Effects of Age and Aerobic Capacity on Arterial Stiffness in Healthy Adults

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Background. It has been well established that arterial stiffness, manifest as an increase in arterial pulse wave velocity or late systolic dilation of the carotid artery pressure pulse, increases with age. However, the populations studied in prior investigations were not rigorously screened to exclude clinical hypertension, occult coronary disease, or diabetes. Furthermore, it is unknown whether exercise capacity or chronic physical endurance training affects the age-associated increase in arterial stiffness.

Methods and Results. Carotid arterial pressure pulse augmentation index (AGI), using applanation tonometry, and aortic pulse wave velocity (APWV) were measured in 146 male and female volunteers 21 to 96 years old from the Baltimore Longitudinal Study of Aging, who were rigorously screened to exclude clinical and occult cardiovascular disease. Aerobic capacity was determined in all individuals by measurement of maximal oxygen consumption (VO2max) during treadmill exercise. In this healthy, largely sedentary cohort, the arterial stiffness indexes AGI and APWV increased approximately fivefold and twofold, respectively, across the age span in both men and women, despite only a 14% increase in systolic blood pressure (SBP). These age-associated increases in AGI and APWV were of a similar magnitude to those in prior studies of less rigorously screened populations. Both AGI and APWV varied inversely with VO2max, and this relationship, at least for AGI, was independent of age. In endurance trained male athletes, 54 to 75 years old (VO2max = 44 ± 3 mL·kg⁻¹·min⁻¹), the arterial stiffness indexes were significantly reduced relative to their sedentary age peers (AGI, 36% lower; APWV, 26% lower) despite similar blood pressures.

Conclusions. Even in normotensive, rigorously screened volunteers in whom SBP increased an average of only 14% between ages 20 and 90 years, major age-associated increases of arterial stiffness occur. Higher physical conditioning status, indexed by VO2max, was associated with reduced arterial stiffness, both within this predominantly sedentary population and in endurance trained older men relative to their less active age peers. These findings suggest that interventions to improve aerobic capacity may mitigate the stiffening of the arterial tree that accompanies normative aging. (Circulation. 1995;88[part 1]:1456-1462.)

Key Words • aging • exercise

Alterations in arterial structure and function occur with advancing age in healthy individuals. These changes comprise an increase in arterial wall thickness secondary to hyperplasia of the intima, addition of medial lamellae, and loss of the orderly arrangement of elastin in the media. Additionally, there is degeneration and disorganization of the medial layers with partial replacement of elastin with less compliant forms of collagen.¹ These changes in the arterial wall with advancing age are accompanied by a progressive dilation of major arteries and a progressive increase in arterial stiffness. The age-associated increase in arterial stiffness manifests itself as a progressive increase in systolic blood pressure with advancing age, a widening of the arterial pulse pressure, and an increase in the pulse wave velocity.² The arterial stiffening with advancing age leads to changes in the arterial pulse wave contour. Older subjects demonstrate an increase in the late systolic pressure peak,³ which is thought to result from early reflected pressure waves secondary to an increase in pulse wave velocity.⁴⁻⁷

In prior studies that have examined the effect of aging on arterial stiffness, the populations examined were not rigorously screened to exclude hypertension, occult coronary disease, or diabetes mellitus.⁵⁻¹¹ Additionally, whether arterial changes with aging differ between men and women has not been addressed. Furthermore, it is unknown whether exercise capacity or physical conditioning status affects the age-associated increase in arterial stiffness.

In the present study, we used the noninvasive techniques of carotid applanation tonometry⁶ and measurements of pulse wave velocity to define age-associated changes in arterial stiffness in healthy men and women from the Baltimore Longitudinal Study of Aging (BLSA). These community dwelling volunteers were

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carefully screened to exclude clinical and occult cardiovascular disease, hypertension, and diabetes. We hypothesized that the aortic pulse wave velocity and the late systolic pressure wave augmentation would increase to a lesser extent in this healthy normotensive population than in prior studies of less rigorously screened subjects. Because maximal oxygen consumption ($V_{O2max}$) is determined in part by the ability of the arterial system to dilate and thereby increase flow to exercising muscle, we further examined whether $V_{O2max}$ is independently related to these arterial stiffness indexes. Additionally, we hypothesized that physical conditioning in older individuals might attenuate the age-associated increase in arterial stiffness. To test this hypothesis, we compared arterial stiffness measurements in endurance trained older athletes with those of age-matched sedentary subjects.

