

Swimming training lowers the resting blood pressure in individuals with hypertension

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Background Despite the fact that swimming is often recommended for the prevention and treatment of hypertension, no study has examined the potential efficacy of regular swimming exercise for lowering the blood pressure in hypertensive humans.

Objective To test the hypothesis that regular swimming exercise lowers the resting blood pressure.

Design A 10-week closely supervised swimming training program compared with a non-exercising control group.

Patients Eighteen previously sedentary men and women [aged 48 ± 2 years (mean \pm SEM)] with stage 1 or 2 essential hypertension.

Results The resting heart rate, an index of cardiovascular adaptation, decreased in the swimming training group from 81 ± 4 to 71 ± 3 beats/min ($P < 0.01$). The body mass and body fat percentage did not show statistically significant changes. The systolic blood pressure of patients in the seated position fell significantly ($P < 0.05$) from 150 ± 5 to 144 ± 4 mmHg. The seated diastolic blood pressure did not change significantly. A similar magnitude of reductions in systolic blood pressure ($P < 0.05$) was also found in patients in the supine position. No significant changes in plasma catecholamine

concentrations, casual forearm vascular resistance, plasma volume and blood volume were observed. There were no significant changes in any of these variables in the control group.

Conclusion Swimming training elicits significant reductions in arterial blood pressure at rest in individuals with hypertension. This is a clinically important finding since swimming can be a highly useful alternative to land-based exercises for hypertensive patients with obesity, exercise-induced asthma, or orthopedic injuries.

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Introduction

A chronically elevated blood pressure is associated strongly with increased risks of coronary heart disease, stroke and congestive heart failure. Cardiovascular mortality and morbidity rates increase with elevations in systolic and diastolic blood pressures [1]. Therefore, the goal of treating hypertension is to reduce the morbidity and mortality associated with a high blood pressure and to lower the blood pressure by the least intrusive means [1,2]. The fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure recommended regular aerobic physical activity as part of an initial lifestyle modification for patients with stage 1 or 2 hypertension [1,2].

Physical activity has been shown to be one of the most effective nonpharmacologic treatments for individuals with high blood pressures [3,4]. Several investigations [3,4] have found significant reductions in resting blood

pressures of hypertensive individuals after habitual physical activities. Most studies to date, however, have employed walking, running, or cycling as activity modes. Despite the fact that swimming is recommended specifically and widely for the prevention and treatment of hypertension by respected national and international health organizations (e.g. the American Heart Association [5], World Hypertension League [6], and World Health Organization [7]), no information on its effectiveness for lowering the blood pressure [4] is available.

There is some evidence to suggest that swimming may not benefit hypertensive individuals. It has been reported that swimmers tend to have chronically higher resting blood pressures than other endurance athletes [8–10]. In addition, compared with that during running, the mean arterial blood pressure tends to be higher during swimming at the same heart rate values, possibly owing to the increased peripheral resistance or to an altered ther-

moregulatory demand, or both [11]. These cross-sectional studies suggest that swimming training might not be effective in modifying the resting blood pressure. No longitudinal studies have addressed the effects of swimming training on the resting arterial blood pressure in patients with hypertension.

Thus, the possible benefit of swimming exercise for the treatment of hypertension remains unknown despite the fact that swimming is one of the most popular and recommended exercises because of its low-impact, rhythmic, and dynamic nature. Since swimming includes the minimal weight-bearing stress, a humid environment, and a reduced heat load, it has potentially important applications for hypertensive individuals who have orthopedic problems or bronchospasms, or who are obese. Therefore, the primary purpose of this study was to determine the effects of swimming training on the resting arterial blood pressure in stage 1 and 2 hypertensive patients. Additionally, we sought to examine several mechanistic variables that could explain training-induced changes, if any were observed, in arterial blood pressure.

