Lack of age-associated elevations in 24-h systolic and pulse pressures in women who exercise regularly

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Lack of age-associated elevations in 24-h systolic and pulse pressures in women who exercise regularly. Am. J. Physiol. 277 (Heart Circ. Physiol. 46): H947–H955, 1999.—We tested the hypothesis that the elevations in 24-h arterial systolic (SBP) and pulse (PP) pressures with age in sedentary adult females are absent or smaller in women who exercise regularly. Four groups of healthy normotensive women were studied: premenopausal (n = 12; 29 ± 1 yr, mean ± SE) and postmenopausal (n = 20; 62 ± 1) sedentary, and premenopausal (n = 14; 30 ± 1) and postmenopausal (n = 12; 58 ± 1) endurance-exercise trained (distance runners). In the sedentary group, 24-h SBP and PP (Spacelabs ambulatory monitor 90207) were ~10 mmHg higher (P < 0.05) in the postmenopausal women than in the premenopausal controls; this was because of higher daytime and nighttime SBP and PP levels in the postmenopausal women. In contrast, 24-h, daytime and nighttime SBP and PP were not different with age in the endurance-trained women. SBP variability and SBP load (% of all recordings > 140 mmHg) generally were greater with age in the sedentary women (e.g., SBP load = 14 ± 4 vs. 3 ± 1%, P < 0.05) but not in the endurance-trained women. In the pooled population, 24-h SBP and PP were related to waist-to-hip ratio (measure of abdominal adiposity) (r = 0.48 and 0.49, respectively, P < 0.001) and carotid augmentation index (measure of arterial stiffness) (r = 0.43 and 0.53, P < 0.005). In the sedentary women, accounting for the influence of either of these factors eliminated the significant age-associated differences in 24-h SBP and PP (P > 0.3). Our results suggest that the elevations in 24-h SBP and PP with age in sedentary adult females may not occur in women who regularly perform endurance exercise. This appears to be related to the absence of age-associated increases in abdominal adiposity and arterial stiffness in the exercising women.

blood pressure variability; abdominal adiposity; arterial stiffness

ARTERIAL BLOOD PRESSURE (BP) increases with advancing age in adult humans, the largest elevations occurring in systolic pressure (SBP) with consequent increases in pulse pressure (PP) (13, 22). The increases in SBP and PP in women from their premenopausal years (e.g., 30–39 yr) to their postmenopausal years (e.g., >50 yr) are approximately twofold greater than those observed in men over the same period (8, 13, 22). These marked increases in SBP and PP with age in adult females can be attributed in part to women who eventually develop clinical hypertension. However, we (5) and others (20, 30, 34) have shown that SBP and PP are higher even in healthy normotensive postmenopausal compared with premenopausal sedentary women.

In light of the important health implications associated with elevated SBP and PP (7, 13), physiological factors or states that may modulate increases with age are of considerable interest. In this context, we recently (26) found that 24-h levels of SBP, a more reliable measure of mean daily levels than casually determined SBP at rest (17, 30, 34), were lower in endurance exercise-trained postmenopausal women than in sedentary postmenopausal women; although not reported, 24-h PP also was lower in the trained women. Moreover, SBP variability and SBP load (% of recordings > 140 mmHg), which along with mean 24-h levels of SBP and PP are important predictors of elevated BP-related target organ damage (14, 16, 19, 33), were lower in the exercising women. These observations suggest that the age-related increases in average daily SBP and PP, as well as SBP variability and load, may be attenuated or even absent in women who exercise regularly. However, because young adult controls were not included in this previous study, no insight was provided regarding age-related differences.

Absent or smaller age-related elevations in 24-h SBP and PP in women who exercise may be related in part to adiposity and/or arterial stiffness. In our previous study (26) the strongest physiological correlates of 24-h SBP were measures of total and abdominal body fat. We recently reported smaller age-related elevations in total and abdominal adiposity in endurance exercise-trained compared with sedentary women (31). Thus we would predict that these smaller body fat differences with age in the exercising women would be associated with more favorable 24-h SBP and PP.