**Methods**

A healthy, sedentary study group comprising 146 individuals was recruited from the BLSA; 96 men 21 to 91 years old (mean, 55±17 years) and 50 women 26 to 96 years old (mean, 53±18 years). These community dwelling volunteers, largely from the Baltimore-Washington area, undergo 2.5 days of extensive medical, physiologic, and psychological testing biennially at the Gerontology Research Center in Baltimore. The population is predominately white, college educated, and of upper-middle-class socioeconomic status. To examine the effects of normative aging on vascular stiffness, we used the following screening criteria: a blood pressure of less than 160/95 mm Hg, absence of cardiovascular disease by history, physical examination, resting and maximal exercise ECG, and if 40 years old or older, by exercise thallium scintigraphy. All individuals were nonsmokers, and none were taking cardiovascular medication. None participated in aerobic exercise on a regular basis (ie, at least 20 minutes at least three times weekly).

To determine whether chronic endurance training is associated with an attenuation of the expected age-associated increase in arterial stiffness, 14 endurance trained men at least 54 years old were recruited, all of whom competed regularly in local distance running events. These men fulfilled the same health and screening criteria described for the sedentary BLSA volunteers and had a $V_{O2max}$ at least 1 SD above the mean value for their age-matched nontrained counterparts, measured during a modified Balke treadmill exercise test.

The study protocol was approved by the joint Gerontology Research Center—Francis Scott Key Medical Center institutional review board. Informed consent was obtained for all subjects.

**Arterial Pressure Pulse Contour**

Arterial pressure waveforms were obtained from the right common carotid artery by applanation tonometry, using a pencil-sized probe over the maximal pulsation of the artery. This probe contains a Millar micromanometer (model SPR-428, Millar Instruments Inc, Houston Tex), which has the same high-fidelity characteristics as Millar intravascular catheters. Applanation tonometry requires the flattening or applanation of the curved surface of a pressure containing structure. When a superficial artery (radial, common carotid, femoral) is

![Fig 1. Schematic of augmentation index (AGI) % derivation from the carotid artery pressure contour. Recordings were made transcutaneously by applanation tonometry using a Millar micromanometer. PP indicates pulse pressure.](image)
**Pulse Wave Velocity**

Simultaneous recordings of the arterial flow waves from the right common carotid artery, or ascending aorta, and right femoral artery were made using non-directional transcutaneous Doppler flow probes (model 810-A, 10 mHz, Parks Medical Electronics Inc, Aloha, Ore). Pulse wave velocity measurements were derived from these waveforms. Ascending aortic flow was measured by angulating the transducer in the suprasternal notch until a characteristic high-amplitude signal was obtained. If no reliable aortic arch signals could be obtained, the Doppler signal was recorded from the right common carotid artery.

The aortic pulse wave velocity (APWV) was determined from the foot-to-foot flow wave velocity. A minimum of 10 simultaneously recorded flow waves were recorded and averaged. The foot of the flow wave was identified visually as the point where systolic flow began. The time delay between the feet of simultaneously recorded flow waves was recorded. The distance between the sampling site over the aortic arch (mid point of the manubrium sterni) and the right femoral artery was measured over the surface of the body with a tape measure. When a flow wave could not be adequately measured in the aorta, the right common carotid artery flow wave was used instead; the distance between the midpoint of the manubrium sterni and the sampling site on the carotid was measured and subtracted from the manubrium-to-femoral artery distance. Measurements of APWV using the carotid artery were highly correlated with those using the aortic arch (r=.79, P<.01). If both measurements were obtained in a given subject, a mean of the two was used.

