Cardiac hypertrophy and function in master endurance runners and sprinters

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CHILD, JOHN S., R. JAMES BARNARD, AND RICHARD L. TAW. *Cardiac hypertrophy and function in master endurance runners and sprinters.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 57(1): 176–181, 1984.—Cardiac enlargement with increased left ventricular mass is a recognized adaptive response to intensive physical conditioning. There have been few reports regarding cardiac hypertrophy and function in the middle-aged or older athlete. Accordingly, we studied 9 male Masters Track endurance distance runners (E) (mean age 54 yr) and 13 male Masters Track sprinters (S) (mean age 47 yr) by M-mode echocardiography, systolic time intervals, and maximal treadmill stress testing with direct measurement of maximal O_2 consumption ($\dot{V}O_{2\,max}$). Left ventricular mass (LVM) index for E (154.0 ± 27.4 g/m^2; mean ± SD) was greater than for S (127.1 ± 15.9 g/m^2) ($P < 0.05$). LVM index was greater for both groups compared with age-range matched controls ($N = 111.7 \pm 23.8$ g/m^2) ($P < 0.02$). $\dot{V}O_{2\,max}$ was greater in E (54 ± 12 ml·kg^{-1}·min^{-1}) than S (47 ± 7 ml·kg^{-1}·min^{-1}) ($P < 0.05$) despite the younger age of S ($P < 0.05$); both E and S had greater $\dot{V}O_{2\,max}$ than predicted for nonathletic males of equivalent age. There was no significant linear relationship for LVM index and $\dot{V}O_{2\,max}$ in the athletes. Left ventricular function at rest was normal. Thus, in well-trained older (≥40 yr) male distance runners and sprinters $\dot{V}O_{2\,max}$ and LVM index are greater in E than S, and each greater than in controls, resting left ventricular function is normal, as judged by fractional shortening and systolic time intervals, and the degree of hypertrophy (LVM index) does not directly correlate with aerobic capacity ($\dot{V}O_{2\,max}$).

aerobic capacity; echocardiography; left ventricular mass

CARDIAC ENLARGEMENT with an increase in left ventricular mass is an adaptive response to prolonged intensive physical conditioning. In recent years, noninvasive diagnostic developments, namely, echocardiography and radionuclide angiography, have been used extensively to study the cardiac effects of training by a variety of athletic endeavors, both in humans and animals (1, 5–7, 9, 11, 16, 18–21, 25, 26, 28, 31, 32, 33, 43, 47, 48).

The long-term effects of athletic cardiac hypertrophy on ventricular pump performance are uncertain, and there have been few reports regarding left ventricular hypertrophy and function in middle-aged and older athletes (20, 29).

Masters Track athletes are 40 yr of age and older and compete in various categories established by decades. We therefore studied 9 Master endurance runners and 13 Master sprinters by M-mode echocardiography and systolic time intervals at rest and by treadmill stress testing with measured $\dot{V}O_{2\,max}$ *1*) to evaluate cardiac anatomy and function in older (≥40 yr of age) men who have continued intense dynamic exercise training, *2*) to compare these effects in long-distance endurance runners vs. sprinters, and *3*) to reassess the correlation of compensatory left ventricular changes (left ventricular mass and dilatation) with aerobic capacity ($\dot{V}O_{2\,max}$) developed by chronic dynamic exercise training.

METHODS

Population. Thirteen male sprinters, aged 47 ± 6 yr (mean ± SD) (range = 41–58 yr), and nine male endurance distance runners, aged 54 ± 11 yr (range = 40–69 yr) who participated in Masters Track competition and had technically satisfactory echocardiograms, were studied. Many of the subjects were quite successful, and some were national or world record holders in their events at the time of the study. Six subjects had been training for ≥20 yr. Their physiological characteristics, including assessment of body composition (lean weight, relative fat), serum lipids, and maximal stress characteristics (heart rate, maximum ventilation, maximum O_2 consumption) have been previously reported (3).

