Effects of endurance training on the cardiovascular system and water compartments in elderly subjects

GISELE P. PICKERING,1 NICOLE FELLMANN,2 BÉATRICE MORIO,2 PATRICK RITZ,2 AIME AMONCHOT,1 MICHEL VERMOREL,2 AND JEAN COUDERT1

1Laboratoire de Physiologie-Biologie du Sport, Faculté de Médecine, and 2Laboratoire de Nutrition Humaine, Centre de Recherche en Nutrition Humaine, Auvergne, 63001 Clermont-Ferrand, France

Pickering, Gisèle P., Nicole Fellmann, Béatrice Morio, Patrick Ritz, Aimé Amonchot, Michel Vermorel, and Jean Coudert. Effects of endurance training on the cardiovascular system and water compartments in elderly subjects. J. Appl. Physiol. 83(4): 1300–1306, 1997.—The effects of endurance training on the water compartments and the cardiovascular system were determined in 10 elderly subjects [age 62 ± 2 yr, pretraining maximal oxygen consumption (VO2max)/kg = 25 ± 2 ml·min⁻¹·kg⁻¹ body wt]. They trained on a cycleergometer 3 times/wk for 16 wk (50–80% VO2max, then 80–85% VO2max). They were checked at 8 wk, 16 wk, and 4 mo after detraining. Training improved VO2max (+16%) and induced plasma volume expansion (+11%). No change in total body water, extracellular fluid, interstitial and intracellular fluid volumes, fat-free mass, and body weight was detected in this small sample with training. Body fat mass decreased (-2.1 ± 2.2 kg). Echocardiography at rest showed increased fractional shortening and ejection fraction and decreased left ventricular end-systolic dimension (P < 0.05). Blood volume expansion correlates with cardiac contractility and has an impact on cardiac function. These improvements are precarious, however, and are completely lost after 4 mo of detraining, when elderly subjects lose the constraints and the social stimulation of the imposed protocol.

AGING IS ASSOCIATED with a continual change in all body systems. Research over the last 20 years has shown that some of these declines, such as that in maximal oxygen consumption (VO2max), can be acted on and slowed down by endurance training (11, 15). Aging is associated with a decrease of blood volume (BV) (4) and total body water (TBW) (23), a proneness to disturbances in water balance, homeostasis impairment while in extreme situations, and alterations in renal function (22), the perception of thirst (17), and cardiovascular performance (8). Although exercise-induced hypervolemia in young people is now well established (3, 7), studies in older subjects are fewer and controversial. Whereas Hagberg et al. (14) did not demonstrate any BV change after 9 mo of training (70–85% VO2max), Carroll et al. (1) showed an increased BV (+12%) in subjects after 26 wk of training (70% VO2max). Information on the global changes in water during chronic endurance exercise is also scarce: in older healthy subjects, Goran and Poehlman (10) showed increased TBW linked to increased fat-free mass (FFM) after 8 wk of endurance training at 60–80% VO2max. On the other hand, endurance training has positive effects on the cardiovascular system, especially on heart rate (HR) (13), ejection fraction (EF), and diastolic filling at rest and during exercise (16). In our global understanding of the effects of training on the water compartments and cardiovascular system in healthy elderly subjects, a few points are still incomplete: 1) water changes in the extra- and intracellular compartments, 2) the link between exercise-induced increased BV and the concomitant cardiac benefits, and 3) the evolution of these parameters with detraining. The purpose of our study was therefore to determine whether 4 mo of individualized training modify global and compartmental hydration in elderly subjects, induce plasma hypervolemia, and if so, how such training affects cardiac function. We were also interested in following the water and cardiovascular consequences of the interruption of a supervised protocol and the detraining, if any, that it may induce.

METHODS

Subjects

Ten healthy subjects [6 women, 4 men; age 62 ± 2 (SD) yr; weight 70.1 ± 14.8 kg; height 164 ± 1 cm] served as volunteers in this study. Recruitment was in response to advertisements for a medically supervised study of the effects of exercise training in older volunteers. Subjects had been sedentary before entry into the study and had no overt history of cardiovascular or pulmonary disease or any orthopedic limitations to exercise testing and training. They were taking no medication and were nonsmokers. All procedures were approved by the National Ethical Committee; written consent was obtained from all subjects.

