood athletes was left ventricular end-diastolic cavity dimension greater than that in control subjects (Fig. 4). Some childhood athletes may possess a genetic predisposition to develop such cardiac morphologic changes or are naturally selected to be athletes and thereby develop features of the athlete heart because of an advantageous genetic constitution.

Geenen et al. (77) showed that in 6 and 7 year old children (nonathletes), left ventricular posterior wall thickness and left ventricular mass increased after an 8 month aerobic exercise program. These two studies, (76,77) which show left ventricular wall thickening to usually occur in the absence of significant left ventricular cavity enlargement, differ considerably from the studies in adult endurance athletes in whom left ventricular cavity enlargement is the most consistent structural alteration (10). Such discrepancies imply that cardiovascular training effects may differ in children and adults.

Figure 5. Effect of a short-term training program on 24 nonathletic subjects. Demonstration by M-mode echocardiography of mildly increased transverse left ventricular (LV) end-diastolic dimension and calculated left ventricular mass after an 11 week walk-jog-run exercise program. Mean values and standard deviation are shown for both the preexercise and postexercise evaluations. (Reprinted from DeMaria AN, Neumann A, Lee G, Fowler W, Mason DT. Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography. Circulation 1978;57:237–44. By permission of the American Heart Association, Inc.)

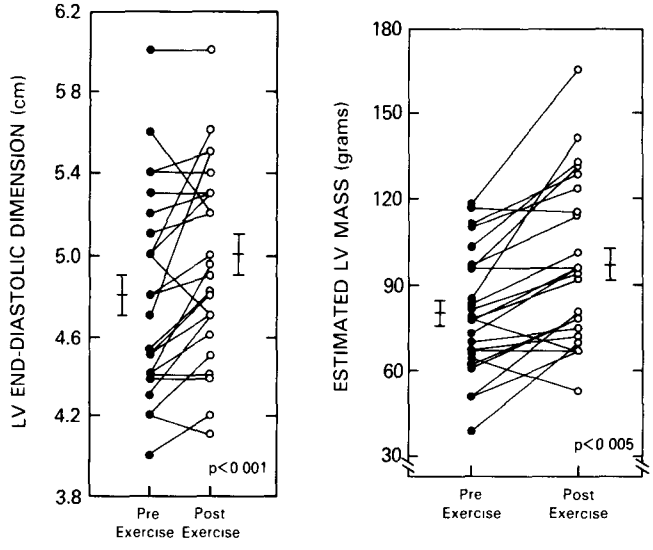


Table 3. Comparison of the Athlete Heart and Hypertrophic Cardiomyopathy

Variable	Athlete Heart	Nonobstructive HCM	"Gray Zone"
Ventricular septal thickness (mm)	Usually < 15	Usually > 15	13 to 15
Septal/free wall ratio	Usually < 1.3 (symmetric)	Usually ≥ 1.3 (asymmetric)	1.2 to 1.4
LV end-diastolic dimension	Normal or ↑	Normal or ↓	Normal
Ejection fraction	Usually normal	Normal or ↑	Normal
Abnormal electrocardiogram	25 to 50%	About 90%	Normal or abnormal

HCM = hypertrophic cardiomyopathy; LV = left ventricular; ↓ = decreased; ↑ = increased.