Methods

Subjects

Male and female hypertensive patients with uncomplicated stage 1 or 2 hypertension [1] were recruited from outpatient clinics and from the surrounding community via advertisements. Prior to baseline measurements, every subject had an average systolic blood pressure at rest in the range 140–179 mmHg or a diastolic blood pressure in the range 90–109 mmHg, or both, on the basis of repeated casual blood pressure readings. Most subjects were capable of swimming continuously for at least 10 min. However, none had participated in regular vigorous activity during the previous year. All of the patients had their medical history assessed and were subjected to a physical examination and treadmill exercise stress tests by a licensed physician to ensure that the study would not be dangerous for them. If the patients developed symptoms other than fatigue, they were excluded from the study. No subjects had clinical or electrocardiographic evidence of coronary artery disease. They also had no orthopedic complications that would have prohibited them from swimming. Only one subject in the training group was being administered antihypertensive medications (diuretics). This subject's responses did not differ from those of the other subjects in the training group, and the results were not affected by the inclusion or exclusion of this subject. Prior to participation, verbal and written explanations of the procedure and its potential risks were provided. Each subject then gave their written consent to participate in the investigation. The experimental procedures were reviewed and approved by the Institutional Review Board of the University of Tennessee, Knoxville, Tennessee, USA.

After the baseline measurements had been completed, subjects were assigned to one of the two groups: swimming training ($n = 12$, seven men and five women) and a control group ($n = 6$, three men and three women). Group assignments were made as randomly as possible, with some regard given to individual preference when subjects strongly objected to their group assignment. As a result, one subject was allowed to switch from the control to the training group. The results were not influenced by the inclusion or exclusion of this particular subject. The mean ages of subjects in the training and control groups were 47 ± 3 and 49 ± 5 years, respectively. Prior to the study, there were no significant differences ($P > 0.05$) in age and physical characteristics between the groups (Table 1). During the course of this investigation, subjects in both groups were instructed to maintain their usual lifestyle and dietary habits.

Training

Subjects in the swimming training group participated in a supervised 10-week swimming training program of 60 min sessions, 3 days per week on alternate days. Each exercise session consisted of a warm-up of 5 min stretching and 5 min swimming followed by a 45 min swimming workout. The last 5 min served as the cooling down period. The duration of the swimming workout was gradually increased from 30 min during the first week to the required 45 min during the fourth week. Most subjects used freestyle as their primary swimming style. Approximately 20% of the swimming workout was comprised of kicking drills using a kick board. The exercise intensity during the 45 min swimming workout was set at a 60% of the maximal heart rate reserve (approximately 60% of maximal oxygen consumption) recorded during a graded exercise test on a treadmill. The target heart rate was adjusted on the basis of the observation that the maximal heart rate during swimming is approximately 10–13 beats/min lower than that during running [12–14]. The water temperature of the swimming pool was held constant at 27–28°C during this investigation. Each subject was instructed to swim continuously during the 45 min swimming workout, except during the time needed for checking a 10 s target heart rate. Attendance, distance swum, and target heart rates were monitored

Table 1 Physical characteristics of subjects

Variable	Training group	Control group
Height (cm)	177 \pm 2	173 \pm 4
Body mass (kg)		
Before	106.7 \pm 8.0	91.8 \pm 6.4
After	105.2 \pm 7.6	91.8 \pm 6.1
Lean body mass (kg)		
Before	67.5 \pm 4.2	57.9 \pm 4.2
After	67.9 \pm 4.1	57.4 \pm 4.4
Body fat percentage (%)		
Before	36 \pm 2	36 \pm 4
After	35 \pm 2	37 \pm 4

Values are expressed as means \pm SEM.

carefully and recorded. Subjects assigned to the control group remained sedentary during the course of this investigation.

Testing protocol

Testing was conducted before and after the 10-week training and control periods. Prior to data collection, the subjects were familiarized with all pertinent laboratory procedures. Subjects did not receive any feedback about the results of the testing until the conclusion of the study. All post-training measurements were performed 24–48 h after the last exercise session to avoid the immediate effects of a single bout of exercise [15,16]. In addition, measurements before and after the training and control periods were obtained at about the same time of day (± 1 h) for each subject.

