# Cardiac output and stroke volume changes with endurance training: The HERITAGE Family Study

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Department of Health and Kinesiology, Texas A&M University, College Station, TX; Physical Activity Sciences Laboratory, Laval University, Quebec City, Quebec, CANADA; Division of Biostatistics, Washington University School of Medicine, St. Louis, MO; School of Kinesiology and Leisure Studies, University of Minnesota, Minneapolis, MN; Department of Kinesiology, Indiana University, Bloomington, IN; and Pennington Biomedical Research Center, Baton Rouge, LA

#### ABSTRACT

WILMORE, J. H., P. R. STANFORTH, J. GAGNON, T. RICE, S. MANDEL, A. S. LEON, D. C. RAO, J. S. SKINNER, and C. BOUCHARD. Cardiac output and stroke volume changes with endurance training: The HERITAGE Family Study. Med. Sci. Sports Exerc., Vol. 33, No. 1, 2001, pp. 99–106. Purpose: The purpose of this study was to determine the magnitude of changes in cardiac output (Qc), stroke volume (SV), and arterial-mixed venous oxygen difference (a-vO, diff) during submaximal exercise following a 20-wk endurance training program, with the primary focus on identifying differences in response by race, sex, and age. Methods: The participants in this study (N = 631) were healthy and previously sedentary men (N = 277) and women (N = 354) of varying age (17-65 yr) and race (blacks, N = 217; whites, N = 414) who had completed the HERITAGE Family Study protocol. After baseline measurements, participants trained on cycle ergometers 3 d·wk<sup>-1</sup> for a total of 60 exercise sessions starting at the HR associated with 55% of maximal oxygen uptake ( $\dot{V}O_{2max}$ ) for 30 min/session and building to the HR associated with 75% of  $\dot{V}O_{2max}$  for 50 min/session, which was maintained during the last 6 wk. HR, Qc (CO2 rebreathing), and SV (Qc/HR) were determined in duplicate at 50 W and at 60% of  $\dot{VO}_{2max}$  on two different days both before and after training. **Results:** After training, there were significant decreases in HR and Qc, and significant increases in SV and a-vO2 diff at 50 W (except for no change in a-vO2 diff in black men). The changes in HR differed by sex and age, and the changes in SV, Qc, and  $a-\bar{v}O_2$  diff differed by race. Qc decreased by 0.6 L·min<sup>-1</sup> at 50 W for the total sample, consistent with the decrease in VO2 at this power output. At 60% of VO2max, HR decreased, and SV, Q, and a-vO2 diff increased. There were small differences in response by sex (HR and SV), race (HR), and age (HR and Qc). Conclusion: It is concluded that the cardiovascular systems of men and women, blacks and whites, and younger and older subjects are not limited in their ability to adapt to endurance training. Key Words: SUBMAXIMAL EXERCISE, VO2, a-vO2 DIFF, RACE, SEX, AGE

uring a single bout of exercise of increasing intensity, it is generally acknowledged that heart rate (HR) and cardiac output (Oc) increase in a near linear manner with increasing power outputs, whereas stroke volume (SV) tends to level off at between 40 and 60% of a person's maximal oxygen uptake ( $\dot{VO}_{2max}$ ) (28). Further, in response to endurance training, for the same absolute power output, Qc remains the same, or decreases slightly, whereas HR decreases and SV increases (28). However, it appears that there are differences in both the magnitude and direction of the cardiovascular responses to acute bouts of exercise and chronic exercise training on the basis of race, sex, age, and state of training. As an example, Spina et al. (27) were unable to find increases in peak Qc and SV in a group of older women, 60-70 yr, who had endurance-trained for 9-12 months despite a 21% increase in  $\dot{VO}_{2max}$ . Using radionuclide ventriculography, they were

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Received for publication April 2000. Accepted for publication April 2000. unable to detect any left ventricular enlargement or enhanced left ventricular systolic function. Previous studies from the same laboratory had found that older men, 60-70 yr, can increase peak SV and improve left ventricular systolic function (9,23), and that young women (mean age = 25 yr) respond similarly to young men (mean age = 25 yr) (26). Studies from our laboratory (JHW) support the similarity in the cardiovascular responses of young men and women (20–30 yr) to endurance training (18,19).

It has been well established that women have a smaller blood volume and heart size than men, even when expressed relative to body mass or fat-free mass (20). Further, women have a lower hemoglobin concentration and arterial oxygen content (20). As a result of these known differences, it is quite likely that women of all ages might adapt differently to endurance exercise training, even if they achieve similar increases in maximal endurance capacity.

Although it appears that both sex and aging can affect the cardiovascular response to endurance training, it is less clear if race is also a factor in modifying this response. Berry et al. (4) reported differences in the responses of black and white male subjects to an acute bout of exercise at 0, 50, and 100 W (W) on a cycle ergometer, with blacks having similar

TABLE 1. Pretraining physical characteristics of the participants.

