

Anger Expression and Incident Hypertension

SUSAN A. EVERSON, PHD, MPH, DEBBIE E. GOLDBERG, MS, GEORGE A. KAPLAN, PHD, JUHANI JULKUNEN, PHD, AND JUKKA T. SALONEN, MD, PHD, MSCPh

Objective: It has long been thought that anger is important in the development of essential hypertension. However, tests of this hypothesis have yielded conflicting findings. This study prospectively examined the relationship between anger expression style and incident hypertension in a population sample of middle-aged men. **Methods:** Participants were 537 initially normotensive men from eastern Finland, who completed a medical examination and series of psychological questionnaires at baseline and at 4-year follow-up. Anger expression was assessed by Spielberger's Anger-out and Anger-in scales. **Results:** At follow-up, 104 men (19.4%) were hypertensive (blood pressure ≥ 165 mm Hg systolic and/or 95 mm Hg diastolic). Age-adjusted logistic regression analyses revealed that each 1-point increase in Anger-out was associated with a 12% increase in risk of hypertension after 4 years of follow-up ($p < .002$), which corresponded to a two-fold increased risk of hypertension among men with scores in the top tertile of the Anger-out scale, relative to those with scores in the bottom tertile (odds ratio = 2.00, 95% confidence interval 1.20–3.38). Each 1-point increase on the Anger-in scale also was related to a 12% increased risk of hypertension ($p < .01$). Adjustments for body mass index, smoking, alcohol consumption, physical activity, a positive parental history of hypertension, and baseline resting diastolic blood pressure had little impact on the findings. **Conclusions:** These data provide strong epidemiological evidence for a positive relationship between anger expression style and subsequent hypertension, independent of known risk factors. Findings support the hypothesis that extreme expression of anger in either direction has adverse cardiovascular consequences. **Key words:** anger expression, hypertension, risk factors, cardiovascular disease.

BP = blood pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; KIHD = Kuopio Ischemic Heart Disease Risk Factor Study.

INTRODUCTION

Anger and hostility have long been considered important psychological factors in the development of essential hypertension (1–3). Indeed, Alexander's (1) classic hypothesis, which postulated that chronic inhibition of anger leads to sustained elevations in BP, continues to motivate research in this area nearly six decades after it was proposed (4, 5). Although research has identified distinct patterns of cardiovascular activation associated with anger, including increased BP and high peripheral resistance (6, 7), the role of anger in the development and progression of hypertension is still unclear (see Refs. 3 and 8 for reviews).

Several studies have examined the influence of *suppressed* hostility or anger ("anger-in") on BP and found that anger-in was positively related to resting BP and/or prevalent hypertension (9–13), particularly under conditions of stress (14–16). Other studies, including early psychodynamic research, have found that hypertensives, including those with borderline hypertension, reported greater intensity of anger and more repressed hostile wishes or anger-in than normotensives (3, 17, 18). However, data from the Framingham study and others do not support this association (19, 20) and some research has found that *expressed* anger and high levels of trait anger are related to higher BP levels (4, 21). Moreover, individuals with high levels of expressive hostility or potential for hostility (behavioral measures of hostility associated with both physical and verbal expressions of anger) have shown exaggerated

BP responses under conditions of stress or harassment (22, 23) and anger expression has been associated with increased risk of fatal and nonfatal coronary heart disease, including myocardial infarction and angina pectoris (24, 25).

