Anger Expression and Risk of Stroke and Coronary Heart Disease Among Male Health Professionals

PATRICIA MONA ENG, SCD, GARRETT FITZMAURICE, SCD, LAURA D. KUBZANSKY, PHD, ERIC B. RIMM, SCD, AND ICHIRO KAWACHI, MD

Objective: Anger expression is a dimension of anger that may be strongly related to coronary heart disease and stroke. To date few cohort studies have evaluated the role of anger coping style in the development of cardiovascular disease. This study prospectively examined the effects of anger expression on incidence of cardiovascular disease. **Methods:** Participants were male health professionals (N = 23,522), aged 50 to 85 years old and without previous cardiovascular disease, who responded to a mailed questionnaire incorporating the Spielberger Anger-Out Expression Scale in 1996. The cohort was followed for 2 years (1996–1998). **Results:** Men with moderate levels of anger expression had a reduced risk of nonfatal myocardial infarction compared with those with lower levels of expression (relative risk: 0.56; 95% confidence interval: 0.32–0.97), controlling for coronary risk factors, health behaviors, use of psychotropic medication, employment status, and social integration. Anger expression was also inversely associated with risk of stroke. The multivariate relative risk of stroke was 0.42 (95% confidence interval: 0.20–0.88), comparing men with higher anger-out scores to men with lower scores. A protective dose-response relationship was observed between anger-out score and risk of stroke (p for multivariate trend test: 0.04). **Conclusions:** Among this cohort of older men with high socioeconomic status and relatively low level of anger expression on average, moderate anger expression seemed to be protective against cardiovascular disease over a limited follow-up period. **Key words:** anger, cerebrovascular accident, coronary disease, prospective studies, male, middle age and older age.

CABG = coronary artery bypass graft surgery; CHD = coronary heart disease; CI = confidence interval; CVD = cardiovascular disease; MI = myocardial infarction; RR = relative risk; SES = socioeconomic status.

INTRODUCTION

Recent prospective studies suggest that anger influences the development of cardiovascular disease (CVD). In a cohort study of 12,986 black and white men and women aged 45 to 64 years old, anger proneness, measured by the Spielberger Trait Anger Scale, was associated with increased risk of coronary heart disease (CHD) in multivariate analyses (1). A strong, angry temperament (eg, tendency to anger quickly with little or no provocation) was identified as a particularly toxic component of trait anger (2). Among 1305 older men followed for an average of 7 years, individuals with higher levels of irritability and impulsive, aggressive anger had elevated rates of CHD (RR: 2.66; 95% CI: 1.26–5.61) (3).

Chronic anger may arouse sympathetic activity and activate the hypothalamic-pituitary-adrenocortical axis, resulting in elevated levels of serum catecholamines that can adversely affect blood pressure, heart rate, and free fatty acids. Repeated episodes of anger are believed to cause endothelial damage and promote arteriosclerosis through hemodynamic stress (4). In addition, intense anger may trigger acute coronary events by initiating vascular (5) and prothrombotic changes (6, 7).

Despite accumulating evidence that anger is linked to CVD, the role of coping style, a key dimension of anger, remains unclear. Outward expression and suppression (eg, conscious inhibition) of anger have each been hypothesized to increase the risk of poor cardiovascular health (8, 9). Much of the research on anger coping style has focused on intermediate outcomes rather than incident CVD. Among provoked persons, extreme anger expression (eg, anger-out) has been related to greater blood pressure and heart rate reactivity (10, 11), though low levels of expression have been linked to increased cardiovascular reactivity as well (12). In addition, high levels of expressed anger have been associated with increased risk of hypertension (13, 14) and unfavorable lipid concentration (15). Yet studies have also shown that anger suppression (eg, anger-in) is related to high blood pressure (13, 16–18), atherosclerosis (19, 20), and adverse lipid profile (15). According to a recent reconciling hypothesis, both extremes in coping style may in fact be detrimental (21, 22). Nonetheless, discordant findings could be driven by the use of different measures and populations as

From the Departments of Epidemiology (P.M.E., E.B.R.), Biostatistics (G.F.), Health and Social Behavior (L.D.K., I.K.), and Nutrition (E.B.R.), Harvard School of Public Health; and the Channing Laboratory (E.B.R., I.K.), Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts.

Address reprint requests to: Ichiro Kawachi, MD, Department of Health and Social Behavior, Harvard School of Public Health, 677 Huntington Avenue, Boston, MA 02115. Email: ichiro.kawachi@channing.harvard.edu

Received for publication August 29, 2001; revision received February 22, 2002.