Effects of Training in Nonathletes

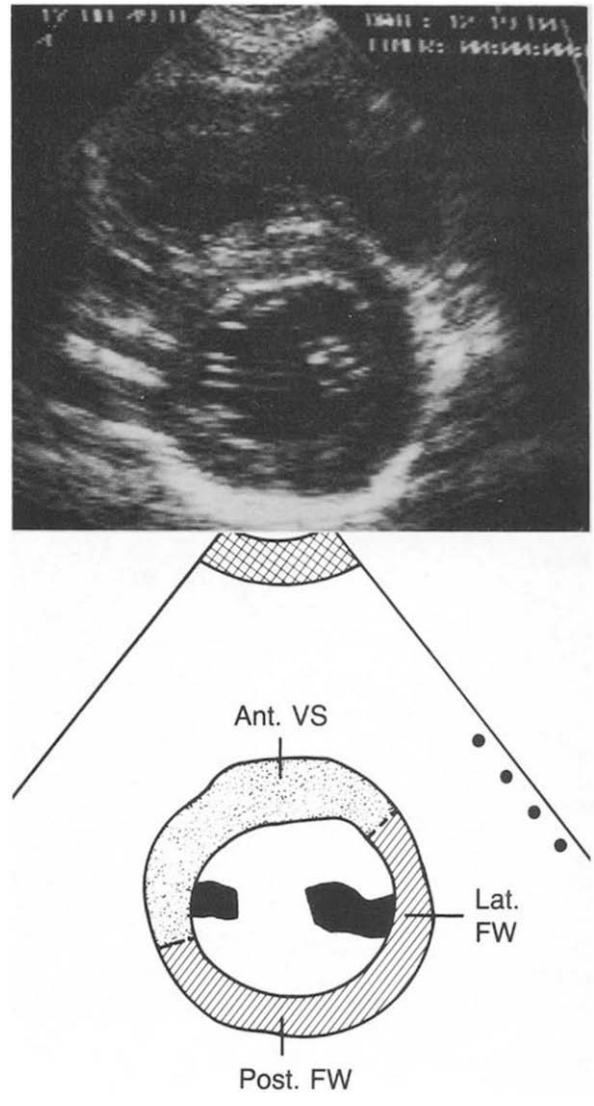
Seven short-term longitudinal echocardiographic studies (53–59) have analyzed the changes in cardiac dimensions produced at rest in nonathletic subjects who were exposed to systematic exercise programs for periods of 6 to 20 weeks. Most subjects were male and young (average age 27 years). The activity programs were diverse in design; most involved running or jogging several times a week, although two consisted of bicycling and one involved isometric strength training.

These studies demonstrated minimal or no changes in cardiac dimensions associated with training (Fig. 5). These alterations were well within the methodologic error of M-mode echocardiography. Furthermore, the concomitant changes in maximal oxygen consumption were also relatively small (average increase of about 20% compared with 40% for competitive athletes). In only three studies (53,55,58) was the increase in transverse left ventricular diastolic dimension statistically significant (average increase about 5%). This magnitude of change is about half of that observed in highly conditioned competitive athletes (Table 1). Only two studies (57,59) showed a statistically significant, albeit small, increase in ventricular septal or posterior free wall thickness, or both (of only about 4%). In three studies (53,57,59) these relatively small changes involving left ventricular dimension and wall thickness were reflected in a significant increase in calculated left ventricular mass. Percent fractional shortening was slightly increased after training in three studies (53,55,57), but in two others (58,59) no change was evident. In each of these longitudinal investigations, however, virtually all the *absolute* values for cardiac dimensions measured at the completion of the training period were still within the accepted normal range, even though they represented a statistically significant change from the pretraining state in some instances.

The Athlete Heart and Hypertrophic Cardiomyopathy

The clinical features of the athlete heart may frequently resemble those of certain cardiac diseases, most often

Figure 6. Stop-frame two-dimensional echocardiogram (at end-diastole) in the short-axis cross-sectional plane from a 22 year old competitive runner. There is a relatively mild increase in thickness of the anterior ventricular septum (Ant. VS) (13 mm); other segments of the left ventricular wall appear to be of normal thickness. Left and right ventricular cavities are of normal size. **Calibration dots** are 10 mm apart. Lat. FW = lateral free wall; Post. FW = posterior free wall.



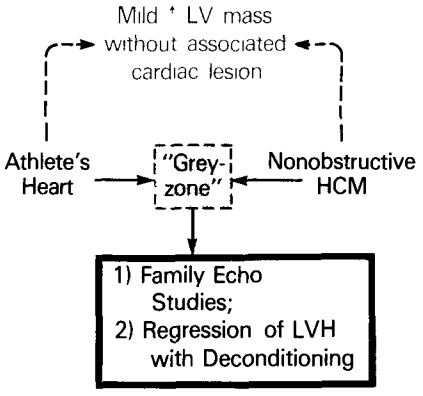


Figure 7. Diagram summarizing the relation between the normal athlete heart, nonobstructive hypertrophic cardiomyopathy (HCM) and the heart of athletes in whom morphologic cardiac findings are intermediate between the two ("Grey-zone"). Echo = echocardiographic; LV = left ventricular; LVH = left ventricular hypertrophy; ↑ = increased.

hypertrophic cardiomyopathy (Table 3). The competitive athlete being evaluated for possible cardiac disease may present a particular diagnostic dilemma when the anterior ventricular septal thickness is only mildly increased to 13 to 15 mm (and the remainder of the left ventricular wall is free of significant hypertrophy), the left ventricular cavity is nondilated and systolic anterior motion of the mitral valve is absent (Fig. 6).