Twelve normal sedentary males, aged 56 ± 8 yr (range = 40–69 yr), acted as an age-range-matched control population. These were nonhospitalized volunteers, who were selected at random from the age of 40–80 yr from our normal population file. Each subject was considered to be normal on the basis of a history and physical by a cardiologist, M-mode echocardiography, phonocardiography, 24-h ambulatory electrocardiography, resting 12-lead electrocardiography, and graded treadmill stress testing.

Echocardiographic measurements. With the subject in the supine position, echocardiograms were obtained with a Smith-Kline Instruments Ekoline 20A Ultrasonoscope using a 10-cm focus 2.25-Mhz transducer with a repetition rate of 1,000/s. Tracings were made with a Honeywell 1856 fiber-optic recorder on Kodak light-sensitive paper at a speed of 50 mm/s. By use of standard techniques, the transducer was placed in the third, fourth, or fifth left parasternal intercostal interspace, and a stand-

ard sweep from apex to base was performed. The echoes of the interventricular septum and left ventricular posterior wall were recorded at the level of the chordae tendineae just below the tip of the anterior mitral leaflet. Damping, reject, and sensitivity controls were carefully adjusted to allow optimal recording for measurement of wall thickness and cavity dimensions. Two independent observers blinded to the identity of each subject used calipers to make the measurements to the closest millimeter; results were averaged for three successive cardiac cycles. Measurements were made using the leading edge method. Left ventricular end-diastolic dimension (EDD) = distance between the left interventricular septum and posterior left ventricular wall endocardium at the peak of the R wave of the simultaneous lead II electrocardiogram (ECG). Left ventricular end-systolic dimension (ESD) = shortest simultaneous systolic distance from the left interventricular septum to the posterior left ventricular wall endocardium. Interventricular septal (IVS) thickness and posterior left ventricular wall (PLVW) thickness were measured at end diastole. Careful attention was paid to correct identification of the right and left septal endocardial surfaces to ensure accurate wall thickness measurement.

By use of these measurements, the following are derived: fractional shortening (FS%) of the left ventricular minor dimension, where FS% = [(EDD − ESD) ÷ (EDD)] × 100, and left ventricular myocardial mass (LVM) (g), where LVM = [(EDD + IVS thickness + PLVW thickness)^3 − (EDD)^3] × 1.05.

Other direct echocardiographic measurements included *1*) left atrial size measured at end systole from the posterior wall of the aortic root to the left atrial wall; *2*) aortic root size measured at end diastole from the anterior aortic wall to the posterior aortic wall at the level of the aortic valve; and *3*) right ventricular end-diastolic dimension measured at the peak of the R wave of the ECG from right ventricular wall endocardium to the right interventricular septum and at end expiration.

Chamber dimensions and wall thickness have a direct relationship to body surface area (BSA) (13, 14), and the BSA was different for each of the three groups studied. Accordingly, measured chamber dimensions and wall thickness and calculated left ventricular mass were corrected for BSA. Fractional shortening was not corrected for BSA because it is independent of BSA (13, 14).

Systolic time intervals. With the subject supine, and during quiet respiration, systolic time intervals were measured from simultaneous recordings of the indirect carotid pulse, ECG, and phonocardiogram from the third left intercostal space, using a Honeywell 3820 echocardiography-phonocardiography system interfaced with the Honeywell 1856 fiber-optic recorder with a paper speed of 100 mm/s and 40-ms time lines. Measurements included heart rate, electromechanical systole (QS_2 = time from the onset of the Q wave of the ECG to the first high-frequency positive deflection of the aortic component of the second heart sound), left ventricular ejection time (LVET = time from the onset of the rapid upstroke of the carotid pulse to the incisura of the dicrotic notch), and preejection period (PEP = QS_2 − LVET). These results were expressed as the PEP-to-LVET ratio because this ratio is unrelated to heart rate and is the single most useful indicator of left ventricular performance by systolic time intervals. A mean value of 10 beats was taken for each measurement.