Nevertheless, the role that anger plays in the development and progression of hypertension remains unclear largely because few prospective studies have adequately tested the anger-hypertension hypothesis (8). Three studies are suggestive, however. Data from the Tecumseh Community Health Study were used to examine the associations between anger coping styles, BP, and all-cause mortality over 12 years of follow-up (26). Suppressed anger was not correlated with high BP in that study but it is unclear whether incident hypertension was examined in relation to anger coping. However, among persons with elevated SBP (≥ 140 mm Hg), those who suppressed their anger were at five times the risk of all-cause mortality over the follow-up period, relative to those who expressed their anger. Unfortunately, death certificate data were available only on a subset of the population and too few deaths occurred to reliably test the interaction between suppressed anger and hypertension in relation to cardiovascular mortality. In the Israeli Ischemic Heart Disease Study (27), initially normotensive men who suppressed their feelings in response to interpersonal conflicts were more likely to be hypertensive at 5-year follow-up. Psychosocial measures used in that study were not validated or standardized before use, however, and hypertension risk factors were not adequately controlled in the statistical models. Finally, Markovitz et al. (5) reported that increases in feelings of anger ("trait anger") were independently associated with increases in SBP and DBP over 3 years of follow-up among a sample of normotensive, middle-aged women. However, anger expression was unrelated to BP changes in that study, and only seven women became hypertensive over the follow-up period.

We examined the relationship between anger expression and incident hypertension over 4 years in a randomly selected, population-based sample of more than 500 middle-aged men. This report is from the KIHD, designed to examine the relationships between various behavioral and psychosocial risk factors and mortality and morbidity due to cardiovascular diseases, as well as other outcomes (28). Available data on health habits, family illness history, and various anthropomet-

From the Department of Epidemiology (S.A.E., G.A.K.), University of Michigan, Ann Arbor, Michigan, Human Population Laboratory (D.E.G.), Public Health Institute, Berkeley, California, Department of Psychology (J.J.), University of Helsinki, Helsinki, Finland, and Research Institute of Public Health and Department of Community Health and General Practice (J.T.S.), University of Kuopio, Kuopio, Finland.

Address reprint requests to: Susan A. Everson, PhD, MPH, Department of Epidemiology, University of Michigan School of Public Health, 109 S. Observatory St., Ann Arbor, MI 48109-2029. E-mail: severson@umich.edu.

Received for publication March 10, 1998; revision received April 24, 1998.

ANGER EXPRESSION AND INCIDENT HYPERTENSION

ric and demographic measures enabled us to examine potential confounding influences of other risk factors for hypertension.

METHODS

Study Population

The KIHJ study is a population-based study of unestablished and traditional risk factors for atherosclerotic vascular disease, ischemic heart disease, mortality, and other outcomes (28, 29) among middle-aged men from the Kuopio region in Eastern Finland, an area of high coronary morbidity and mortality (30). A total of 2682 participants (82.9% of those eligible), aged 42, 48, 54, or 60 years, were enrolled in the study between March 1984 and December 1989. Follow-up examinations were conducted between March 1991 and December 1993 on men who had undergone ultrasound examination of the right and left carotid arteries at baseline. A total of 1229 were eligible for the follow-up study; of these, 52 had died, were suffering severe illness, or had migrated from the region, and 139 could not be contacted or refused to participate. Thus, the follow-up study included 1038 participants or 88.2% of those eligible. Average time to follow-up was 4.2 years (range 3.8–5.2 years).

For the present analyses, subjects were excluded if they had missing BP data at baseline or follow-up ($N = 4$), were hypertensive according to World Health Organization criteria (ie, BP $\geq 165/95$ mm Hg, or on antihypertensive medications; $N = 396$), had incomplete information on the measures of anger expression ($N = 94$), or had missing data on the covariates at baseline ($N = 7$). Thus, results are based on 537 initially normotensive men (BP $< 165/95$ mm Hg) who completed the anger expression scales at baseline and had complete information on covariates at baseline and hypertension status at follow-up. Comparison of the 101 KIHJ participants with missing data on the anger expression scales and/or baseline covariates with the 537 subjects with complete data revealed no significant differences in baseline age, smoking, weekly physical activity, or parental history of hypertension (p values $> .14$), although those included in the present study had lower resting BP, lower BMI, and lower weekly alcohol consumption (p values $< .004$) than those excluded because of missing data. Baseline subject characteristics are shown in Table 1.

Baseline and Follow-Up Examinations

Examinations were performed over 2 days, 1 week apart, at both baseline and follow-up, and consisted of a wide variety of biochemical, physiological, anthropometric, and psychosocial measures. Medical history and medication use were checked during a medical examination and interview at both baseline and follow-up.