In such a circumstance, it may be extremely difficult to discriminate between the "physiologic" increase in left ventricular mass produced by athletic training and the relatively mild (but pathologic) hypertrophy observed in some patients with nonobstructive hypertrophic cardiomyopathy (78,79); hence, such athletes appear to fall into an equivocal "gray zone" between these two diagnostic possibilities (Fig. 7). In addition, athletes with the normal athlete heart and patients with nonobstructive hypertrophic cardiomyopathy may show a variety of similarly abnormal (or even normal) patterns on the 12 lead electrocardiogram (1–4,20–22, 25,26,29,31,36–38,43,47,80–84).

To clarify the diagnosis in the occasional athlete whose heart appears to fall within the morphologic gray zone between the normal athlete heart and hypertrophic cardiomyopathy, it may be necessary to assemble additional clinical data (Fig. 7). For example, because hypertrophic cardiomyopathy is often genetically transmitted (85,86), echocardiographic identification of a relative with the morphologic expression of hypertrophic cardiomyopathy would make this diagnosis likely in the athlete. However, negative echocardiographic studies in relatives of an athlete would not definitively exclude the diagnosis of hypertrophic cardiomyopathy since this disease may occur without apparent familial transmission (85). An additional clinical manipulation that may prove useful in discriminating the normal athlete heart from hypertrophic cardiomyopathy is prospective deconditioning of the athlete (for a period of about 3 to 6 months) to ascertain whether regression of left ventricular mass occurs. A decrease in left ventricular wall thickening or cavity dimension, or both, would probably result if the hypertrophy was primarily due to athletic conditioning, but would not be expected to change significantly if the hypertrophy was a manifestation of cardiac disease. If, however, a definitive diagnosis cannot be ascertained in such a "gray zone" athlete, we recommend that athletic training and competition be allowed to continue (assuming that important ventricular arrhythmias are not present on 24 hour ambulatory monitoring) and periodic evaluation and follow-up be undertaken.

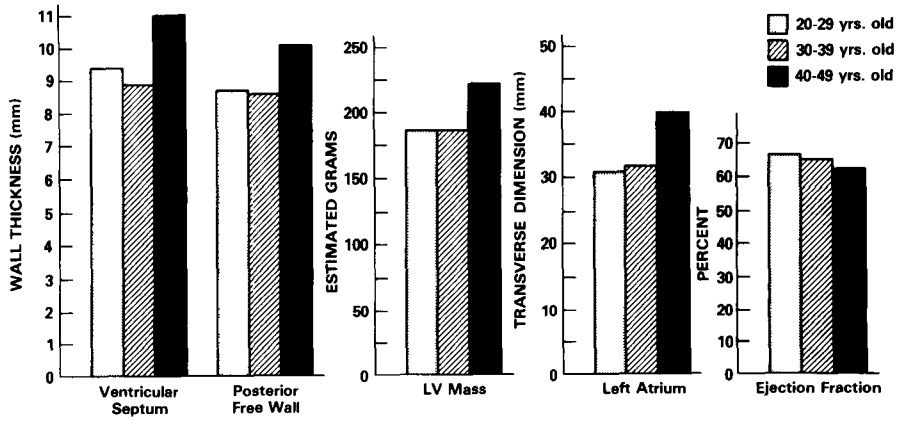


Figure 8. Alterations in cardiac dimensions resulting from particularly prolonged (lifelong) athletic conditioning. Adapted from the M-mode echocardiographic data of Nishimura et al. (31) in 60 competitive bicyclists. Mean ventricular septal and posterior free wall thicknesses, calculated left ventricular (LV) mass, left atrial dimension and ejection fraction are compared for bicyclists in three different age groups (20 to 29, 30 to 39 and 40 to 49 years of age). Values for 40 to 49 year old bicyclists differ significantly from those for both 20 to 29 and 30 to 39 year old bicyclists. (Reprinted from Nishimura T, Yamada Y, Kawai C. Echocardiographic evaluation of long-term effects of exercise on left ventricular hypertrophy and function in professional bicyclists. Circulation 1980;61:832–40. By permission of the American Heart Association, Inc.)