In our earlier study on 24-h BP (26) measures of central arterial stiffness, an important determinant of age-associated increases in SBP and PP (6, 9, 29), were not obtained. In a subsequent investigation (28), however, we demonstrated that central arterial stiffness was higher in postmenopausal compared with pre-

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menopausal sedentary women, but was not different with age in endurance-trained women. These observations were consistent with earlier findings of lower central arterial stiffness in endurance exercise-conditioned compared with sedentary middle-aged and older men (29). Therefore, it is possible that this factor may also contribute mechanistically to habitual exercise-related differences in 24-h SBP and PP with age in women.

In the present study we tested the hypothesis that compared with healthy sedentary women, 24-h SBP and PP, SBP variability, and SBP load either would not be elevated or would be elevated to a lesser extent with age in women who regularly perform endurance exercise. If so, we further hypothesized that this would be related to more favorable age-associated differences in adiposity and/or arterial stiffness in the exercising women.

METHODS

Subjects

Fifty-eight healthy Caucasian women were studied: 14 premenopausal and 12 postmenopausal endurance exercise-trained, and 12 premenopausal and 20 postmenopausal sedentary controls. The endurance-trained premenopausal and postmenopausal women were distance runners who had been training for 16.4 ± 1.7 and 8.6 ± 1.0 yr (mean ± SE), respectively. The sedentary women had performed no regular physical exercise for at least the previous 2 yr. All subjects were free of overt coronary artery disease as assessed by medical history, physical examination, and resting and maximal exercise electrocardiograms (postmenopausal women only). Postmenopausal status was documented by plasma follicle-stimulating hormone (FSH) levels exceeding 30 mU/ml (25). Estrogen-based hormone supplementation was used for at least the entire preceding year by 7 of the 12 (58%) endurance-trained women and by 11 of the 20 (55%) sedentary postmenopausal women. None of the subjects smoked or took other medications that could affect BP. The experimental protocol was approved by the Human Research Committee at the University of Colorado at Boulder; voluntary written informed consent was obtained from each subject after the nature, purpose, and risks of the study had been explained to them.

Measurements

Maximal oxygen consumption was measured using on-line computer assisted open circuit spirometry during incremental treadmill exercise, as previously described (3), and was used as a measure of maximal aerobic capacity. Weekly running mileage was used as a measure of exercise training volume in the exercising women (3, 28, 31). Estimated physical activity-related energy expenditure was assessed in the sedentary women using the Stanford Physical Activity Questionnaire (21) to document an absence of age-group differences. Body mass index (BMI) was calculated from body mass and height (kg/m²). Total body density was determined by hydrodensitometry. Residual volume of the lungs was measured using the oxygen dilution technique (2). Body fat percentage was calculated using the equation of Brozek et al. (2). Fat mass and fat-free mass were estimated from percent fat and body mass. Waist circumference and waist-to-hip ratio, which have been validated against computed tomographic and magnetic resonance imaging measures of abdominal adiposity (1, 4), were used as measures of total abdominal body fat. Three-day dietary records were analyzed to provide an estimate of sodium intake as described previously (26, 27, 31), and sodium excretion was determined from an overnight urine collection.

Casual BP during quiet supine rest was measured by conventional sphygmomanometry using guidelines established by the American Heart Association (15) as described previously by our laboratory (24, 26). BP recordings over a 24-h period of normal daily activity were made using a noninvasive ambulatory monitor (model 90207, Spacelabs, Redlands, WA) as described in detail previously (23, 24, 26, 27). The ambulatory system was calibrated against a mercury sphygmomanometer and the cuff was programmed to inflate automatically every 15 min from 6 AM to 11 PM and every 20 min between 11 PM and 6 AM. For each individual subject, the nighttime period was defined as the time when the subject went to bed at night until she arose the following morning; daytime was determined as the remainder of the 24-h period. SBP and diastolic BP (DBP) variabilities were assessed as the standard deviations of the individual BP recordings (19, 26, 27, 34). SBP load was determined as percentage of SBP recordings >140 mmHg, and DBP load the percentage of DBP recordings >90 mmHg (26, 27, 33). PP was calculated as SBP – DBP. To eliminate the effects of acute postexercise hypotension (10) while preserving as much as possible the normal physiological state of the runners (i.e., endurance training on most days of the week), all of the above BP measures were obtained ~20 h after their last bout of exercise (26, 27).

