Suppressed Anger Is Associated With Increased Carotid Arterial Stiffness in Older Adults

David E. Anderson, E. Jeffrey Metter, Hidetaka Hougaku, and Samer S. Najjar

Background: Anger and hostility have been implicated in the pathogenesis of heart disease, but the extent to which the large conduit arteries play an intermediate role in this relationship remains to be clarified. The present study investigated associations of anger frequency and expression style with carotid artery intima–media thickness (IMT) and stiffness in healthy adults older than 50 years.

Methods: Two hundred participants (95 men) in the Baltimore Longitudinal Study of Aging completed the Spielberger Anger Expression Inventory, which assesses anger frequency (trait anger), anger expression (anger-out), and anger suppression (anger-in). The carotid artery IMT was assessed by ultrasonography. Carotid stiffness was determined from the log of systolic over diastolic blood pressure (BP) as a function of carotid distensibility.

Results: In univariate correlational analysis, a significant positive association of anger-in with stiffness was observed \((P < .01)\), together with a less significant association of anger-in with carotid artery IMT \((P < .05)\). Neither anger-out nor trait anger was significantly associated with carotid artery IMT or stiffness. Moreover, none of the anger measures was significantly associated with resting BP in this normotensive sample. As expected, carotid artery IMT, stiffness, and systolic BP were all positively associated. In multivariate analysis, anger-in remained a determinant of stiffness independent of BP, and a marginally significant determinant of carotid artery IMT.

Conclusions: This is the first known finding that high anger-in is a significant independent determinant of carotid artery stiffness. These results suggest that high anger-in can potentiate the effects of age on stiffening of the central arteries. Am J Hypertens 2006;19:1129–1134 © 2006 American Journal of Hypertension, Ltd.

Key Words: Age, anger, arterial stiffness, blood pressure, carotid artery.

The available evidence supports the view that anger and hostility are determinants of all-cause mortality and coronary heart disease.1–3 The mechanism by which they contribute to adverse cardiovascular outcomes remains to be clarified. Evocation of anger experience in laboratory settings can elicit hemodynamic changes that elevate blood pressure (BP) acutely.4–7 The duration of the response depends on whether the anger is expressed or suppressed. Whether anger and hostility participate in the age-associated increases in conduit artery structure and function remains to be clarified. To date, three epidemiologic studies have used the Spielberger Anger Expression Inventory (SAEI)8 to investigate the role of trait anger and anger expression (anger-out or anger-in) in carotid artery intima–media thickness (IMT). One study found that high trait anger and high anger-in predicted increases in carotid artery IMT of women during a 10-year period.9 Another found that high trait anger predicted an increase in carotid artery IMT of healthy women during a 3-year period, as did the risk for developing the metabolic syndrome.10 A more recent study found that an antagonistic disposition and a propensity to express anger outwardly were associated with increased carotid atherosclerosis in men with untreated hypertension.11

Whether trait anger or anger expression style plays a role in arterial stiffness has not been investigated to date. The present study examined associations between scores on the SAEI and both carotid artery IMT and carotid artery stiffness in a sample of healthy older adults in the Baltimore Longitudinal Study of Aging (BLSA). It was hypothesized that high scores on the SAEI would be associated with increased carotid stiffness.

Methods

Study Population

Two hundred men and women in the BLSA volunteered for participation in this study, including 75 white and 20
African-American men, 77 white and 28 African-American women. The BLSA is composed of community-dwelling, mostly college-educated volunteers who are studied approximately every 2 years for 2.5 days of extensive medical, physiologic, and psychologic examinations. In addition to the ongoing systematic effort to characterize aspects of normal aging, the BLSA provides investigators at the Gerontology Research Center of the National Institute on Aging with samples of healthy subjects to test specific hypotheses concerned with aging. We collected data opportunistically for more than 1 year on a subset of the BLSA population of approximately 1000 persons. Exclusion criteria included a history of treatment for hypertension, coronary artery disease, stroke, diabetes, or cigarette smoking within the past year. Only persons aged 50 years and older were included in the present analysis after preliminary examination of a larger cohort indicated that the results were specific to older subjects. The protocol was approved by the Institutional Review Board of the National Institute on Aging, and informed consent was obtained from each participant in this study.