Effects of "Lifelong" Athletic Activity

The study of Nishimura et al. (31) is unique in providing data on cardiac dimensions in a group of 60 chronically trained competitive athletes (professional bicyclists) of widely different ages (20 to 49 years) (Fig. 8). The younger bicyclists had trained for an average of 5 years and for as many as 35 hours/week. The older bicyclists had been competing in races consistently for an average of 27 years and continue to train for up to 10 hours/week. Nishimura and his coworkers found that those bicyclists 20 to 29 and 30 to 39 years of age showed similar increases in left ventricular cavity size, wall thicknesses and mass. However, older bicyclists (40 to 49 years of age) showed greater ventricular septal and posterior free wall thicknesses and left ventricular mass than did the younger bicyclists. The older athletes also demonstrated increased left atrial size and slightly decreased percent fractional shortening. In addition, Heath et al. (32) observed that endurance athletes at the master level (average age 59 years) had significantly greater left ventricular end-diastolic volume index and calculated mass than did anthropometrically matched younger competitive athletes (average age 22 years); however, in contrast to the findings of Nishimura et al. (31), ventricular wall thicknesses were not greater in the older masters athletes.

These findings imply that particularly long-term (virtually lifelong) participation in competitive athletics may result in progressive alteration of cardiac dimensions well into middle age (although the absolute dimensions in such athletes were only slightly greater than those in normal control subjects). These observations are pertinent to the cardiovascular evaluation of older individuals with suspected organic heart disease who may have participated in competitive or highly vigorous athletic activities most of their lives. The life-style of such individuals must be considered when assessing whether cardiac disease associated with left ventricular hypertrophy is present.

Long-Term Significance of the Athlete Heart

As emphasized by Oakley (87), the morphologic features of the athlete heart have been well defined with M-mode echocardiography over the past decade. However, these echocardiographic observations are derived largely from cross-sectional studies in which cardiac dimensions are assessed in athletes at one point in time. Such studies tell us little regarding the long-term significance of the "physiologic" left ventricular hypertrophy present in well conditioned athletes. Because the hypertrophy of the athlete's heart may be rapidly reversible with cessation of conditioning (28,43), those individuals who participate in an intense training program for a relatively short portion of their life (for example, <5 to 10 years) would not seem to be at any particular risk for cardiovascular disease solely by virtue of once having been conditioned athletes when younger. The study of Nishimura et al. (31) suggests that persistent conditioning through middle life may progressively increase left ventricular mass after age 40 years. At present, however, there is no convincing evidence to suggest that even these alterations in cardiac structure importantly affect longevity or are detrimental to the athlete in any way.

Sudden death. Premature sudden death in competitive athletes is usually due to a variety of structural cardiovascular diseases, each distinguishable from the normal "athlete heart." For example, in young athletes congenital cardiovascular diseases (particularly hypertrophic cardiomyopathy) (88,89) are most commonly responsible for these catastrophes, while virtually all sudden deaths in older athletes are due to coronary heart disease (89–92). Occasional young athletes have been reported (88,89) who died suddenly and at autopsy were found to have a moderate increase in left ventricular mass characterized by a symmetric pattern of wall thickening in the presence of nondilated ventricular cavities, normal myocardial architecture and no evidence of genetic transmission of cardiomyopathy to relatives. It is conceivable (although unlikely) that such individuals with "idiopathic left ventricular hypertrophy" represent rare malignant expressions of the athlete heart that for undefined pathophysiologic reasons result in premature sudden death.

Follow-up. The medical status of former athletes many years after completion of the competitive periods of their lives has been analyzed in several investigations (reviewed in detail by Crawford and O'Rourke [3]); However, each of these studies contains important flaws in design that limit their usefulness. Hence, well designed, long-term follow-up studies will be necessary to determine the ultimate clinical significance of the athlete heart.

