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Ten-Year Echo/Doppler Determination of the Benefits of Aerobic Exercise after the Age of 65 Years

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Abstract

As the human lifespan becomes progressively extended, potential health-related effects of intense aerobic exercise after age 65 need evaluation. This study evaluates the cardiovascular (CV), pulmonary, and metabolic effects of competitive distance running on age-related deterioration in men between 69 (± 3) and 77 (± 2) years (mean $\pm$ SD). Twelve elderly competitive distance runners (ER) underwent oxygen consumption and echo/Doppler treadmill stress testing (Balke protocol) for up to 10 years. Twelve age-matched sedentary controls (SC) with no history of CV disease were similarly tested and the results compared for the initial three series of the study. CV data clearly separated the ER from SC. At entry, resting and maximal heart rate, systolic/diastolic blood pressure, peak oxygen consumption (VO_{2max}), and E/A ratio of mitral inflow were better in the ER ($P < 0.05$ vs. SC). With aging, ER had a less deterioration of multiple health parameters. Exceptions were VO_{2max} and left ventricular diastolic function (E/A, AFF, IVRT) that decreased ($P < 0.05$, Year 10 vs. Year 1). Health advantages of high-level aerobic exercise were demonstrated in the ER when compared to SC. Importantly, data collected in ER over 10 years confirm the benefit of intensive exercise for slowing several negative effects of aging. However, the normative drop of exercise capacity in the seventh and eighth decades reduces the potential athleticism plays in prevention of CV events.

Keywords

aging; exercise; cardiac function; echocardiography

Age 65 is often designated as the beginning of old age. During “young” old age (65–74 years) motivated healthy seniors continue with prior athletic activities.¹ Physically active lifestyle can extend into the “middle” old age (75–84 years), although with limitations. After age 85 (“old” old age), however, even the most motivated seniors find it difficult to exercise at higher levels. Muscle tissue loss (sarcopenia), atherosclerosis, “presbycardia,” and other effects of senescence take their toll.²

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Well before age 65, however, participation in organized aerobic exercise drops precipitously. Consequently, contribution of habitual exercise to primary prevention of cardiovascular morbidity may be lost. After the age of 65, participation in competitive aerobic activities becomes further reduced. Performance drops primarily due to decrease in muscle power and lower aerobic capacity. Switching to lower intensity exercise (walking) is associated with 30% longer disease-free life.³

In this study, we evaluated the significance of cardiopulmonary fitness in slowing the effects of aging in senior men. Twelve “young old” and “middle old” competitive distance runners were evaluated over a span of 10 years. A series of 6 treadmill stress tests (12–24 months apart) including echo/Doppler echocardiography, oxygen consumption, and lipid profiling were performed between 1995 and 2005. Twelve age-matched sedentary seniors with no previous history or current clinical evidence of cardiovascular disease were recruited as controls for the initial three series.

Data were collected in 2 phases:

Phase 1 (initial 3 years). The treadmill stress/echo protocol data with several health indicators were collected from both the athletes and the sedentary controls. Study of control subjects after three initial series had to be discontinued due to adverse health and/or motivational issues.

Phase 2 (total duration 10 years). The athletes continued with the same treadmill stress/echo protocol for longitudinal analysis data individually to examine the changes in exercise capacity and cardiac function in athletes as they age.

Methods

Study Population

Twelve male competitive runners (entry age 65 years or older) who consistently placed at the top of their age groups in road races ranging from 5 km to the marathon were recruited in 1995. On the average, these men ranked in the 75th percentile of age-related standards for long distance running established by the World Association of Veteran Athletes (WAVA) (now World Masters Athletics). For several years, these participants averaged 40 miles of training per week prior to enrolling in the study and continued this amount of training over the years of the study. Twelve age-matched health-screened nonathletic men served as controls. Entry screening included echo/Doppler echocardiography, 12-lead electrocardiogram (ECG), heart rate, blood pressure, and personal health history. Cholesterol profile was determined but not used at entry screening. No participant had a history of heart disease, hypertension, or vascular disease at entry. Similarly, no participant was previously or currently taking medications that have an effect on the tested cardiovascular parameters.

Participation of controls ended after the first three consecutive evaluations due to health or motivational issues. Runners committed to follow up for as long as able to perform the treadmill protocol. By the 10th year (2005), one runner had moved and three became incapacitated, thus eight out of twelve originally recruited athletes completed the study.

