# Comparison of Central Artery Elasticity in Swimmers, Runners, and the Sedentary

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**Although swimming is one of the most popular, most practiced, and most recommended forms of physical activity, little information is available regarding the influence of regular swimming on vascular disease risks. Using a cross-sectional study design, key measurements of vascular function were performed in middle-aged and older swimmers, runners, and sedentary controls. There were no group differences in age, height, dietary intake, and fasting plasma concentrations of glucose, total cholesterol, and low-density lipoprotein cholesterol. Runners and swimmers were not different in their weekly training volume. Brachial systolic blood pressure and pulse pressure were higher ($p < 0.05$) in swimmers than in sedentary controls and runners. Runners and swimmers had lower ($p < 0.05$) carotid systolic blood pressure and carotid pulse pressure than sedentary controls. Carotid arterial compliance was higher ($p < 0.05$) and β-stiffness index was lower ($p < 0.05$) in runners and swimmers than in sedentary controls. There were no significant group differences between runners and swimmers. Cardiovagal baroreflex sensitivity was greater ($p < 0.05$) in runners than in sedentary controls and swimmers and baroreflex sensitivity tended to be higher in swimmers than in sedentary controls ($p = 0.07$). Brachial artery flow-mediated dilation was significant greater ($p < 0.05$) in runners compared with sedentary controls and swimmers. In conclusion, our present findings are consistent with the notion that habitual swimming exercise may be an effective endurance exercise for preventing loss in central arterial compliance.**  **(Am J Cardiol 2011;107:783–787)**

Swimming can be an ideal mode of exercise for those at increased risk of vascular disease including elderly patients, obese patients, and patients with arthritis. Although swimming is widely promoted and recommended as a mode of aerobic exercise by national and international organizations,[1–3] research focusing on influences of swimming on vascular disease risks is lacking.[4] The aims of the present investigation were to determine (1) whether swimmers would demonstrate higher levels of key vascular function measurements (i.e., central artery compliance, arterial baroreflex sensitivity, and endothelial-dependent vasodilation) than sedentary controls and (2) if levels of such vascular measurements are different from runners who are matched for age and exercise training status.

## Methods

We studied 75 apparently healthy middle-aged and older adults (37 to 75 years of age). They were swimmers, runners, or sedentary controls. All subjects were nonobese, nonsmoking, normotensive (<140/90 mm Hg), normolipidemic, and free of overt cardiovascular and other chronic diseases as assessed by medical history questionnaire, blood chemistry, and hematologic evaluation. None of the subjects were taking cardiovascular-acting medications including hormone replacement therapy. Physical activity status was verified by a modified physical activity questionnaire[5] and maximal oxygen consumption. In average, runners and swimmers had been exercising 4.1 ± 2.2 times/week for 9 ± 2 years and 4.8 ± 1.1 times/week for 9 ± 2 years, respectively. Sedentary participants had been sedentary at least for the previous 12 months. All subjects gave their written informed consent to participate. The study was reviewed and approved by the institutional review board.

Before they were tested, subjects abstained from food, alcohol, and caffeine for ≥4 hours (overnight 12-hour fast for metabolic risk factors). Premenopausal women were tested during the early follicular phase of the menstrual cycle. All testing was performed 24 to 48 hours after the last exercise bout.[6]

Body composition was assessed using dual-energy x-ray absorptiometry (Lunar DPX, GE Medical, Fairfield, Connecticut). A 3-day dietary intake record was obtained and analyzed by a registered dietitian using Nutritionist Pro software (Axxya Systems, Stafford, Texas). A blood sample was collected by venipuncture after an overnight fast. Fasting plasma concentrations of glucose, lipids, and lipoproteins were determined using a Vitros DT60 analyzer (Ortho-Clinical Diagnostics, Raritan, New Jersey). Graded exercise testing was undertaken using a metabolic cart during a

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modified Bruce protocol. After a 5-minute warm-up, subjects walked or ran while the treadmill slope was gradually increased 2% every 2 minutes until volitional exhaustion.