**Maximal Oxygen Consumption**

Maximal exercise testing was performed according to a modified Balke protocol on a motor-driven treadmill at a constant speed, usually 3.5 mph for men and 3.0 mph for women. The treadmill incline was increased by 3% every 2 minutes starting from the level, until the subject was exhausted. The ECG was monitored continuously in leads I, aVF, and V5. Brachial arterial cuff pressure and a 12-lead ECG were obtained during the final 30 seconds of each exercise stage and at 2, 4, and 6 minutes into recovery. The subjects’ expired gases were measured using a Rayfield Equipment Corporation (model REP-9200) gas meter. O2 and CO2 concentrations in expired air were analyzed using a mass spectrometer, which was calibrated daily using standard gases. Ventilation, O2 uptake, CO2 production, and respiratory exchange ratio were measured continuously during each test by a computerized data acquisition system, which interfaced the gas meter and mass spectrometer. Values were calculated every 30 seconds and recorded for analysis. The highest 30-second value for O2 uptake defined the VO2max. Individuals who did not achieve at least 85% of their age-predicted maximal heart rate and those who demonstrated at least 1-mm horizontal or downsloping ST segment responses (Minnesota Code 4:1) were excluded from all analyses.

**Statistical Analysis**

In the 146 sedentary BLSA participants, simple linear regression was used to determine the effects of age and  

\[\text{VO}_{2\text{max}} = a + bx\]

\[\text{APWV} = a + bx\]

\[\text{AGI} = a + bx\]

where \(a\) and \(b\) are regression coefficients, and \(x\) is age. The effect of gender on these regressions was assessed using analysis of covariance. The independent effects of \(\text{VO}_{2\text{max}}, \text{age}, \text{and gender on systolic blood pressure, PWV, and AGI as well as interactions among these were assessed in a multiple regression model (SAS Institute, Inc, Cary, NC). The effect of chronic endurance training on arterial stiffness indices was assessed by comparing a group of senior athletes with age-matched sedentary individuals and younger sedentary individuals, using analysis of variance with the Bonferroni correction for multiple comparisons. Data are expressed in these groups as the mean±SEM. P<.05 was considered statistically significant.**

**Results**

Fig 2 shows that in this carefully screened healthy population, there was only a 14% increase in systolic
blood pressure (Fig 2A) within the clinically normal range between the ages of 20 and 91 years. Over this age, range pulse pressure (ie, systolic blood pressure minus diastolic blood pressure) increased 36.8% in men \((r=.52, P<.001)\) and 47.3% in women \((r=.39, P<.005)\). By comparison, the APWV increased twofold (Fig 2B) and AGI increased fivefold (Fig 2C) over this age range. Analysis of covariance revealed no gender differences in the slopes of the age regressions of systolic blood pressure, pulse pressure, APWV, or AGI. While the systolic blood pressure was higher in men than in women across the age range \((P=.005)\), the AGI was significantly lower in men \((P=.009)\).

\[ V_{O_{2\text{max}}} \] demonstrated a progressive decline with increasing age. The age-associated decline in \( V_{O_{2\text{max}}} \) was steeper in women \((y=49.6−0.40 \text{ age})\) than men \((y=47.4−0.25 \text{ age}, \text{covariance } P=.04)\). Each of the arterial stiffness indexes was inversely related to \( V_{O_{2\text{max}}} \) (Fig 3). Analysis of covariance demonstrated that at any given \( V_{O_{2\text{max}}} \) value, both systolic blood pressure and APWV are higher in men than in women \((P=.006 \text{ and } P=.03\text{, respectively})\). In the overall sample, both APWV and AGI correlated only weakly with systolic blood pressure \((r=.45 \text{ and } r=.33\text{, respectively})\).