All participants signed an informed consent, received report of test results, and were advised to discuss the findings with their primary care physician.

Athletic Data

Five-kilometer (3.1 miles) running race results between ages 65 and 75 from runners who completed the study were compiled, analyzed, and tabulated for rate of age-related slowing of athletic performance.

Study Protocol

Treadmill stress test—Treadmill activity was aimed at reaching maximal heart rate and blood pressure response. Each subject signaled when no longer able to continue with exercise. Stress echo recording began 1-minute postexercise. To suit both the untrained and trained subjects, the Balke treadmill protocol based on a steady 3.3 mph speed and an increase in incline from 2% to a maximum of 22% every 2 minutes was selected.⁴ If anaerobic threshold was not reached, treadmill speed was increased until inability to continue. Continuous breath-by-breath gas analysis using a one-way valve mouthpiece and apparatus (Medgraphics, St. Paul, MN, USA) was employed to obtain oxygen consumption (VO_2), anaerobic threshold (AT), and respiratory exchange ratio. A 12-lead ECG was recorded continuously, and upper arm blood pressure was measured by cuff and stethoscope at baseline, every change in treadmill incline and every 2-minutes during recovery.

M-mode and two-dimensional echo/Doppler recording—Imaging was recorded onto videotape from reclining subjects prior to the treadmill test, immediately after maximal effort and throughout recovery (Hewlett-Packard Sonos 5500, Andover, MA, USA). Left ventricular (LV) dimension, LV wall, and septal thickness as well as LV inflow Doppler velocities were quantitated. LV function parameters were calculated using established methodology.⁵

Cholesterol profile—Venous blood for 12-hour fasting cholesterol profile was drawn just prior to exercise.

Measurements

M-mode, two-dimensional, and Doppler echocardiographic measurements—

All subjects had echo Doppler evaluation in the left lateral decubitus position. From the echocardiographic study, videotape and digital data were acquired. Five cardiac cycles of high quality were identified for each echocardiographic parameter. LV internal dimensions were identified. Valvular regurgitant lesions were identified and graded. Transmitral filling dynamics was obtained.

Transmitral filling dynamics—Supine LV diastolic filling was assessed at three stages (resting, immediately after exercise within the first minute, and cessation of exercise within 3–5 minutes following) using two-dimensional guided transmitral, pulsed-wave, Doppler echocardiography. Echocardiographic studies were recorded using a Hewlett-Packard Sonos 5500 by the same operator from the apical four-chamber view using standard techniques. All subjects had initial pulsed- and continuous-wave Doppler recording of transmitral inflow and aortic outflow patterns with either the sample volume or the focal point (pulsed/continuous wave) placed on the ventricular side of the mitral annulus. After ensuring that the continuous-wave Doppler technique gave nearly identical Doppler waveforms and velocities, this was used for the collection of all data immediately after exercise and at 3–5 minutes into recovery because it allowed faster determination of Doppler waveforms than did the pulsed-wave method. Four indices of LV diastolic filling were determined in all three stages: peak early (E) filling velocity (cm/s), peak late or atrial (A) filling velocity (cm/s), peak E/A velocity ratio and atrial filling fraction, assessed by dividing the velocity-time integral of the A-wave (the area beneath the A-wave) by the total diastolic velocity-

time integral (the area beneath both E- and A-waves). The isovolumic relaxation time (IVRT; aortic valve closure to beginning of transmitral inflow) was determined using the Doppler waveform from the point that aortic outflow velocity fell to zero to the point that mitral inflow velocity increased above zero.

Respiratory parameters—Respiratory parameters were evaluated during the study including maximal oxygen consumption ($\text{VO}_{2\text{max}}$), maximum oxygen consumption percent predicted ($\text{VO}_{2\% \text{pred}}$), and oxygen consumption at anaerobic threshold ($\text{VO}_{2\text{thresh}}$). These parameters gave some evaluation of oxygen consumption and metabolism during exercise.

Statistical Analysis

SigmaStat 3.5 (Systat Software Inc., Point Richmond, CA, USA) was utilized for data analysis with the use of the Mann-Whitney rank sum test with a two-tailed $P \leq 0.05$ value to assess differences in parameters, oxygen consumption, and stress/Doppler echocardiographic indices between and within the exercise group and the sedentary control subjects. Data are reported as mean $\pm$ SD.

