International Journal of Aquatic Research and Education

Volume 8 | Number 4 Article 3

11-1-2014

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International Journal of Aquatic Research and Education, 2014, 8, 310-320
http://dx.doi.org/10.1123/ijare.2014-0048

International Journal of
Aquatic Research and Education
www.IJARE-Journal.com
RESEARCH

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Arterial stiffening (AS) is characterized by the loss of the elasticity of arteries and is an independent risk factor for cardiovascular (CV) mortality. We tested the hypothesis that shallow water aerobics can lower AS in older individuals with CV risk factors. Participants were randomized into two groups. The Con group remained inactive, whereas the ExT group exercised for 10 weeks. Carotid-to-femoral pulse wave velocity was used to measure AS and pulse wave analysis was used to determine central blood pressures pre- and postintervention. A significant group by time interaction was identified for AS in favor of the ExT group. No differences were found pre- and post-training in brachial or central systolic pressures; however, a reduction in brachial and central diastolic pressures was evident in the ExT group. These findings indicate that the introduction of shallow water aerobic exercise training positively influences AS and blood pressure of individuals with CV risk factors.

Keywords: arterial stiffness; aquatic exercise; aquatic fitness

Arterial stiffening describes the reduced capability of an artery to expand and contract in response to changes in pressure. Structural changes to the large elastic arteries such as increased fibrosis, collagen deposition along with collagen cross-linking, and a reduction in elastin content and integrity, increase the stiffness of the arteries (Dao, Essalihi, Bouvet, & Moreau, 2005; Zieman, Melenovsky, & Kass, 2005). Such changes are evident with aging even in the absence of cardiovascular disease (CVD) (AlGhatrif et al., 2013; Vaitkevicius et al., 1993); however, the degree of arterial stiffening with aging is exacerbated in the presence of CVD (Fournier et al., 2014; Scuteri et al., 2014). Arterial stiffness (AS) can be measured noninvasively as pulse wave velocity and used to characterize the cardiovascular risk for a given individual. AS is a major contributor to the age-related increase in the prevalence of hypertension and also serves as an independent predictor of cardiovascular morbidity and mortality. Indeed, a 1 m/s increase in AS corresponds

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to an age-, sex-, and risk factor-adjusted risk increase of 15% in cardiovascular and all-cause mortality (Vlachopoulos, Aznaouridis, & Stefanadis, 2010). Therefore, it is essential to identify effective interventions intended for reducing AS.

Land-based aerobic exercise training has been shown to effectively improve AS in healthy individuals and in patients with CVD (Currie, Thomas, & Goodman, 2009; Donley et al., 2014; Moreau, Donato, Seals, DeSouza, & Tanaka, 2003; Hirofumi Tanaka, DeSouza, & Seals, 1998; Tanaka et al., 2000). Further, aquatic-based exercise training (swimming) has been shown to improve arterial compliance and blood pressure (Nualnim et al., 2012).

Water-based activities are an ideal form of exercise for obese patients, individuals who suffer from joint dysfunction, and the elderly due to the nonweight bearing nature of the underwater environment. However, swimming-related exercise training requires specific skills and techniques to achieve steady exercise intensity. In contrast, shallow water aerobic exercise training does not require any specific skill set and is a form of exercise that is popular among individuals who lack confidence or proficiency in swimming.

Surprisingly, little is known about the effects of aquatic exercise on arterial health. We examined whether shallow water aerobic exercise training can improve AS and central hemodynamics in older individuals with CVD risk factors. We hypothesized that shallow water aerobic exercise training would reduce AS compared with the nontrained control group.

Method

Study Population

Thirty-nine men and women (ages 59–86) were recruited by flyers and advertisements and participated in the study. Inclusion criteria required each subject to acquire medical clearance from their physician to participate in regular, aerobic exercise training. Exclusion criteria included pulmonary disease, angina, atrial fibrillation, myocardial infarction, or coronary revascularization, and no neurological defect (e.g., possible dementia, self-report of neurological disease such as multiple sclerosis, brain tumor, and Parkinson's disease) as assessed by a detailed medical history. Subjects who participated in regular exercise, defined as > 30 min, 3 times/week were excluded. All subjects provided written informed consent to participate that was submitted to, and approved by, WVU Institutional Review Board.