A maximal exercise test, body composition, water compartment volumes, and echocardiography, determined before training (T1) and described below, were repeated after 2 mo (T2), at the end of the 4 mo of training (T3), and 4 mo after subjects had stopped training (T4).

Maximal Exercise Test and Anaerobic Threshold

Before acceptance of the protocol, the volunteers underwent a full medical examination and a test to exhaustion on a cycle ergometer (Ergometra) connected to a gas and volume analyzer (CPX/D Medical Graphics, St. Paul, MN). A satisfactory medical history and examination and a negative maximal test were preconditions to starting the protocol. Starting at 30 W, 30-W increments were applied every 2 min 30 s to exhaustion, with blood pressure and electrocardiographic recordings and strong verbal encouragement throughout the test. Oxygen consumption (VO2) was measured continuously by open-circuit spirometry and averaged every 30 s with the use of the automated one-line system. The test was considered as being maximal when three of the following four conditions were fulfilled: constant VO2 despite 30-W increment increase; maximal HR (HRmax) near theoretical HRmax [HRmax = 220 – age (yr)]; respiratory quotient >1.1; and exhaustion of the subject.
Earlobe blood samples were taken at the end of each step and 3 min after the end of the test for blood lactate concentration ([La]), measurements (Analox LMS). Measured maximal variables \( \text{VO}_{2\text{max}} \), maximal aerobic power (MAP), maximal expiratory ventilation (Ve\text{max}), and maximal [La] ([La\text{max}]) were used as baseline results (T1). Lactate threshold ([La]) was determined, and the corresponding HR threshold (HRt) was used as a guideline for subsequent individualized training. Each subject came to the laboratory on a second occasion for determination of body composition and water compartment volumes.

Anthropometry

Body weight (BW) was measured on a mechanical scale (Ceba) to the nearest 0.1 kg, with subjects wearing light underwear. Height was measured with the greatest 0.001 m. Skinfold thicknesses were measured at four sites on the left side of the body (triceps, biceps, subscapular, suprailiac) in triplicate with a skinfold caliper by the same person, as described by Durnin and Womersley (5). Body density was estimated by using these results in the equation of Durnin and Womersley, adapted to >50-yr-old subjects (5).

\[
\text{Density in men} = 1.1715 - 0.0779 \times \log \sum \text{skinfold thicknesses}
\]

\[
\text{Density in women} = 1.1339 - 0.0645 \times \log \sum \text{skinfold thicknesses}
\]

Percent body fat (BF) was then estimated with Siri's equation:

\[
\%\text{BF} = (4.95/\text{density} - 4.5) \times 100 (26) \text{ and converted to FFM: FFM (kg)} = [1 - (%\text{BF}/100)] \times \text{BW (kg)}.
\]

Blood Parameters

Fasting blood samples were taken for hematologic and biochemical analyses. Hemoglobin concentration ([Hb]), hematocrit (Hct), mean cell volume (MCV), and mean cell hemoglobin concentration (MCHC) were measured on an H3 Technicon (Beckman). Red cell mass (RCM) was calculated as (BV \times Hct) and total hemoglobin (THb) as BV \times [Hb]. Plasma Na\(^+\) concentration ([Na\(^+\)]) was measured by specific electrode and protein concentration ([Prot]) by the biuret method (Hitachi 911, Boehringer). Glucose and urea were measured by glucose oxidase and urea oxidase, respectively (Hitachi 911). Plasma osmolarity (mosmol/l) was calculated as 2 \times [Na\(^+\)] + urea concentration ([urea]) + glucose concentration ([glucose]).

Water Compartments

Plasma volume (PV), extracellular water (ECW), and TBW were measured as follows.

PV measurement. A catheter was inserted into an antecubital vein of fasting volunteers, and PV was measured with use of the Evans blue dye technique (9). An accurately weighed amount of 2.5 ml Evans Blue solution from sterile 5-ml ampules (at 5 mg/ml concentration) was then injected. Ten milliliters of saline were flushed to rinse the catheter, and blood samples were drawn 5, 7, 10, 15, and 20 min after injection. Evans blue concentrations of standard and plasma solutions were calculated from 620-nm and corrected 760-nm optical densities measured on a Unicam 8625 UV/VIS spectrophotometer (Unicam, Cambridge, UK). Time 0 concentration was calculated by back extrapolation of the dilution curve with time (T) and was used to calculate PV. BV was calculated as PV/(1 − Hct).