Results

Phase 1 (Athletes and Controls for Initial 3 Years)

Mean cholesterol profiles of athletes and sedentary controls, summarized for 1995–1998 period, are compared in Table I. Athletes and controls had comparable profiles on the whole. High-density lipoprotein (HDL) was relatively low for runners when compared to previously reported data.⁶ Table II demonstrates exercise data for the athletes and controls at baseline. During running or walking rapidly on treadmill, athletes scored better in duration of exercise, oxygen consumption, heart rate, and blood pressure when compared to controls. Echo/Doppler data including LV diastolic function indices (E/A ratio and IVRT) for the athletes and controls at baseline are shown in Table III.^{7,8} Similar to the treadmill data, mitral inflow velocities were better for athletes when compared to controls at rest and in recovery. The IVRT was shorter in the athletes than controls and cardiac dimensions were larger in the athletes when compared to controls.

Phase 2 (Athletes Only for 10 Years)

Age, BMI, and cholesterol profile (mean and 1 SD) at the study beginning (1995) and at conclusion (2005) are presented in Table IV. BMI and cholesterol profile did not change over the 10 years (LDL cholesterol <150 mg/dL was in the acceptable range throughout the study). Table V and Figure 1 illustrate race results and oxygen consumption treadmill data for the athletes over the 10 year study. Five-kilometer (3.1 miles) race times slowed during the 10 years as the athletes aged and the weekly training mileage decreased substantially. The training intensity and frequency also decreased. Race results were unchanged when age adjusted (WAVA/WMA standards). Peak exercise heart rate and oxygen consumption dropped, but were above average when normalized for age. The capacity to lower diastolic blood pressure during exercise was preserved throughout the study period. In Table VI, the echo/Doppler data over the 10 years for the athletes are summarized. After 10 years, diastolic LV function indices (E/A ratio and IVRT) deteriorated and cavity volume decreased. This is compatible with decreased cardiovascular diastolic function. Valvular indices and rhythm observations over the study period were determined. Regurgitation of mitral, aortic, and tricuspid valves increased, but aortic regurgitation was mild and rare. A small pressure gradient was noted across the aortic orifice in three out of eight subjects at the 10-year examination. Arrhythmias both at rest and during exercise became more frequent. The incidence of premature atrial contractions and premature ventricular

contractions increased during exercise, and short runs of asymptomatic supraventricular tachycardia were also observed.

Discussion

Beginning well before “young” old age, participation of men in competitive aerobic activities drops sharply. In dropouts, the role of habitual aerobic exertion in primary prevention of cardiovascular morbidity is diminished or absent. After age 65, participation in competitive activities, such as distance running, is markedly reduced. At that stage, muscular force and aerobic capacity are noticeably on a downward slope, arterial wall becomes stiffer, and musculoskeletal injuries heal slower. As well, age-related arterial dysfunction is greater when associated with sedentary lifestyle compared to a physically active lifestyle.⁹

This study points to health benefits of higher levels of aerobic exercise in the elderly runners. Compared to age-matched healthy sedentary controls, athletes had superior health indicators in all categories except for lipid profile (including HDL) and systolic blood pressure. These benefits, however, were more apparent when individuals are stressed. In the 10-year longitudinal study, the effect of aging on athletic performance and health indicators became evident. The high level of aerobic fitness was unable to appreciably slow deterioration of LV diastolic function. Cardiac valves stiffened and became progressively more regurgitant. Nevertheless, LV diastolic dimension and ejection fraction diminished presumably due to reduced training mileage and frequency of competitive events (reversal of “athletes heart” effect).

In the majority of our athletes, the decrease in achievement level is attributed to normative aging and appears to occur exponentially with increasing age. Drop in oxygen utilization and quicker muscle fatigue are major contributing factors. Additionally, LV diastolic function diminished (reversal of E/A ratio). Athletes were compensating for decreased diastolic filling by amplifying atrial contraction (A-wave), especially during strenuous exercise.