TABLE 1. Baseline Subject Characteristics

<i>N</i>	537
Age (yr)	51.0 (6.6) ^a
Anger expression scales	
Anger-out	10.6 (2.9) ^a
Anger-in	7.6 (2.3) ^a
Anger-control	21.0 (4.3) ^a
Resting SBP (mm Hg)	126 (11.0) ^a
Resting DBP (mm Hg)	83 (7.3) ^a
BMI (kg/m ²)	25.8 (2.9) ^a
Current smokers (%)	31.7
Alcohol consumption:	
Abstainers	61 (11.4%)
>2 drinks/day	103 (19.2%)
Physical activity (hr/wk)	2.2 (2.5) ^a
Parental hypertension	212 (39.5%) ^b

^a Values are mean (SD).

^b Parental hypertension includes maternal and paternal history of hypertension.

Blood Pressure Measurement

BP was measured with a random-zero muddler sphygmomanometer (Hawksley, London, UK) by a trained observer. The measurement protocol, which was completed on the first examination day at both baseline and follow-up, was as follows: 15 minutes supine rest with BP measured at minutes 5, 10, and 15; standing rest with one reading taken after 1 minute; and 10 minutes seated rest with BP measured at minutes 5 and 10. For the present analyses, the averages of the last two supine readings and the two seated measurements were considered resting SBP and DBP, respectively.

Measurement of Anger Expression

The Spielberger Anger Expression Scales (31) were used to measure self-reported levels of outwardly expressing ("anger-out"), inwardly containing ("anger-in"), and controlling ("anger-control") anger. Previous research indicates that these measures are valid and reliable in Finnish samples (32). Each scale consists of eight items with statements such as "I say nasty things," "I tend to harbor grudges that I don't tell anyone about," and "I keep my cool." Participants indicated the extent to which each statement describes their general feelings or action when angry or mad with responses coded from 1 ("hardly ever") to 4 ("almost always"). Previous confirmatory factor analyses (33) of these scales with the KIHJ cohort revealed that 7 of the original 24 items on these two scales had inadequate goodness-of-fit indices (eg, Tucker-Lewis coefficient) and thus the anger scales were recalculated, excluding two items from the Anger-out scale, four items from the Anger-in scale, and one item from the Anger-control scale. Separate scores for each scale were obtained by summing across the items in each scale. Cronbach's α coefficients were .80, .76, and .90 for anger-out, anger-in, and anger-control, respectively. Correlations between the modified and original scales were $> .90$.

Hypertensive Status at Follow-up

The BP measurement protocol at follow-up was identical with the baseline protocol with resting SBP and DBP calculated as the averages of two supine and two seated measurements. A subject was considered hypertensive at the 4-year follow-up examination if his resting SBP ≥ 165 mm Hg or his resting DBP ≥ 95 mm Hg, or if he was currently taking antihypertensive medications. A total of 104 men (19.4%) met these criteria.

Baseline Covariates

Baseline covariates included BMI, calculated as weight (kg) divided by height (m) squared; cigarette smoking, assessed by self-report of never, former, or current smoking (pack-years); alcohol consumption, assessed by a questionnaire on drinking behavior over the previous 12 months and by a 4-day dietary record, and calculated as grams of alcohol per week; physical activity, assessed by self-report of leisure-time activities for the previous 12 months, and calculated as total duration in hours per year; maternal and paternal history of hypertension, measured by participant self-report; and resting DBP (described above). Each of these factors is known to be importantly associated with hypertension.

Data Analyses

Associations between anger expression style and hypertensive status were assessed using a series of age-adjusted logistic regression models with anger scores modeled both continuously and categorically. Subsequent age-adjusted regression models examined potential confounding by BMI, smoking, alcohol consumption, physical activity, positive parental history of hypertension, and baseline resting DBP. Statistical analyses were performed with LOGISTIC and GLM procedures from SAS (SAS Institute, Cary, NC), version 6.12.