Resting heart rate and blood pressure

The resting heart rate and casual blood pressure were measured after the subject had sat resting for at least 10 min under quiet, comfortable laboratory conditions. The resting heart rate was measured with palpation in duplicate, and the average was used in statistical analysis. All of the blood pressure measurements were performed according to the guidelines established by the American Heart Association [17]. Briefly, after the arm circumference had been measured to determine the proper cuff size, the resting blood pressure was obtained in triplicate for both arms using a conventional mercury sphygmomanometer. To permit the release of blood trapped in the arm vein, each blood pressure measurement was separated from the next by a 1–2 min resting period. The blood pressure obtained from the arm with the higher readings was used for statistical analysis. Korotkoff phases I and V were taken as the systolic blood pressure (SBP) and diastolic blood pressure (DBP) values, respectively. All of the blood pressure measurements before and after the 10-week period were performed by the same trained observer who was blinded with respect to the group assignment. The supine blood pressure was measured during the 20 min rest before the casual forearm blood flow measurements in a quiet, temperature-controlled (27–30°C) laboratory. The mean arterial blood pressure (MABP) was determined from the following formula:

$$\text{MABP} = \text{DBP} + [(\text{SBP} - \text{DBP})/3]$$

Blood collection and analysis

A blood sample was extracted from an antecubital vein through an indwelling catheter after the patient had rested supine for at least 15 min under quiet, comfortable laboratory conditions. Each subject abstained from caffeine consumption and fasted overnight for at least 12 h before the determination of plasma catecholamines. Blood samples for epinephrine and norepinephrine determinations were placed in tubes containing EGTA and glutathione. Plasma epinephrine and norepinephrine con-

centrations were determined in duplicate according to the radioenzymatic technique described by Passon and Peuler [18]. Catechol-*o*-methyl transferase required for the catecholamine assay was extracted from rat livers using the purification procedure of Axelrod and Tomchick [19]. Each sample analyzed was accompanied by an internal standard for epinephrine and norepinephrine. All samples from a subject were analyzed within a single assay in an attempt to minimize the variability. The coefficients of variation of catecholamine assays in our laboratory averaged 9% for epinephrine and 11% for norepinephrine [20].

Plasma and blood volumes

The plasma volume was determined using a modification of Evans blue dye dilution method, as described previously [21]. After the subject had rested supine for 20 min, a control blood sample was extracted before injection of Evans blue dye; 4.0–4.5 ml Evans blue dye (New World Trading Corp., DeBary, Florida, USA) was injected from a preweighed syringe into an antecubital vein through an indwelling catheter. Exactly 10 min after the dye injection had begun, a 10 ml blood sample was extracted from an antecubital vein in the contralateral limb. These blood samples were analyzed with a spectrophotometer at wavelength 610 nm to determine the plasma volume. Venous hematocrit values were determined by the microcapillary method. The blood volume was determined subsequently from the plasma volume and venous hematocrit values [21].

Casual forearm vascular resistance

The casual forearm vascular resistance was measured using venous occlusion plethysmography and a mercury-in-Silastic strain gauge apparatus as described previously [22]. Subjects were supine for 20 min in a quiet, temperature-controlled (27–30°C) laboratory. One minute prior to casual forearm blood flow measurement the wrist cuff (TMC-7; Hokanson, Bellevue, Washington, USA) was inflated to 200 mmHg to occlude the circulation to the hand. A venous occlusion cuff placed on the upper arm was inflated to 50 mmHg for approximately 10 s (Hokanson E-20 rapid cuff inflator) for determination of the casual blood flow. During the blood flow measurement, the arterial blood pressure was measured simultaneously in the contralateral arm using the auscultatory method. The forearm vascular resistance was derived by dividing the resting MABP by the forearm blood flow, and expressed in arbitrary units. In our laboratory, the average difference between two within-day forearm blood flow measurements was 9% [22].

Body composition

The body composition was determined by the hydrostatic weighing technique using a submersion tank fitted with a submerged electronic scale. The measurements were repeated several times until a clear plateau of underwater weight had been noted or the highest underwater weight

had been reproduced. Immediately before hydrostatic weighing, the residual lung volume was determined by the oxygen dilution method using a calibrated nitrogen analyzer (505 Nitralyzer; MedScience, St Louis, Missouri, USA) and a 91 spirometer as described previously [23]. The percentage of body fat was estimated subsequently from the body density using the equation published by Siri [24].