Table 1 presents means and standard deviations of age, anger, and cardiovascular measures for the 200 subjects in this study.

### Anger Expression Questionnaire

The SAEI is a 34-item questionnaire that assesses the frequency of anger experience (trait-anger), as well as the extent to which the respondent expresses anger (anger-out) and suppresses anger (anger-in), using a four point scale (1 = never; 2 = sometimes; 3 = often, and 4 = almost always). Responses to the items are summed to yield a score for each dimension. Previous studies have shown that reliabilities of the anger expression scores ranged from 0.70 to 0.89.12

### Carotid Arterial Stiffness

**Index and Intima–Media Thickness**

Measurement of carotid stiffness was performed after the subjects lay in a supine position in a dark and quiet room for 10 min of rest. Bilateral common carotid arteries (CCA) were examined by high-resolution B-mode ultrasonography with a linear array, 5- to 10-MHz duplex-type scanner (Ultramark 9 HDI, Advanced Technology Laboratories, Inc., Cherry Hill, NJ). The ultrasound unit has a cineloop image review system that allows for storage of sequential images into the system memory. The transducer was manipulated so that the near and far walls of the artery were parallel to the transducer footprint and the lumen was maximized in the longitudinal plane. Minimal pressure was applied on the skin by the transducer, and acoustic coupling was achieved with gel. A region approximately 1.5 cm proximal to the bulge of the carotid bifurcation was identified, and the B-mode image was recorded for several seconds.

By immediately reviewing the images stored in the memory, maximal systolic and minimal diastolic diameters were identified with the help of electrocardiography. Using electronic calipers, the CCA diameters were measured between the blood–intima boundaries (ie, both endothelial layers), perpendicular to the course of the vessel. This procedure was repeated on three different cardiac cycles for the left and right CCAs, and the six measurements were averaged for systolic and diastolic diameters. Diameter measurements were made by a trained sonographer, who was unaware of the anger scale scores or hypotheses under study.

Stiffness of the CCA was calculated as \( \ln(\text{Systolic BP/Diastolic BP})/(\Delta d/D) \), in which systolic BP was divided by diastolic BP, and the change in carotid diameter between systole and diastole (\( \Delta d \)) was divided by the diastolic diameter (\( D \)) of the mean of left and right carotid arteries.13 Intrarater correlation between repeated stiffness measurements from 10 subjects was 0.96 (\( P < .01 \)), with similar averages for the two sets of readings (6.37 ± 2.59 vs 6.43 ± 2.58, \( P = \) not significant [NS]), and met Bland-Altman criteria for reproducibility.14

The carotid artery IMT of the CCA far wall was defined as the distance between the luminal–intimal surface and the medial–adventitial interface. The carotid artery IMT was observed on the frozen frame of a suitable longitudinal image with the image magnified to achieve resolution of detail. The carotid artery IMT measurement was obtained from five contiguous sites at 1-mm intervals from both the right and left CCA, and the average of the 10 measurements calculated.

### Blood Pressure

An appropriately sized inflatable cuff for measurement of systolic and diastolic BP was placed around the nondominant arm and connected to an automated oscillometric system (Critikon, 1846SXP/P, version 085, Dinamap, Tampa, FL). Blood pressure was recorded after 15 min of rest in the supine posture (that was required for carotid measurement), and within 5 min before the carotid measurement.
Data Analysis

Linear correlations coefficients were calculated to assess the significance of the associations among the anger and cardiovascular variables. The independence of the associations of anger and cardiovascular variables with carotid artery IMT and carotid stiffness were evaluated by multiple regression analyses, using successive backward elimination of nonsignificant variables. The initial models included age, gender, ethnicity, and all anger and cardiovascular measures. The significance of mean differences in carotid artery IMT and carotid stiffness between groups above and at or below the medians for trait anger, anger-in, and anger-out was determined by two-tailed *t* test. All analyses were performed using SPSS software (version 11, Chicago, IL).