RESULTS

Table 2 shows the odds ratios (OR) and 95% confidence intervals (CI) from the age-adjusted logistic regression models with anger expression scores modeled continuously.

Similar results were obtained for anger-out and anger-in, with each 1-point increase on either scale associated with a 12% increased risk of hypertension at the 4-year follow-up (p values $< .01$). Adjustments for BMI, smoking, alcohol consumption, physical activity, a positive parental history of hypertension, and resting DBP showed no confounding by these risk factors. Also, each 1-point increase on the anger-control scale was associated with a 5% decrease in risk of hypertension ($p < .03$), with no evidence of confounding in the risk factor-adjusted model.

Subsequent age-adjusted logistic regression analyses that modeled anger-out and anger-in scores categorically revealed an apparent threshold effect such that men in the top tertile of either scale were at increased risk for hypertension, although results were strongest for anger-out. Men scoring in the top tertile of anger-out were at two-fold greater risk of hypertension after 4 years of follow-up, relative to those with low scores on the anger-out scale (OR = 2.00, 95% CI 1.20–3.38; $p < .009$). Men with scores in the top tertile of the anger-in scale were at 66% greater risk of hypertension, compared with men whose scores were in the bottom tertile (OR = 1.66, 95% CI 0.98–2.82; $p < .06$). Men with scores in the middle tertile of either scale were not at increased risk of hypertension. Results were relatively unchanged after additional adjustments for known hypertension risk factors. Figure 1 presents the findings from the risk factor-adjusted models.

Modeling anger-control scores in approximate tertiles showed that both the middle and high tertiles were at approximately 35% decreased risk of hypertension, relative to the low tertile, although these decreases were only marginally significant (OR = 0.63, 95% CI 0.37–1.05, and OR = 0.64, 95% CI 0.37–1.10, respectively; p values $< .11$). Results were similar following adjustment for risk factors for hypertension.

Influence of Anger Frequency

We then sought to determine whether frequency of anger influenced the relation between anger expression style and hypertension. Self-reported frequency of anger was not directly assessed in the KIH D study; thus, we used a measure of cynical hostility to approximate anger frequency. Research suggests that hostile individuals tend to experience more frequent and intense episodes of anger (34, 35).

To test our hypothesis, we examined the interaction between anger expression (anger-in and anger-out) and hostility in relation to hypertensive status at follow-up in a series of logistic regression analyses. Hostility was measured at base-

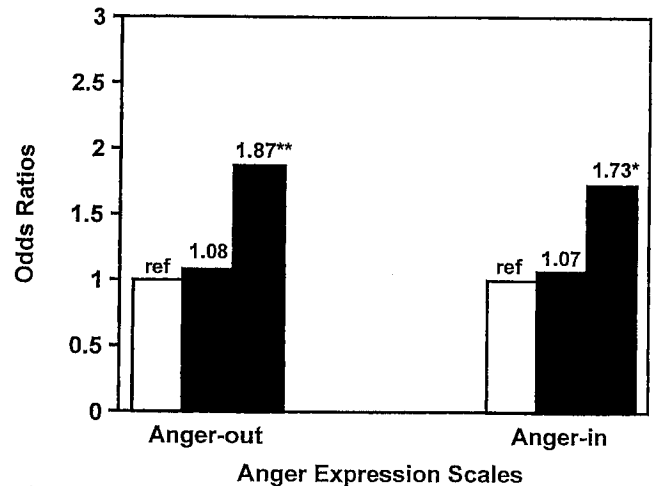


Figure 1. Association between scores on the measures of anger-out and anger-in and 4-year incidence of hypertension. ORs are adjusted for age, BMI, smoking, alcohol consumption, physical activity, parental history of hypertension, and baseline resting DBP. ** $p < .027$; * $p < .06$.

line by the 8-item cynical distrust scale (36) that was factor analytically derived from the Cook-Medley hostility scale (37) and which has established reliability and validity (32, 36). The cynical distrust scale has been shown to predict cardiovascular and all-cause mortality and incident myocardial infarction in the KIH D population (38). A total of 531 of the participants in the present study had completed the cynical distrust scale at baseline and were included in these analyses. Because we had identified a threshold effect for anger expression, we used dichotomous variables for our interaction terms, with the top tertile of scores on anger-out and anger-in, respectively, coded as "1" and the upper half (median-split) of scores on the cynical distrust scale coded as "1." Thus, our models included dummy-coded variables for the interaction term and main effects. In both sets of analyses, 107 men were coded "high" on anger-out or anger-in and hostility, and there were 103 incident cases of hypertension.