Participants	Age (yr)	Height (cm)	Weight (kg)	BMI (kg·m <sup>−2</sup> )	BSA (m²)
All participants ( $N = 631$ )	34.6 ± 13.2	$169.5\pm9.6$	75.8 ± 17.1	$26.3\pm5.2$	$1.89 \pm 0.25$
By sex					
Men ( $N = 277$ )	35.7 ± 14.1	$177.5 \pm 6.4$	84.1 ± 15.9	$26.7 \pm 4.8$	$2.04 \pm 0.21$
17-29  yr (N = 126)	$22.8 \pm 3.8$	$178.3 \pm 6.3$	78.9 ± 15.6	$24.8 \pm 4.7$	$1.98 \pm 0.21$
30-49  yr (N = 89)	$39.2 \pm 6.6$	$177.4 \pm 6.6$	$89.9 \pm 14.9$	$28.6 \pm 4.5$	$2.12 \pm 0.20$
50-65  yr (N = 63)	56.1 ± 4.1	$176.2 \pm 6.0$	86.2 ± 14.7	27.7 ± 4.1	$2.06 \pm 0.20$
Women ( $N = 354$ )	$33.8 \pm 12.5$	$163.2 \pm 6.5$	69.3 ± 15.1	$26.0 \pm 5.5$	$1.78 \pm 0.21$
17-29 yr ( $N = 164$ )	$22.6 \pm 3.6$	$163.3 \pm 6.2$	64.5 ± 15.0	$24.1 \pm 5.4$	$1.71 \pm 0.21$
30-49  yr (N = 141)	$39.7 \pm 6.1$	$163.5 \pm 6.8$	72.9 ± 13.4	$27.3 \pm 5.0$	$1.83 \pm 0.19$
50-65  yr (N = 49)	$54.8 \pm 3.7$	$162.3 \pm 6.4$	75.5 ± 15.1	$28.7 \pm 5.3$	$1.86 \pm 0.21$
By race					
Blacks - total ( $N = 217$ )	$33.0 \pm 10.6$	$167.2 \pm 9.4$	76.9 ± 16.8	$27.5 \pm 5.7$	$1.90 \pm 0.24$
Men $(N = 77)$	$34.3 \pm 11.3$	$176.2 \pm 6.5$	83.4 ± 15.2	$26.8 \pm 4.6$	$2.03 \pm 0.21$
Women $(N = 140)$	$32.3 \pm 10.2$	$162.2 \pm 6.5$	73.3 ± 16.6	$27.9 \pm 6.2$	$1.83 \pm 0.23$
Whites - total $(N = 414)$	$35.5 \pm 14.4$	$170.7 \pm 9.5$	75.3 ± 17.2	$25.7 \pm 4.8$	$1.89 \pm 0.26$
Men $(N = 200)$	$36.2 \pm 15.0$	$178.0 \pm 6.2$	84.3 ± 16.2	$26.6 \pm 4.9$	$2.05 \pm 0.22$
Women ( $N = 214$ )	$34.9\pm13.7$	$163.9\pm6.4$	$66.8\pm13.5$	$24.8\pm4.7$	$1.75\pm0.20$

 $^{\star}$  Data are expressed as mean  $\pm$  SD.

Qc values, but lower HRs and a trend for higher SVs at each power output. It is possible that the observed differences in response to an acute bout of exercise between blacks and whites could also be present in the response of blacks and whites to endurance exercise training. To the best of our knowledge, this has never been studied.

Therefore, the purpose of this study was to determine the effects of a highly controlled, 20-wk endurance training program on Qc, SV, and arterial-mixed venous oxygen difference (a- $\bar{v}O_2$  diff) during submaximal exercise in a previously sedentary population of black and white participants in the HERITAGE Family Study. The HERITAGE Family Study is a large multicenter clinical trial investigating the possible genetic basis for the large variability in the responses of physiological measures, as well as risk factors for cardiovascular disease and type 2 diabetes mellitus, to endurance exercise training. This study includes four Clinical Centers [Indiana University (formerly at Arizona State University), the Pennington Biomedical Research Center (formerly Laval University, Québec, Canada), the University of Minnesota, and Texas A&M University (formerly at The University of Texas at Austin)] and a Data Coordinating Center (Washington University School of Medicine, St. Louis, MO). Details of the HERITAGE Family Study aims, experimental design, and measurement protocols have been presented in detail in a previous publication (5).

## **METHODS**

**Participants.** The HERITAGE Family Study subject population consisted of families, including the natural father and mother ( $\leq 65$  yr of age) and generally three offspring 17 yr of age or older for white families, or at least two firstdegree relatives for black families. Inclusion and exclusion criteria were summarized in detail in a prior publication (5). Specific criteria of importance to this paper included the fact that participants were sedentary at baseline, normotensive or mildly hypertensive (<160/100) without antihypertension medication, and had a body mass index (BMI) < 40.0kg·m<sup>-2</sup>. Participants were included in the study with BMIs slightly in excess of this value if they were considered by the supervising physician to be "healthy" and able to perform the required exercise tests and exercise prescription. A total of 744 participants finished all HERITAGE testing and training protocols. Of this total, 633 had complete cardiovascular data during submaximal exercise and constitute the sample for this paper. Their characteristics are presented in Table 1. Each Clinical Center's Institutional Review Board had previously approved the study protocol, and informed consent was obtained from each participant.