Physical work capacity of the Master endurance and sprint runners. Subjects reported to the laboratory after a 12-h fast. After informed consent and a resting 12-lead supine ECG were obtained, subjects were prepared for the exercise stress test using a bipolar V_5 electrode configuration. Graded exercise testing was performed using a Quinton motor-driven treadmill on a protocol described previously (3). Blood pressure (indirect sphygmomanometry) and heart rate were recorded at rest and every minute throughout the test. O_2 uptake, ventilation, and respiratory exchange ratios (R) were obtained each minute and during the final seconds of the tests using a Beckman Metabolic Measurement Cart as described by Wilmore (33). Maximal O_2 uptake ($\dot{V}O_{2\,max}$) was determined as the peak $\dot{V}O_2$ during exercise with a corresponding R ≥ 1.10. The R values were used to estimate the extent to which the subjects were willing to push themselves. (The normal control subjects did not undergo measurement of $\dot{V}O_2$ during stress testing.)

Statistical analyses. Results for each group are expressed as means ± SD. Analysis of variance was done to detect a significant difference among the three groups. A one-tailed unpaired Student's *t* test was used to detect significant differences between each group. Chosen level of significance was $P \leq 0.05$. Correlation between left ventricular mass index and $\dot{V}O_{2\,max}$ was examined by least-squares linear regression analysis and a correlation coefficient (*r*) was generated for the endurance runners and sprinters separately; significance of each *r* value was tested by the *t* test. Statistics were performed on a programmable Texas Instruments calculator (TI-58) using the Advanced Statistics Package.

RESULTS

Physiological characteristics. Training for the distance runners at the time of testing ranged from 67 to 200 km/wk with some men alternating long distance with interval work. Six of the distance runners had been training for ≥20 yr. All subjects, except for two sprinters, participated in formal training programs. Table 1 reveals pertinent physiological data, with sprinters (S) and endurance (E) distance runners compared to each other and to the normal controls. The sprinters were younger (47 ± 6 yr) than the endurance runners (54 ± 11 yr) or the normal controls (56 ± 8 yr). Blood pressure at rest was equivalent for the three groups. $\dot{V}O_{2\,max}$ (Fig. 1) was higher in endurance runners (56.0 ± 8.0 ml·kg^{-1}·min^{-1}) than in the sprinters (47.2 ± 7.2 ml·kg^{-1}·min^{-1}) despite the older age of the endurance runners. Endurance distance runners and sprinters had higher measured values for $\dot{V}O_{2\,max}$ than previously noted for nonathletes in an equivalent age range (27, 29). Resting heart rate was lower in endurance runners (49.0 ± 7.2 beats/min). However, maximum heart rate attained on treadmill testing was slightly less for endurance runners (161.8 ± 9.7 beats/

TABLE 1. *Physiological and left ventricular function data*

	Athletes		*t* Test *P* Value Between Athletes	Controls ($n = 12$)	*t* Test *P* Values		ANOVA *P* Value
	Endurance runners ($n = 9$)	Sprinters ($n = 13$)			Endurance runners vs. controls	Sprinters vs. controls	
Age, yr	53.7 ± 10.6 (40–69)	46.5 ± 6.0 (41–58)	<0.03	56.3 ± 7.8 (40–69)	NS	<0.001	<0.0001
BSA, m	1.78 ± 0.13 (161–1.96)	1.91 ± 0.11 (1.80–2.18)	<0.02	1.93 ± 0.08 (1.83–2.10)	≤0.001	NS	<0.001
HR (rest)	49.0 ± 7.2 (37–58)	64.5 ± 5.3 (54–73)	<0.001	62.2 ± 11.2 (53–90)	<0.003	NS	<0.001
HR (max)	161.8 ± 9.7 (146–177)	168.2 ± 7.6 (154–178)	≤0.05	Sheffield* (175–189) for ages 40–49, untrained, male			
$\dot{V}O_{2\,max}$, ml·kg^{-1}·min^{-1}	56.0 ± 8.0 (44.4–71.0)	47.2 ± 7.2 (38.6–62.9)	≤0.01	Robinson; Åstrand† (35 ± 5) for ages 40–70, untrained, male			
PEP/LVET	0.34 ± 0.08 (0.19–0.44)	0.32 ± 0.04 (0.25–0.38)	NS	0.34 ± 0.05 (0.25–0.41)	NS	NS	NS
FS%	37.8 ± 2.6 (33–43)	34.6 ± 9.8 (17–51)	NS	39.1 ± 4.7 (29–49)	NS	NS	NS