Results

Univariate Linear Correlations of Anger and Cardiovascular Measures

Table 2 shows that trait anger was positively correlated with both anger-in and anger-out, and that neither trait anger nor anger-out was significantly correlated with any cardiovascular measure.

Table 2 also shows, however, that anger-in was significantly correlated with carotid stiffness (*P* < .01) and, to a lesser extent, carotid artery IMT (*P* < .05), although not with systolic or diastolic BP. As expected, carotid artery IMT was positively associated with systolic BP and stiffness, whereas stiffness was also positively associated with heart rate. Fig. 1 illustrates this graphically, showing means and standard deviations of carotid artery IMT and stiffness for groups above and at or below the median for anger-in. High anger-in subjects showed a less significant difference in carotid artery IMT (*t* = 2.38; *P* < .05), and a more significantly increased carotid stiffness (*t* = 3.23; *P* < .002).

Independent Determinants of Carotid Artery Intima–Media Thickness and Carotid Stiffness

Table 3 shows the results of multiple regression analyses of age, anger-in, and all cardiovascular measures on carotid artery IMT and stiffness. Age was a significant independent determinant of carotid artery IMT (*P* < .01), and diastolic BP and anger-in were less significant independent determinants (*P* < .05) accounting for 20% of the variance. Anger-in, as well as systolic BP, diastolic BP, and heart rate, were all significant independent determinants of stiffness (*P* < .01), accounting for 31% of the variance.

Discussion

The main finding of this study was that anger-in was a significant independent determinant of carotid stiffness.
in a sample of healthy older participants in the BLSA. Anger-in was found to be a less significant independent determinant of carotid artery IMT. Neither trait anger nor anger-out was significantly correlated with either carotid artery IMT or stiffness in this cohort.

In younger persons, carotid artery IMT and carotid stiffness tend to be largely independent. Both tend to increase with age, and large individual differences in the extent to which arterial stiffness increases with age have been observed in longitudinal studies. In the present cross-sectional study, relatively large individual differences in carotid stiffness and carotid artery IMT were observed in a population of older adults with normal BP, in which only a small (although statistically significant) proportion of the variance of stiffness and carotid artery IMT was shared. The conditions under which carotid artery IMT and stiffness both increase with age in the same individuals remain to be clarified. Previous research has shown that decreased respiratory fitness during an 18-year interval was associated with increased arterial stiffness, but not carotid artery IMT. Moreover, dietary cholesterol has been found to increase carotid artery IMT, but not stiffness. The finding in the present study that anger-in is associated with increased carotid stiffness, but only marginally with carotid artery IMT, raises the possibility that the association of anger-in with carotid artery IMT in previous studies might be mediated by a common association of both with increased stiffness.

Arterial stiffness in nonatherosclerotic persons is thought to be due largely to the fatigue effects of cyclic stress on the elastic fibers of the vessel wall, resulting in progressive dilatation, and changes in the proportions of collagen and elastin. Previous research showing that suppressed anger was associated with increased lymphocyte β-adrenergic receptor sensitivity and systolic BP response to a standardized mathematics stressor is consistent with the view that anger may impact vascular structure and function by sympathetic nervous system activity. However, the results of the present study show that the effects of anger-in on carotid stiffness are independent of resting BP and heart rate, suggesting that mechanisms other than sympathetic nervous system activation could also be involved in the amplification of the age-associated increase in carotid arterial stiffness in older adults.

A distinction should be made between the acute expression of anger and an underlying psychophysiologic state that might heighten reactivity to anger-provoking events. For example, one experimental study found that the magnitude of the pressor response to anger evocation was greater in subjects rated high in hostility. Previous studies also found that recovery of basal cardiovascular function was more rapid in those who expressed anger than in those who suppressed it, although this response depended on cultural influences. Recently, it has been reported that a tendency to suppress anger interacted with BP level to predict cardiovascular mortality 17 years later.