Experimental design and exercise test protocols. Participants were screened by the Clinical Center's supervising physician and staff, and only those who were previously sedentary, free of preexisting disease, and not taking medications that would affect any of the outcome variables were allowed to enter the study (5). A comprehensive battery of tests was administered before starting the training program, which included the following: health, medical, and nutrition questionnaires; maximal and submaximal exercise tests; blood tests for lipids, lipoproteins, and sex steroids; intravenous glucose tolerance test; resting blood pressure; and body composition tests. After the initial test battery, subjects completed a 20-wk endurance training program (3 d·wk<sup>-1</sup> for a total of 60 exercise sessions) on cycle ergometers that were computer-controlled to maintain the participants' HRs at levels associated with fixed percentages of their aerobic capacity ( $\dot{VO}_{2max}$ ). The training program started at 55% of  $\dot{V}O_{2max}$  for 30 min/session and gradually increased to 75% of  $\dot{V}O_{2max}$  for 50 min/session, which was maintained during the last 6 wk of training. The full test battery was administered again at the conclusion of the training program.

Subjects completed a total of three exercise tests, each on a different day, both before and after the period of exercise training. These included the following: a maximal test (MAX), a submaximal test (SUBMAX), and a submaximal to maximal test (SUBMAX/MAX). All exercise tests were conducted on a cycle ergometer (SensorMedics Ergo-Metrics 800S, Yorba Linda, CA). Subjects completed the initial MAX exercise test using a graded exercise test protocol, starting at 50 W for 3 min. The rate of work was then increased by 25 W every 2 min thereafter to the point of exhaustion. For older, smaller, or less fit subjects, the test was started at 40 W and increased by 10- to 20-W increments. Using the results of this initial maximal test, subjects then performed the SUBMAX exercise test on a second day at 50 W and at a power output equivalent to 60% of their initial  $\dot{VO}_{2max}$ . The SUBMAX/MAX exercise test was then performed on a third day, starting with the SUBMAX protocol, i.e., 50 W and 60% of the initial  $\dot{VO}_{2max}$ , and continuing with the MAX test protocol to a maximal level of exertion. The results of the three exercise tests were used to determine the reproducibility of measurements (24,31), as well as to establish the endurance training program power outputs and to quantify the magnitude of the training response (5).

Cardiovascular and metabolic measurements. For the SUBMAX and SUBMAX/MAX tests, two HR and Qc values were obtained and averaged both at 50 W and at 60% of the initial VO<sub>2max</sub>, pre- and post-training. Subjects exercised for approximately 12-15 min at each power output, with a 4-min period of seated rest between power outputs. The values presented in this paper represent the mean of the responses at each power output for the two submaximal tests, both before and after training. Qc was determined using the Collier  $CO_2$  rebreathing technique (6), as described by Wilmore et al. (30). Each Clinical Center used the same electronic mixing system to assure the proper volume and concentration of CO<sub>2</sub> for rebreathing dependent on each subject's steady state  $\dot{VO}_2$  and end-tidal pCO<sub>2</sub>. SV was derived by dividing the estimated Qc by the measured HR at the time of the Qc determination (i.e., SV = Qc/HR). The a- $\bar{v}O_2$  diff was determined by dividing the  $\dot{V}O_2$  for a given power output by the Qc for that power output (values expressed in mL of  $O_2 \cdot 100 \text{ mL}^{-1}$  of blood).

For all three tests,  $\dot{VO}_2$ ,  $\dot{VCO}_2$ , expiratory minute ventilation ( $\dot{VE}$ ), and the respiratory exchange ratio (RER) were determined every 20 s and reported as a rolling average of the three most recent 20-s values, using a SensorMedics 2900 metabolic measurement cart. The  $\dot{VO}_{2max}$  was defined as the peak  $\dot{VO}_2$  obtained during the test. Determined by electrocardiography, HR was recorded during the last 15 s of each stage of the MAX test and once steady state had been achieved at each of the submaximal power outputs during the SUBMAX and SUBMAX/MAX tests.

Quality assurance, quality control, and statistical methodology. Important quality assurance and quality control procedures were instituted across all four Clinical Centers, as described by Gagnon et al. (11). Staff from all Clinical Centers were trained centrally on several occasions, and all staff had to be certified on each technique for which they were responsible. A detailed Manual of Procedures (MOP) was developed, and staff were required to review those sections of the MOP for which they were responsible every 6 months. The reproducibility of measurements in this study was very high as has been previously reported (31). Coefficients of variation (CVs) for the HR, blood pressure, and Qc at 50 W and 60% of  $\dot{V}O_{2max}$  ranged from 4.4 (HR at 60%) to 7.6 (SV at 50 W), and intraclass correlation coefficients (ICCs) ranged from 0.76 (Qc at 50 W) to 0.93 (Qc at 60%).