Maintaining physical fitness at older ages is hindered by progressive musculoskeletal deterioration and drop in tissue oxygen utilization. Consequently, pursuit of fitness levels achievable by sustained increases in heart and respiratory rates as a means of primary disease prevention will have limited appeal. Age group competitions offered by annual Senior State Games and Senior Olympics have the potential of increasing athletic participation, but need to be better promoted by the media. In 2005, 86 million Americans walked regularly for exercise.¹⁰

Runners in this study were competitive amateur athletes. Health benefits were considered secondary to their pursuit of fitness and generally taken for granted. Two participants (ages 80 and 83), while still athletic, experienced serious cardiovascular complications and were excluded from the final testing. Elderly men who score well on treadmill stress test are not necessarily immune from unexpected cardiovascular morbidity.¹¹ Absolute values of diastolic parameters are important in defining LV compliance, especially when differentiating between athletes’ heart and cardiomyopathy.¹² Test results were made available to each participant’s personal physician. Most athletes declined treatment of borderline or abnormal findings for fear of interfering with race performance.

There are some potential difficulties with our study. This study was based on a small segment of a large metropolitan population and its statistical power is low. The pool of volunteers suitable for our exercise protocol was limited. Assembling a larger cohort of elderly athletes and especially controls for a long-term treadmill follow-up is expected to

remain a formidable task. However, given these difficulties some parameters of cardiovascular fitness remain preserved in the elderly elite athletes.

Conclusion

Twelve male athletes (mean entry age 66 years) were tracked for 10 years to determine the value of vigorous aerobic exercise in preserving athletic stamina and cardiovascular diastolic function. Eight of the twelve recruited athletes completed the study.

The study confirms effectiveness of habitual aerobic exercise in health maintenance after the age of 65. However, its effectiveness diminishes as aging progresses. During the seventh and eighth decades, aging alone represents a serious cardiovascular risk factor.

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Figure 1.

Fastest 5-km (3.1 miles) race times (mean and 1 SD) of the 8 senior runners who remained in the study for a decade. On average, no appreciable slowing occurred until age 68. Thereafter, slowing rate averaged 30 seconds per year (10 seconds/mile). The more gifted the athlete, the slower the rate of aging.

Three-Year Summary Data on BMI and Lipid Profile in Athletes and Sedentary Controls: There Is No Difference in HDL Values

TABLE I

	Age (Years)	BMI* (kg/m ²)	Cholesterol* (mg/dL)	HDL (mg/dL)	LDL* (mg/dL)	Triglycerides* (mg/dL)
Athletes (n = 12)						
M	70	23	215	54	141	92
SD	1.5	0.4	3.0	10.0	5.0	1.0
Controls (n = 12)						
M	70	27	202	57	124	99
SD	0.9	0.8	16.0	7.0	22.0	1.0
P value	NS	0.001	0.012	NS	0.016	0.001

M = mean; SD = standard deviation; BMI = body mass index; HDL = high-density lipoproteins; LDL = low-density lipoproteins.

* P < 0.05 (athletes vs. controls).

Summary of 3-Year Treadmill Stress Test Data: Athletes Scored Better in Nearly All Categories Tested

TABLE II

	Exercise Duration (min)*	VO ₂ [*] max	VO ₂ [*] thresh	HR [*]		SBP [*]		DBP [*]	
				Rest	Max	Rest	Max	Rest	Max
Athletes (n = 8)									
M	24	44	38	56	170	140	190	75	67
SD	1.0	1.0	4.0	3.0	1.0	4.0	4.0	3.0	7.0
Controls (n = 8)									
M	13	28	25	69	159	139	179	79	81
SD	1.0	1.0	2.0	2.0	4.0	6.0	4.0	1.0	9.0
P value	0.001	0.001	0.001	0.001	0.001	0.06	0.001	0.001	0.001

M = mean; SD = standard deviation; VO₂max = maximal oxygen consumption (mL/kg per minute); VO₂thresh = oxygen consumption at anaerobic threshold (mL/kg per minute); HR = heart rate (beats/min); SBP = systolic blood pressure (mmHg); DBP = diastolic blood pressure (mmHg).

* P < 0.05 (athletes vs. controls).

Echo/Doppler Results Summarized for 3 Years: At Rest, Immediately Postexercise, and in Early Recovery

TABLE III

	E/A [*] Rest	E/A [*] Max	E/A [*] Recovery	IVRT [*] Rest	IVRT [*] Recovery	LVDD [*]	LVSD [*]
Athletes (n = 12)							
M	1.1	0.9	1.1	102	82	5.2	3.2
SD	0.03	0.07	0.04	5.0	3.5	0.10	0.10
Controls (n = 12)							
M	1.0	0.9	1.0	164	143	4.9	2.9
SD	0.03	0.15	0.04	53.0	45.0	0.10	0.10
P value	0.001	NS	0.001	0.001	0.001	0.001	0.001

M = mean; SD = standard deviation; E = rapid inflow; A = atrial systole; IVRT = isovolumic relaxation (ms); LVDD = left ventricular diastolic dimension (cm); LVSD = left ventricular systolic dimension (cm); NS = not significant.