Dietary intake

Subjects were instructed to follow their customary eating habits throughout the course of training, including on recording days. In an attempt to document this, dietary data were collected using 3-day food intake records before training and during the final week of the 10-week training period. The dietary records were analyzed for nutrient content using Nutritionist IV (N-Squared Computing, Salem, Oregon, USA).

Statistics

Statistically significant differences were determined by two-way (treatment versus time) analysis of variance with repeated measures. In the case of a significant interaction effect, a post-hoc test using Tukey's procedure was used to identify significant differences among mean values. Univariate correlation was performed to determine the relation between changes in arterial blood pressure and those in selected physiologic variables. $P < 0.05$ was considered statistically significant. Values are expressed as means \pm SEM.

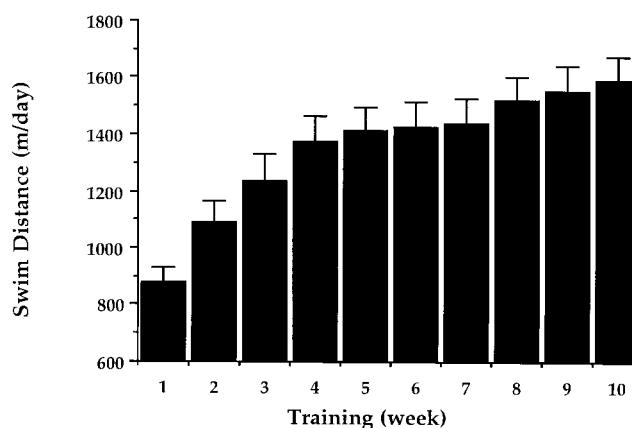
Results

Subjects in the training group were able to gradually increase their daily distance swum from the start of this investigation (Fig. 1). For the first 3 weeks (30–40 min per session), subjects swam 879 ± 54 to 1235 ± 93 m/day. During the final week, subjects averaged 1591 ± 80 m/day. The mean distance swum during week 10 was significantly ($P < 0.001$) greater than that during week 4 (the start of the 45 min per session swimming). The swimming training group completed an average of 94% of the scheduled exercise sessions.

The energy intake and dietary composition estimated from dietary records did not differ between before and after swimming training. There were no significant changes in total energy intake and macronutrient intake. Potentially confounding variables for arterial blood pressure, including alcohol (5 ± 3 versus 7 ± 4 g/day) and sodium (3346 ± 390 versus 3440 ± 359 mg/day) intakes, also did not differ between before and after training.

The body composition data are presented in Table 1. Although obesity was not an inclusion criterion, all of the subjects were obese. Their mean body fat percentages prior to the study were $36 \pm 2\%$ for the training group and

Fig. 1



Mean distances swum during the 10-week swimming training program. There were gradual and significant increases ($P < 0.05$) in daily distances swum. Bars, SEM.

$36 \pm 4\%$ for the control group. There were no significant differences in body mass and body composition between the groups. Neither training nor control group members experienced significant changes in body mass, lean body mass and relative percentage of body fat during the study.

Swimming training resulted in a significant ($P < 0.05$) reduction in resting heart rate whereas the mean heart rate value for the control group was not altered (Table 2). Systolic, diastolic, and mean arterial blood pressures in the control group at seated rest did not change during the study. In contrast, there was a significant ($P < 0.05$) reduction in seated systolic blood pressure in the swimming training group after 10 weeks of swimming training. It is important to note that the seated systolic blood pressure decreased in 11 of 12 subjects in the training group. The reduction in systolic blood pressure was approximately 6.6 mmHg from an initial value of 150 ± 5 mmHg. Seated

Table 2 Changes in resting heart rate and blood pressure

Variable	Training group	Control group
Resting heart rate (beats/min)		
Before	80.8 ± 3.8	74.0 ± 3.6
After	$70.7 \pm 3.2^*$	72.3 ± 5.1
Seated blood pressure (mmHg)		
Systolic		
Before	150 ± 5	140 ± 5
After	$144 \pm 4^*$	139 ± 4
Diastolic		
Before	96 ± 4	93 ± 3
After	94 ± 3	93 ± 3
Supine blood pressure (mmHg)		
Systolic		
Before	141 ± 5	132 ± 4
After	$135 \pm 5^*$	130 ± 4
Diastolic		
Before	90 ± 4	86 ± 3
After	85 ± 3	83 ± 3

Values are expressed as means \pm SEM. * $P < 0.05$, versus before.