All data were analyzed using the SAS statistical package (version 6.12; SAS Institute Inc. Cary, NC). Data are expressed as mean  $\pm$  SD except where noted. For all comparisons across groups, Qc and SV were statistically adjusted for size using body surface area as determined by the equation of DuBois and DuBois (8). A matched-pair *t*-test was used to determine significant differences between preand post-training data. A multiple testing analysis of variance (ANOVA) was implemented by using the general linear models procedure to determine the influence of sex, age (younger: 17–29 yr; middle-aged: 30–49 yr; older: 50–65 yr), and race (blacks vs whites) on the magnitude of change in any given variable. Statistical significance was established at the 0.05 level.

## RESULTS

The mean increase in  $\dot{VO}_{2max}$  consequent to the 20-wk endurance training program was 16.2%, with a variation among subjects of from 0 to 51%. Decreases in exercise HR at each power output posttraining compared with pretraining further confirmed a substantial training response.

The pre- and posttraining data for the cardiovascular responses to submaximal exercise are presented in Table 2 (50 W data) and Table 3 (60% of  $\dot{V}O_2$  max data). At the same absolute power output (50 W), there were significant decreases in HR and Qc, and significant increases in SV and  $a-\bar{v}O_2$  diff for the total sample and all subgroups, with the exception of no increase in  $a-\bar{v}O_2$  diff in blacks. Within subgroups, women had a much greater decrease in HR at 50 W than men, but their mean pretraining value was 23 beats·min<sup>-1</sup> greater. Further, older women had a greater decrease in HR than the younger and middle-aged women. Blacks had a greater increase in SV than whites, but this was significant only in men. Whites had a greater decrease in Qc compared with blacks, but this was significant only in men.

The same relative power output (60% O<sub>2max</sub>) represented a higher power output posttraining compared with pretraining. There were significant decreases in HR and significant increases in SV, Qc, and  $a-\bar{v}O_2$  diff posttraining for the total sample and all subgroups. With respect to HR, men experienced a greater decrease compared with women, and older women experienced a greater decrease compared with middle-aged women. A racial difference was also noted, but this was significant only for women, with white women experiencing a greater decrease in HR compared with black women. SV increased substantially in the total group (+10.8%) and in all subgroups (range: mean from 10.1% to 11.1%), with men experiencing a small, but significantly greater increase than women (11.1% vs 10.6%). Qc increased equally in all groups (range: 6.9-8.4%), with the exception of the older women (2.8%). There was a similar increase in  $a-\bar{v}O_2$  diff posttraining across all groups.

### DISCUSSION

For the total sample, the changes in HR, SV, Qc, and  $a-\bar{v}O_2$  diff at 50 W consequent to training were in the