Determination of carotid augmentation index (AI), a measure of central arterial stiffness and arterial wave reflection (9), was obtained with the subjects in the supine position as previously described (28). The pressure waveform and amplitude were obtained from the right common carotid artery with a pencil-type probe incorporating a high-fidelity strain gauge transducer (Millar Instruments, Houston, TX). The pressure waveform consists of both a "forward" or "incident" wave, and a "reflected" wave that is returning from a peripheral site. The reflected wave is superimposed on the incident wave such that the pulse and systolic pressures are increased. This increase is defined as the carotid AI, and it is calculated as the pressure wave above its systolic shoulder divided by pulse pressure. The shoulder was defined as the first concavity on the upstroke of the wave. All the analyses were performed by the same investigator who was blinded to the group assignment.

Data Analysis

Group differences were examined by two-way analysis of variance with Newman-Keuls post hoc analysis if the significance level was P < 0.05. Simple correlation and regression analyses were used to assess relations of interest. Forward stepwise multiple regression analysis was used to assess the independent and cumulative contributions of factors of interest to the individual variance in BP variables. Analysis of covariance (ANCOVA) was used to examine age-group differences in BP after accounting for the independent effects of related factors. Data are presented as means ± SE.

RESULTS

Subjects

Selected subject characteristics are presented in Table 1. Body mass, BMI, waist circumference, and waist-to-hip ratio were higher in the postmenopausal sedentary (all P < 0.05), but not in the endurance-trained women compared with their respective premenopausal controls. Height was not different with age in either
population. Body fat was higher in both postmenopausal groups (P < 0.05) but the age-related difference was twice as great in the sedentary women. Maximal oxygen consumption was similarly lower in the postmenopausal women in both groups (P < 0.05). Estimated physical activity-related energy expenditure was similar in the two sedentary age groups. Weekly running mileage was 30% lower in the postmenopausal compared with the premenopausal endurance-trained women (P < 0.05). There were no age-associated differences in dietary sodium intake or urinary sodium excretion in either population.

Casually Determined BP Under Resting Conditions.

Mean levels of BP at rest are shown in Table 2. Both SBP and DBP were within the normotensive range for all groups. SBP and PP were higher (P < 0.05) in the postmenopausal women regardless of exercise status. However, the mean age-associated differences were approximately two- to threefold greater in the sedentary (+13–18 mmHg) compared with the exercising (+5–10 mmHg) women. DBP was not different between age groups in either population.