The inverse relationship of systolic blood pressure, APWV and AGI with \( V_{O_{2\text{max}}} \) may be confounded by the fact that the former three variables increase with age, whereas the latter decreases with age. We therefore used a multiple regression model to determine the independent effects of age, \( V_{O_{2\text{max}}} \), gender, and relative weight (body mass index) on these vascular stiffness variables, as well as the interactions among the variables (Table 1). This analysis indicated a marked effect of \( V_{O_{2\text{max}}} \) on AGI over and above the effect of age. Furthermore, the effect of age on AGI persisted after adjustment for \( V_{O_{2\text{max}}} \), gender, and body mass index. It is noteworthy that while the AGI was independently related to \( V_{O_{2\text{max}}} \) in the multiple regression model, systolic blood pressure was not; APWV demonstrated a borderline relationship. There was no independent effect of gender or body mass index on AGI over and above those of age and \( V_{O_{2\text{max}}} \). Finally, both APWV and AGI correlated strongly with age \((P<.0001)\), even after adjusting for systolic blood pressure, providing additional evidence that these stiffness were not merely surrogates of systolic blood pressure.

In the multivariate analysis of APWV, we observed a \( V_{O_{2\text{max}}}-\text{age} \) interaction (Table 1). We therefore divided the population into tertiles by age \((20 \text{ to } 44 \text{ years}, 45 \text{ to } 70 \text{ years, and more than } 70 \text{ years})\) and examined the relationship of the variables within each age group. The tertiles were not analyzed by gender because multiple regression analysis did not indicate a significant gender difference in the age-associated increase in APWV. It was found that the \( V_{O_{2\text{max}}}-\text{age} \) interaction was due to a difference between the older and the two younger age groups. In the older cohort, there was a significant decrease in APWV with increasing \( V_{O_{2\text{max}}} \) \((r=.52, P<.02)\), whereas in the young and middle-aged groups, APWV and \( V_{O_{2\text{max}}} \) were not related \((r=.03 \text{ and } .17\text{, respectively})\).

The effect of physical conditioning on the age-associated increase in arterial stiffness was examined by comparing the arterial stiffness indexes in senior athletes with those of age-matched and young sedentary BLSA male controls (Fig 4). The senior athletes weighed less than the old and young controls and had a lower body mass index than the two control groups (Table 2). As anticipated by the study design, the \( V_{O_{2\text{max}}} \) of the senior athletes greatly exceeded that of the old controls and was similar to that of the young controls. The systolic blood pressure did not differ between old controls and senior athletes; it was significantly higher in old controls than in young controls (Fig 4A). Pulse pressure followed a pattern similar to systolic blood pressure; values were 55.2±2.1, 55.8±2.7, and 45.7±2.3 mm Hg in the three groups, respectively \((P<.05\text{ for young controls versus both older groups})\). APWV (Fig 4B) was 25.9% less in the senior athletes than in their sedentary age peers; values for the young controls and the athletes were similar (Fig 4B). Finally, the AGI was

![Graph showing scatterplots of systolic blood pressure (SBP), aortic pulse wave velocity (APWV), and augmentation index (AGI) as a function of maximal oxygen consumption (VO2max).](http://circ.ahajournals.org/)

**FIG 3.** Scatterplots of systolic blood pressure (SBP), aortic pulse wave velocity (APWV), and augmentation index (AGI) as a function of maximal oxygen consumption (VO2max). A, SBP: men: \(y=140.2−0.55 \text{ VO2max}, r=.30, P<.01; \) women: \(y=126.6−0.41 \text{ VO2max}, r=.22, P=\text{NS}\). B, APWV: men: \(y=1234−13.8 \text{ VO2max}, r=.34, P<.01; \) women: \(y=1144−14.3 \text{ VO2max}, r=.49, P<.01\). C, AGI: men: \(y=34.6−0.64 \text{ VO2max}, r=.54, P<.0001; \) women: \(y=37.7−0.76 \text{ VO2max}, r=.74, P<.0001\).
TABLE 1. Multiple Regression Determinants of Vascular Stiffness Indices in Healthy Sedentary Men and Women