DOI: 10.1097/01.PSY.0000040949.22044.C6

0033-3174/03/6501-0100

well as temporal ambiguity related to study design. As with anger in general, the effect of coping style on incident CVD may be ideally studied using a prospective design and standardized measures. To date few cohort studies have specifically examined the role of anger coping style in the development of CHD and stroke (23–25). High levels of outwardly expressed anger have been related to increased risk of stroke among 481 men with preexisting ischemic heart disease (23). Other researchers have detected associations between low anger-out and high anger-in and incident CHD using relatively crude scales (24, 25).

Therefore, we prospectively examined the effects of anger expression as assessed by the Spielberger Anger-Out Expression Scale (26) on incidence of CHD and stroke. The Spielberger scale measures a trait rather than a state—essentially the frequency that a person outwardly expresses anger toward other persons or objects when provoked in daily life. Hence, we sought to examine the chronic atherogenic effects of anger as opposed to its acute triggering effects. We also examined the effects of anger frequency on CVD. We hypothesized that high levels of expressed anger and greater frequency of feeling angry would each elevate risk of CHD and stroke.

METHODS

The Health Professionals Follow-up Study

The Health Professionals Follow-up Study is a prospective study of chronic disease among 51,529 male health professionals aged 40 to 75 years old in 1986. Cohort members are dentists (58%), veterinarians (20%), pharmacists (8%), optometrists (7%), osteopaths (4%), and podiatrists (3%). Data on medical history and risk factors were obtained from the participants by mailed questionnaire at enrollment. Every 2 years follow-up questionnaires have been sent to update information on risk factors and newly diagnosed diseases.

Study Population

In 1996 the Spielberger Anger-Out Expression Scale was incorporated into the biennial questionnaire. Because preexisting illness could affect anger levels, we excluded from follow-up 14,591 men with myocardial infarction (MI), angina, coronary artery bypass graft surgery (CABG), angioplasty, heart-rhythm disturbance, and stroke before 1996. We also excluded 4152 men who died before 1996. A total of 9264 men did not provide data on anger expression. The study population for the current analyses therefore consisted of 23,522 men. Respondents to the anger questions were similar in age to nonrespondents (mean age 61.9 vs. 62.4 years) but were less likely to be current smokers (5.6% vs. 7.2%). Although only 14% of nonrespondents provided data on social networks, levels of social integration assessed by the Berkman-Syme Social Network Index (27, 64) seemed to be similar between response groups. Risk of CHD between 1996 and 1998 was slightly higher among respondents (1.2% vs. 0.8%).

Assessment of Anger Expression and Frequency

The Spielberger Anger-Out Scale assesses an anger coping style in which one engages in outwardly expressive behavior, such as slamming doors and insults, in response to feeling angry. The eight items of the Anger-Out Scale are listed in Appendix. Participants reported how often they behaved in a particular manner when angry using a 4-point response scale: 1 = almost never; 2 = sometimes; 3 = often; and 4 = almost always. Ratings for the items were summed to obtain a total anger-out score; high scores indicate greater frequency of outwardly expressed anger. We categorized scores a priori into three levels using cutoff points similar to those of previous studies of anger and CVD (3, 23). Categories were as follows: 1 = 8 or 9 (19.5%); 2 = 10 to 12 (41.5%); and 3 = 13 and above (39.0%). We attempted to isolate the effects of very high expression by using a more stringent cutoff point for our high-risk category: 1 = 8 to 11 (48.7%); 2 = 12 to 16 (43.3%); and 3 = 17 and above (8.0%). For this latter variable, we followed Spielberger's guidelines for interpreting scores and used cutoff points close to the 25th and 90th percentiles of normative adult male samples (eg, 12 and 18) in creating low- and very-high-risk categories (26). Cronbach's coefficient of internal consistency was acceptably high ($\alpha = 0.74$) when calculated for participants. Further details on the Spielberger Anger Expression Scale, including validity, have been described elsewhere (26, 28). We also asked participants how often they felt angry; response choices ranged from 1 (almost never) to 6 (two or more times per day). We categorized responses into tertiles corresponding to the following cutoff points: almost never, 1 to 2 times per month, and 1 or more times per week. The correlation between anger frequency and the Anger-Out Scale (0.51, $p < .0001$) was comparable to the previously reported correlation between the Spielberger Trait Anger and Anger-Out Scales in males (0.52, $p < .001$) (26), suggesting that our one-item assessment of anger frequency had similar convergent validity as the Trait Anger Scale, a standardized measure of anger frequency.