Study Design

After the measurements of body anthropometry, AS and pulse wave analysis were measured in a quiet, temperature-controlled room, after a minimum 15 min of quiet supine rest.

Body anthropometry. Height and weight, along with waist and hip circumferences were measured using standard laboratory procedures. Fat distribution was assessed by measuring the waist circumference at the site of the smallest circumference between the rib cage and the ileac crest, with the subjects in standing position. Hip circumference was measured at the site of the largest circumference between waist and thighs. Body composition was measured using air displacement

plethysmography (BodPod; Life Measurement, Concord, CA, USA; Dempster & Aitkens, 1995). During assessment of body composition subjects wore tight-fitting bathing suits and a swim cap. Body mass index (BMI) was calculated as weight (kg)/height $(m)^2$.

Arterial function. Brachial systolic (SBP) and diastolic (DBP) blood pressures were measured with an automated, oscillometric sphygmomanometer (Critikon Dinamap Compact BP Monitor, GE Medical, Tampa, FL, USA) and pulse pressure (PP) was calculated from SBP-DBP. Pulse wave analysis was performed noninvasively on the radial artery (SphygmoCor System, AtCor Medical, Sydney, Australia). All measurements were made in triplicate and the mean values used for subsequent analysis. The SphygmoCor system synthesizes a central (ascending aortic) pressure waveform from the radial pressure waveform that does not differ from that of an intra-arterially recorded wave (Chen et al., 1996) using a validated generalized transfer function (Chen et al., 1997) that has good reproducibility under major hemodynamic changes (Sharman et al., 2006). These waveforms were calibrated against brachial mean arterial and diastolic pressure to estimate aortic pressures.

The characteristics of the aortic pulse wave were determined using established guidelines (Laurent et al., 2006; Van Bortel et al., 2002). Augmented pressure (AP), a measure of the contribution of wave reflections to SBP, was defined as the difference between aortic SBP and the pressure at the forward wave peak. Augmentation index (AGI) provides a measure of the contribution of wave reflection pressure (i.e., AP) to SBP relative to total PP. AGI was calculated as the ratio of amplitude of the pressure wave above its systolic shoulder (i.e., the difference between the early and late systolic peaks of the arterial waveform), to the total PP expressed as a percentage (P_2–P_1/PP*100; Chen et al., 1996). Since AGI varies with heart rate, AGI is commonly adjusted to a standard heart rate of 75 beats per minute (AGI@75; Wilkinson et al., 2000). Systolic-to-diastolic pressure shifts were assessed by the systolic and diastolic pressure-time integrals. The Buckberg subendocardial viability ratio (SEVR) index, which correlates with LV subendocardial:subepicardial flow ratio (an apparent marker of subendocardial ischemia; Buckberg, Fixler, Archie, & Hoffman, 1972), was calculated as the percentage ratio of the diastolic and systolic pressure-time integral.

Carotid to femoral pulse wave velocity (cfPWV; central arterial stiffness) and carotid to radial pulse wave velocity (crPWV: peripheral arterial stiffness) were measured by applanation tonometry (AtCor Medical, Sydney, Australia; O'Rourke, Pauca, & Jiang, 2001). ECG-gated waveforms were sequentially recorded. Aortic distance (D) was calculated as the difference in the distances from the carotid to the suprasternal notch and from the suprasternal notch to the femoral artery or radial artery. Time delay was calculated using a foot-of-the-wave method. Further, we also estimated vascular age based on published sex-specific algorithms for cfPWV (PWVage; McEniery et al, 2005).