Values are means ± SD with ranges given in parentheses. ANOVA, analysis of variance; BSA, body surface area; FS%, fractional shortening of the left ventricular minor axis; HR, heart rate; PEP/LVET, preejection period to left ventricular ejection time ratio; $\dot{V}O_{2\,max}$, maximal O_2 consumption. * Sheffield and Roitman (30). † Robinson et al. (27); Åstrand (2).

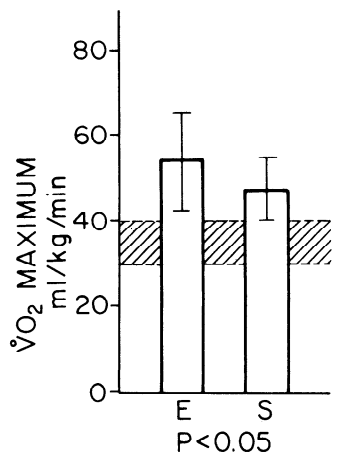

FIG. 1. Maximal O_2 consumption ($\dot{V}O_{2\,maximum}$) in Master Track male endurance (E) runners and sprinters (S). *Top of bar*, mean; *bracket*, ±SD. Previously reported (27) values for nonathletic males, age 40–70 yr, are represented by *crosshatched area.*

min) than sprinters (168.2 ± 7.6 beats/min) and of borderline significance ($P = 0.05$); these values correspond to 95–100% of predicted maximum heart rate (30). The endurance runners had a significantly smaller BSA than either sprinters or controls when corrections were made for BSA, primarily due to the lesser body weight.

For endurance runners, sprinters, and controls, absolute measurements of echocardiographic posterior left ventricular wall thicknesses were 10.3 ± 0.9, 9.8 ± 1.2, and 9.9 ± 0.8 mm, respectively; interventricular septal thicknesses were 11.2 ± 2.2, 10.8 ± 1.4, and 10.5 ± 1.5 mm, respectively; left ventricular end-diastolic dimensions were 52.1 ± 3.7, 50.9 ± 3.1, and 48.3 ± 4.5 mm, respectively.

When corrections were made for BSA, the endurance runners had significantly greater diastolic thickness of the posterior left ventricular wall and interventricular septum (PLVW index = 5.8 ± 0.5 mm/m^2; IVS index = 6.3 ± 1.0 mm/m^2) than the sprinters (PLVW index = 5.2 ± 0.4 mm/m^2; IVS index = 5.5 ± 0.9 mm/m^2); sprinters did not differ significantly from controls (Table 2). Endurance runners also had larger end-diastolic left ventricular dimensiona/BSA (29.3 ± 2.7 mm/m^2) than sprinters (26.7 ± 1.7 mm/m^2); sprinters had larger EDD/BSA than the control group (25.1 ± 2.1 mm/m^2). Left ventricular mass index (Fig. 2) was greater in endurance runners (153.9 ± 27.4 g/m^2) than sprinters (127.1 ± 15.9 g/m^2): sprinters had larger LVM index than control (111.7 ± 23.8 g/m^2). For both EDD index and LVM index, sprinters were statistically significantly greater than controls though the overlap was considerable. Because Henry et al. (13) found that EDD varied with the cube root of BSA, and PWT and IVS varied with the square root of BSA, we also compared each group using those data. The difference among the groups remained significant, and thus because of the ease of using BSA in comparing with other reports, we include in Table 2 only those values indexed for BSA.