Variations of PV (%) were also calculated from Hct and [Hb] according to the equation (27)

\[
(1 - \text{Hct}T/1 - \text{Hct}T1) \times [\text{Hb}]T1/\text{[Hb]}T - 1
\]

where T1 corresponded to baseline and T to T2, T3, or T4 as defined earlier.

Extracellular fluid volume measurement. ECW volume was measured with the bromide-dilution technique. After collection of baseline blood samples, an accurately weighed amount of potassium bromide syrup, provided by the Regional Hospital Pharmacy, was taken orally by the volunteer. Blood samples were then taken through the inserted catheter after 4, 5, and 6 h, and plasma samples were frozen at −20°C. Volunteers continued fasting for 6 h but were permitted light activity within the laboratory (reading, watching TV, and so on). Samples were later analyzed by means of high-performance liquid chromatography as described by Miller et al. (18) by using a diode array detector (Partisl 10 SAX column, Whatman International, Maidstone, UK). Protein-free plasma samples were obtained after centrifugation by using a MPS1 micropartition system (Amicon, Eperon, France); ECW was calculated from the plateau concentration of plasma bromide (18).

TBW measurement. TBW was measured with the $^2$H$_2$O dilution technique (20, 24). Urine specimens were collected before oral administration of an accurately weighed dose (0.15 g/kg BW) of $^2$H$_2$O (99.9% enriched, Sigma Chemical, Poole, UK) and 4, 5, and 6 h afterward. $^2$H enrichments were measured with the zinc-reduction technique on an Optima dual-inlet mass spectrometer (VG Isotech). $^2$H dilution space was calculated from plateau enrichments in $^2$H, and TBW was considered 4% smaller than the dilution space.

Interstitial fluid volume (ITW) and intracellular water (ICW) were calculated as

\[
\text{ITW} = \text{ECW} - \text{PV}
\]

and

\[
\text{ICW} = \text{TBW} - \text{ECW}
\]

Echocardiography

Echocardiographic and pulsed-wave Doppler studies were performed on six subjects only at rest by using a Vibmed 750 model imaging system with electrocardiographic recording electrodes. Parasternal measurements were made through parasternal slits and aortic flux Doppler measurements through an apical slit. All measurements were averaged from at least three successive beats. All echocardiographic and Doppler measurements were made by a single observer, who was unaware of the fitness level and age of the subjects. The following variables were measured or derived: fractional shortening (FS; %); EF (%); left ventricle end-diastolic and end-systolic diameters (EDD and ESD, respectively), interventricular septal diastolic dimension (IVSD), and left ventricular posterior wall diastolic thickness (PVD), all in millimeters; stroke volume (SV; ml); and cardiac index (CI; l/min/m$^2$ body surface area).

Training

The exercise program comprised supervised bicycling on a Monark cycloergometer 3 times/wk for 16 wk and consisted of a moderate-intensity training period (T1-T2), followed by a high-intensity training period (T2-T3). All training sessions began with a 10-min warm-up and ended with a 10-min cooling-down session at the HR corresponding to 50% of the subject's VO$_{2\text{max}}$. After the first 2 wk with subjects training at 50% of VO$_{2\text{max}}$ for 20 min, a 25-min-long interval training period (with 5 min at a HR 50% of VO$_{2\text{max}}$ and 5 min at HR, respectively) was applied for 6 wk. From week 9 to week 16, intensity and length of training were gradually increased, with the same interval training pattern at a higher intensity.
for 35 min (with 5 min at HR, and 5 min above it, respectively), with care taken, because of the high value of the thresholds, that subjects always stayed ~10 beats below HR\textsubscript{max} as a safety measure.

HR was continuously recorded with a commercially available device (Sport Tester PE4000 Polar Electro, Kemple, Finland), and blood pressure was checked before and after exercise.

**Detraining**

After 4 mo of training (T3), subjects were completely discharged and strongly recommended to keep training as regularly as possible. Some of the subjects even purchased an ergocycle, and all were very enthusiastic and willing to maintain vigorous physical activity. They came back to the laboratory 4 mo later for reassessment. Their activity between T3 and T4 was evaluated via a questionnaire that showed that they had not succeeded in maintaining vigorous physical activity and that they had replaced it for leisure activity and that they had replaced the vigorous physical activity. They came back to the laboratory 4 mo later for reassessment. Their activity between T3 and T4 was evaluated via a questionnaire that showed that they had not succeeded in maintaining vigorous physical activity and that they had replaced it for leisure activity and that they had replaced it for leisure.