An interaction was noted between anger-out and cynical distrust such that hostile men who reported a high degree of outward anger expression were at more than 2.5-fold risk of becoming hypertensive by the 4-year follow-up, relative to men who were less hostile and less prone to outward displays of anger, and after adjustment for age and risk factors for hypertension (OR = 2.61, 95% CI 1.38–4.97; $p < .003$). No interaction between anger-in and hostility was observed, and separate analysis of the relation between cynical distrust and incident hypertension revealed no significant association.

TABLE 2. Association Between Anger Expression Style and 4-Year Incident Hypertension: Kuopio Ischemic Heart Disease Risk Factor Study

	Model 1 ^a			Model 2 ^b		
	OR	95% CI	p	OR	95% CI	p
Anger-out	1.12	1.04–1.20	<0.002	1.12	1.04–1.21	<0.004
Anger-in	1.12	1.03–1.23	<0.01	1.13	1.03–1.25	<0.01
Anger-control	0.95	0.90–0.99	<0.03	0.95	0.90–1.00	<0.05

^a Model 1 adjusted for age.

^b Model 2 adjusted for age, BMI, smoking, alcohol consumption, physical activity, parental history of hypertension, and baseline resting DBP.

ANGER EXPRESSION AND INCIDENT HYPERTENSION

Anger Expression and 4-Year Change in BP

We then conducted a series of age-adjusted regression models to examine the impact of anger expression style on 4-year change in BP, regardless of hypertensive status at follow-up. Results were weaker than our primary analyses using hypertensive status as the outcome, with a marginally significant association observed between anger-out and 4-year increases in SBP and DBP (β values = 0.284 and 0.193, respectively; p values < .07), after adjusting for age and baseline resting BP. Anger-in and anger-control were not significantly associated with 4-year change in BP (p values > .13).

DISCUSSION

This study identified a significant prospective association between anger expression style and subsequent hypertension, independent of known risk factors for hypertension, in a population-based sample of middle-aged men. To our knowledge, this study offers the strongest epidemiological evidence to date that expression of anger is related to the development of high BP. Similar patterns of association were seen between anger-out and anger-in and risk of hypertension 4 years later, with increasingly higher levels of outward expression of anger or increasingly greater anger-in associated with increasingly greater risk. Anger-out was more strongly related to hypertension risk than anger-in, however, when categorized into tertiles (Figure 1), suggesting that the effects of extreme outward expression of anger on cardiovascular function are particularly pronounced. Also, men who reported both a high degree of anger-out and a cynical outlook were at more than two and a half times greater risk of becoming hypertensive than men without these characteristics, and men who reported controlling their anger tended to be at somewhat lower risk of hypertension.

The pattern of findings observed here is consistent with the idea that expressions of anger or hostility that deviate from the norm in either direction (ie, withholding or repressing feelings and outright displays of anger and aggression) may be related to elevated risk of hypertension or other cardiovascular disorders (39, 40). Similarly, our results support the theory postulated by Harburg et al. (13) that distinguished between resentful versus reflective styles of coping with anger, and noted that both anger-out and anger-in can be characterized as resentful styles that serve to prolong feelings of anger and thus sustain elevations in BP. Effects of these coping styles also could be due to distinct interpersonal processes. For example, anger-in may contribute to perceptions of recurring mistreatment and related brooding and resentment, whereas anger-out may provoke repeated episodes of conflict, and both could serve to sustain feelings of anger and contribute to BP elevations. Alternatively, shared variance between anger-out and anger-in could drive the association between anger expression and hypertension risk. In our sample, these two scales were moderately correlated ($r = .57$). People who are easily provoked and express their anger also may harbor grudges and it could be the combination of these characteristics that contributes to elevated BP. We did not observe an interaction between anger-in and anger-out in relation to hypertension risk, however. Nonetheless, some or all of these factors may help explain the equivocal findings reported in the literature. That is, several reviews and meta-analyses indicate some support for Alexander's (1) original hypothesis, which postulated that chronic inhibition of angry feelings led to sustained