TABLE 2.	Changes	in	cardiovascular	variables	and	a-v02	diff	at	50	Watts
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Variable	Pretraining (Mean $\pm$ SD)	Posttraining (Mean $\pm$ SD)	Absolute Difference (post - pre)	Significant Difference* ( $P < 0.05$ )
Heart rate (beats/min $^{-1}$ )				
Total	$120.2 \pm 18.4$	$108.8 \pm 14.4$	-11.4	a
Men - total	$10/.1 \pm 11.8$	98.3 ± 9.1	-8.8	a, b
30-49 yr	$100.0 \pm 10.0$ $107.8 \pm 12.0$	$90.3 \pm 0.0$ 98 9 + 9 3	-0.5 -8 9	
50–65 yr	$107.0 \pm 13.8$	97.2 ± 9.8	-9.8	_
5	107.0 ± 13.8	97.2 ± 9.8	-9.8	_
Women - total	$130.5 \pm 16.1$	117.0 ± 12.1	-13.5	a, b
17–29 yr	$132.0 \pm 15.6$	$118.9 \pm 11.4$	-13.1	g
30-49 yr 50-65 yr	$120.9 \pm 17.0$ 129.6 + 14.8	110.0 ± 12.7 113.5 + 12.1	- 12.9 	y e f
Blacks - total	125.8 + 19.4	$113.5 \pm 12.1$ $113.6 \pm 14.5$	-12.2	a.
Men	$108.9 \pm 11.8$	$99.9 \pm 8.3$	-9.0	a
Women	135.2 ± 16.3	$121.2 \pm 11.3$	-14.0	а
Whites - total	$117.2 \pm 17.2$	$106.2 \pm 13.6$	-11.0	a
Men	$106.4 \pm 11.7$	$97.6 \pm 9.4$	-8.8	a
WUITEIT	127.4 ± 15.2	114.3 ± 11.9	- 13.1	d
Stroke volume (mL/beat <sup>-1</sup> )				
Total	95.9 ± 18.9	99.8 ± 18.6	+3.9	а
Men - total	$108.7 \pm 16.7$	$112.4 \pm 16.9$	+3.7	a
17-29 yr 30-49 yr	112.0 ± 15.4 107.2 + 16.2	110.4 ± 10.2 112.0 + 17.6	+2.0	
50–65 vr	$107.2 \pm 10.2$ $103.1 \pm 18.4$	$106.8 \pm 15.9$	+3.7	_
Women - total	85.9 ± 13.7	90.0 ± 13.2	+4.1	а
17–29 yr	84.5 ± 14.2	88.3 ± 12.9	+3.8	—
30–49 yr	88.1 ± 13.0	91.7 ± 13.3	+3.6	—
50–65 yr	$84.6 \pm 13.6$	$90.6 \pm 13.3$	+6.0	—
Men	94.4 ± 10.1 110.1 + 15.0	99.7 ± 10.9 116.5 + 17.3	+5.3 +6.4	a, c a d
Women	85.8 + 13.3	$905 \pm 124$	+47	a, u a
Whites - total	96.7 ± 19.2	99.8 ± 18.5	+3.1	a, c
Men	108.1 ± 17.3	$110.7 \pm 16.5$	+2.6	a, d
Women	86.0 ± 14.0	89.6 ± 13.7	+3.6	a
Cardiac output (L/min <sup>-1</sup> )				
Total	$11.3 \pm 1.4$	10.7 ± 1.4	-0.6	а
Men - total	$11.5 \pm 1.5$	$11.0 \pm 1.4$	-0.5	а
17–29 yr	$11.9 \pm 1.4$	$11.3 \pm 1.4$	-0.6	—
30–49 yr	$11.4 \pm 1.3$	$11.0 \pm 1.5$ 10.2 $\pm$ 1.2	-0.4	—
SU-OS YI Women - total	10.9 ± 1.7 11.0 + 1.3	$10.3 \pm 1.3$ $10.4 \pm 1.3$	-0.0 -0.6	
17–29 vr	$11.0 \pm 1.0$	$10.4 \pm 1.3$ $10.4 \pm 1.2$	-0.6	<u> </u>
30–49 yr	$11.2 \pm 1.2$	$10.5 \pm 1.3$	-0.7	_
50–65 yr	$10.8 \pm 1.5$	$10.2 \pm 1.4$	-0.6	—
Blacks - total	$11.6 \pm 1.3$	$11.1 \pm 1.4$	-0.5	a, c
Men Women	$11.9 \pm 1.3$	$11.6 \pm 1.5$ 10.0 $\pm$ 1.2	-0.3	a, d
Women Whites - total	$11.0 \pm 1.3$ 11.1 + 1.5	$10.9 \pm 1.3$ $10.4 \pm 1.3$	-0.0	a
Men	$11.4 \pm 1.6$	$10.7 \pm 1.0$	-0.7	a, d
Women	$10.8\pm1.3$	$10.1 \pm 1.2$	-0.7	a
$a-\bar{v}O_{-}$ diff (ml /100 ml $^{-1}$ )				
Total	92 + 12	94 + 11	+0.2	а
Men - total	$9.6 \pm 1.3$	9.7 ± 1.2	+0.1	a
17–29 yr	9.0 ± 1.0	9.2 ± 1.1	+0.2	—
30–49 yr	9.9 ± 1.2	$10.0 \pm 1.3$	+0.1	—
50-65 yr Women - total	$10.2 \pm 1.5$ 80 + 10	10.3 ± 1.1 01 + 10	+0.1	
17–29 vr	0.5 ± 1.0 87 + 0.0	5.1 ± 1.0 89 + 10	+0.2	<u>a</u>
30–49 vr	$9.0 \pm 0.9$	$9.2 \pm 1.0$	+0.2	_
50–65 yr	$9.5 \pm 1.1$	9.7 ± 1.1	+0.2	
Blacks - total	9.1 ± 1.1	9.1 ± 1.1	0.0	C
Men	9.3 ± 1.1	$9.3 \pm 1.3$	0.0	
Women Whitee - total	8.9 ± 1.0	9.0 ± 1.0 0.5 ± 1.1	+0.1	—
Men	9.3 ± 1.2 9.7 + 1.4	9.5 ± 1.1 9.9 + 1.2	+0.2	a, u a
Women	9.0 ± 1.0	9.2 ± 1.0	+0.2	a

\* a, significant difference pre- to posttraining; b, significant difference between men and women; c, significant difference between blacks and whites; d, significant difference between black men and white men; e, significant difference compared with the 17 to 29-year-old group; f, significant difference compared with the 30 to 49-year-old group; g, significant difference compared with the 50 to 65-year-old group.

direction and of the magnitude expected on the basis of the existing research literature with one notable exception: black men did not increase  $a-\bar{v}O_2$  diff posttraining. To achieve the same  $\dot{V}O_2$  at 50 W posttraining, black men had a greater increase in SV (+6.4 vs +2.6 mL·beat<sup>-1</sup>) and a

smaller decrease in Qc  $(-0.3 \text{ vs} -0.7 \text{ L} \cdot \text{min}^{-1})$  compared with white men. Thus, black men achieved the appropriate  $\dot{VO}_2$  at 50 W posttraining by maintaining Qc rather than increasing  $a-\bar{v}O_2$  diff, whereas white men increased  $a-\bar{v}O_2$ diff, allowing Qc to decrease. A similar trend was noted for