Measurement of Potential Confounders and Effect Modifiers

Information on covariates was also obtained by mailed questionnaire in 1996. Data were collected on health behaviors, including smoking history, combined leisure time and routine physical activity, and body mass index. Participants reported whether they had a routine physical exam in the last 2 years. They also reported medical diagnoses of hypertension, diabetes, and hypercholesterolemia; parental history of MI before age 60; and use of β-blockers, tranquilizers, and antidepressants. Alcohol consumption and energy-adjusted intakes of total and saturated fats, folate, and fiber were measured in 1994 using a semiquantitative food frequency questionnaire (29). Data on vitamin supplement use were also obtained. Subjects provided information on employment status and level of social integration as assessed by the Berkman-Syme Social Network Index (27, 64).

Previous reports have suggested that the effects of anger and hostility may be greater among individuals with preexisting CHD (30). Effects may also be heightened among younger (31, 32) or socially isolated persons (33, 34). Therefore, we conducted analyses stratified by the following potential effect modifiers: preexisting CHD (yes vs. no), age (<65 years vs. ≥65 years), social network index (lower vs. higher levels of social integration), and frequency of feeling angry (one to two times per month or less vs. once a week or more).

CVD End Points

Incident cases of CHD and stroke occurred between return of the 1996 questionnaire and January 31, 1998. Medical records for reported CHD and stroke were reviewed by a physician in a blinded manner. Nonfatal MI was classified as "definite" if World Health Organization criteria (35) were met, and required symptoms were noted along with typical electrocardiographic changes or high cardiac enzyme levels. Stroke was confirmed if characterized by a typical neurological defect of sudden or rapid onset lasting at least 24 hours and attributable to a cerebrovascular event; stroke was subclassified using criteria of the National Survey of Stroke (36). Strokes resulting from infection or neoplasia were excluded. Nonfatal MI and stroke were classified as "probable" when hospital records were not available but the event required hospital admission and the diagnosis was corroborated in additional contact by letter or telephone. We included both definite and probable cases in counts of nonfatal MI and nonfatal stroke. Angina was confirmed by angiogram or if symptoms were reported and nonfatal MI or CABG was concomitant. In most instances we were informed of a subject's death by next of kin, postal authorities, or work associates. The National Death Index was used to ascertain vital status of repeat nonrespondents to the mailed questionnaires. It is estimated that at least 98% of cohort deaths are ascertained with the National Death Index (37). Deaths from MI and stroke were confirmed with hospital records and autopsy reports. Additionally, fatal CHD and stroke were confirmed using death certificates if listed as the underlying cause of death and, in the case of CHD deaths, preexistence of CHD was supported by hospital records or interviews with next of kin. We included all incident events of nonfatal MI, fatal CHD, and CABG in counts for total CHD. Total incident strokes, including nonfatal and fatal cases, were added to total CHD to obtain total incident CVD. Angina cases were not included in total counts unless concomitant CHD was present.

Data Analysis

Each subject contributed person-time from return of the 1996 questionnaire until the time of first event, death, or January 31, 1998. Preliminary analyses were conducted on the basis of complete anger-out data (eg, excluding men with missing data) and with missing values imputed as the average of nonmissing items if at least 50% of items were completed. The two approaches yielded similar relative risk estimates and response rates (72% with complete data vs. 74% with imputed values); therefore, we proceeded with analyses using complete data. Proportional hazards models were used to examine the impact of anger, controlling for potential confounders measured at baseline. The Mantel extension test was used to examine linear trends in risk with increasing levels of anger (38). We also conducted stratified analyses to examine the impact of potential effect modifiers.

RESULTS

The mean Spielberger anger-out score of the cohort was 12.1 (SD: 3.1; range: 8–32), which was lower than means previously reported for older male populations (eg, 14–15) (26, 39). The distribution of anger-out scores is shown in Figure 1. Extremely high levels of anger expression were rare in our cohort; only 5.2% scored at or above the 90th percentile of normative adult male samples (eg, 18 or more). Half of our sample fell below the normative 25th percentile (eg, 12), indicating that many participants were relatively low expressers (26). Item distribution analyses (data not shown) indicated that high frequency responses were rare for items reflecting strongly antagonistic behavior. Only 1.0% reported that they "almost always" expressed anger by slamming doors or striking out. High frequency responses were more common for items that could reflect milder expression; for example, 9.4% reported they "almost always" express anger. At baseline (Table 1), anger expression was associated with a number of potential confounders, such as age and alcohol intake. Relative risks of CHD for traditional risk factors such as smoking were similar in effect magnitude and direction to previously observed estimates (data not shown). Men who felt angry more frequently also tended to express their anger outwardly (Table 1). The average score for the anger frequency assessment was 2.3 (SD:1.2; range: 1–6), or between 1 to 4 times per month.