Ambulatory-Recorded BP

Mean levels. Ambulatory-determined levels of SBP and PP are presented in Figs. 1 and 2, respectively. In the sedentary group, 24-h SBP and PP were higher (P < 0.05) in the postmenopausal women than in the premenopausal controls; this was because of higher (P < 0.05) daytime and nighttime SBP and PP in the postmenopausal women. In contrast, 24-h, daytime and nighttime DBP were not different between age groups in either population.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Sedentary</th>
<th>Endurance Trained</th>
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<tbody>
<tr>
<td></td>
<td>Premenopausal (n = 12)</td>
<td>Postmenopausal (n = 20)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>29 ± 1</td>
<td>62 ± 1*</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>62.9 ± 3.5</td>
<td>69.9 ± 2.6*</td>
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<tr>
<td>Height, cm</td>
<td>166.4 ± 1.4</td>
<td>163.0 ± 2.2</td>
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<tr>
<td>BMI, kg/m²</td>
<td>22.8 ± 1.3</td>
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</tr>
<tr>
<td>% Body fat</td>
<td>26 ± 2</td>
<td>35 ± 1*</td>
</tr>
<tr>
<td>Waist circum., cm</td>
<td>75 ± 2</td>
<td>89 ± 2*</td>
</tr>
<tr>
<td>WHR</td>
<td>0.75 ± 0.01</td>
<td>0.84 ± 0.01*</td>
</tr>
<tr>
<td>Na⁺ excretion, mmol/day</td>
<td>98 ± 12</td>
<td>120 ± 13*</td>
</tr>
<tr>
<td>Na⁺ intake, mg/day</td>
<td>2,937 ± 124</td>
<td>2,782 ± 146*</td>
</tr>
<tr>
<td>Running, km/week</td>
<td>34.3 ± 1.6</td>
<td>22.6 ± 0.9*</td>
</tr>
<tr>
<td>PAEE, kcal·kg⁻¹·day⁻¹</td>
<td>35 ± 1</td>
<td>35 ± 1</td>
</tr>
<tr>
<td>V̇O₂max, ml·kg⁻¹·min⁻¹</td>
<td>98 ± 12</td>
<td>120 ± 13*</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, number of subjects. BMI, body mass index; Waist circum., waist circumference; WHR, waist-to-hip ratio; Na⁺ excretion, urinary sodium excretion; Na⁺ intake, estimated dietary sodium intake; PAEE, physical activity-related energy expenditure; V̇O₂max, maximal oxygen consumption. *P < 0.05 vs. premenopausal women of same physical activity status.

Table 2. Casually determined levels of arterial blood pressure at rest across age in sedentary and exercising women

<table>
<thead>
<tr>
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<th>Sedentary</th>
<th>Endurance Trained</th>
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<tbody>
<tr>
<td></td>
<td>Premenopausal (n = 12)</td>
<td>Postmenopausal (n = 20)</td>
</tr>
<tr>
<td>Pressure, mmHg</td>
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<td></td>
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<tr>
<td>Systolic</td>
<td>99 ± 2</td>
<td>117 ± 4*</td>
</tr>
<tr>
<td>Diastolic</td>
<td>73 ± 2</td>
<td>76 ± 2</td>
</tr>
<tr>
<td>Pulse</td>
<td>26 ± 2</td>
<td>39 ± 2*</td>
</tr>
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</table>

Values are means ± SE; n = no. of women/group. *P < 0.05 vs. premenopausal women of same physical activity status.

Fig. 1. 24-h (A), daytime (B), and nighttime (C) systolic arterial blood pressure (SBP) in premenopausal and postmenopausal sedentary (S) and endurance exercise-trained (ET) women. NS, not significant. Data are means ± SE.
nighttime levels of SBP and PP were not different across age in the endurance-trained women. Age \times exercise status interactions for these comparisons ranged from $P < 0.12$–$0.13$. Table 3 shows ambulatory-recorded levels of DBP. There were no significant age-associated differences in 24-h, daytime, or nighttime DBP in either the sedentary or the endurance-trained women.

Variability. SBP and DBP variabilities are depicted in Table 4. In the sedentary group, daytime SBP and DBP variabilities were greater ($P < 0.05$), as was nighttime SBP variability, in the postmenopausal women. No age-related differences in daytime or nighttime BP variabilities were observed in the endurance-trained women. Age \times exercise status interactions for these variables ranged from $P = 0.03$–$0.19$. 24-h BP variabilities were not significantly different between age groups in either population.

Load. SBP loads are presented in Fig. 3. In the sedentary group, 24-h SBP load was approximately fourfold greater ($P < 0.05$) in the postmenopausal women than in the premenopausal controls; this was because of markedly higher ($P < 0.05$) daytime and nighttime levels in the postmenopausal women. In contrast, no age-associated differences were observed for 24-h, daytime, and nighttime SBP load in the endurance-trained women. The age \times exercise status 24-h SBP load interaction was $P = 0.11$. Table 5 shows group data for DBP load. In the sedentary group, there was a consistent, albeit nonstatistically significant, trend for greater 24-h, daytime, and nighttime DBP loads in the postmenopausal women. No such age-associated trends were observed, however, in the endurance-trained women.