Table 3 Casual forearm blood flow and vascular resistance in resting subjects

Variable	Training group	Control group
MABP (mmHg)		
Before	105 ± 4	99 ± 2
After	103 ± 4	96 ± 2
CFBF (ml/min per 100 ml)		
Before	3.9 ± 0.4	3.6 ± 1.2
After	3.5 ± 0.4	3.9 ± 0.9
CFVR (U)		
Before	31 ± 4	38 ± 7
After	34 ± 5	31 ± 6

Values are expressed as means ± SEM. MABP, mean arterial blood pressure; CFBF, casual forearm blood flow; CFVR, casual forearm vascular resistance.

Table 4 Changes in plasma and blood volumes and plasma catecholamines

Variable	Training group	Control group
Total plasma volume (ml)		
Before	3454 ± 165	2898 ± 188
After	3521 ± 161	2919 ± 167
Relative plasma volume (ml/kg)		
Before	33.2 ± 1.4	32.2 ± 2.5
After	34.4 ± 1.7	32.2 ± 1.9
Blood volume (ml)		
Before	5579 ± 291	4793 ± 374
After	5600 ± 280	4823 ± 319
Relative blood volume (ml/kg)		
Before	53.4 ± 2.1	53.2 ± 4.6
After	54.6 ± 2.6	53.3 ± 3.7
Plasma epinephrine (pg/ml)		
Before	53.9 ± 12.0	38.1 ± 10.5
After	42.4 ± 10.3	45.9 ± 13.1
Plasma norepinephrine (pg/ml)		
Before	373.5 ± 79.8	267.8 ± 122.2
After	369.4 ± 111.9	336.2 ± 105.9

Values are expressed as means ± SEM.

diastolic and mean arterial blood pressures were reduced by 2.5 and 3.9 mmHg, respectively. Swimming training resulted in reductions in supine systolic, diastolic, and mean arterial blood pressures similar to those measured in seated subjects. There were no significant changes in supine blood pressure in the control group. There were no obvious systematic differences between male and female subjects in terms of the direction and magnitude of changes in arterial blood pressure.

Table 3 presents the resting forearm blood flow and resistance data. The casual forearm blood flow and vascular resistance did not change significantly in the training and control groups during the study.

Table 4 shows plasma and blood volume data obtained using Evans blue dye. Hypertensive subjects in the training and control groups had no significant changes in plasma or blood volume, whether expressed in absolute terms or relative to their body mass, after 10 weeks of the respective intervention. There were no significant training-induced changes in epinephrine or norepinephrine concentrations in the training group.

Univariate correlation analyses were performed to determine which physiologic variables were associated most closely with changes in blood pressure. There were no significant correlations between changes in training-induced blood pressure and those in other physiologic variables (e.g. body mass, dietary intake, forearm vascular resistance, blood volume, and catecholamines).

Discussion

Despite the fact that swimming is widely recommended for the prevention and treatment of hypertension [5–7], it has not received much focus as a training mode for individuals with hypertension. Since a substantial number of patients choose a non-weight-bearing activity such as swimming as their primary form of physical activity, it is important to determine whether this type of exercise exerts antihypertensive effects. The present study is the first to have examined the effect of swimming training on the arterial blood pressure in individuals with hypertension. The primary new finding of this study was that swimming training produced a significant reduction in resting arterial blood pressure in individuals with hypertension. Training-induced reductions in resting blood pressure were independent of changes in body mass and dietary intake.