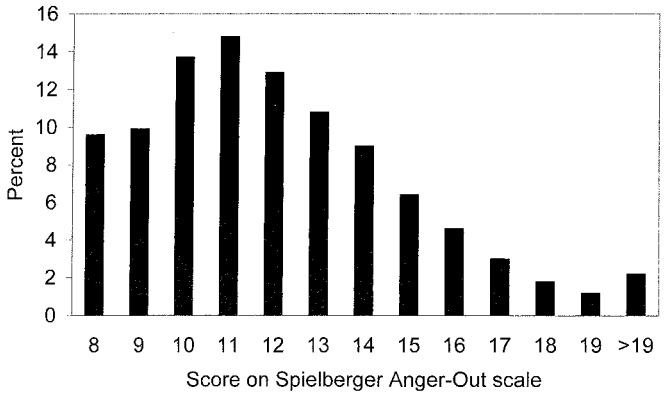

Fig. 1. Distribution of scores on the Spielberger Anger-Out Expression Scale.

During 2 years 328 incident cases of total CVD, including 86 nonfatal MIs, 18 cases of fatal CHD, 167 CABGs, and 57 strokes, occurred among 23,522 men. Of 63 angina cases, 48 also suffered nonfatal MI or had CABG and were included in total counts. In Table 2 we present the age-adjusted and multivariate RRs of CHD by level of anger-out expression. Higher anger-out scores were not related to increased risk of total CHD. Contrary to our hypothesis, anger expression seemed to be somewhat protective against nonfatal MI. Compared with men with low scores on the Anger-Out Scale, men with moderate levels of anger-out expression had nearly half the risk of nonfatal MI (multivariate RR: 0.56; 95% CI: 0.32–0.97). In contrast, expression of anger was associated, albeit not significantly, with increased risk of incident angina. Controlling for frequency of feeling anger did not affect RR estimates (data not shown).

Results from analyses of anger-out scores and CVD

TABLE 1. Age-Adjusted Characteristics, by Level of Anger-Out Expression, of Study Participants in 1996

	Level of Anger-Out Expression			p^a
	8, 9	10–12	13–32	
No. of subjects (%)	4587 (19.5)	9752 (41.5)	9183 (39.0)	
Age, mean (SD), y	65.2 (9.2)	62.2 (8.8)	59.9 (8.0)	<.0001
Current smoker, %	5.7	6.0	5.7	.74
Alcohol intake,[b] mean (SD), g/d	10.1 (14.8)	10.9 (14.3)	12.0 (15.1)	<.0001
Body mass index, mean (SD), kg/m^2	25.7 (3.5)	25.8 (3.5)	26.2 (3.6)	<.0001
Physical activity, mean (SD), METs/wk	33.7 (37.0)	34.7 (36.6)	36.8 (38.3)	<.0001
Hypertension, %	19.5	20.2	21.2	.40
High serum cholesterol, %	21.0	22.6	24.1	<.001
Diabetes, %	4.0	4.4	4.5	.78
Myocardial infarction in parent <60 y, %	10.5	11.3	12.2	.003
β-Blocker use, %	5.3	5.4	5.5	.98
Antidepressant use, %	1.5	2.4	2.9	<.001
Tranquilizer use, %	1.2	1.9	2.3	<.001
Recent routine physical exam, %	65.8	66.6	65.7	.30
Total fat,[b] mean (SD), g/d	67.4 (15.0)	67.3 (15.0)	67.5 (15.1)	.67
Saturated fat,[b] mean (SD), g/d	22.4 (6.3)	22.1 (6.2)	22.2 (6.2)	.03
Folate,[b] mean (SD), mcg/d	508.9 (276.2)	521.9 (279.7)	522.3 (281.1)	.05
Fiber,[b] mean (SD), g/d	22.3 (7.9)	22.3 (7.2)	22.1 (7.3)	.29
Multivitamin use, %	53.6	53.1	53.3	.37
Vitamin E use, %	39.3	41.4	43.4	<.001
Full-time employment, %	63.0	64.0	63.1	<.001
Low social network index, %	7.7	7.5	8.2	.10
Angered at least once per week, %	13.4	30.8	58.4	<.001

[a] p values from analysis of variance (continuous variables) and the Cochran-Mantel-Haenszel test (dichotomous variables), adjusted for age (except where age is the dependent variable).
[b] Assessed in 1994.