### Statistical Analyses

Data analysis was performed with the Statview statistical package (Abacus Concept, Statview). Statistical differences were established by using a repeated-measures analysis of variance design with a confidence level of $P < 0.05$. All data were expressed as means $\pm$ SD. **RESULTS**

**Anthropometry (Table 1)**

BW was maintained constant all through training and detraining. FFM did not increase significantly ($+1.3 \pm 2.9 \text{ kg}; P = 0.09$), but BF decreased between T1 and T3 ($-2.1 \pm 2.2 \text{ kg}, i.e., -2.3 \pm 2.9\%; P < 0.05$). After 4 mo of detraining, BF was back to its pretraining value.

**Bioenergetics (Table 2)**

$V_{O2\text{max}}$ (l/min and ml·min$^{-1}$·kg$^{-1}$ BW) did not vary significantly between T1 and T2, but increases between T2 and T3 were significant: $+175 \pm 123 \text{ ml/min (i.e., } +10 \pm 8\%; P < 0.01)$ and $+2.6 \pm 2.0 \text{ ml}·\text{min}^{-1}·\text{kg}^{-1} \text{ BW (i.e., } +10 \pm 8\%; P < 0.01)$. The overall $V_{O2\text{max}}$ (l/min and ml·min$^{-1}$·kg$^{-1}$ BW) training gain between T1 and T3 was $+237 \pm 152 \text{ ml/min (i.e., } +14 \pm 8\%; P < 0.01)$ and $+3.8 \pm 2.1 \text{ ml}·\text{min}^{-1}·\text{kg}^{-1} \text{ BW (i.e., } +16 \pm 8\%; P < 0.01)$, respectively. There was no correlation between pretraining $V_{O2\text{max}}$ (ml·min$^{-1}$·kg$^{-1}$ BW), which indicates the initial degree of fitness, and the percentage $V_{O2\text{max}}$ (ml·min$^{-1}$·kg$^{-1}$ BW) gain from T1 to T3.

MAP (15 W, i.e., $+16 \pm 10\%; P < 0.01$) and $V_{E\text{max}}$ ($P < 0.05$) increased, whereas HR\textsubscript{max} and [La]\textsubscript{max} were maintained constant. La\textsubscript{max} did not change significantly between T1 and T3 ($\%V_{O2\text{max}} 77 \pm 9\%$) nor did corresponding HR ($133 \pm 12$ beats/min).

After 4 mo of detraining, $V_{O2\text{max}}$ (ml·min$^{-1}$·kg$^{-1}$ BW) decreased by 14% ($P < 0.01$) and came back to its T1 value, as did MAP and $V_{E\text{max}}$.

### Water Compartments (Table 3)

Between T1 and T3, PV, hence BV, increased with training: $+263 \pm 80 \text{ ml (i.e., } +11 \pm 3\%; P < 0.01)$ and $+286 \pm 120 \text{ ml (i.e., } +7 \pm 3\%; P < 0.01)$. Calculated PV change ($+10 \pm 4\%$) was similar to the change ($+11 \pm 3\%) measured with Evans blue dye dilution. PV and