Physiological Correlates of 24-h SBP and PP, SBP Variability, and SBP Load: General Subject Characteristics.

In the present study, main effects of age and habitual exercise status were confined largely to SBP and PP. In the pooled subject sample, significant (all $P < 0.05$) univariate correlations were observed between 24-h SBP and: waist-to-hip ratio ($r = 0.47$; Fig. 4), waist circumference ($r = 0.43$), maximal oxygen consumption

**Figure 2.** 24-h (A), daytime (B), and nighttime (C) arterial pulse pressure (PP) in premenopausal and postmenopausal sedentary and endurance exercise-trained women. Data are means ± SE.

**Table 3.** Ambulatory-recorded levels of diastolic arterial blood pressure across age in sedentary and exercising women

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<th>Endurance Trained</th>
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<tr>
<td></td>
<td>Premenopausal (n=12)</td>
<td>Postmenopausal (n=20)</td>
</tr>
<tr>
<td>24-h</td>
<td>74 ± 2</td>
<td>72 ± 2</td>
</tr>
<tr>
<td>Daytime</td>
<td>78 ± 2</td>
<td>75 ± 2</td>
</tr>
<tr>
<td>Nighttime</td>
<td>61 ± 1</td>
<td>63 ± 2</td>
</tr>
</tbody>
</table>

Values are means ± SE (in mmHg).

**Table 4.** Arterial blood pressure variability assessed by the standard deviation of individual 24-h ambulatory recordings across age in sedentary and exercising women

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<th>Sedentary</th>
<th>Endurance Trained</th>
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<tr>
<td></td>
<td>Premenopausal (n=12)</td>
<td>Postmenopausal (n=20)</td>
</tr>
<tr>
<td>24-h SD</td>
<td>11 ± 1</td>
<td>12 ± 1</td>
</tr>
<tr>
<td>SBP</td>
<td>10 ± 1</td>
<td>10 ± 1</td>
</tr>
<tr>
<td>DBP</td>
<td>8 ± 1</td>
<td>10 ± 1*</td>
</tr>
<tr>
<td>Daytime SD</td>
<td>7 ± 1</td>
<td>8 ± 1*</td>
</tr>
<tr>
<td>SBP</td>
<td>7 ± 1</td>
<td>9 ± 1*</td>
</tr>
<tr>
<td>DBP</td>
<td>7 ± 1</td>
<td>7 ± 1</td>
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</tbody>
</table>

Values are means ± SE (in mmHg); n = no. of women/group. SD, standard deviation; SBP, systolic blood pressure; DBP, diastolic blood pressure. *$P < 0.05$ vs. premenopausal women of same physical activity status.
Arterial Stiffness: Relation to 24-h SBP and PP, SBP Variability and SBP Load.

In the sedentary group, carotid AI was markedly higher in the postmenopausal women (20.36 ± 1.7 vs. 16.5 ± 1.8%, P < 0.05). In contrast, carotid AI was not significantly different with age in the endurance-trained women (2.9 ± 2.5 vs. 8.0 ± 2.2%, P > 0.10). In
the pooled population, carotid AI was directly related to 24-h SBP ($r = 0.43$, $P < 0.005$) and PP ($r = 0.53$, $P < 0.001$) (Fig. 5). Accounting for the influence of carotid AI via ANCOVA abolished the significant age-group differences in 24-h SBP and PP in the sedentary women ($P \geq 0.60$). Finally, carotid AI also was related to both 24-h SBP variability ($r = 0.34$, $P < 0.05$) and 24-h SBP load ($r = 0.41$, $P < 0.01$) in the pooled population.

DISCUSSION

The primary findings from the present study were as follows. First, 24-h SBP and PP were higher in healthy sedentary women with age, but not in endurance-trained women. Second, the higher 24-h SBP and PP in the postmenopausal sedentary women were caused by higher daytime and nighttime levels. Third, in general SBP variability and especially SBP load were higher with age in the sedentary, but not in the exercising women. Fourth, the elevations in 24-h SBP and PP, SBP variability and SBP load with age in the sedentary women, and the absence of such elevations in the endurance-trained women, were related to abdominal adiposity and arterial stiffness.