Bilateral brachial and ankle blood pressures, carotid and femoral pulse waves, and heart rate were measured by an automated vascular testing device (VP-2000, Omron Healthcare Bannockburn, Illinois)[7] after a subject had been lying in a supine position for ≥15 minutes. Ankle–brachial pressure index was calculated as ankle systolic blood pressure divided by brachial systolic blood pressure. Carotid and femoral artery pulse waves were recorded by arterial applanation tonometry incorporating an array of 15 micropiezoresistive transducers placed on the carotid and femoral arteries. Time delay was measured automatically with the foot-to-foot method, and pulse wave velocity was subsequently calculated. Augmentation index, an index of arterial wave reflection, was obtained using an arterial tonometry placed on the carotid artery as previously described.[8]

Arterial compliance and $\beta$-stiffness index were measured noninvasively by a combination of ultrasound imaging on the carotid artery and simultaneous applanation tonometry on the contralateral artery.[9] A longitudinal image of the common carotid and femoral artery were acquired 1 to 2 cm proximal to the bifurcation using an ultrasound machine equipped with a high-resolution linear array transducer (Philips iE33 Ultrasound System, Philips, Bothel, Washington). All data were analyzed by the same investigator who was blinded to group assignment and used image analysis software (Vascular Research Tool Carotid Analyzer, Medical Imaging Applications, Coralville, Iowa). Pressure waveform and amplitude were obtained from the contralateral artery using arterial applanation tonometry (VP-2000, Omron Healthcare) and analyzed by waveform browsing software (WinDaq 2000, Dataq Instruments, Akron, Ohio).

Brachial artery flow-mediated dilation (FMD) was measured using a standard procedure as described previously.[10] Briefly, a pneumatic blood pressure cuff was positioned 2 inches below the antecubital fossa. Brachial diameter and blood flow velocity were acquired from a Doppler ultrasound machine equipped with a high-resolution linear array transducer (Philips iE33 Ultrasound). After baseline images were obtained, the cuff was inflated to 100 mm Hg above a subject's systolic blood pressure for 5 minutes. All ultrasound-derived blood flow and diameter data were analyzed by the same investigator using image analysis software (Brachial Analyzer, Medical Imaging Applications). FMD was calculated as (maximal artery diameter minus baseline artery diameter)/baseline artery diameter × 100.[11]

Cardiovagal baroreflex sensitivity (BRS) was determined using the Valsalva maneuver as previously described.[12,13] Subjects performed 3 Valsalva maneuvers by forcibly exhaling against a closed airway. Subjects were asked to maintain an expiratory mouth pressure of 40 mm Hg for 10 seconds. Data for cardiovagal BRS were recorded and analyzed by waveform browsing software (WinDaq 2000) during the phase IV overshoot. Systolic blood pressure values were linearly regressed against corresponding RR intervals from the point where the RR intervals began to lengthen to the point of maximal systolic blood pressure increase.[14]