### Table 2. Maximal exercise test parameters

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
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</thead>
<tbody>
<tr>
<td>$V_{O2\text{max}}$\text{ l/min}</td>
<td>1.78$\pm$0.54</td>
<td>1.84$\pm$0.53</td>
<td>2.01$\pm$0.56</td>
<td>1.76$\pm$0.62</td>
</tr>
<tr>
<td>$V_{O2\text{max}}$\text{ ml}·\text{min}^{-1}·\text{kg}^{-1}$</td>
<td>25.0$\pm$3.2</td>
<td>26.3$\pm$3.2</td>
<td>29.0$\pm$3.7</td>
<td>25.0$\pm$4.5</td>
</tr>
<tr>
<td>HR\textsubscript{max} beats/min</td>
<td>161$\pm$36</td>
<td>158$\pm$14</td>
<td>161$\pm$17</td>
<td>161$\pm$15</td>
</tr>
<tr>
<td>MAP, W</td>
<td>123$\pm$36</td>
<td>129$\pm$39</td>
<td>134$\pm$45</td>
<td>141$\pm$44</td>
</tr>
<tr>
<td>$V_{E\text{max}}$</td>
<td>71.4$\pm$19</td>
<td>73.5$\pm$22.1</td>
<td>80.6$\pm$24.4</td>
<td>74.2$\pm$24.6</td>
</tr>
<tr>
<td>$[\text{La}]\text{max}$ mmol/l</td>
<td>7.0$\pm$18</td>
<td>7.8$\pm$2.0</td>
<td>7.5$\pm$19</td>
<td>7.6$\pm$14</td>
</tr>
<tr>
<td>SBP\textsubscript{max}, mmHg</td>
<td>177$\pm$18</td>
<td>181$\pm$18</td>
<td>181$\pm$20</td>
<td>174$\pm$32</td>
</tr>
<tr>
<td>DBP\textsubscript{max}, mmHg</td>
<td>91$\pm$8</td>
<td>89$\pm$6</td>
<td>86$\pm$10</td>
<td>86$\pm$7</td>
</tr>
<tr>
<td>HR\textsubscript{max}, beats/min</td>
<td>133$\pm$12</td>
<td>128$\pm$13</td>
<td>135$\pm$16</td>
<td>133$\pm$10</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SD; $n = 10$ subjects. $V_{O2\text{max}}$, maximal O\textsubscript{2} consumption (l/min), uptake (ml·min$^{-1}$·kg$^{-1}$ BW); HR\textsubscript{max}, maximal heart rate; MAP, maximal aerobic power; $V_{E\text{max}}$, maximal expiratory ventilation; $[\text{La}]\text{max}$, maximal lactate concentration; SBP\textsubscript{max}, maximal systolic blood pressure; DBP\textsubscript{max}, maximal diastolic blood pressure; HR\textsubscript{max}, anaerobic threshold heart rate. *Different from T1, $P < 0.01$. †Different from T2, $P < 0.01$ (nonparametric paired Wilcoxon test).

### Table 3. Water compartments

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>PV, liters</td>
<td>2.54$\pm$0.59</td>
<td>2.69$\pm$0.59</td>
<td>2.81$\pm$0.64</td>
<td>2.57$\pm$0.62</td>
</tr>
<tr>
<td>BV, liters</td>
<td>4.35$\pm$1.19</td>
<td>4.51$\pm$1.19</td>
<td>4.64$\pm$1.25</td>
<td>4.36$\pm$1.21</td>
</tr>
<tr>
<td>ECW, liters</td>
<td>16.9$\pm$3.5</td>
<td>16.0$\pm$3.7</td>
<td>16.0$\pm$3.3</td>
<td>16.0$\pm$3.3</td>
</tr>
<tr>
<td>TBW, liters</td>
<td>35.5$\pm$10.2</td>
<td>35.5$\pm$9.9</td>
<td>35.9$\pm$11.3</td>
<td>34.4$\pm$9.3</td>
</tr>
<tr>
<td>ITW, liters</td>
<td>14.3$\pm$3.1</td>
<td>13.3$\pm$3.3</td>
<td>13.2$\pm$2.8</td>
<td>13.7$\pm$2.8</td>
</tr>
<tr>
<td>ICW, liters</td>
<td>19.2$\pm$7.0</td>
<td>19.3$\pm$7.1</td>
<td>19.5$\pm$8.0</td>
<td>19.5$\pm$8.0</td>
</tr>
</tbody>
</table>

Values are means $\pm$ SD; $n = 10$ subjects. PV, plasma volume measured by Evans blue dye dilution; BV, blood volume; ECW, extracellular fluid measured by bromide dilution; TBW, total body water measured by $^3$H\textsubscript{2}O dilution; ITW, interstitial fluid volume calculated from ECW-PV; ICW, intracellular water space calculated from TBW – ECW. *Different from T1, $P < 0.05$. †Different from T1, $P < 0.01$ (nonparametric paired Wilcoxon test).
ECW were not measured at T4 to lighten the tests, and therefore ICW and ITW could not be calculated. We calculated PV at T4 on the basis of changes of Hct and Hb from T1 values.