Intervention. Forty-four subjects meeting the inclusion criteria initially volunteered to participate in this study. Subjects were arranged into matched pairs by utilizing age and fitness parameters. One participant from each pair was then randomly assigned to either a 10-week shallow water aerobic exercise intervention group (ExT) or a 10-week nonexercise control (Con) group. However, three

individuals were later identified to have overt CVD and thus were excluded from analysis. Further, eight individuals did not complete the study (four in the Con group and four in the ExT group). Of the 44 individuals, 33 (17 ExT and 16 Con) completed the study in full, demonstrating an attrition of 20% from the start of the study to completion. Reasoning for attrition was noted to fall under two underlying circumstances: (1) medical reasons (25%, including hip replacements), and (2) time constraints (75%). Data were only analyzed on subjects who completed the study. The Con group was instructed to maintain their normal lifestyle activities. The ExT group performed 10 weeks of supervised, instructed shallow water aerobic exercise, 3 days/week for 60 min/day at a fixed exercise intensity performed in the Natatorium at West Virginia University. The intensity of prescribed exercise was based on the Karvonan formula with the application of the Kruel reduction model. A ramp exercise protocol was used whereby the exercise intensity started at 50% of heart rate reserve with the Kruel deduction and increased by 5% every week until reaching 75% of the heart rate reserve. Duration of time spent in this target heart rate was then increased for the remaining 4 weeks of the study. Adherence to the exercise prescription was documented through the use of Portable heart rate monitors (E600, Polar Electro OY, Oulu, Finland) and a rate of perceived exertion (RPE) scale. The participants were instructed to maintain their normal diet for the duration of the intervention. All post-training measurements were performed at least 48 hours after the last exercise session to avoid the immediate effects of a single bout of exercise.

Statistical analysis. Our sample size calculation for our primary outcome (cfPWV) assumed a power of 90% and an alpha error probability of 0.05. We required a minimum of 11 subjects to detect a clinically significant effect of exercise training on arterial stiffness reflecting a difference in cfPWV of 1 ± 1 (SD) m/s. However, we recruited up to 21 subjects per group to account for potential dropouts. Normality was evaluated by the Kolmogorov-Smirnov test. Categorical variables were compared by the chi-square test. Continuous variables were log transformed as necessary and compared between groups (Con vs. ExT) with an independent sample *t* test. To evaluate the effects of exercise training, paired *t* tests and two-way repeated-measures ANOVA were used. All analyses were performed with the statistical package SPSS version 21 (SPSS, Chicago, IL). Values shown in the tables represent means ± SEM unless otherwise stated. $p \leq .05$ was defined as significant.

Results

Age, anthropometric, and clinical characteristics of the participants are shown in Table 1. Both the Con and ExT groups were well-matched with no significant differences in age, sex, height, weight, % body fat, or BMI (Table 1). Similarly, no significant differences in baseline arterial parameters were evident between Con vs. ExT groups before the intervention.

Effects of Exercise Training

Body anthropometry. Shallow water exercise training did not significantly alter body anthropometrics, highlighted by a similar body weight, percentage body fat, BMI and BSA between pre- and postintervention (Table 2).

Table 1 Clinical Characteristics of the Subject Cohorts

	Con (*n* = 16)	ExT (*n* = 17)	*p* value
Age, years	69 ± 2	68 ± 2	0.90
Sex, % female	81	75	0.46
Height, cm	165 ± 2	163 ±3	0.52
Weight, kg	86 ± 7	84 ± 5	0.86
Body fat %	40 ± 2	40 ± 2	0.73
BSA, m^2	1.92 ± 0.07	1.87 ± 0.05	0.89
BMI, kg/m^2	31 ± 2	32 ± 2	0.52
Waist circumference, cm	102 ± 7	102 ± 5	0.92
Hips circumference, cm	116 ± 5	116 ± 4	0.97
SBP, mmHg	131 ± 5	126 ± 4	0.39
DBP, mmHg	67 ± 3	68 ± 2	0.72
Hypertensive, %			0.59
Normal	19	29	
PRE	13	12	
HTN	69	59	
Smoker,%			0.56
Never	63	71	
Former	31	29	
Current	6	0	
Type 2 Diabetes, %	6	18	0.32
Medications, %			
Diuretic	6	13	0.52
Cardiac	56	69	0.59
Statin	31	0	< 0.01

Notes. Values are mean ± SEM. BMI = body mass index; BSA = body surface area; DBP = diastolic.