TABLE 2. Age-Adjusted and Multivariate[a] Relative Risks (With 95% Confidence Intervals) of Coronary Heart Disease by Level of Anger-Out Expression

	Level of Anger-Out Expression			Trend, p
	8, 9	10–12	13–32	
Nonfatal MI				
Cases	24	30	32	
Age-adjusted RR	1.00	0.65 (0.38–1.12)	0.81 (0.47–1.39)	.98
Multivariate RR	1.00	0.56 (0.32–0.97)	0.70 (0.40–1.21)	.77
Angina				
Cases	9	30	24	
Age-adjusted RR	1.00	1.78 (0.84–3.76)	1.80 (0.83–3.91)	.38
Multivariate RR	1.00	1.59 (0.75–3.40)	1.63 (0.74–3.59)	.46
Total CHD[b]				
Cases	69	107	95	
Age-adjusted RR	1.00	0.84 (0.62–1.13)	0.92 (0.67–1.26)	.97
Multivariate RR	1.00	0.81 (0.60–1.10)	0.85 (0.62–1.18)	.72

[a] Multivariate relative risks adjusted for age in years; smoking history (never, past, and current in categories of 1–14, 15–24, and 25 or more cigarettes/d); alcohol intake (0, 0.01–9.9, 10–19.9, 20–29.9, or 30 or more g/d); quintiles of body mass index; quintiles of physical activity; history of hypertension; high serum cholesterol; diabetes; history of MI in parent aged less than 60 (yes/no); β-blocker use (yes/no); antidepressant use (yes/no); tranquilizer use (yes/no); routine physical exam in last 2 years (yes/no); quintiles of energy-adjusted intakes of total fat, saturated fat, folate and fiber; multivitamin and vitamin E supplement use (yes/no); employment status (full-time, part-time, retired/disabled); and Berkman-Syme Social Network Index (levels I–IV).
[b] Includes nonfatal MI, fatal CHD, and CABG.

are shown in Table 3. For total CVD we observed nonsignificant reductions in risk with increasing expression. We detected significant protective effects of anger expression on risks of nonfatal and total stroke. The multivariate RR of total stroke was 0.42 (95% CI: 0.20–0.88), comparing men with high anger-out scores

TABLE 3. Age-Adjusted and Multivariate[a] Relative Risks (With 95% Confidence Intervals) of Stroke and Cardiovascular Disease by Level of Anger-Out Expression

	Level of Anger-Out Expression			Trend, *p*
	8, 9	10–12	13–32	
Nonfatal stroke				
Cases	17	20	11	
Age-adjusted RR	1.00	0.66 (0.35–1.27)	0.46 (0.21–1.00)	.09
Multivariate RR	1.00	0.64 (0.32–1.26)	0.44 (0.20–0.98)	.09
Total stroke				
Cases	20	25	12	
Age-adjusted RR	1.00	0.71 (0.39–1.28)	0.43 (0.21–0.89)	.04
Multivariate RR	1.00	0.68 (0.37–1.25)	0.42 (0.20–0.88)	.04
Total CVD[b]				
Cases	89	132	107	
Age-adjusted RR	1.00	0.81 (0.61–1.06)	0.81 (0.61–1.08)	.40
Multivariate RR	1.00	0.78 (0.59–1.02)	0.76 (0.56–1.01)	.21

[a] Multivariate relative risks adjusted for age in years; smoking history (never, past, and current in categories of 1–14, 15–24, and 25 or more cigarettes/d); alcohol intake (0, 0.01–9.9, 10–19.9, 20–29.9, or 30 or more g/d); quintiles of body mass index; quintiles of physical activity; history of hypertension; high serum cholesterol; diabetes; history of MI in parent aged less than 60 (yes/no); β-blocker use (yes/no); antidepressant use (yes/no); tranquilizer use (yes/no); routine physical exam in last 2 years (yes/no); quintiles of energy-adjusted intakes of total fat, saturated fat, folate, and fiber; multivitamin and vitamin E supplement use (yes/no); employment status (full-time, part-time, retired/disabled); and Berkman-Syme Social Network Index (levels I–IV).
[b] Includes total CHD and stroke.

to low scorers. A significant dose-response relationship between anger-out score and risk of total stroke was noted (*p* for multivariate trend test: 0.04). In additional analyses, RR estimates did not change when the term for hypertensive status was dropped from multivariate models, suggesting that hypertension was not a mediator between anger expression and stroke. The protective effects of anger expression on stroke were independent of anger frequency.

We repeated analyses of anger expression using a higher cutoff point to isolate extremely high anger-out expressers (data not shown). We observed attenuation of (nonsignificant) protective effects on total coronary and cardiovascular disease. Comparing very high anger-out expressers to low expressers, the multivariate RR of total CHD was 1.01 (95% CI: 0.64–1.59). The corresponding RR of total CVD was 0.88 (95% CI: 0.57–1.36). Protective effects on nonfatal MI were also attenuated. Although stroke cases were scarce among extreme expressers, protective effects were not attenuated and remained significant.