References

1 Gott PH, Roselle HA, Crampton RS. The athletic heart syndrome. Five-year cardiac evaluation of a champion athlete. Arch Intern Med 1968;122:340–4

2. Raskoff WJ, Goldman S, Cohn K. The "athletic heart." Prevalence and physiological significance of left ventricular enlargement in distance runner. JAMA 1976,236:158–62.

3. Crawford MH, O'Rourke RA. The athlete's heart. Adv Intern Med 1979.311–29 (Stollerman GH, ed Yearbook Medical Publication; vol 24)

4 Rost R, Hollmann W Athlete's heart—A review of its historical assessment and new aspects. Int J Sports Med 1983;4:147–65.

5 Beckner GL, Winsor T. Cardiovascular adaptations to prolonged physical effort. Circulation 1954;9:835–45.

6. Reindell H, Roskamm H, Stein H. The heart and blood circulation in athletes. Med Welt 1960,31:1557–63.

7. Bevegard S, Holmgren A, Jonsson B. Circulatory studies in well-trained athletes at rest and during heavy exercise with special reference to stroke volume and the influence of body position. Acta Physiol Scand 1963;57:26–50

8. Scheuer J, Tipton CM. Cardiovascular adaptations to physical training. Annu Rev Physiol 1977;39:221–51

9 Blomqvist CG, Saltin B. Cardiovascular adaptations to physical training Annu Rev Physiol 1983;45:169–89.

10. Schaible TF, Scheuer J. Cardiac adaptations to chronic exercise Prog Cardiovasc Dis 1985;27:297–324.

11 Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Cardiovascular responses to static exercise in distance runners and weight lifters. J Appl Physiol (Respir Environ Exerc Physiol) 1980;49:676–83

12. Henschen S. Skilauf and Skiwettlauf. Eine medizinische Sportstudie. Mitt Med Klinik Uppsala 1899;2:15.

13. Marach JH Physiological and pathological effects of severe exertion, marathon race, on circulatory and renal system. Arch Intern Med 1910;5:382–405

14. Herxheimer H. Unterschungen uber die Anderung der Hergro unter dem Einflu bestinmter Sportarten. Z Klin Med 1929;111:376–93.

15. Keys A, Friedell HL. Size and stroke of the heart in young men in relation to athletic activity. Science 1938;88.456–8.

16. Gordon B, Levine SA, Welmaers A A group of marathon runners with special reference to circulation. Arch Intern Med 1924;33:425–534.

17. Klemola E. Electrocardiographic observations of 650 Finnish athletes. Ann Med Intern Fenn 1951;40:121–32.

18 Karvonen MJ, Rautaharju P, Rousteenoja R. Heart size of champion skiers. Ann Med Intern Fenn 1957;46:169–78.

19. Bulychev VV, Khmelevskii VA, Rutman IV. Roentgenological and instrumental examination of the heart in athletes. Klin Med (Mosk) 1965;43:108–14.

20. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes Ann Intern Med 1975;82:521–4.

21. Roeske WR, O'Rourke RA, Klein A, Leopold G, Karliner JS. Noninvasive evaluation of ventricular hypertrophy in professional athletes. Circulation 1976;53:286–92.

22. Zoneraich S, Rhee JJ, Zoneraich O, Jordan D, Apple J. Assessment of cardiac function in marathon runners by graphic noninvasive techniques. Ann NY Acad Sci 1977;301:900–17.

23. Gilbert CA, Nutter DO, Felner JM, Perkins JV, Heymsfield SB, Schlant RC. Echocardiographic study of cardiac dimensions and functions in the endurance-trained athlete. Am J Cardiol 1977;40:528–33.

24. Laurenceau JL, Turrat J, Dumesnil J. Echocardiographic findings in Olympic athletes (abstr). Circulation 1977;56(suppl III):III-25.

25. Underwood RH, Schwade JL. Noninvasive analysis of cardiac function in elite distance runners—echocardiography, vectorcardiography, and cardiac intervals. Ann NY Acad Sci 1977;301:297–309.