Relationship of hypertrophy to training. Using LVM index as a measure of hypertrophy and $\dot{V}O_{2\,max}$ as a measure of aerobic capacity (or training effect), the endurance runners had a "statistically significant" negative correlation ($r = -0.61$); sprinters revealed no correlation (Fig. 3). When all runners were considered together, no correlation was statistically evident. Testing the individual measurements in the LVM equation vs. the $\dot{V}O_{2\,max}$, there was no significant correlation (whether corrected or uncorrected for BSA).

Cardiac function at rest. Using echocardiographic fractional shortening of the left ventricular minor axis (FS%) and the ratio of preejection period to left ventricular ejection time (PEP/LVET) as indicators of resting left ventricular function, neither the endurance runners nor sprinters were significantly different from normal controls (Table 1).

Other echocardiographic measurements. End-systolic left atrial (LA) dimension, aortic root (AO) end-systolic dimension, and right ventricular (RV) cavity end-dia-

TABLE 2. *Cardiac cavity size and wall thickness data*

	Athletes		*t* Test *P* Value Between Athletes	Controls ($n = 12$)	*t* Test *P* Values		ANOVA *P* value
	Endurance runners ($n = 9$)	Sprinters ($n = 13$)			Endurance runners vs. controls	Sprinters vs. controls	
PWT/BSA, mm/m^2	5.8 ± 0.5 (5.1–6.8)	5.2 ± 0.8 (3.7–6.6)	<0.025	5.2 ± 0.4 (4.7–6.0)	≤0.003	NS	<0.0001
IVS/BSA, mm/m^2	6.3 ± 1.0 (5.3–8.7)	5.6 ± 0.8 (4.8–7.2)	≤0.05	5.5 ± 0.9 (4.2–6.4)	<0.03	NS	<0.0001
EDD/BSA, mm/m^2	29.3 ± 2.7 (24.6–33.2)	26.7 ± 1.7 (24.3–30.1)	≤0.005	25.1 ± 2.1 (20.5–27.9)	<0.001	<0.025	<0.0001
LVM/BSA, g/m^2	153.9 ± 27.4 (126.6–208.7)	129.7 ± 17.9 (103.3–161.3)	<0.02	111.7 ± 23.8 (67.9–137.7)	<0.001	<0.05	<0.0001
LA/BSA	21.4 ± 2.5 (17.9–25.5)	18.4 ± 4.1 (11.2–25.8)	<0.02	18.5 ± 1.6 (15.8–20.8)	<0.003	NS	<0.0001
AO/BSA, mm/m^2	18.8 ± 2.4 (14.3–21.8)	17.0 ± 2.3 (13.2–21.5)	<0.05	16.0 ± 1.9 (12.8–19.8)	<0.01	NS	<0.0001
RV/BSA, mm/m^2	15.1 ± 2.9 (8.2–18.0)	11.4 ± 3.6 (4.3–16.3)	<0.01	9.8 ± 2.7 (6.9–14.1)	<0.001	<0.01	<0.0001

Values are means ± SD with ranges given in parentheses. ANOVA, analysis of variance; BSA, body surface area; AO, aortic root; EDD, end-diastolic dimension of left ventricle; IVS, interventricular septum; LA, left atrium; LVM, left ventricular mass; PWT, posterior wall thickness of left ventricle; RV, right ventricular end-diastolic dimension.