TABLE 3.	Changes i	n c	ardiovascular	variables	and	a-v02	diff	at	60%	of	V0 <sub>2m</sub>	

Variable	Pretraining (Mean $\pm$ SD)	Posttraining (Mean ± SD)	Absolute Difference (post - pre)	Significant Difference* ( $P < 0.05$ )
Heart rate (beats/min $^{-1}$ )				
Total	140.1 ± 16.3	135.8 ± 14.8	-4.3	а
Men - total	138.1 ± 15.6	132.8 ± 13.3	-5.3	a, b
17–29 yr	145.7 ± 12.5	139.6 ± 10.3	-6.2	—
30–49 yr	135.6 ± 13.4	131.4 ± 10.6	-4.2	—
50–65 yr	126.4 ± 15.6	121.1 ± 13.5	-5.3	—
Women - total	141.7 ± 16.7	138.2 ± 15.5	-3.5	a, b
17–29 yr	150.9 ± 13.8	147.1 ± 12.5	-3.8	—
30–49 yr	135.9 ± 14.8	134.0 ± 12.6	-1.9	g
50–65 yr	127.4 ± 13.4	120.2 ± 11.2	-7.2	f
Blacks - total	137.4 ± 14.6	$134.7 \pm 13.0$	-2.7	a, c
Men	134.6 ± 13.1	$130.7 \pm 11.5$	-3.9	а
Women	138.9 ± 15.2	136.9 ± 13.3	-2.0	a, d
Whites - total	141.5 ± 17.0	$136.4 \pm 15.6$	-5.1	а, с
Men	139.4 ± 16.2	$133.6 \pm 13.9$	-5.8	а
Women	143.5 ± 17.5	139.0 ± 16.7	-4.5	a, d
Stroke volume (ml /beat $^{-1}$ )				
Total	98.6 + 22.2	109 2 + 23 6	+10.6	а
Men - total	$114.8 \pm 19.8$	$100.2 \pm 20.0$ $1275 \pm 192$	+10.0	ah
17–29 vr	$119.0 \pm 13.0$ $119.1 \pm 17.7$	$127.0 \pm 10.2$ 132.1 + 18.5	+12.7	a, b
30-49 yr	$115.0 \pm 20.7$	$128.0 \pm 17.7$	+13.0	_
50-45 yr	$105.8 \pm 10.8$	$120.0 \pm 17.7$ $1175 \pm 19.3$	+11.7	_
Women - total	$85.9 \pm 14.5$	$95.0 \pm 15.0$	+91	a h
17–29 vr	$845 \pm 148$	$93.8 \pm 15.0$	+9.3	<u> </u>
30–49 vr	$87.9 \pm 14.5$	$97.3 \pm 15.7$	+9.4	_
50–65 vr	85 2 + 12 7	$924 \pm 154$	+72	_
Black - total	$96.5 \pm 21.3$	$106.5 \pm 22.1$	+10.0	а
Men	$115.1 \pm 19.8$	$127.8 \pm 17.4$	+12.7	a
Women	86.2 ± 13.9	94.7 ± 14.2	+8.5	a
Whites - total	99.7 ± 22.6	110.7 ± 24.2	+11.0	а
Men	114.6 ± 19.9	127.3 ± 19.9	+12.7	а
Women	85.8 ± 14.8	95.1 ± 16.2	+9.3	a
Cardiac output (L/min <sup>-1</sup> )				
Total	137 + 31	147 + 31	+10	а
Men - total	$15.8 \pm 2.9$	$169 \pm 29$	+11	a
17–29 vr	$17.3 \pm 2.3$	$18.4 \pm 2.4$	+1.1	_
30–49 vr	$15.5 \pm 2.8$	$16.8 \pm 2.3$	+1.3	_
50–65 yr	$13.2 \pm 2.2$	$14.1 \pm 2.2$	+0.9	_
Women - total	$12.0 \pm 2.0$	$13.0 \pm 2.2$	+1.0	а
17–29 vr	12.6 ± 1.9	$13.7 \pm 2.0$	+1.1	q
30–49 yr	11.9 ± 2.0	$12.9 \pm 2.1$	+1.0	ğ
50–65 yr	10.7 ± 1.6	$11.0 \pm 1.7$	+0.3	e, f
Blacks - total	13.1 ± 2.8	$14.2 \pm 2.7$	+1.1	а
Men	$15.4 \pm 2.6$	$16.6 \pm 2.3$	+1.2	а
Women	11.9 ± 1.9	$12.9 \pm 1.9$	+1.0	а
Whites - total	14.0 ± 3.2	$15.0 \pm 3.3$	+1.0	а
Men	$15.9 \pm 3.0$	$17.0 \pm 3.0$	+1.1	a
Women	$12.2 \pm 2.0$	$13.1 \pm 2.3$	+0.9	a
$a-\bar{v}O_{2}$ diff (mL/100 mL <sup>-1</sup> )				
Total	$10.3 \pm 1.6$	$10.9 \pm 1.6$	+0.6	а
Men - total	$11.4 \pm 1.3$	$12.1 \pm 1.3$	+0.7	а
17–29 yr	$11.4 \pm 1.3$	$12.1 \pm 1.3$	+0.7	
30–49 yr	11.6 ± 1.2	12.3 ± 1.3	+0.7	_
50–65 yr	$11.3 \pm 1.5$	11.9 ± 1.4	+0.6	—
Women - total	9.5 ± 1.2	10.1 ± 1.2	+0.6	a
17–29 yr	9.6 ± 1.1	$10.1 \pm 1.1$	+0.5	—
30–49 yr	9.3 ± 1.2	$10.0 \pm 1.2$	+0.7	—
50–65 yr	9.4 ± 1.2	$10.1 \pm 1.3$	+0.7	—
Blacks - total	9.7 ± 1.5	$10.3 \pm 1.5$	+0.6	а
Men	$10.8 \pm 1.3$	$11.5 \pm 1.4$	+0.7	а
Women	9.1 ± 1.2	9.7 ± 1.2	+0.6	a
Whites - total	10.b ± 1.5	$11.3 \pm 1.5$	+0.7	a
women	11.0 ± 1.3 97 + 1 0	12.3 ± 1.2 10.3 + 1.1	+0./ +0.6	d
WOITIGH	$J_{11} \doteq 1.0$	10.0 - 1.1	10.0	ci (i