elevations in BP, yet other studies have shown that expression of anger or hostility is associated with higher BP levels (3, 4, 8, 21-23, 41).

Specific mechanisms by which anger expression increases risk for hypertension remain to be delineated, although the well documented physiological effects of anger and mental stress make this association biologically plausible. Several lines of evidence indicate that anger and mental stress activate the sympathetic nervous system and hypothalamic-pituitary-adrenal axis, producing increases in heart rate and BP, higher levels of vascular resistance, and secretion of cortisol, catecholamines, glucose, and insulin (6, 7, 42, 43), all of which may contribute to the development or progression of hypertension (44). Moreover, emerging evidence indicates that local growth factors and endothelial mechanisms, which could be influenced by psychosocial characteristics and stress factors, play an important role in hypertension (44). We were unable to examine these mechanisms in the present study; however, it is possible that such mechanisms were enhanced in men who reported the highest levels of anger-out or anger-in, particularly if they also experienced anger more frequently, and thus contributed to their elevated risk of hypertension over the 4 years of follow-up.

We previously have reported that the excess risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction associated with high levels of cynical hostility in the KIH population is largely mediated through behavioral factors (38). The current data are not consistent with this, however. We observed relatively little confounding due to BMI, an indicator of obesity, cigarette smoking, physical activity, or average weekly alcohol consumption. Socioeconomic status, as indicated by education and income, also did not explain our findings (data not shown). Additional research is needed to determine why behavioral factors seem to be more important mediators of hostility and anger in relation to myocardial infarction and mortality than to hypertension.

The majority of men in our study who met the criteria for hypertension at follow-up were not taking antihypertensive medications. In other words, most of these men had an average BP at follow-up ≥ 165 mm Hg, which was determined by an average of two supine and two seated readings over a 30-minute rest on one occasion. Current recommendations are that hypertension be diagnosed only after high BP readings are obtained on at least two clinic visits (45); therefore, the participants in this study cannot be diagnosed with hypertension on the basis of their follow-up BP alone. However, comparison of the resting SBP and DBP measured at follow-up with a 6-day average of resting BP measured at home via a portable, oscillometric BP monitor, available for 94% of our participants, indicates that the average BP obtained during the follow-up examination was a good indicator of average daily BP (age-adjusted r values = .68, p values < .0001). Thus, it is likely that many of the men in our study whose resting BP $\geq 165/95$ mm Hg at follow-up would meet the criteria for diagnosed hypertension.

Our findings are based on a relatively homogeneous sample of middle-aged white men from a single geographic region (eastern Finland). Future research will need to determine whether similar relationships are observed in younger, non-white populations, women, or among other racial or ethnic groups. The differential socialization of behavior and emo-

tions that men and women experience, and cultural differences in how anger is expressed or experienced could lead to differential health effects of anger in varied populations.

In summary, this study provides important epidemiological evidence for the influence of anger expression on risk for hypertension among middle-aged men. The excess risk associated with extremes of anger expression was noted over a relatively short follow-up period of 4 years and was independent of lifestyle factors known to contribute to hypertension risk, as well as parental history of hypertension and initial resting BP level. Future research should test these associations in populations of women and minorities and also should explore the physiological mechanisms by which anger can lead to sustained elevations in BP.

This work was supported by Grant HL44199 from the National Heart, Lung, and Blood Institute and by grants from the Academy of Finland and the Finnish Ministry of Education.