TBW, ECW, calculated ITW, and calculated ICW did not change significantly with training. BV (liters) and VO$_{2\text{max}}$ (l/min) at T1, T2, T3, and T4 show a good correlation ($P < 0.01, r = 0.814$ at T1), but we did not show a relationship between the increases in BV ($\%$) and VO$_{2\text{max}}$ ($\%$) from T1 to T3.

After 4 mo of detraining, PV and BV decreased ($-239 \pm 204$ ml, i.e., $-9 \pm 6\%; P < 0.01$) and ($-276 \pm 185$ ml, i.e., $-6 \pm 4\%; P < 0.01$), respectively, and were back to pretraining values. There were no significant correlations between %changes from T1 to T3 in TBW and VO$_{2\text{max}}$ and TBW and BV.

Blood Parameters (Table 4)

From T1 to T3, [Na$^+$], [Prot], and plasma osmolarity were maintained, whereas total circulating Na$^+$ and protein increased ($P < 0.01$ and $P < 0.05$, respectively). RCM ($1.81 \pm 0.6$ liter), MCV ($91.4 \pm 4.8$ fl), MCHC ($32.9 \pm 0.08$ g/dl), and Hb ($597 \pm 215$ g) were maintained, whereas [Hb] and Hct decreased ($P < 0.01$).

After 4 mo of detraining, [Na$^+$], [Prot], and plasma osmolarity did not change, but Na$^+$ and protein masses, Hct, and [Hb] were back to pretraining values.

Cardiovascular Parameters (Table 5)

Resting ($136 \pm 84 \pm 8$ mmHg) and maximal (Table 2) systolic and diastolic blood pressures did not change with training and detraining. The resting HR decrease was not significant ($86 \pm 14$ beats/min (T1) vs. $82 \pm 14$ beats/min (T3)). Echocardiography (T1-T3) showed a significant increase in FS ($+20\%; P < 0.05$) and EF ($P < 0.05$) and a concomitant decrease of ESD ($P < 0.05$). All other cardiac parameters studied were maintained. At T4, ESD, FS, and EF were not significantly different from pretraining values.

Variations of FS (%) and EF (%) correlated well with BV changes (%) between T1 and T3 ($r = 0.885, P < 0.05$ and $r = 0.904, P < 0.05$, respectively) (Figs. 1 and 2).

Table 4. Blood parameters

<table>
<thead>
<tr>
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<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>[Na$^+$], mmol/l</td>
<td>142±1</td>
<td>142±2</td>
<td>141±2</td>
<td>143±2</td>
</tr>
<tr>
<td>Na$^+$ mass, mmol</td>
<td>360±66</td>
<td>375±84</td>
<td>394±89†</td>
<td>367±94</td>
</tr>
<tr>
<td>[Prot], g/l</td>
<td>71±5</td>
<td>70±3</td>
<td>69±3</td>
<td>73±3</td>
</tr>
<tr>
<td>Prot mass, g</td>
<td>188±45</td>
<td>189±37</td>
<td>203±43*</td>
<td>190±45</td>
</tr>
<tr>
<td>[Urea], mmol/l</td>
<td>6.3±1.0</td>
<td>6.3±0.7</td>
<td>5.8±1.5</td>
<td>6.1±0.7</td>
</tr>
</tbody>
</table>
| [GlC], mmol/l     | 5.2±0.6| 5.4±0.9| 4.8±0.7| 4.8±0.8|†
| Plasma osmolarity, mosmol/l | 294±3  | 296±4  | 293±4  | 295±3  |
| Hct, %            | 41.0±3.0| 39.4±4.0| 39.0±3.1*| 40.6±2.9|
| [Hb], g/dl        | 13.5±1.2| 13.1±1.4| 12.7±1.1*| 13.6±1.1|

Values are means ± SD; n = 10 subjects. [Na$^+$], plasma Na$^+$ concentration; [Prot], plasma protein concentration; [GlC], plasma glucose concentration; plasma osmolarity = 2 $\times$ [Na$^+$] + [urea] + [GlC]; Hct, hematocrit; [Hb], hemoglobin concentration. *Different from T1, $P < 0.05$; †Different from T1, $P < 0.01$; ‡Different from T2, $P < 0.05$ (nonparametric paired Wilcoxon test).