26. Parker BM, Londeree BR, Cupp GV, Dubiel JP. The noninvasive cardiac evaluation of long-distance runners. Chest 1978;73:376–81.

27. Zeldis SM, Morganroth J, Rubler S. Response of the heart to isotonic conditioning in female athletes. A correlation between echocardiographically determined left ventricular size and exercise performance J Appl Physiol 1978;44:849–52.

28. Ehsani AA, Hagberg JM, Hickson RC. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning Am J Cardiol 1978;42:52–6.

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

30. Blair NL, Youker JE, McDonald IG, Jelinek VM. Echocardiographic assessment of cardiac chamber size and left ventricular function in aerobically trained athletes. Aust NZ J Med 1980;10:540–7.

31. Nishimura T, Yamada Y, Kawai C. Echocardiographic evaluation of long-term effects of exercise on left ventricular hypertrophy and function in professional bicyclists. Circulation 1980;61.832–40.

32. Heath GW, Hagberg JM, Eshani AA, Holloszy JO. A physiological comparison of young and older endurance athletes. J Appl Physiol (Respir Environ Exerc Physiol) 1981;51:634–40.

33. Keul J, Dickhuth H-H, Simon G, Lehmann M. Effect of static and dynamic exercise on heart volume, contractility, and left ventricular dimensions. Circ Res 1981;48(suppl I):I-162–70.

34. Dickhuth H-H, Simon G, Kindermann W, Wildberg A, Keul J. Echocardiographic studies on athletes of various sport-types and non-athletic persons. Z Kardiol 1979;68:449–53.

35. Paulsen W, Boughner DB, Cunningham DA, Persaud JA Left ventricular function in marathon runners: echocardiographic assessment. J Appl Physiol (Respir Environ Exerc Physiol) 1981;51.881–6

36. Bekaert I, Pannier JL, Van De Weghe C, Van Durme JP, Clement DL, Pannier R Non-invasive evaluation of cardiac function in professional cyclists. Br Heart J 1981;45:213–8.

37. Mumford M, Prakash R Electrocardiographic and echocardiographic characteristics of long distance runners. Comparison of left ventricular function with age- and sex-matched controls Am J Sports Med 1981;9:23–8.

38. Wieling W, Borghols EAM, Hollander AP, Danner SA, Dunning AJ Echocardiographic dimensions and maximal oxygen uptake in oarsmen during training. Br Heart J 1981;46:190–5.

39. Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Chronic training with static and dynamic exercise. cardiovascular adaptation and response to exercise. Circ Res 1981;48(suppl I):I-171–8.

40. Longhurst JC, Kelly AR, Gonyea WJ, Mitchell JH. Echocardiographic left ventricular masses in distance runners and weight lifters. J Appl Physiol (Respir Environ Exerc Physiol) 1980;48:154–62.

41. Snoeckx LHEH, Abeling HFM, Lambregts JAC, Schmitz JJF, Verstappen FTJ, Reneman RS. Echocardiographic dimensions in athletes in relation to their training programs. Med Sci Sports Exerc 1982;14.428–34.

42. Rost R. The athlete's heart. Eur Heart J 1982.3(suppl A).193–8.

43. Fagard R, Aubert A, Lysens R, Staessen J, Vanhees L, Amery A. Noninvasive assessment of seasonal variations in cardiac structure and function in cyclists. Circulation 1983;67:896–901

44 Brown S, Byrd R, Jayasinghe, Jones D. Echocardiographic characteristics of competitive and recreational weight lifters. JCU 1983;2:163–5

45. Sugishita Y, Koseki S, Matsuda M, Yamaguchi T, Ito I. Myocardial mechanics of athletic hearts in comparison with diseased hearts. Am Heart J 1983;105:273–80.

46. Spirito P, Maron BJ, Bonow RO, Epstein SE. Prevalence and significance of an abnormal S-T segment response to exercise in a young athletic population. Am J Cardiol 1983;51:1663–6.

47. Fagard R, Aubert A, Staessen J, VanDen Eynde E, Vanhees L, Amery A. Cardiac structure and function in cyclists and runners. Comparative echocardiographic study. Br Heart J 1984;52:124–9.