Arterial parameters. There was a significant time (pre vs. post) by group (Con vs. ExT) interaction for cfPWV, indicating that 10 weeks of shallow water aerobic exercise training significantly reduced cfPWV (-8%), whereas no change was found in the Con group (+4%); see Figure 1. When examining the effects of exercise training on blood pressure, no differences in brachial or central SBP were found (Table 3). However, a significant time by group interaction was found for brachial diastolic blood pressure (bDBP), central diastolic blood pressure (cDBP), and brachial pulse pressure (bPP). Whereby a 2 mmHg decrease in bDBP and cDBP was noted in the ExT group and a 3 mmHg increase in bDBP and cSBP was noted in the Con group. As a result of the changes in bDBP a corresponding change was

Table 2 Body Anthropometrics

	Con		ExT	
	Pre	Post	Pre	Post
Body weight, kg	86 ± 7	87 ± 6	84 ± 5	84 ± 5
Body fat, %	40 ± 2	41 ± 2	41 ± 2	42 ±2
Lean mass, kg	51 ± 3	51 ± 4	49 ± 3	48 ± 3
BSA	1092 ± 0.07	1.93 ± 0.07	1.87 ± 0.05	1.87 ± 0.05
BMI	31 ± 2	33 ± 1	32 ± 2	33 ± 1
Waist, inches	102 ± 7	99 ± 6	102 ± 5	100 ± 5
Hips, inches	116 ± 5	116 ± 5	116 ± 4	116 ± 4

Note. Values are mean ± SEM. BSA = body surface area; BMI = body mass index.

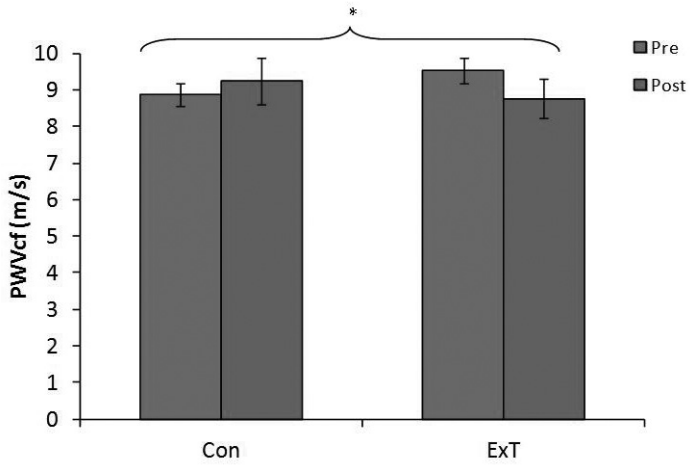

Figure 1 — Effects of aquatic exercise training on arterial stiffness. The change in arterial stiffness was measured by carotid to femoral pulse wave velocity before (pre) and after (post) the intervention in inactive controls (Con) and in the exercise training (ExT) aquatics group. * denotes a significant ($p \leq .05$) time (pre vs. post) by group interaction (Con vs. ExT). Values are mean ± SEM.

noted in bPP, with a 2 mmHg increase and a 3 mmHg decrease in bPP the Con group, respectively (Table 3). In contrast, there was no significant time by group interaction for cPP. Importantly, a significant group by time interaction was observed for vascular age, whereby exercise training significantly reduced vascular age (by 5 years), while no differences were evident in the Con group (Table 3).

When examining the central pressure wave, exercise training did not affect AGI (or AGI@HR75), augmented pressure (AP), travel time (AoTr), ejection duration (ED), or subendocardial viability ration (SEVR). Further, no differences in central pressure wave mechanics were found in the Con group between pre- and postassessments (Table 3).