Because of limited numbers, we could examine the impact of potential effect modifiers on total coronary and cardiovascular disease only. Among men less than 65 years old, high and moderate levels of anger expression were each associated with significantly reduced risk of total CVD (RR, 95% CI: 0.54, 0.34–0.87 vs. 0.55, 0.34–0.89, respectively). Among men 65 and older, corresponding reductions in risk were attenuated (RR: 0.86 and 0.90, respectively) and not significantly different from 1. For total CHD, similar findings were observed in age-stratified analyses, though associations were not as marked or significant among younger men. Stratification by frequency of feeling angry and level of social network index did not yield evidence of effect modification.

Findings on the relation between anger frequency and total coronary and cardiovascular disease are shown in Table 4. Among men free of CVD, frequency of feeling angry was not associated with risk of total coronary or cardiovascular disease. We also conducted analyses of specific CVD end points (data not shown). In contrast to findings on anger-out scores, frequent feelings of anger were not associated with risk of stroke or nonfatal MI, controlling for anger expression.

Because anger may have a more pronounced effect among persons with preexisting CHD, we examined the impact of anger frequency and expression among men with coronary disease. Neither expression nor frequency of anger seemed to vary by preexisting disease status since mean scores and frequencies were similar for diseased and nondiseased men (data not shown). During 2 years of follow-up of 2287 men who had preexisting CHD and provided data on anger, 123 cardiovascular events occurred, including 102 recurrent coronary events and 21 cases of total stroke. Increased frequency of feeling angry was significantly associated with elevated risk of recurrent coronary disease (Table 4). Men who reported feeling angry one to two times a month had 1.83 times the risk of recur-

TABLE 4. Age-Adjusted and Multivariate[a] Relative Risks (With 95% Confidence Intervals) of Total Coronary and Cardiovascular Disease,[b] According to Frequency of Feeling Angry, for Men With and Without Preexisting Coronary Heart Disease

	Frequency of Feeling Angry			Trend, *p*
	Almost Never	1–2 Times/Month	≥1 Time/Week	
No preexisting CHD				
Total CHD				
Cases	103	80	87	
Age-adjusted RR	1.00	0.88 (0.65–1.18)	0.92 (0.69–1.23)	.81
Multivariate RR	1.00	0.87 (0.65–1.18)	0.89 (0.66–1.20)	.66
Total CVD				
Cases	126	102	99	
Age-adjusted RR	1.00	0.92 (0.71–1.20)	0.87 (0.66–1.13)	.37
Multivariate RR	1.00	0.91 (0.70–1.19)	0.83 (0.63–1.10)	.25
Preexisting CHD				
Total CHD				
Cases	28	39	35	
Age-adjusted RR	1.00	1.80 (1.11–2.94)	1.47 (0.89–2.45)	.58
Multivariate RR	1.00	1.83 (1.10–3.03)	1.24 (0.73–2.10)	.78
Total CVD				
Cases	39	46	38	
Age-adjusted RR	1.00	1.55 (1.01–2.39)	1.16 (0.74–1.84)	.86
Multivariate RR	1.00	1.55 (0.99–2.42)	0.98 (0.61–1.58)	.32

[a] Multivariate relative risks adjusted for age in years; smoking history (never, past, and current in categories of 1–14, 15–24, and 25 or more cigarettes/d); alcohol intake (0, 0.01–9.9, 10–19.9, 20–29.9, or 30 or more g/d); quintiles of body mass index; quintiles of physical activity; history of hypertension; high serum cholesterol; diabetes; history of MI in parent aged less than 60 (yes/no); β-blocker use (yes/no); antidepressant use (yes/no); tranquilizer use (yes/no); routine physical exam in last 2 years (yes/no); quintiles of energy-adjusted intakes of total fat, saturated fat, folate, and fiber; multivitamin and vitamin E supplement use (yes/no); employment status (full-time, part-time, retired/disabled); and Berkman-Syme Social Network Index (levels I–IV).

[b] Total CHD includes nonfatal MI, fatal CHD, and CABG; total CVD includes total CHD and stroke. Case numbers are slightly different from those in Tables 2 and 3 because one individual did not provide data on anger frequency.

rent coronary disease compared with men who claimed to almost never feel angry (95% CI: 1.10–3.03). The significant elevation in risk remained after adjusting for level of anger expression (data not shown). However, even higher frequency of anger was not associated with significantly elevated risk. Anger expression was not related to risk of recurrent CHD or total CVD among men with preexisting disease. Case numbers were too sparse to examine specific end points.

DISCUSSION

Our findings suggest a more complex pattern of associations between anger and CVD than previously described. Contrary to our expectations, we observed a protective dose-response relationship between anger expression and risk of stroke. Moderate levels of anger expression were protective against nonfatal MI, though we did not observe a dose-response relationship. We did not find that anger expression was significantly related to risk of total coronary or cardiovascular disease in the overall cohort; however, anger expression was significantly protective against total CVD among participants less than 65 years old. Among men with preexisting coronary disease, more frequent feelings of anger significantly increased the risk of recurrent coronary disease in multivariate analyses.