REFERENCES

- Alexander F: Emotional factors in essential hypertension. *Psychosom Med* 1:173-179, 1939
- Ayman D: The personality type of patients with arteriolar essential hypertension. *Am J Med Sci* 186:213-223, 1933
- Diamond EL: The role of anger and hostility in essential hypertension and coronary heart disease. *Psychol Bull* 92:410-433, 1982
- Harburg E, Gleiberman L, Russell M, et al: Anger coping styles and blood pressure in blacks and whites. *Psychosom Med* 53:153-164, 1991
- Markovitz JH, Matthews KA, Wing RR, et al: Psychological, biological and health behavior predictors of blood pressure changes in middle-aged women. *J Hypertens* 9:399-406, 1991
- Schwartz GE, Weinberger DA, Singer JA: Cardiovascular differentiation of happiness, sorrow, anger, and fear following imagery and exercise. *Psychosom Med* 43:343-367, 1981
- Sinha R, Lovallo WR, Parsons OA: Cardiovascular differentiation of emotions. *Psychosom Med* 54:422-435, 1992
- Suls J, Wan C, Costa PT Jr: Relationship of trait anger to resting blood pressure: A meta-analysis. *Health Psychol* 14:444-456, 1995
- Cottingham EM, Brock BM, House JS, et al: Psychosocial factors and blood pressure in the Michigan statewide blood pressure survey. *Am J Epidemiol* 121:515-529, 1985
- Esler M, Julius S, Zweifler A, et al: Mild high-renin essential hypertension: Neurogenic human hypertension? *N Engl J Med* 296:405-411, 1977
- Gentry WD, Chesney AP, Gary HE Jr, et al: Habitual anger-coping styles. I. Effect on mean blood pressure and risk for essential hypertension. *Psychosom Med* 44:195-202, 1982
- Goldstein HS, Med D, Edelberg R, et al: Relationship of resting blood pressure and heart rate to experienced anger and expressed anger. *Psychosom Med* 50:321-329, 1988
- Harburg E, Blakelock EH, Roeper PJ: Resentful and reflective coping with arbitrary authority and blood pressure: Detroit. *Psychosom Med* 41:189-202, 1979
- Cottingham EM, Matthews KA, Talbott E, et al: Occupational stress, suppressed anger, and hypertension. *Psychosom Med* 48:249-260, 1986
- Gentry WD: Relationship of anger-coping styles and blood pressure among black Americans. In Chesney MA, Rosenman RH (eds), *Anger and Hostility in Cardiovascular and Behavioral Disorders*. New York, McGraw-Hill, 1985
- Harburg E, Erfurt JC, Hauenstein LS, et al: Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosom Med* 35:276-296, 1973
- Johnson EH, Spielberger CD: Assessment of the experience, expression, and control of anger in hypertension research. In Johnson EH, Gentry WD, Julius S (eds), *Personality, Elevated Blood Pressure, and Essential Hypertension*. Washington, DC, Hemisphere Publishing, 1992, 3-24
- Schneider RH, Egan BM, Johnson EH, et al: Anger and anxiety in borderline hypertension. *Psychosom Med* 48:242-248, 1986
- Haynes SG, Levine S, Scotch N, et al: The relationship of psychosocial factors to coronary heart disease in the Framingham study. *Am J Epidemiol* 107:362-383, 1978
- Costa PT Jr, McCrae RR, Andres R, et al: Hypertension, somatic complaints, and personality. In Elias MF, Streeten DHP (eds), *Hypertension and Cognitive Processes*. Mount Desert, ME, Beech Hill, 1980, 95-110
- Laude D, Girard A, Consoli S, et al: Anger expression and cardiovascular reactivity to mental stress: A spectral analysis approach. *Clin Exp Hypertens* 19:901-911, 1997
- Miller SB, Dolgoy L, Friese M, et al: Dimensions of hostility and cardiovascular response to interpersonal stress. *J Psychosom Res* 41:81-95, 1996
- Everson SA, McKey BS, Lovallo WR: Effect of trait hostility on cardiovascular responses to harassment in young men. *Intl J Behav Med* 2:172-191, 1995
- Mittleman MA, Maclure M, Sherwood JB, et al: Triggering of acute myocardial infarction onset by episodes of anger. *Circulation* 92:1720-1725, 1995
- Kawachi I, Sparrow D, Spiro A, et al: A prospective study of anger and coronary heart disease. The Normative Aging Study. *Circulation* 94:2090-2095, 1996
- Julius M, Harburg E, Cottingham E, et al: Anger-coping types, blood pressure, and all-cause mortality: A follow-up in Tecumseh, Michigan (1971-1983). *Am J Epidemiol* 124:220-233, 1986
- Kahn HA, Medalie JH, Neufeld HN, et al: The incidence of hypertension and associated factors: The Israeli ischemic heart disease study. *Am Heart J* 84:171-182, 1972
- Salonen JT: Is there a continuing need for longitudinal epidemiologic research? The Kuopio Ischemic Heart Disease Risk Factor Study. *Ann Clin Res* 20:46-50, 1988
- Salonen JT, Lakka TA, Lakka H-M, et al: Hyperinsulinemia is associated with the incidence of hypertension and dyslipidemia in middle-aged men. *Diabetes* 47:270-275, 1998
- Keys A: Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease. Cambridge, MA, Harvard University Press, 1980
- Spielberger CD, Johnson EH, Russell SF, et al: The experience and expression of anger: Construction and validation of an anger expression scale. In Chesney MA, Rosenman RH (eds), *Anger and Hostility in Cardiovascular and Behavioral Disorders*. New York, Hemisphere, 1985, 5-30
- Julkunen J: Suppressing your anger: Good manners, bad health? In Spielberger CD, Sarason IG (eds), *Stress and Emotion*, Vol 16. Washington, DC, Taylor & Francis, 1996, 227-240
- Miller TQ, Jenkins CD, Kaplan GA, et al: Are all hostility scales alike? Factor structure and covariation among measures of hostility. *J Appl Soc Psychol* 25:1142-1168, 1995
- Suls J, Sanders GS: Why do some behavioral styles place people at coronary risk? In Siegman AW, Dembroski TM (eds), *In Search of Coronary-Prone Behavior: Beyond Type A*. Hillsdale, NJ, Erlbaum, 1989, 1-20