Table 1
Selected subject characteristics

| Variable | Sedentary | Runners | Swimmers |
|---|---|---|---|
| Men/women | 16/9 | 17/8 | 17/8 |
| Age (years) | 54 ± 2 | 52 ± 2 | 56 ± 2 |
| Height (cm) | 170 ± 2 | 173 ± 2 | 173 ± 2 |
| Body mass (kg) | 74 ± 2 | 67 ± 2* | 76 ± 2† |
| Body mass index (kg/m$^2$) | 26 ± 1 | 22 ± 1* | 25 ± 1† |
| Body fat (%) | 30 ± 2 | 18 ± 2* | 24 ± 2*† |
| Maximal oxygen consumption (ml/kg/min) | 31 ± 2 | 50 ± 2* | 41 ± 2*† |
| Physical activity score (units) | 11 ± 4 | 58 ± 3* | 57 ± 4* |
| Total caloric intake (kcal/day) | 2,370 ± 212 | 2,343 ± 169 | 2,160 ± 150 |
| Carbohydrate intake (%) | 46 ± 3 | 49 ± 3 | 47 ± 2 |
| Fat intake (%) | 36 ± 2 | 32 ± 2 | 33 ± 2 |
| Protein intake (%) | 13 ± 1 | 14 ± 1 | 16 ± 1 |
| Alcohol intake (%) | 5 ± 2 | 4 ± 1 | 3 ± 1 |
| Sodium intake (mg/day) | 3,472 ± 348 | 2,968 ± 278 | 3,014 ± 246 |
| Total cholesterol (mg/dl) | 193 ± 9 | 179 ± 9 | 194 ± 11 |
| Low-density lipoprotein cholesterol (mg/dl) | 120 ± 8 | 105 ± 8 | 123 ± 9 |
| High-density lipoprotein cholesterol (mg/dl) | 47 ± 3 | 61 ± 3* | 52 ± 4† |
| Triglyceride (mg/dl) | 125 ± 14 | 70 ± 14* | 97 ± 15 |
| Plasma glucose (mg/dl) | 94 ± 3 | 95 ± 3 | 99 ± 3 |

Values are means ± SEMs.
* $p < 0.05$ versus sedentary.
† $p < 0.05$ versus runners.

Table 2
Hemodynamic measurements at rest

| Variable | Sedentary | Runners | Swimmers |
|---|---|---|---|
| Heart rate (beats/min) | 60 ± 2 | 50 ± 2* | 58 ± 2† |
| Systolic blood pressure (mm Hg) | 119 ± 3 | 119 ± 3 | 128 ± 3*† |
| Mean blood pressure (mm Hg) | 89 ± 2 | 88 ± 2 | 93 ± 2 |
| Diastolic blood pressure (mm Hg) | 71 ± 1 | 72 ± 1 | 74 ± 2 |
| Pulse pressure (mm Hg) | 48 ± 2 | 47 ± 2 | 54 ± 2*† |
| Carotid systolic pressure (mm Hg) | 116 ± 3 | 104 ± 3* | 104 ± 3* |
| Carotid pulse pressure (mm Hg) | 47 ± 2 | 36 ± 2* | 37 ± 2* |
| Brachial-ankle pulse wave velocity (cm/s) | 1,336 ± 36 | 1,230 ± 35* | 1,334 ± 36† |
| Carotid augmentation index (%) | 15 ± 5 | 13 ± 4 | 13 ± 5 |
| Carotid artery diameter at end-diastole (mm) | 6.77 ± 0.16 | 6.0 ± 0.15* | 6.67 ± 0.15† |
| Brachial artery diameter at end-diastole (mm) | 4.2 ± 1.9 | 4.2 ± 1.8 | 4.6 ± 1.8 |

Values are means ± SEMs.
* $p < 0.05$ versus sedentary.
† $p < 0.05$ versus runners.

Analysis of covariance was used for statistical analysis to determine significant group differences. Correlation and regression analyses were performed to determine the relation