Previous cohort studies have reported that anger expression is inversely related to CHD risk, but only when measured by the Framingham Anger-Out Scale, a two-item scale. Among white collar men aged 45 to 64 years, Haynes et al. (24) observed that lower Framingham anger-out scores were related to significantly increased risk of CHD in multivariate analyses. More recently, in the Caerphilly study of 2890 men aged 49 to 65 years, investigators detected an increased risk of ischemic heart disease among low scorers on the Framingham scale (RR: 1.70; 95% CI: 1.26–2.29) (25). However, it is unclear how the Framingham scale relates to standardized measures such as the Spielberger scale. Only one study has prospectively examined the impact of anger expression as measured by the Spielberger scale on CVD. Everson et al. (23) used a truncated six-item version of the Spielberger Anger-Out Scale to examine the association between anger expression and stroke in a population-based cohort of Finnish men (mean age, 53 years). Among men with

preexisting ischemic heart disease, higher anger-out scores (eg, top tertile) were related to a nearly sevenfold increase in risk of stroke (RR: 6.87; 95% CI: 1.50–31.4), controlling for biological risk factors and income. Other prospective studies finding a link between anger and CHD have used scales that measure trait anger or capture aspects of anger beyond expression, such as irritability (1–3).

Anger and its expression may have different effects on the onset vs. progression of disease (40). In our cohort moderate anger expression seemed to protect against development of disease but was not related to prognosis. However, frequent angry feelings were linked to poorer prognosis. Other studies have shown that anger may be especially toxic among individuals with established CHD. A 3-year follow-up study found that irritability and easy arousal of anger were associated with elevated rates of CHD-related hospitalization and mortality among men with preexisting disease (30). As noted, adverse effects of anger-out expression on stroke have been prospectively detected among individuals with preexisting CHD (23). In the present study there were too few cases to study the specific relation between expression and stroke among men with preexisting disease. In terms of underlying physiology, it has been hypothesized that severity of arteriosclerosis may increase vulnerability to anger among those with preexisting disease (30). More advanced pathological processes (eg, atherosclerosis) may also counter any potentially beneficial effects of moderate anger expression. A caveat to findings based on individuals with preexisting disease is that severity of disease could have biased self-reports of anger and expression, either attenuating or exaggerating effects. In addition, individuals with existing CHD might be less likely to admit expressing anger than feeling anger; such a scenario could account for the null findings on expression among men with preexisting disease.

Underlying differences in study populations may explain disparate findings between studies. First, participants in our study may have expressed anger at lower levels compared with previous populations. Although we set out to study the adverse effects of extreme anger expression, few individuals may have actually expressed anger at toxic levels. According to Siegman (8), it is the full-blown expression of anger rather than moderate expression that may increase risk of CVD. Although anger is often viewed negatively, anger expression can be used to communicate disagreement and assert power (41). Mild expression may not be associated with CVD and could even be protective compared with extremes in anger coping style (eg, anger-out vs. anger-in) (21, 22). Furthermore, low Spielberger anger-out scores have been linked to increased resting blood pressure (42) and blood pressure reactivity (12, 43). Interestingly, in the current study anger-out expression was most protective against stroke, though hypertension did not seem to mediate the relationship. It is also important to note that the Spielberger Anger-Out Scale measures how often individuals express anger when they feel angry. Although it is difficult to compare frequency of feeling angry across studies, the average frequency of angry feelings within our cohort was lower than previously reported frequencies (eg, several times a day to several times a week) (44). It is possible that expression is detrimental only if anger is experienced above a threshold level of frequency.

The older age of our cohort may have precluded adverse effects of anger and hostility that are more consistently observed among younger individuals, particularly those under 55 years (31, 32, 45). Selective survival and/or adaptive development of collateral circulation are believed to contribute to attenuation of adverse effects and null associations among the elderly (32, 45). Most of the cohort studies reviewed here reported deleterious effects of anger among middle-aged individuals (1, 2, 23–25), though adverse effects have also been detected among older men (3). Because the average age of our cohort was more than 60 years at baseline, many events related to anger may have occurred before follow-up. To address this issue, we further examined age-related differences in effect within the cohort. Although we observed significantly protective effects of expression on CVD among men under 65 years, effects were attenuated and no longer significant among men less than 60 years, though numbers were sparse (data not shown). Because of the limited age range of our cohort, it was not possible to study younger men (eg, <55 years). Nonetheless, effect modification by age can readily account for only lack of adverse effects in the present study and not the significantly protective effects on stroke and nonfatal MI observed.