24-h BP: Mean Levels

Higher levels of 24-h SBP and PP with age in normotensive sedentary men and women have been reported previously (30, 34). To our knowledge, however, the present study is the first to report 24-h BP across age in physically active adults. Our findings indicate that, in contrast to their sedentary normotensive peers, 24-h SBP and PP may not increase with age in women who regularly perform vigorous endurance exercise. These results support and extend our earlier findings of lower 24-h SBP in habitually exercising compared with sedentary postmenopausal women (26). Ambulatory-determined 24-h, daytime, and nighttime DBP were not different across age in either the sedentary or the endurance-trained women in the present study. Collectively, our findings suggest that the main effect of primary (i.e., healthy) sedentary aging on 24-h BP is elevations in SBP and PP, and that habitual endurance exercise may prevent such increases.

In the present study, we found that casually determined resting SBP and PP were higher with age in both populations, although the age-related differences in the endurance-trained women were only ~40–55% as large as those in the sedentary women. We have reported this pattern of resting SBP across age previously (5). We can only speculate on the reason(s) for the modestly higher resting, but not 24-h, SBP (and, therefore, PP) in the postmenopausal endurance-trained women. Perhaps ambulatory-recorded 24-h levels of SBP are a more robust measure than casually determined levels in this population. In our experience, these physically conditioned women tend to be more active than their sedentary counterparts, independent of their endurance training per se. If so, the large number of measurements obtained with 24-h ambulatory recordings should provide a more precise SBP profile than casually determined values in such populations. Whatever the reason, it is important to note that dissociations between resting and 24-h SBP values have been commonly observed (12, 30), including in response to differing exercise states (23, 24).

SBP Variability and SBP Load

SBP variability and SBP load increase with age in sedentary women, although less so in strictly normotensive populations (34, 35). In the present study, daytime and nighttime SBP variability as well as 24-h SBP load were higher with age in the sedentary women. In contrast, there were no age-related differences in SBP variability or SBP load in our endurance-trained women. In fact, the SBP loads in the postmenopausal exercising women were as low as those observed in either the sedentary or the endurance-trained premenopausal controls (Fig. 3). Together, the present findings suggest that the reductions in precision of BP control with advancing age, as reflected by increases in SBP variability and load, may not occur in habitually exercising women.

Fig. 5. 24-h arterial SBP (A) and PP (B) as functions of carotid augmentation index (AI) in the pooled study population.
Physiological Correlates of 24-h SBP

In agreement with our previous observations (26, 27), in the present study we found that measures of adiposity were significant physiological correlates of 24-h SBP and PP. In particular, a well-accepted measure of total abdominal adiposity, waist-to-hip ratio (1, 4), was the sole significant independent correlate of 24-h SBP and PP in the pooled population. Moreover, 24-h SBP and PP were higher with age only in the population (sedentary women) who demonstrated corresponding elevations in waist-to-hip ratio; neither 24-h SBP, 24-h PP, nor waist-to-hip ratio were higher with age in the endurance-trained women. Accounting for the influence of waist-to-hip ratio abolished the significant age-related differences in 24-h SBP and PP in the sedentary women. Collectively, these observations support the idea that increases in abdominal adiposity with age contribute to elevations in 24-h SBP and PP in sedentary women, and that the lack of such increases in abdominal fat are related to the absence of elevated 24-h SBP and PP in endurance-trained women.