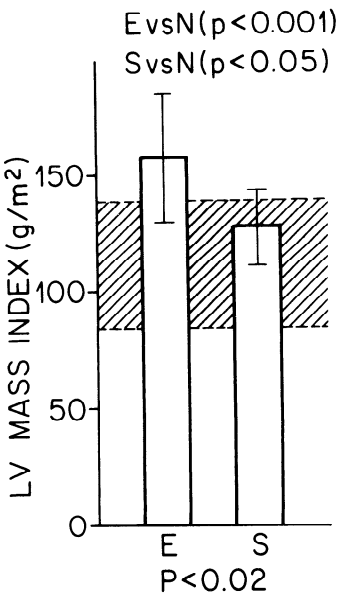

FIG. 2. Echocardiographic left ventricular (LV) mass index (mean ± SD) in Masters Track male endurance (E) runners and sprinters (S). Normal male controls, age-range matched, are shown in *crosshatched area* (mean ± SD).

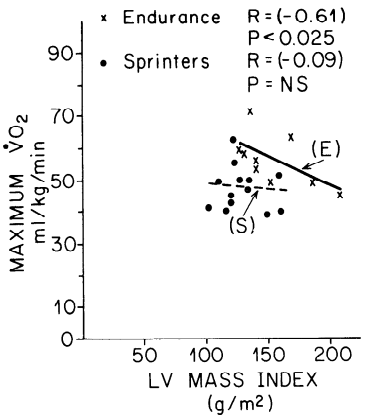

FIG. 3. Relationship of maximal O_2 consumption ($\dot{V}O_2$) and left ventricular (LV) mass index for Masters Track male endurance (E) runners and sprinters (S). Linear regression analysis by least-squares method generated a correlation coefficient (R) for both E and S.

stolic dimension, each corrected for BSA, were larger in the endurance runners than either the sprinters or the controls, but only RV/BSA was larger in the sprinters than in the controls (Table 2).

DISCUSSION

The adaptation of the human heart to vigorous exercise continues to be a subject of intense interest. Furthermore, as the number and percentage of aged people increases in our society, characterization of the normal physiological changes in the heart associated with the aging process becomes more crucial. Echocardiography is particularly suited for such a study because it gives anatomic information unobtainable by other techniques in living subjects (10, 14). Henry et al. (14) and Gerstenblith et al. (10) found that with increasing age there are progressive increases in ventricular septal and left ventricular free wall thickness and in estimated left ventricular mass. Aging either does not effect or causes a slight decrease in left ventricular internal dimensions; fractional shortening of the minor axis is not effected (10, 14). Of note, these changes cannot be explained by differences in blood pressure. Few reports have used echocardiography to study the long-term effects of exercise in older athletes. Recently, Nishimura et al. (20) reported that professional bicyclists showed increased echocardiographic left ventricular end-diastolic dimensions and wall thicknesses when compared to age-matched controls, but an older subgroup of bicyclists, aged 40–49 yr, had a greater increase in wall thickness than the athletes ≤39 yr. In addition, the older bicyclists compared either

with athletes ≤39 yr old or with the control group had "significantly depressed" resting left ventricular function (franctional shortening, mean velocity of circumferential fiber shortening), apparently unrelated to heart rate.

Masters Track endurance runners and sprinters, by definition ≥40 yr old, afford the scientific community an opportunity to study highly motivated and well-trained individuals who continue to train into middle age, and in some cases into old age. Data obtained from these older athletes can provide insight into the biological aging process by eliminating the variable of inactivity. Our distance endurance runners represented a wide age range (40–69 yr) of highly trained athletes. At the time of testing, weekly training for the endurance runners ranged from 67 to 200 km, and their $\dot{V}O_{2\,max}$ was 56.0 ± 8.0 $ml \cdot kg^{-1} \cdot min^{-1}$ (range 44.4–71.0). These $\dot{V}O_{2\,max}$ values are similar to values for athletes of the same age as reported by Saltin and Grimby (29) and Pollock et al. (23). In comparison with the data reported for 55-yr-old nonathletes (31–36 $ml \cdot kg^{-1} \cdot min^{-1}$) by Robinson et al. (27) and others (2, 8, 29), our values for both sprinters and endurance runners are significantly higher. They also exceed the values of $\dot{V}O_{2\,max}$ reported by others (12) for middle-aged men participating in physical fitness programs, indicating that our distance runners were indeed exceptionally trained. Barnard et al. (3) previously noted that $\dot{V}O_{2\,max}$ decreased with age in this group of subjects, as did the maximum exercise heart rate, even though it was evident that training allowed achievement of a higher $\dot{V}O_{2\,max}$ for any given age group.