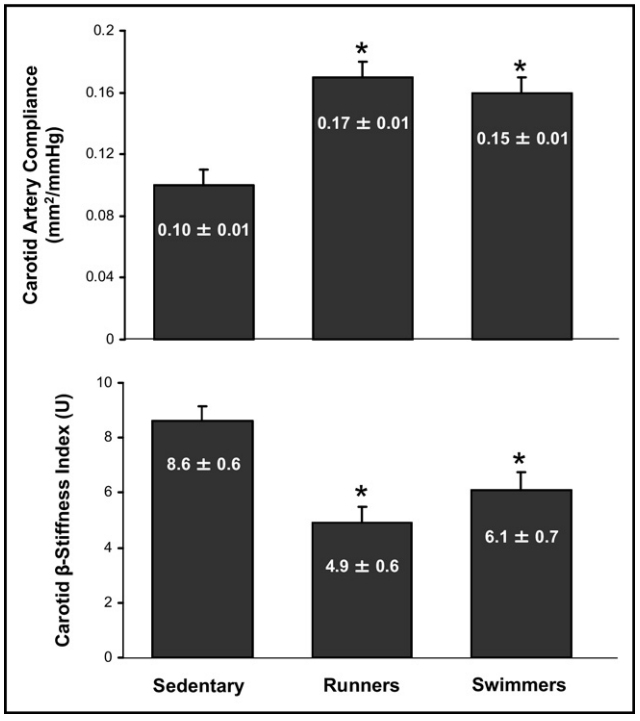


Figure 1. Carotid artery compliance and $\beta$-stiffness index. Values are means ± SEMs. *p <0.05 versus sedentary.

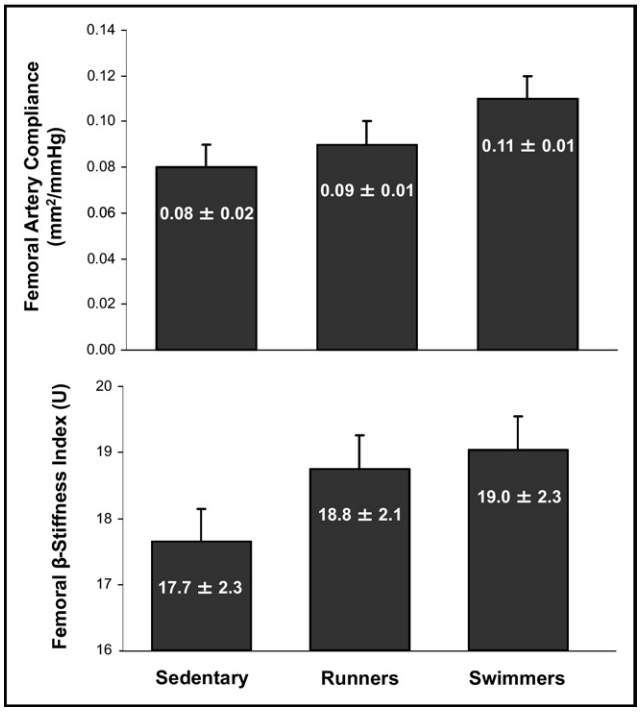


Figure 2. Femoral artery compliance and $\beta$-stiffness index. Values are means ± SEMs.

between carotid arterial compliance and cardiovagal BRS. All variables were expressed as mean ± SEM.

## Results

As presented in Table 1, there were no group differences in age, height, and dietary intakes. Body mass and body

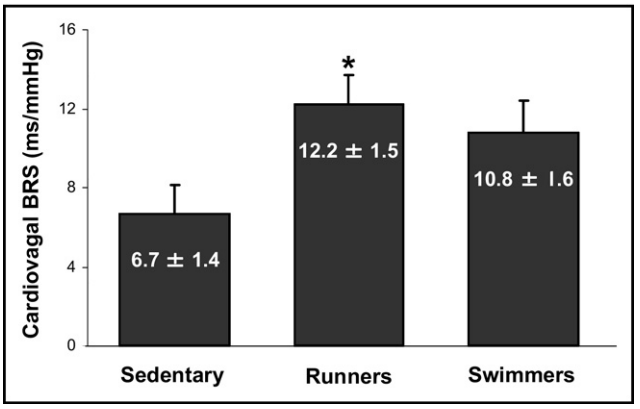


Figure 3. Cardiovagal baroreflex sensitivity. Values are means ± SEMs. *p <0.05 versus sedentary.