The finding that none of the anger measures was associated with resting BP in this study is consistent with reviews of the literature on anger and hypertension. Previous reviews have found only low and inconsistent associations between trait anger and hypertension, and a more recent review concluded that an absence of expected anger is characteristic of hypertensives, implicating repression rather than suppression of anger. Subconscious anger would not manifest itself on the SAEI. Evidence for such a psychophysiologic pathway was provided in a study finding that high resting end-tidal CO₂ was positively associated with resting BP in healthy older women, but only in those with low trait anger.

Trait anger tends to decrease with age, whereas direction of anger expression tends to remain stable. A previous study showing that anger expression style of children tends to resemble that of their parents suggested that anger expression style might be learned early in life. Although the present correlational study does not permit a causal inference regarding anger expression style and carotid stiffness, the findings are consistent with the view that

| Table 3. Regression of age, gender, ethnicity, trait anger, anger-in, anger-out, systolic and diastolic blood pressure, and heart rate on carotid–intima media thickness and carotid stiffness in 200 men and women in the Baltimore Longitudinal Study on Aging |
|---------------------|-------|-----|-----|
| Carotid–intima media thickness |       |     |     |
| Age                  | 0.403 | 5.89| .000|
| Anger-in             | 0.138 | 2.02| .045|
| Diastolic blood pressure | 0.175 | 2.56| .011|
| F<sub>3,175</sub> = 14.69; P < .000; r² = 0.20 |
| Carotid stiffness    |       |     |     |
| Age                  | 0.291 | -2.64| .001|
| Anger-in             | 0.220 | 3.55| .004|
| Systolic blood pressure | 0.344 | 2.96| .002|
| Diastolic blood pressure | -0.303 | -2.90| .004|
| Heart rate           | 0.251 | 3.35| .001|
| F<sub>5,126</sub> = 11.45; P < .000; r² = 0.31 |

AJH–November 2006–VOL. 19, NO. 11
tonic biological effects of suppressed anger could participate in the increases in stiffness with age.

Limitations of the present study include the sample size, the cross-sectional design, and the use of self-report measures to assess anger expression style. The sample size was not sufficient, for example, to permit analysis of gender differences, which might be of interest, considering previous studies of anger suppression.6,7 The correlation of self-report to anger expression style needs further documentation, although the available evidence suggests that self-reports of anger expression style are predictive of behavior in naturalistic situations.30 In this regard, it is worth noting that two of the statements on the SAEI were themselves independently associated with increased arterial stiffness (“I boil inside, but don’t show it,” and “I’m irritated a great deal more than people are aware of”). Thus, anger-in, in which feelings are consciously experienced but suppressed, should be differentiated from anger repression or alexithymia, for which a stronger case can be made in the pathogenesis of primary hypertension.31

Another limitation of the present study was the measurement of BP in the brachial, rather than carotid, artery. Because of pressure amplification across the arterial tree, brachial pressures can overestimate carotid arterial pressures, but the overestimation decreases with advancing age.32 Finally, additional research with other measures of arterial stiffness, such as pulse wave velocity and arterial tonometry, are needed to assess the generality of the relationship of anger expression style to arterial stiffness in various regions.

In conclusion, the present study found that the tendency to report anger suppression as a characteristic behavioral style was associated with amplified arterial stiffness in older persons, independent of the effects of BP and heart rate. Because arterial stiffness is a risk factor for cardiovascular mortality,33 it would be worthwhile to explore the extent to which behavioral interventions that influence anger management can prevent or retard the age-associated progression of central arterial stiffness and its role in cardiovascular morbidity and mortality.

Acknowledgments
We thank Veena Shetty for assistance with the analysis of the data, as well as the participants and staff of the Baltimore Longitudinal Study of Aging.

References