Our echocardiographic assessment of these Masters athletes revealed that intensive training is associated with an increase in left ventricular mass index when compared with a sedentary normal control group of similar age. Though older than the sprinters, the group of endurance runners exhibited a higher aerobic capacity ($\dot{V}O_{2\,max}$) and higher left ventricular mass index. The increase in left ventricular mass index in the endurance runners appeared to be due to both increased left ventricular end-diastolic dimension index and wall thickness index (both interventricular septum and posterior left ventricular wall), whereas in the sprinters increased left ventricular mass index was predominately due to increased left ventricular end-diastolic dimension index. Blood pressure did not differ among the three groups (runners, sprinters, control).

These findings corroborate those reported for younger endurance athletes (1, 6, 7, 9, 11, 16–21, 25, 26, 28, 31). Our mean values of left ventricular posterior wall thickness index, interventricular septal index, end-diastolic dimension index, as well as left ventricular mass index, are comparable to the data of Gilbert et al. (11) or Ikaheimo et al. (16) for younger (<40 yr old) distance runners and sprinters. The difference in the relative contributions of cavity size vs. wall thickness of left ventricular mass in distance runners vs. sprinters was noted by Ikaheimo et al. (16). They postulated that these differences were due to the nature of the left ventricular stress imposed by each type of training—namely, short-distance training causes extreme, but transient, tachycardia and volume overload leading to left ventricular dilatation; regular long-distance running causes sustained marked increases in systolic blood pressure in addition to volume overload resulting in left ventricular hypertrophy as well as dilatation.

Exercise-induced hypertrophy of the left ventricle is generally considered to be benign (4, 5, 22, 32). Left ventricular ejection phase indices at rest in endurance runners have generally been found to be normal (7, 9, 11, 16) or increased (21, 31).

As shown by the higher left ventricular mass index and $\dot{V}O_{2\,max}$ for endurance (distance) runners as a group vs. the sprinters as a group vs. normal subjects we confirmed that cardiac hypertrophy, i.e., left ventricular mass index, is an important adaptive response to sustained exercise and that the degree of cardiac hypertrophy has a general relationship to the degree of training. Linear regression analysis of the relationship of $\dot{V}O_{2\,max}$ and left ventricular mass index revealed a significant negative r value for endurance runners and no significant statistical relationship for sprinters; for the whole group, there was no statistical relationship. This was unexpected, but it seems logical to assume that although cardiac hypertrophy is an adaptive response to exercise, the degree of hypertrophy in each individual is not directly related to his ability to achieve a given level of $\dot{V}O_{2\,max}$. Rather, differences among individuals probably exist as to the amount of peripheral adaptation that they may develop for any given level of aerobic capacity ($\dot{V}O_{2\,max}$). Whether such individual differences reflect superior physical endowment due to genetic factors (cardiac and/or skeletal muscle) or differences in modes of training is unclear. That very large hearts are reported for some athletes even many years after the cessation of training supports a genetic factor because studies show rapid regression of training-induced hypertrophy after cessation of training (15, 17, 32).

In conclusion, our study of well-trained older (≥40 yr old) male athletes showed *1*) $\dot{V}O_{2\,max}$ and left ventricular mass index are greater in distance runners than in sprinters, and in each greater than control subjects; *2*) resting left ventricular function is normal as judged by fractional shortening and systolic time intervals; and *3*) aerobic capacity ($\dot{V}O_{2\,max}$) does not directly correlate with left ventricular mass index, though both clearly have a general relationship to degree of training.

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