DISCUSSION

The short and intense endurance-training protocol we proposed to our healthy sedentary subjects improved their aerobic and cardiac functions. VO$_{2\text{max}}$ improvement ($+16 \pm 8\%$, T1-T3) is within the range of published studies [from 10 to 28% (15)]. However, our protocol is shorter than most, extending up to 1 yr of training and/or with more training sessions per week. Studies report different training programs, with the intensity on the basis of VO$_{2\text{max}}$ or HR reserve, and exact comparison with our protocol is difficult because its intensity is based on the anaerobic threshold HR and not on VO$_{2\text{max}}$ alone. Our protocol has shown that a short but individualized training period leads quickly to improvements in exercise capacity and a significant gain in MAP of 20 W, corresponding almost to one step of the maximal exercise test protocol. Most of the VO$_{2\text{max}}$ increase took place during the high-intensity (T2-T3) period ($+10 \pm 8\%$); this stresses the fact that a high level of intensity is required to get any valuable VO$_{2\text{max}}$ improvement. We studied the influence of the initial level of fitness (pretraining VO$_{2\text{max}}$) on the VO$_{2\text{max}}$ improvement and found it was not a determining factor.

![Fig. 1. Correlation between fractional shortening variation (FS; %) and changes in blood volume (BV; %) with 4 mo of endurance training.](image-url)
in these sedentary subjects as in the study of Kohrt et al. (15). Among central and peripheral mechanisms proposed for the increase of exercise capacity with training, we focused on the concomitant changes of resting cardiovascular and BV parameters. FS and EF increased with training and correlated well with exercise-induced BV expansion. Detraining, however, led to a return to pretraining values of VO2max, BV, FS, and EF, thus stressing the benefits and the responsibility of the imposed protocol in changing cardiovascular and BV variables.

With training, the cardiovascular axis has to accommodate progressively a 7% BV expansion. Echocardiographic assessment of the left ventricle at rest, apart from increases of FS and EF, shows a concomitant decrease of ESD. This implies a better emptying of the ventricle with each systole at rest and improved inotropic effect. Age-associated incomplete emptying of the older heart can thereby be improved by short-term training for an elderly sedentary population. The good correlation between resting FS and EF and exercise-induced BV changes translates the direct and proportional impact of the increased preload and stretch on the cardiac myocyte. The increased preload was probably accompanied by an increased end-diastolic pressure (not measured), and although resting EDD shows only a tendency to increase and SV was not significantly modified, the results reflect the Frank-Starling relationship. The heart, with its permissive role, accommodates the increased BV by an augmentation of contractility, probably also through improved local cardiac oxidative mechanisms. Studies have shown that elderly subjects rely more than the young on the Frank-Starling mechanism, especially to increase cardiac output during exercise (21): reliance is more marked in elderly men than in women (8) because men make greater use of this mechanism at rest (8).

On the other hand, ESD, HR, and EF are catecholamine-mediated cardiovascular parameters, especially during exercise (21). Resting HR did not decrease significantly, probably because of the shortness of the protocol and also because of the size of the sample. The resting decreased ESD and increased EF might reflect the evolution of the resting sympathetic nervous system activity with training toward an enhancement of this sympathetic control. Increased resting circulating catecholamine levels and decreased β-adrenergic responsiveness are associated with aging and/or adaptations to physical inactivity. Repeated solicitations of the sympathetic system with adaptation to chronic physical exercise must modify resting sympathetic nervous system activity. Norepinephrine and epinephrine concentrations have been reported as being maintained (1) or increased (19) with training. Modifications of catecholamine receptor number or affinity and Ca2+-coupling proteins could also be adaptations to training and induce improvement of the heart’s β-adrenoceptor responsiveness. Training would thereby shift, even partially, the reliance of elderly subjects on the Frank-Starling mechanism to an enhanced catecholamine-mediated control. The study of Ehsani et al. (6), with a 12-mo-long protocol, gives different resting cardiovascular results with increased EDD and maintained EF and ESD, implying an enhanced Frank-Starling mechanism. These results might differ from ours because of the length of the study, a larger BV increase (not measured), or the gender of the population (11 men).

Maintained thicknesses of the PWD and the IVSD rule out any heart hypertrophy with this short training protocol. Training had no detrimental effect on resting and maximal blood pressure, despite attenuation of high-pressure baroreceptor sensitivity with aging (2). There might also have been a reduction in peripheral resistance because of increased muscle vasodilation. Most of the FS increase took place when subjects were training at moderate intensity, tending to indicate that training at and around anaerobic threshold improves cardiac function better than strenuous high-intensity exercise.