\* a, significant difference pre- to posttraining; b, significant difference between men and women; c, significant difference between blacks and whites; d, significant difference between black women and white women; e, significant difference compared with the 17 to 29-year-old group; f, significant difference compared with the 30 to 49-year-old group; g, significant difference compared with the 50 to 65-year-old group.

black and white women, but it was not statistically significant. There is no obvious explanation for this difference in response between black and white men. Further, this difference was not present at 60% of  $\dot{V}O_{2max}$ . Older women had a greater decrease in HR than the middle-aged and younger women, but the greatest difference was only 3.2 beats min<sup>-1</sup>. There were no other sex, race or age differences in response to training at 50 W.

The decrease of 0.6 L·min<sup>-1</sup> in Qc (-5.3%) at 50 W posttraining was not unexpected. Although most studies

TABLE 4. Changes in maximal heart rate and estimated maximal cardiac output.

Variable	Pretraining (Mean ± SD)	Posttraining (Mean $\pm$ SD)	Absolute Difference (post - pre)	Significant difference* (P < 0.05)
Heart rate (beats/min $^{-1}$ )				
Total	184.9 ± 13.9	184.3 ± 12.8	-0.6	а
Men - total	$185.3 \pm 14.3$	$183.2 \pm 13.4$	-2.1	a. b
17–29 vr	$194.0 \pm 9.1$	190.8 ± 8.8	-3.2	
30–49 yr	$182.2 \pm 12.1$	$181.2 \pm 11.1$	-1.0	_
50–65 yr	172.4 ± 14.4	170.7 ± 13.7	-1.7	_
Women - total	184.6 ± 13.6	185.1 ± 12.3	+0.5	b
17–29 yr	192.0 ± 9.5	$192.2 \pm 8.9$	+0.2	—
30–49 yr	180.9 ± 12.0	181.6 ± 10.4	+0.7	—
50–65 yr	170.0 ± 13.5	171.2 ± 11.3	+1.2	—
Blacks - total	183.0 ± 14.0	183.3 ± 12.7	+0.3	С
Men	182.1 ± 14.1	181.8 ± 13.3	-0.3	d
Women	183.5 ± 14.0	184.2 ± 12.2	+0.7	—
Whites - total	185.9 ± 13.8	184.7 ± 12.9	-1.2	a, c
Men	186.5 ± 14.3	183.7 ± 13.4	-2.8	a, d
Women	185.3 ± 13.3	185.6 ± 12.4	+0.3	_
Cardiac output $(I/min^{-1})$				
Total	$18.2 \pm 4.3$	$20.1 \pm 4.4$	+1.9	а
Men - total	$21.3 \pm 4.0$	$23.3 \pm 3.9$	+2.0	a, b
17–29 vr	$23.1 \pm 3.4$	$25.2 \pm 3.3$	+2.1	
30–49 yr	$20.9 \pm 3.9$	$23.2 \pm 3.3$	+2.3	_
50–65 yr	18.1 ± 3.2	$20.0 \pm 3.2$	+1.9	_
Women - total	$15.8 \pm 2.7$	$17.5 \pm 2.8$	+1.7	a, b
17–29 yr	$16.2 \pm 2.8$	18.0 ± 2.7	+1.8	
30–49 yr	15.9 ± 2.7	17.6 ± 2.8	+1.7	_
50–65 yr	$14.4 \pm 2.2$	$15.7 \pm 2.4$	+1.3	_
Blacks - total	17.6 ± 3.9	$19.5 \pm 4.0$	+1.9	а
Men	$20.9 \pm 3.7$	$23.2 \pm 3.2$	+2.3	а
Women	15.8 ± 2.6	17.4 ± 2.6	+1.6	а
Whites - total	18.5 ± 4.4	$20.4 \pm 4.6$	+1.9	а
Men	21.4 ± 4.1	23.4 ± 4.1	+2.0	а
Women	$15.9 \pm 2.8$	17.6 ± 2.9	+1.7	а