* P < 0.05 (athletes vs. controls).

Effectiveness of habitual exercise on diastolic LV function indices is evident.

TABLE IV

Effects of Aging on Lipid Profiles of Athletes for Year 1 and Year 10: Results over the 10 Years Are Not Different

	Age (Years)*	BMI	Cholesterol (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	Triglycerides (mg/dL)
1995 (Year 1) (n = 8)						
M	66	23	214	49	147	96
SD	4.0	3.0	48.0	8.0	42.0	52.0
2005 (Year 10) (n = 8)						
M	77	24	204	52	130	107
SD	2.0	4.0	38.0	18.0	33.0	75.0
P value	0.001	NS	NS	NS	NS	NS

M = mean; SD = standard deviation; BMI = body mass index (kg/m^2); HDL = high-density lipoproteins; LDL = low-density lipoproteins; NS = not significant.* $P < 0.05$ (Year 1 vs. Year 10).

Slowing of 5 Kilometer (3.1 miles) Running Race Results in 10 Years

TABLE V

	5K [*] Race	5K % WAVA	HR Rest	HR [*] Max	SBP Rest	SBP Max	DBP Rest	DBP Max	VO ₂ [*] max	VO ₂ %pred	VO ₂ [*] thresh	METs
1995 (Year 1) (n = 8)												
M	23	76	59	172	144	189	75	68	43	158	35	12
SD	2.0	5.0	9.0	9.0	12.0	9.0	7.0	11.0	4.0	23.0	7.0	1.7
2005 (Year 10) (n = 8)												
M	30	75	58	156	141	181	78	70	38	169	22	11
SD	4.0	14.0	7.0	5.0	10.0	26.0	10.0	9.0	6.0	43.0	5.0	0.1
P value	0.001	NS	NS	0.001	NS	NS	NS	NS	0.05	NS	0.001	0.03

M = mean; SD = standard deviation; 5K race (minutes); WAVA = World Association of Veteran Athletes; % WAVA = % of world record; HR = heart rate (beats/min); SBP = systolic blood pressure (mmHg); DBP = diastolic blood pressure (mmHg); VO₂max = maximal oxygen consumption (mL/kg per minute); VO₂%pred = maximum oxygen consumption percentage predicted; VO₂thresh = oxygen consumption at anaerobic threshold (mL/kg per minute); METs = metabolic equivalents; NS = not significant.

* P < 0.05 (Year 1 vs. Year 10).

Group data summarized for the eight athletes include treadmill stress test data. Race times are shown in minutes and as percent of world records (WAVA standards). Peak heart rate and VO₂max declined as anticipated. Exercise intensity (METs) remained high.

TABLE VI

Cardiac Echo/Doppler Flow Measurements at Rest, after Maximal Effort, and during Early Recovery

	E/A* Rest	E/A* Max	E/A Recovery	AFF* Rest	AFF* Max	AFF Recovery	IVRT* Rest	IVRT* Recovery	LVDD*	LVSD*
1995 (Year 1) (n = 8)										
M	1.1	1.2	1.0	0.4	0.6	0.5	50	40	5.0	2.9
SD	0.18	0.37	0.21	0.07	0.11	0.12	10.0	10.0	0.50	0.30
2005 (Year 10) (n = 8)										
M	0.9	0.7	0.9	0.5	0.6	0.5	80	80	4.3	2.0
SD	0.15	0.10	0.25	0.04	0.05	0.04	20	10	0.50	0.80
P value	0.015	0.003	NS	0.003	NS	NS	0.001	0.001	0.004	0.001

M = mean; SD = standard deviation; E = rapid inflow; A = atrial systole; AFF = atrial filling fraction; IVRT = isovolumic relaxation (ms); LVDD = left ventricular diastolic dimension (cm); LVSD = left ventricular systolic dimension (cm); NS = not significant.

* P < 0.05 (Year 1 vs. Year 10).

The data confirmed the decline in diastolic LV function with aging. A decade of participation in distance training and racing did not preserve the LV function.