<table>
<thead>
<tr>
<th></th>
<th>Systolic Blood Pressure, mm Hg</th>
<th></th>
<th>Pulse Wave Velocity, cm/s</th>
<th></th>
<th>Augmentation Index, %</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>β</td>
<td>P</td>
<td>β</td>
<td>P</td>
<td>β</td>
<td>P</td>
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<tr>
<td>Age</td>
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<td>.041</td>
<td>20.0</td>
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<td>.22</td>
<td>.0001</td>
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<tr>
<td>VO₂max</td>
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<td>NS</td>
<td>15.8</td>
<td>.082</td>
<td>–.35</td>
<td>.0014</td>
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<tr>
<td>Gender*</td>
<td>–5.56</td>
<td>.049</td>
<td>–22.9</td>
<td>NS</td>
<td>1.96</td>
<td>NS</td>
</tr>
<tr>
<td>Body mass index</td>
<td>1.22</td>
<td>.0002</td>
<td>4.93</td>
<td>NS</td>
<td>.11</td>
<td>NS</td>
</tr>
<tr>
<td>Age* VO₂max</td>
<td>–.006</td>
<td>NS</td>
<td>–.34</td>
<td>.030</td>
<td>–.001</td>
<td>NS</td>
</tr>
<tr>
<td>Age* body mass index</td>
<td>.024</td>
<td>NS</td>
<td>–.11</td>
<td>NS</td>
<td>.013</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Male=0, Female=1 in model.

significantly (35.5%) lower in the senior athletes than in old controls but was higher than in the young controls (Fig 4C).

Discussion

Age-associated stiffening of the arterial tree has been recognized for nearly a century from both clinical and pathologic studies. Although such stiffening was originally regarded as a benign concomitant of the aging process, recent epidemiological studies have shown elevated arterial pressure1–7 to be a potent predictor of cardiovascular morbidity and mortality, even in older populations. At least some of this increased cardiovascular risk derives from the adverse effect of increased arterial pressure (and presumably stiffness) on the left ventricle.8–11 In the Framingham study, every 20 mm Hg increase in systolic blood pressure was associated with a 10.6-g increase in left ventricular mass in male subjects 60 to 90 years old.12 Left ventricular hypertrophy, in turn, was a powerful independent risk factor for subsequent cardiac events.13 Even in normotensive populations, an age-associated increase in left ventricular mass has been demonstrated,14,15 which may be due, at least in part, to the increase in arterial stiffness with age.

In the present study, two vascular stiffness indexes, APWV and AGI, that have been previously described and validated by invasive methods16–18 were used to assess noninvasively the age-associated change in arterial stiffness in highly screened sedentary volunteers. There was a progressive increase in APWV with advancing age, with a 2.5-fold increase in APWV over an age range from 20 to 91 years. Avolio et al19 demonstrated a similar percentage increase in the APWV across age in urban and rural Chinese populations. The absolute level of APWV at any given age was similar in their rural cohort to our values. However, APWV values in their urban population, who consumed a much larger sodium intake and had a greater prevalence of hypertension than their rural counterparts, were considerably higher. In another population advised to inject low quantities of sodium for a 2-year period, the expected age-associated increases in aortic, arm, and leg APWV values did not occur.20 Thus, several factors in addition to age per se probably exert significant influence on arterial stiffness, including ethnic and genetic differences, dietary and activity habits, as well as the prevalence of elevated blood pressure and other vascular diseases.

Arterial pressure waveforms were obtained by applanation tonometry and demonstrated an approximate
fivefold increase in the augmentation of the late systolic pressure peak. In a study of 1005 subjects, Kelly et al. demonstrated a 15-fold increase in the AGI of the carotid arterial pressure waveform from the first to the eighth decade. In their study, there was approximately a fivefold increase in the AGI in the age range from 21 to 70 years, similar to that observed in the present study between these ages. Our study thus extends the finding of an age-associated stiffening of the arterial tree to a population intensively screened for the absence of hypertension or occult coronary artery disease, strongly suggesting that such arterial stiffening accompanies aging in Western society even in the absence of demonstrable cardiovascular disease. Additionally, there were no differences in the indexes of arterial rigidity between the sexes in our population, an issue not addressed by these earlier studies.

Even in the present study of normotensive volunteers, systolic blood pressure rises with age as commonly observed in the general population within the clinically normal age range. It is noteworthy that the age-associated increase in arterial stiffness, measured by either AGI or APWV, greatly exceeds that for systolic blood pressure or pulse pressure. In our study population, systolic blood pressure rose only 14% between ages 20 and 90 years, whereas AGI and APWV increased approximately fivefold and twofold, respectively. One possible reason for this finding is the fact that the pressures in peripheral arteries underestimate the age-associated augmentation in central systolic blood pressure because they do not incorporate the characteristic exaggeration of the late systolic peak observed in the central arteries with advancing age.