<sup>\*</sup> a, significant difference pre- to posttraining; b, significant difference between men and women; c, significant difference between blacks and whites; d, significant difference between black men and white men; e, significant difference compared with the 17 to 29-year-old group; f, significant difference compared with the 30 to 49-year-old group; g, significant difference compared with the 50 to 65-year-old group.

have reported no change in Qc at the same absolute submaximal power output posttraining (3), some have not (1,10). Andrew et al. (1) and Ekblom et al. (10) reported posttraining decreases in Qc of 1.5 and 1.6 L·min<sup>-1</sup>, respectively, at approximately the same power output. The decline in Qc posttraining was associated with a decline of similar magnitude in  $\dot{VO}_2$  (-3.5%). Several investigators have reported a decline in  $\dot{V}O_2$  at the same absolute submaximal power output posttraining (12,29,32), but others have not (13,15,16). The subjects in the present study obviously became more efficient over the course of the 20-wk cycle ergometer training period. We had attempted to prevent a "learning effect" by providing the subjects with practice trials on the cycle ergometer before the initial tests and by having the subjects perform three exercise tests before starting their training programs. It is likely that this represents a true increase in efficiency.

At 60% of  $\dot{VO}_{2max}$ , subjects were exercising at higher power outputs posttraining, as reflected by a significantly higher  $\dot{VO}_2$  posttraining (1.63 L·min<sup>-1</sup> vs 1.43 L·min<sup>-1</sup>). This increase in  $\dot{VO}_2$  was the result of an increase in both Qc and a- $\bar{vO}_2$  diff. The increase in Qc was solely the result of a large increase in SV, as HR actually decreased. There were statistically significant differences in the response to training by sex (HR and SV), race (HR for women), and age (HR and Qc for older women), but these differences, with one exception, were small and of little physiological significance. For older women, the increase in Qc was less than one-third of that observed in the middle-aged and younger women, yet their HR decreased to a much greater extent.

Although values for Qc, SV, and  $a-\bar{v}O_2$  diff were not obtained at each subject's maximal power output, Qc max for each subject was estimated by multiplying SV at 60%  $\dot{VO}_{2max}$  by HR max. This approach has been used by others in the past based on previous research that demonstrates that SV values plateau between 40% and 60%  $\dot{V}O_{2max}$  in untrained or moderately trained subjects (2,17,21). Highly trained subjects, however, do appear to be able to increase their SV up through maximal power outputs when compared with untrained or moderately trained subjects (7,14,22). Estimated Qc max increased from 18.2 to 20.1 L·min<sup>-1</sup> for the total sample (10.4%). Men had a slightly greater absolute increase than women (2.0 vs 1.7 L·min<sup>-1</sup>), whereas women had a greater relative increase (10.8% vs 9.4%). There were no other significant differences between subgroups (see Table 4).

Spina et al. (26) have reported that SV may decline in untrained individuals between 50% and 100% of  $\dot{V}O_{2max}$ , and this might be reversed with training. Thus, recognizing these limitations in estimating Qc max, it appears that the there were no differences by sex, age, or race in the ability to increase Qc max with training. Spina et al. have reported in two studies of older women, 60–70 yr of age, that 9–12 months of endurance training increased  $\dot{V}O_{2max}$  21% (27) and 22% (25). However, the increase in both studies was the

result of an increase in  $a-\bar{v}O_2$  diff, and not Qc. SV max remained unchanged in both studies. The authors postulated that the inability of these older women to increase SV could be related to estrogen deficiency. The older women in the present study were younger, and not all were postmenopausal. Further, some were on estrogen replacement therapy. This could explain why the older women in the present study increased their SV at 60% of  $\dot{VO}_{2max}$  and their estimated Qc at maximal power output consequent to training. Their absolute (and relative) increase in estimated Oc max was 1.3 L·min<sup>-1</sup> (9.0%) compared with 1.8 and 1.7 L·min<sup>-1</sup> (11.1% and 10.7%) in the younger and middle-aged groups, respectively. Therefore, the trend for an attenuated response in estimated Qc max was there, but it was not statistically significant.

In summary, the alterations in cardiovascular function resulting from 20 wk of endurance training were very similar across sex, age, and race at both the same absolute (50 W) and relative (60% of  $\dot{V}O_{2max}$ ) rates of work, with few exceptions. Estimated SV max and Qc max also increased by a similar amount in all subgroups. The most notable

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difference between subgroups was the failure of black men to increase  $a-\bar{v}O_2$  diff at 50 W compared with white men. They compensated for this by increasing their SV to a greater extent than the white men. There was a substantial decrease in Qc at 50 W after training, which was associated with a decrease of similar magnitude in  $\dot{V}O_2$ .

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