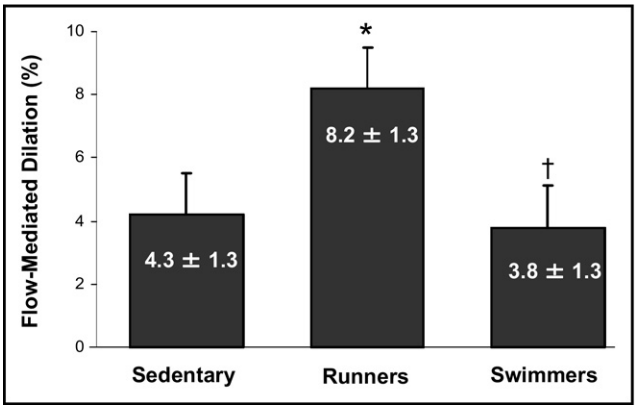


Figure 4. Flow-mediated dilation. Values are means ± SEMs. *p <0.05 versus sedentary; †p <0.05 versus runners.

mass index were lower ($p < 0.05$) in runners than in sedentary controls and swimmers. Percent body fat of swimmers was lower ($p < 0.05$) than of sedentary controls but higher ($p < 0.05$) than of runners. Maximal oxygen consumption of swimmers was greater than of sedentary controls but lower than of runners. Fasting plasma concentrations of glucose, total cholesterol, and low-density lipoprotein cholesterol were not different among groups. Runners had significantly lower plasma triglyceride and higher high-density lipoprotein cholesterol concentrations than sedentary controls.

Heart rate at rest was lower ($p < 0.05$) in runners than in sedentary controls and swimmers (Table 2). Brachial systolic blood pressure and pulse pressure were higher ($p < 0.05$) in swimmers than in sedentary controls and runners. Runners and swimmers had lower ($p < 0.05$) carotid systolic blood pressure and carotid pulse pressure than sedentary controls. Brachial ankle pulse wave velocity was significantly lower in runners than in swimmers and sedentary controls. There were no group differences in carotid artery augmentation index.

Carotid arterial compliance was higher ($p < 0.05$) and $\beta$-stiffness index was lower ($p < 0.05$) in runners and swimmers than in sedentary controls (Figure 1). Unlike measurements of central artery stiffness, measurements of peripheral artery stiffness, femoral artery compliance, and femoral $\beta$-stiffness index were not different among the 3 groups (Figure 2). Cardiovagal BRS was greater ($p < 0.05$) in

runners than in sedentary controls and swimmers (Figure 3). Cardiovagal BRS of swimmers tended to be higher than in sedentary controls but this did not achieve statistical significance ($p = 0.07$). Cardiovagal BRS was positively associated with carotid arterial compliance in the pooled population ($r = 0.44$, $p < 0.01$).

Brachial artery FMD was significant greater ($p < 0.05$) in runners compared with sedentary controls and swimmers (Figure 4). Peak brachial artery blood flow and calculated shear rate were not different among the 3 groups so FMD values were not adjusted for shear rate.

## Discussion

This is the first study, to our knowledge, to determine whether swimmers would exhibit a similar phenotype of vascular function to runners. The salient finding of the present study is that central artery compliance was greater in swimmers than in age-matched sedentary controls, and the level of arterial compliance was not different from runners, suggesting that high levels of regular swimming exercise may prevent arterial stiffening similar to land-based exercises.

In the present study, middle-aged and older swimmers demonstrated higher levels of brachial systolic and pulse pressures than runners and sedentary controls. Our findings are consistent with previous cross-sectional studies showing that cardiovascular risk profiles, in particular arterial blood pressure, of swimmers are less favorable than those of runners.[15,16] Previous exercise intervention studies have also shown that swimming exercise intervention may induce effects on blood pressure that are smaller in magnitude than running exercise[17] and may even increase blood pressure.[18]

Interestingly, in the present study, "central" blood pressures were significantly lower in swimmers than in sedentary controls. Central blood pressure is determined by several factors including aortic diameter, arterial wave reflection, and left ventricular ejection characteristic[19] and is a better predictor of cardiovascular disease risks than brachial blood pressure.[20] Lower central blood pressure in swimmers was associated with greater arterial compliance. Taken together, these results are consistent with the notion that regular swimming exercise plays an important role in preventing arterial stiffening.