Our results suggest that differences in arterial stiffness with age also may play a role in 24-h SBP and PP. The stiffness of the central arteries is thought to contribute importantly to age-associated elevations in SBP and PP (6, 9, 28, 29). In the present study, carotid AI, a well-recognized index of the integrated stiffness of the large central and intermediate-sized arteries (9), was higher with age only in the sedentary women and correlated with 24-h SBP and PP. As with waist-to-hip ratio, accounting for the effects of carotid AI eliminated the significant age-group difference in 24-h SBP and PP in the sedentary women. Parenthetically, our observations related to arterial stiffness are consistent with the results of Vaitkevicius et al. (29) who reported lower levels of aortic pulse wave velocity and carotid AI in endurance exercise-conditioned men compared with sedentary middle-aged and older men. The present findings, considered together, support the view that the absence of any obvious elevations in 24-h SBP and PP in the endurance-trained postmenopausal women was related, at least in part, to their relatively favorable levels of arterial stiffness.

The lack of significant elevations in abdominal adiposity and arterial stiffness also may have been involved in the absence of age-associated increases in SBP variability and SBP load in the endurance-trained women. Significant positive relations were observed in each population (sedentary women) who demonstrated corresponding elevations in waist-to-hip ratio; neither 24-h SBP, 24-h PP, nor waist-to-hip ratio were higher with age in the endurance-trained women. Accounting for the influence of waist-to-hip ratio abolished the significant age-related differences in 24-h SBP and PP in the sedentary women.

Physiological Significance and Experimental Considerations

Because of the high variability in casual BP measurements, 24-h recordings provide a more reliable indicator of BP during typical daily activities (6, 11, 17, 30, 34). Moreover, mean levels of ambulatory-recorded SBP, DBP, and PP are more closely linked to BP-associated target organ damage and clinical prognosis than their respective casually determined levels at rest (6, 11, 14, 16, 32). Importantly, 24-h PP is a strong predictor of target organ effects and future cardiovascular risk independent of SBP and DBP (6, 11, 32). Ambulatory-determined BP variability and load can be even better predictors of target organ damage than mean 24-h BP levels (16), and also predict BP-related left ventricular hypertrophy and vascular structural changes (14, 19, 33). Thus these ambulatory-recorded measures of BP behavior are critical for assessing the influence of putative physiological factors or states that may modulate long-term BP regulation in humans. As such, the present findings provide new experimental support for the hypothesis that habitual and vigorous endurance exercise exerts an important influence on age-associated elevations in daily SBP and PP in healthy women.

There are at least three important experimental considerations regarding the present study. First, although cross-sectional study designs are commonly used to provide insight into the effects of aging on BP and its associated cardiovascular determinants (5, 9, 28–30, 34, 35), it is possible that genetic or other constitutional factors, independent of age or habitual exercise, influenced our results. It is possible, for example, that the absence of obvious elevations in 24-h SBP and PP with age in our endurance-trained women was not due to their habitual exercise per se, but to some other factor. Second, because we studied only healthy normotensive women it is likely that we underestimated, perhaps substantially, the true strength of the relation between the endurance-trained state and age-associated elevations in SBP and PP. In particular, inclusion of less healthy women in our sedentary population undoubtedly would have resulted in more marked age-related differences in BP compared with our endurance-trained women. In support of this concept, the relations between regular physical activity and resting BP were much stronger in a population of middle-aged and older women that included hypertensive subjects (18) than we found in our previous investigation on normotensive women of similar age (26). Finally, in most cases our age × exercise status interactions for the 24-h SBP- and PP-related dependent variables did not achieve statistical significance. In our experience, this is a common occurrence in laboratory-based physiological studies on humans because of a combination of intersubject variability and sample-size limitations. We believe, however, that despite this the directionality and magnitudes of the age-related
differences within our sedentary and endurance-trained populations for these variables are obvious. More importantly, the differences are both physiologically and clinically significant.

In conclusion, the results of the present study suggest that the elevations in 24-h SBP and PP observed with age in sedentary adult females may not occur in endurance exercise-trained women. Moreover, our findings indicate that age-associated elevations in 24-h SBP and PP in sedentary women and the absence of such changes in endurance-trained women are related to abdominal body fat and arterial stiffness. As such, our data provide the experimental basis for the hypothesis that prevention of increases in abdominal adiposity and arterial stiffness with age may attenuate or even prevent age-associated elevations in 24-h SBP and PP in healthy women.

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REFERENCES