48. Granger CB, Karimeddini MK, Smith V-E, Shapiro HR, Katz AM, Riba AL Rapid ventricular filling in left ventricular hypertrophy. I. Physiologic hypertrophy. J Am Coll Cardiol 1985;5:862–8

49. Colan SD, Sanders SP, MacPherson D, Borow KM. Left ventricular diastolic function in elite athletes with physiologic cardiac hypertrophy. J Am Coll Cardiol 1985;6:545–9.

50 Shapiro LM. Physiological left ventricular hypertrophy. Br Heart J 1984;52:130–5.

51. Peronnet F, Ferguson RJ, Perrault H, Ricci G, Lajoie D. Echocardiography and the athlete's heart. Phys Sports Med 1981;9:102–12.

52. Howald H, Maire R, Heirli B, Follath F. Echocardiographische Befunde bei trainierten Sportlern. Schweiz Med Wochenschr 1977;197:1662–6.

53. DeMaria AN, Neumann A, Lee G, Fowler W, Mason DT. Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography. Circulation 1978;57:237–44.

54. Wolfe LA, Cunningham DA, Rechnitzer PA, Nichol PM. Effects of endurance training on left ventricular dimensions in healthy men. J Appl Physiol (Respir Environ Exerc Physiol) 1979;47:207–12.

55. Stein RA, Michielli D, Diamond J, Horwitz B, Krasnow N The cardiac response to exercise training: echocardiographic analysis at rest and during exercise. Am J Cardiol 1980;46:219–25.

56. Peronnet F, Perrault H, Cleroux J, et al. Electro- and echocardiographic study of the left ventricle in man after training. Eur J Appl Physiol 1980;45:125–30.

57. Kanakis C, Hickson RC. Left ventricular response to a program of lower-limb strength training. Chest 1980;78:618–21.

58. Adams TD, Yanowitz FG, Fisher AG, Ridges JD, Lovell K, Pryor TA. Noninvasive evaluation of exercise training in college-age men. Circulation 1981;64:958–65.

59 Shapiro LM, Smith RG Effect of training on left ventricular structure and function. An echocardiographic study. Br Heart J 1983;50:534–9

60. Gardin JM, Henry WL, Savage DD, et al Echocardiographic measurements in normal subjects: evaluation of an adult population without clinically apparent heart disease. JCU 1979;7:439–47

61. Henry WL, Gardin JM, Ware JH. Echocardiographic measurements in normal subjects from infancy to old age Circulation 1980;62:1054–61

62 Troy BL, Pombo J, Rackley CE. Measurement of left ventricular wall thickness and mass by echocardiography Circulation 1972;45:602–11

63. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man Anatomic validation of the method. Circulation 1977;55:613–8

64 Hirshleifer J, Crawford M, O'Rourke RA, Karliner JS Influence of acute alterations of heart rate and systemic arterial pressure of echocardiographic measures of left ventricular performance in normal human subjects. Circulation 1975;52:835–41.

65. Rerych SK, Scholz PM, Sabiston DC, Jones RH Effects of exercise training on left ventricular function in normal subjects: a longitudinal study by radionuclide angiography. Am J Cardiol 1980;45:244–52

66 Finkelhor RS, Hanak LJ, Bahler RC Diastolic function in endurance trained subjects (abstr). J Am Coll Cardiol 1985;5:540

67. Eichhorn P, Grimm J, Koch R, Hess O, Carroll J, Krayenbuehl HP Left ventricular relaxation in patients with left ventricular hypertrophy secondary to aortic valve disease Circulation 1982;65 1395–1404.

68 Inouye I, Massie B, Loge D, et al. Abnormal left ventricular filling: an early finding in mild to moderate systemic hypertension Am J Cardiol 1984;53:120–6

69. Fifer MA, Borow KM, Colan SD, Lorell BH Early diastolic left ventricular function in children and adults with aortic stenosis J Am Coll Cardiol 1985;5:1147–54

70. Stewart S, Mason DT, Braunwald E Impaired rate of left ventricular filling in idiopathic hypertrophic subaortic stenosis and valvular aortic stenosis. Circulation 1968;37:8–14

71. Hanrath P, Mathey DG, Siegart R, Bleifeld W Left ventricular relaxation and filling pattern in different forms of left ventricular hypertrophy An echocardiographic study. Am J Cardiol 1980;45 15–23

72. Bonow RO, Bacharach SL, Green MV, et al. Impaired left ventricular diastolic filling in patients with coronary artery disease: assessment with radionuclide angiography. Circulation 1981;64:315–23

73 Sanderson JE, Traill TA, St John Sutton MG, Brown DJ, Gibson DG, Goodwin JF. Left ventricular relaxation and filling in hypertrophic cardiomyopathy. an echocardiographic study Br Heart J 1978;40:596–601.