Table 3 Differences in Arterial Parameters Pre- and Postintervention in MetS and Controls

	Con		ExT	
	Pre	Post	Pre	Post
bSBP, mmHg	131 ± 5	131 ± 5	127 ± 3	124 ± 4
bDBP, mmHg*	67 ± 3	70 ± 3	68 ± 2	66 ± 3
bPP, mmHg*	64 ± 3	61 ± 3	56 ± 3	58 ± 4
MAP, mmHg	90 ± 4	92 ± 3	90 ± 2	87 ± 3
cSBP, mmHg	118 ± 5	121 ± 5	118 ± 4	115 ± 3
cDBP, mmHg*	69 ± 3	71 ± 3	69 ± 2	67 ± 2
cPP, mmHg	50 ± 3	50 ± 3	47 ± 3	48 ± 3
AP, mmHg	15 ± 2	16 ± 2	16 ± 2	16 ± 2
AGI, %	29 ± 3	30 ± 3	32 ± 2	33 ± 2
AGI@75HR,%	27 ± 3	28 ± 3	28 ± 2	28 ± 2
HR, bpm	73 ± 3	69 ± 3	66 ± 2	64 ± 3
SEVR, %	129 ± 8	134 ± 5	141 ± 6	145 ± 6
ED, m/s	321 ± 8	328 ± 10	331 ± 7	335 ± 9
AoTr, m/s	133 ± 1	134 ± 2	133 ± 3	134 ± 2
PWV Age, years*	62 ± 2	65 ± 2	65 ± 4	60 ± 4

Notes. Values are mean ± SEM. * Denotes a significant ($p \leq .05$) time (pre vs. post) by group interaction (Con vs. ExT). bSBP = brachial systolic blood pressure; bDBP = brachial diastolic blood pressure; bPP = brachial pulse pressure; MAP = mean arterial pressure; cSBP = central systolic blood pressure; cDBP = central diastolic blood pressure; cPP = central pulse pressure; AP = augmented pressure; AGI = augmentation index; AGI@75 = augmentation index at 75 beats per minute; HR = heart rate; SEVR = subendocardial viability ratio; ED = ejection duration; AoTr = travel time; PWV = pulse wave velocity.

Discussion

The major new findings from the study are as follows. Shallow water exercise training in older individuals with CV risk factors reduced central AS and vascular age. To the best of our knowledge this is the first study to examine and demonstrate that shallow water exercise is able to improve arterial function.

Current recommendations for the prescription of exercise in older individuals with CV risk factors focus on land-based aerobic and resistance training (Garber et al., 2011). Indeed, we and others have shown that land-based aerobic exercise training is an effective way to improve arterial stiffening in patients with the metabolic syndrome (Donley et al, 2014) and in other populations (Beck, Martin, Casey, & Braith, 2013; Collier et al., 2008). However, we have shown that shallow water aerobic exercise training, performed for 10 weeks, 3 times/week for 1 hr/visit, may also be used as an effective intervention to improve arterial stiffness in older individuals with CVD risk factors. This finding may provide a benefit for individuals that have an aversion to land-based training due to joint dysfunction,

balance disturbances, obesity, or generalized discomfort as it represents an effective alternative form of exercise.

Arterial stiffening is an important clinical risk factor that progresses during the normal aging process (AlGhatrif et al., 2013; Vaitkevicius et al., 1993) and is accelerated in the presence of CVD (hypertension, diabetes, metabolic syndrome; Fournier et al., 2014; Scuteri et al., 2014). Specifically, a 1 m/s increase in cfPWV translates to a 15% increase in cardiovascular and all-cause mortality that is independent of age, sex, and cardiovascular risk factors (Vlachopoulos et al., 2010). Of particular importance, cfPWV decreased on average by 0.8 m/s after 10 weeks of shallow water training which likely reflects a significant reduction in CV risk. Furthermore, given the association between increased AS and cerebral small vessel disease, decreased cognitive function, and dementia (Singer, Trollor, Baune, Sachdev, & Smith, 2014), a reduction in AS has several important consequences. Regular exercise training initiated early in life may prevent or delay the age-associated increase in AS and the corresponding cognitive impairments.