A novel finding of the present study is that all three indexes of arterial stiffness are inversely related to \( VO_{2\text{max}} \) and that this inverse relationship with \( VO_{2\text{max}} \) at least for APWV, was strongest in individuals more than 70 years old. Thus, in this sedentary population, differences in exercise capacity among individuals may relate to differences in arterial rigidity. In this regard, arterial stiffness could be a determinant of differences in aerobic capacity among sedentary individuals, sometimes attributed to "genetic" factors. Alternatively, differences in physical activity habits, diet, or other unidentified factors associated with activities of daily living, and not ascertained by our screening techniques, may account for heterogeneity of arterial stiffness among untrained individuals comprising the population.

To examine the effect of intensive physical conditioning on arterial stiffness indexes in older individuals, we compared sedentary older and younger men with endurance trained senior athletes. The senior athletes had AGI and APWV measurements that were 36% and 26% lower, respectively, than their sedentary age peers, despite a similar systolic blood pressure and pulse pressure. Thus, systolic blood pressure and pulse pressure again proved insensitive to differences in arterial stiffness between sedentary and endurance trained older men compared with measurements of AGI and APWV, just as observed across age in our sedentary volunteers. Since the age-associated increase in arterial stiffness contributes substantially to the work load of the left ventricle, the reduced vascular stiffness noted in the senior athletes demonstrates a potential for endurance training to improve the suboptimal ventricular-vascular coupling relationship that occurs with advancing age. In support of this hypothesis, a recent study has demonstrated an augmentation of exercise ejection fraction response in older men after 12 months of intensive aerobic training. Although physically conditioned older individuals demonstrated lower indexes of arterial stiffness than their sedentary age peers in our study, our results do not permit differentiation of cause and effect. Arterial stiffness and exercise capacity may in part be genetically predetermined. Thus, the more compliant vessels of endurance trained senior athletes may be a factor that allows them to exercise so extensively and successfully. Longitudinal studies using training or detraining interventions are needed to clarify the causality of these relationships.

**Conclusions**

These results indicate that arterial stiffness, whether measured by AGI or APWV, demonstrates a progressive increase with advancing age in healthy normotensive adults. The magnitude of the age-associated increase in stiffness was similar in men and women and resembled values previously reported in less intensively screened samples. Arterial stiffness in these sedentary individuals was inversely related to exercise capacity, as indexed by \( VO_{2\text{max}} \). Endurance trained older men demonstrated lower APWV and AGI values than their sedentary age peers, despite similar systolic blood pressure. These findings suggest that the age-associated augmentation of arterial stiffness may be mitigated by regular aerobic exercise.

### TABLE 2. Anthropometric and Aerobic Capacity Measurements in Sedentary Younger and Older Men and in Older Endurance Trained Athletes

<table>
<thead>
<tr>
<th></th>
<th>Senior Athletes</th>
<th>Old Controls</th>
<th>Young Controls</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>14</td>
<td>38</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>69±2.5</td>
<td>67±1.3</td>
<td>29±1.0</td>
<td></td>
</tr>
<tr>
<td>Maximal oxygen consumption, mL · kg⁻¹ · min⁻¹</td>
<td>45±1.1</td>
<td>30±1.0*</td>
<td>42±1.7</td>
<td>.001</td>
</tr>
<tr>
<td>Height, cm</td>
<td>175±1.5</td>
<td>177±1.0</td>
<td>179±1.3</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>68±1.9*</td>
<td>79±1.8</td>
<td>81±2.9</td>
<td>.003</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>22±0.6*</td>
<td>25±0.4</td>
<td>25±0.9</td>
<td>.007</td>
</tr>
</tbody>
</table>

*Values are mean ± SEM.*

*Differs from other two groups by ANOVA at P<.05.*
Acknowledgments
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