It remains unclear how regular aerobic exercise improves arterial compliance. One possibility is that regular physical activity may act on the elasticity of the artery through endothelium-dependent vasodilation.[21] FMD serves as an index of nitric oxide–mediated endothelium-dependent vasodilator function in humans. In the present study, greater arterial compliance in swimmers was not associated with a higher FMD. These results in swimmers are consistent with our previous pharmacologic study showing that nitric oxide does not appear to play a role in increasing arterial compliance through regular walking exercise.[22] Other possibilities to explain the beneficial effects of regular exercise on macrovascular function include decreases in vascular vasoconstrictor tone,[22] endothelin-1,[23] and collagen crosslinking.

In contrast to the central arteries, compliance of peripheral arteries does not appear to change much with different interventions or states including aging and endurance training.[8,9] Consistent with this, we found that femoral arterial compliance was not different among the 3 groups. A lack of influence of regular exercise on peripheral arterial compliance is attributed to the fact that peripheral arteries do not exhibit the same extent of pulsatile changes in diameter compared with central (cardiothoracic) arteries.

Accumulating evidence has indicated that habitual aerobic exercise favorably modulates age-associated decreases in cardiovagal BRS.[12,14] Consistent with previous findings, results from the present study showed that cardiovagal BRS is increased in middle-aged and older endurance-trained runners compared with sedentary controls. Cardiovagal BRS was ~25% greater in swimmers than in sedentary controls, and there was a trend ($p = 0.07$) for the difference to be significant. Moreover, cardiovagal BRS was significantly associated with arterial compliance in the pooled population. Thus, regular swimming appears to be associated with a greater level of cardiovagal BRS.

Is regular swimming associated with a lower risk of cardiovascular and all-cause mortalities? Only 2 studies are available to answer this question. In 1 epidemiologic study, swimming was not associated with a lower risk of cardiovascular disease, although walking and running examined in the same study demonstrated significant associations.[24] A more recent epidemiologic study, however, reported a smaller relative risk of developing cardiovascular disease in swimmers than in sedentary populations,[25] and the relative risk of swimmers were even lower than those of walkers and runners. Thus, at present, it remains highly controversial as to whether swimming is equally cardioprotective to land-based exercise. Similar to these epidemiologic studies, our findings are somewhat divergent. Central arterial compliance was greater in swimmers than in sedentary controls. However, endothelium-dependent vasodilation as assessed by FMD was not different between swimmers and sedentary controls. Clearly, more research effort should be directed toward the influence of swimming exercise on vascular disease risks.

Swimming is an attractive form of exercise because it is easily accessible, inexpensive, and isotonic.[26] Because of the buoyancy of water, compressive stress on joints is low and orthopedic injury rate is low.[27] Due to cold temperature and increased thermoconductivity of surrounding water, heat-related illness is extremely low.[28] Thus, swimming can be an ideal form of exercise for those at increased risk of vascular disease including elderly patients, obese patients, and patients with arthritis. Results of the present study indicate that swimming is also associated with destiffening effects on the central artery. However, it should be noted that the beneficial impact of regular swimming may be smaller than land-based exercises as summarized in a recent review.[26] Even within our subject sample, swimmers did not exhibit phenotypes in lipid profiles and brachial blood pressure similar to runners.

In addition to use of a cross-sectional study design, the present study has other limitations that should be discussed. Although swimmers and runners were matched well for exercise training volume, maximal oxygen consumption was significantly lower in swimmers than in runners. However, based on the principles of specificity of training, this is an expected finding because maximal oxygen consumption

was assessed on a treadmill. Transfer of cardiovascular training benefits is very limited for maximal oxygen consumption on a treadmill when swimming exercise is performed as a training method.[29]


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