74. Maron BJ, Arce J, Bonow RO, Wesley Y Noninvasive assessment of left ventricular relaxation and filling by pulsed Doppler echocardiography in hypertrophy cardiomyopathy (abstr) Circulation 1984, 70(suppl II):II-18

75. Matsuda M, Sugishita Y, Koseki S, Ito I, Akatsuka T, Takamatsu K Effect of exercise on left ventricular diastolic filling in athletes and nonathletes J Appl Physiol (Respir Environ Exerc Physiol) 1983;55 323–8

76. Allen HD, Goldberg SJ, Sahn DJ, Schy N, Wojcik R. A quantitative echocardiographic study of champion childhood swimmers Circulation 1977;55 142–5.

77 Geenen DL, Gilliam TB, Crowley D, Moorehead-Steffens C, Rosenthal A. Echocardiographic measures in 6 to 7 year old children after an 8 month exercise program Am J Cardiol 1982;49:1090–5.

78 Maron BJ, Epstein SE Hypertrophic cardiomyopathy: a discussion of nomenclature. Am J Cardiol 1979;43:1242–4

79 Maron BJ, Gottdiener JS, Epstein SE Patterns and significance of the distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy: a wide-angle, two-dimensional echocardiographic study of 125 patients Am J Cardiol 1981;48:418–28.

80 Smith WG, Cullen KJ, Thorburn IO Electrocardiograms of marathon runners in 1962 Commonwealth Games. Br Heart J 1964;26:469–76.

81 Van Ganse W, Versee L, Eylenbosch W, Vuylsteek K The electrogram of athletes. comparison with untrained subjects. Br Heart J 1970;32:160–4

82 Hanne-Paparo N, Wendkos MH, Brunner D T wave abnormalities in the electrocardiograms of top-ranking athletes without demonstrable organic heart disease Am Heart J 1971;81:743–7.

83 Oakley DG, Oakley CM. Significance of abnormal electrocardiograms in highly trained athletes Am J Cardiol 1982;50:985–9.

84 Maron BJ, Wolfson JK, Ciró E, Spirito P Relation of electrocardiographic abnormalities and patterns of left ventricular hypertrophy identified by two-dimensional echocardiography in patients with hypertrophic cardiomyopathy Am J Cardiol 1983;51:189–94

85 Maron BJ, Nichols PF, Pickle LW, Wesley YE, Mulvihill JJ. Patterns of inheritance in hypertrophic cardiomyopathy: assessment of M-mode and two-dimensional echocardiography. Am J Cardiol 1984;53:1087–94

86 Clark CE, Henry WL, Epstein SE. Familial prevalence and genetic transmission of idiopathic hypertrophic subaortic stenosis. N Engl J Med 1973;289:709–14.

87 Oakley D Cardiac hypertrophy in athletes. Br Heart J 1984;52:121–3.

88 Maron B, Roberts WC, McAllister HA, Rosing DR, Epstein SE. Sudden death in young athletes Circulation 1980;62:218–29.

89 Maron BJ, Epstein SE, Roberts WC Causes of sudden death in competitive athletes. J Am Coll Cardiol 1986;7:204–14.

90 Thompson PD, Stern MP, Williams P, Duncan K, Haskell WL, Wood PD Death during jogging or running A study of 18 cases. JAMA 1979;242:1265–7.

91 Waller BF, Roberts WC Sudden death while running in conditioned runners aged 40 years or over. Am J Cardiol 1980;45:1292–300.

92 Virmani R, Robinowitz M, McAllister HA. Nontraumatic death in joggers A series of 30 patients at autopsy Am J Med 1982;72:874–81.