It was once postulated by an English physician, Thomas Sydenham, MD, that a "Man is as old as his arteries" an expression that declares that the health of one's blood vessels plays a critical role in the aging process (Sydenham, 1928). From this the phrase, *vascular age* was born and reflects the physical stress and fatigue that occurs to the arteries with normal aging (in the absence of CVD risk factors). However, the stress and fatigue subjected on the arteries is impacted by poor lifestyle habits (physical inactivity, smoking, etc.) and the presence of CVD risk factors (hypertension, diabetes). As such, vascular age may be a better reflection of one's age. Arterial stiffening is an integral part of the process of normal vascular aging, as it reflects the present condition of the arterial wall. Therefore, AS is a good method for the evaluation of the vascular age. The reduction in AS observed after aquatic exercise training may translate into a reversal of age-related arterial stiffening of 5 years (Avolio et al., 1983).

In the current study, a reduction in AS was noted following exercise training and occurred in the absence of significant improvements in weight and body fat composition. This finding suggests that the effects of aerobic exercise training on AS may be independent of other well-established benefits of exercise training. Similar to our findings, three months of walking in healthy middle-aged men produced a reduction in AS in the absence of improvements in body weight (Tanaka et al., 2000). In our study, a reduction in AS coincided with a significant group by time interaction in brachial and central DBP, and brachial PP. Endurance (land-based) training has been shown to lower both resting SBP and DBP (Fagard, 2006; Whelton, Chin, Xin, & He, 2002); however, in our study, a reduction in SBP was noted but the decrease was not statistically significant. In contrast, we found that 10 weeks of exercise training produced a 2 mmHg reduction in brachial and central DBP. The average reduction in DBP with land-based aerobic exercise training is reported between 2–5 mmHg and is independent of body weight and race (Fagard, 2006; Whelton et al., 2002). Because PP is the mathematical difference between SBP and DBP, we found that ExT increased bPP by 2 mmHg, whereas the Con group noted a 3 mmHg reduction in bPP. However, PP measured at the brachial artery with the use of the cuff method is not an accurate representation of the proximal aortic PP. Indeed, we found that cPP did not significantly change in either group after the intervention, which highlights the importance of examining central blood pressures when performing interventional studies.

The stiffness of the arterial system is dependent on numerous factors, in particular the dynamic (autonomic tone, smooth muscle cell contractility, endothelial function) and material properties (elastin content and integrity, collagen content, fibrosis, elastin and collagen cross-linking, calcification) of the artery (Dao et al., 2005; Zieman et al., 2005). Changes in distending pressure affect arterial stiffness, where at higher pressures the force on the arterial wall is transferred to the stiffer collagen limiting distention (Dao et al., 2005). In our study, MAP did not change. Thus, it is unlikely that a reduction in distending pressure contributed to the improved arterial compliance. Future research should examine the mechanism behind the improved arterial stiffness after aquatic based exercises. In particular, changes to the autonomic system, structural remodeling of the conduits and the microcirculation, and changes to arterial composition (or their blood biomarkers) warrant further examination.

Limitations

There are several limitations to note. Sex specific differences in the effects of exercise training on AS may exist that could not be detected given the small number of male vs. female subjects. In addition, the short duration of exercise training, 10 weeks, may have been insufficient to alter arterial structure and body composition. Thus, longer aquatic exercise training programs that incorporate different exercise modalities (shallow vs. deep water aerobic exercise training), and comparisons between land-based vs. aquatic-based exercise training are critical for a full understanding of the role of exercise training in improving arterial structure and function in patients with CVD.

In conclusion, the results from the current study demonstrate that shallow water aerobic exercise training is an effective approach to reduce arterial stiffening in elderly individuals with CVD risk factors.

Acknowledgments

This study was supported in part by the National Swimming Pool Foundation (Dr Chantler), American Heart Association 11CRP7370056 (Dr Chantler), National Heart, Lung, Blood Institute T32- HL090610 (Sara Fournier), and the National Institute of General Medical Sciences of the National Institutes of Health under Award Number U54GM104942.

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