Another physiological adaptation to endurance training is the lowered [Hb] and Hct. Expanded PV without change of RCM is the major contributor to this hemodilution. This hemodilution decreases the oxygen-carrying capacity of the arterial blood, but compensatory mechanisms have been proposed, such as an increased oxygen delivery to the muscles because of the lower affinity of Hb for oxygen, decreased blood viscosity that favors perfusion, and improved blood flow to muscles with training. The good correlation between VO2max (l/min) and BV (liters) (i.e., T1 = P < 0.01, r = 0.81) at each stage of the protocol shows the concomitant increases of VO2max and of hemodilution with training; however, we did not find any correlation between the relative increases of BV and VO2max (as percentages of the pretraining values), but the wide individual variability and the size of the sample might be explanatory factors.

The exercise-induced plasmatic hypervolemia (+11%), hence BV expansion (+7%), represents the only fluid modification with training among the water compartments. It is accompanied by maintained [Na+] and [Prot] and increased total circulating protein and Na+. Na+ conservation was very effective in our study de-
Despite literature (22) describing progressive renal Na⁺ conservation defects and reduced aldosterone levels with age, which argue in favor of a limitation of water and electrolyte retention. Na⁺ retention via an aldosterone-feedback mechanism must have diluted intravascular proteins, triggered liver control on oncotic pressure, and increased albumin synthesis, holding water in the intravascular compartment. In our study, PV expansion is definitely not the consequence of increased global water expansion, as suggested by some authors (3).

We did not use a control group of young subjects in our study but assumed that the same techniques used by other investigators (4, 17) provide results comparable to those in our study. When our elderly volunteers in the pretraining stage are compared with younger populations (4, 17), they are in a hyperosmotic hypovolemic state, with higher plasma osmolality (although within physiological range) and lower BV. Elevation of thirst plasma osmolality threshold and impaired perception of thirst (17), attenuation of cardiopulmonary volume-pressure baroreflex function (2, 12, 25), and loss of interaction between cardiopulmonary and aortic carotid reflexes (25) have been described in connection with aging. Despite this age-associated decline of functions, elderly subjects can adapt to training as well as do the young. Exercise-induced hypervolemia is accompanied by maintained electrolyte concentration and also hormone concentration (1) and is perhaps associated with a resetting of volume receptors (1, 3). Plasma osmolality has been maintained with training: this could be accompanied by a decrease in the thirst plasma osmolality threshold, as observed in young athletes with training, but we have no data on thirst ratings and perception with training in our population.

Although the good electrolyte and protein homeostasis proves the adaptation of elderly subjects to training, no changes in TBW, ECW, ICW, and FFM were detected. These results are probably linked to the small size of our sample. Although the skinfold-thickness measurement method is weak in the elderly population and prone to random error because of variations in skin elasticity and compressibility, a decrease of BF (~2.1 ± 2.2 kg; P < 0.05) took place with training. The maintained TBW can only attest that the weight changes with training, although not significant, are largely because of fat loss.

After patients were outside the protocol for 4 mo, evaluation showed that they had completely lost all their aerobic (VO₂max), BV (PV), body composition (BF), and cardiovascular (FS, EF, ESD) improvements, with results similar to those in the T1 stage. Although high motivation and good will were evident at the end of the endurance training period, it is difficult for sedentary healthy elderly subjects to maintain training outside the frame of the protocol, and the 3 h/wk devoted to training are rapidly replaced by nonphysical leisure activities. BV and thus cardiac improvements are rapidly lost, and the subjects return to their initial state after 4 mo of detraining. The maintenance of exercise is especially difficult in the sedentary population, which is instinctively slower and more reluctant to join training groups on a long-term basis.

In conclusion, our study has shown that 4 mo of individualized training that enhances VO₂max improves the resting systolic function of the heart in healthy sedentary elderly subjects. This improved cardiac contractility is correlated with the exercise-induced BV expansion. Global and intracellular body hydration are not modified by training in this small sample, but elderly subjects manage to adequately preserve their electrolyte homeostasis. These gains are of a precarious nature and cannot be maintained outside the social stimulation and friendly competitiveness of the imposed protocol. The subjects rapidly lose all the cardiovascular and aerobic benefits strenuously acquired over 4 mo of vigorous individualized training.

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