Since swimming is a rhythmic, dynamic form of endurance exercise involving a large muscle mass, it is a potentially useful alternate to land-based exercises insofar as the efficacy and safety of swimming can be assured. No subjects in the training group experienced musculoskeletal injuries during the course of this investigation. In addition, the incidence of injury among swimmers has been reported to be significantly lower than that among endurance exercisers in running and cycling [25]. In one rehabilitation center report, no mortality or complications were caused by swimming training during cardiac rehabilitation after myocardial infarction during a 10-year period [26]. Thus, it appears that swimming exercise can be prescribed safely in a manner similar to that for other activity modes.

The relative magnitude of the blood pressure reduction observed after swimming training was slightly smaller than that typically reported for land-based physical activity [27,28]. Studies using training programs equivalent (of similar intensity, frequency, and duration) to ours but employing walking/jogging [28] and cycling [27] reported 12 and 6 mmHg reductions in resting systolic and diastolic blood pressures, respectively. The reductions in systolic and diastolic blood pressures observed in the present study averaged 6.6 and 2.5 mmHg, respectively. It is not clear why the antihypertensive effect of swimming training was relatively smaller than that of land-based physical activity. Similarly to running and cycling, swim training can be considered aerobic exercise. Swimming is, however, inherently different from land-

based exercise in many respects due to water immersion and the prone body position. Physiologic responses to swimming are affected by many factors including the hydrostatic pressure, facial immersion, and the high thermal conductivity of water. The mean arterial blood pressure is higher during swimming despite cardiac output values being similar during swimming and during running [11]. Similarly, the mean arterial blood pressure during maximal swimming is significantly higher than that during maximal running despite the maximal cardiac output during swimming being lower [11]. The greater response of the blood pressure during swimming can be explained in terms of an increased total peripheral resistance caused by the lower skin temperature or recruitment of the smaller exercising musculature, or both [11]. Nevertheless, it is important to recognize that a 5–6 mmHg decrease in arterial blood pressure similar to that observed in this study has been associated with a 42% reduction in stroke incidence and a 14% reduction in coronary heart disease in a meta-analysis of several major epidemiologic studies [29].

Changes in blood volume and in the volume-regulating hormones have been suggested as potential mechanisms responsible for the antihypertensive effects of exercise training [27,30]. We did not observe significant changes in plasma or blood volumes after the swimming training program. This finding differs from those of a series of Japanese studies reporting that volume depletion could be the primary mechanism for the antihypertensive effects of exercise training [27,30]. The substantially higher sodium intake by the Japanese population (as high as 34 g/day) [31] makes interpretation of their results difficult. Conversely, our finding is in agreement with a study using American patients with hypertension. Hagberg *et al.* [32] reported no changes either in plasma or in blood volume after endurance training despite the fact that the training program resulted in a significant reduction both in systolic and in diastolic blood pressure.

Several studies attributed the antihypertensive effects of exercise training to the lowered vascular resistance [33]. A decreased vasodilatory capacity of forearm resistance vessels in hypertensive patients has been reported [34]. Since the primary source of propulsive force in swimming is the upper body muscles [35], it was of particular interest to examine changes in the 'forearm' vascular resistance after swimming training. Cross-sectional studies comparing dominant with nondominant arms of tennis players [36] indicate that exercise training lowers the forearm vascular resistance, and that the training effects may be localized. The present study found no reduction in resting forearm vascular resistance after swimming training. It is likely that the difference arose from the study design (i.e. cross-sectional versus longitudinal) or the use of resting flow versus maximal vasodilatation. We also can not exclude the possibility that, although the forearm

vascular resistance was not changed, the total peripheral resistance might have been reduced, just like in previous training studies in hypertension [33]. A significant reduction in total peripheral resistance has been observed without changes in forearm vascular resistance after 10 weeks of bicycle training [37].

In conclusion, the major new finding of the present study was that swimming training elicited a significant reduction in arterial blood pressure in individuals with hypertension. The blood pressure reduction occurred independently of changes in body weight and dietary intake. These results suggest that swimming training can be prescribed to patients with hypertension in a manner similar to that for other exercise modes. This is a clinically important finding because swimming can be a highly useful alternate to land-based exercises for obese patients and for those with exercise-induced asthma or orthopedic injuries.

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