ANGER EXPRESSION AND INCIDENT HYPERTENSION

35. Williams RB Jr, Barefoot JC, Shekelle RB: The health consequences of hostility. In Chesney MA, Rosenman RH (eds), *Anger and Hostility in Cardiovascular and Behavioral Disorders*. Washington, DC, Hemisphere, 1985, 173-185
36. Greenglass ER, Julkunen J: Construct validity and sex differences in Cook-Medley Hostility. *Person Ind Diff* 10:209-218, 1989
37. Cook WW, Medley DM: Proposed hostility and Pharisic-virtue scales for the MMPI. *J Appl Psychol* 38:414-18, 1954
38. Everson SA, Kauhanen J, Kaplan GA, et al: Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *Am J Epidemiol* 146:142-152, 1997
39. Linden W: We may be able to do even better than that: A rejoinder to Friedman and Booth-Kewley (1987). *Am Psychol* 43:749-750, 1988
40. Linden W, Lamensdorf AM: Hostile affect and casual blood pressure. *Psychol Health* 4:343-349, 1990
41. Jorgensen RS, Johnson BT, Kolodziej ME, et al: Elevated blood pressure and personality: A meta-analytic review. *Psychol Bull* 120:293-320, 1996
42. Muller JE, Tofler GH, Stone PH: Circadian variation and triggers of onset of acute cardiovascular disease. *Circulation* 79:733-743, 1989
43. Vitaliano PP, Scanlan JM, Krenz C, et al: Insulin and glucose: Relationships with hassles, anger, and hostility in nondiabetic older adults. *Psychosom Med* 58:489-499, 1996
44. Kaplan NM: *Clinical Hypertension*, 7th Edition. Baltimore, Williams & Wilkins, 1998, 41-99
45. Joint National Committee. The Sixth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC-VI). *Arch Intern Med* 157: 2413-2439, 1997