In addition to being older, our cohort was composed of well-educated male health professionals with high socioeconomic status (SES). Because the definition and meaning of anger may vary according to social context (46), it is plausible that high-SES respondents could interpret items on the Spielberger scale differently from lower-SES respondents depending on their unique experience of anger (eg, differential item functioning). Higher education level may be related to greater cognitive flexibility and problem solving in dealing with anger (47). In addition, well-educated persons are more likely to perceive their anger as appropriate, a perception that may be linked to greater responsibility and authority within occupational set-

tings (47). Conceivably, items such as "I express my anger" and "If someone annoys me, I'm apt to tell him how I feel" may have tapped protective styles of coping (eg, reflective, constructive verbal or socially assertive expression) within our cohort even though the scale was not intended to do so. Reflective coping style has been linked to lower blood pressure levels relative to resentful (eg, anger-out or anger-in) styles of coping (48). Similarly, constructive anger expression may be inversely related to resting blood pressure (49). Furthermore, socially assertive expression has been negatively correlated with serum cholesterol and low-density/high-density lipoprotein ratio (50). Item analyses revealed that the item "I express my anger" was significantly protective against total stroke in a dose-response relationship, whereas items associated with sarcasm or hostility (eg, "I make sarcastic remarks" and "I say nasty things") were positively associated with stroke risk (data not shown). Furthermore, high frequency responses ("often" or "almost always") were most common for these less explicit items. Therefore, some high anger-out scores could reflect higher levels of positive and constructive expression that may be protective against CVD. On the other hand, although validity data specific for high-SES groups are not available, relationships between the Spielberger Anger-Out Scale and other psychosocial factors within the cohort seem to be similar to those observed in previously studied populations. As reported in the literature, the Anger-Out Scale was moderately correlated with anger frequency but not with level of social integration (26, 51). Furthermore, the Anger-Out Scale has been used in previous studies of relatively well-educated individuals, including college students (52–54).

Finally, social context (eg, social status and roles) may modify the social and physiological consequences of anger expression. Individuals with lower social status may more often be in situations where expression, however desirable, is neither appropriate nor tolerated. Conflict over expression among lower-status individuals may be related to increased cardiovascular reactivity or slower recovery (54–58). Engebretson et al. (54) reported that Spielberger anger-out expressers experienced more rapid decline in reactivity when they were permitted to express their anger by writing negative evaluations of a provoking confederate. No decrease in reactivity was observed if they were made to write positive evaluations (ie, suppressed their anger). In Hokanson's classic studies, subjects experienced reductions in blood pressure elevations when counter-aggressing against provocateurs of equal or lower status but not against those of higher status (56). Hence, recovery seemed to be related to whether expression was considered appropriate or socially sanctioned. Using the Spielberger scale, Shapiro et al. (58) have noted that anger-out expressers have elevated systolic blood pressure in work settings that do not allow overt confrontation. They suggested that conflicting attitudes on expression and attempts to inhibit anger could have contributed to a larger stress response. Addressing the inconsistent findings on anger expression and suppression, Engebretson et al. (54) noted that "the direction of effects between anger expression and CVD should vary according to whether the study population can consistently express and are reinforced for doing so in a preferred or nonpreferred mode" (eg, anger-out vs. anger-in coping styles).

High-SES individuals often have the power and status to freely express anger, particularly within occupational settings where they may hold supervisory positions. In discourse with subordinates, anger expression may represent a means for the expresser to assert authority, achieve goals, and increase efficiency by regulating relationships (47). Thus, because of their position in society, high-SES individuals may be able to display anger without conflict. Among high-SES individuals then, anger-out expression may be related to release of stress without negative social or physiological consequences. Furthermore, because expressing may be socially advantageous for higher-SES individuals, they may express anger at *lower* levels of intensity. This could also contribute to different effects depending on SES since expression may be most toxic when anger is intense. Taken together, it is plausible that anger expression may have differential effects depending on the SES of study populations. Notably, a recent study reported no significant correlation between hostility and coronary artery calcification among asymptomatic, highly educated subjects aged 39 to 45 years (59). This finding provides some additional support for the notion that high SES may modify potentially toxic effects of anger or hostility.

Lifestyle factors related to SES may also have contributed to our findings. High-SES individuals have lower average blood pressure and less hypertension and hyperlipidemia (60–62). They are also less likely to smoke and to be inactive or overweight (60–62). The healthier lifestyle of our cohort may have buffered against any adverse effects of anger expression. In post hoc analyses (data not shown), anger expression was protective against total CVD only among men without high blood pressure. Normotensive men with high levels of expression had a 34% decrease in risk compared with less expressive counterparts (RR: 0.66; 95% CI: 0.46–0.96). Anger expression was not related to CVD among hypertensives. Anger expression did not have different effects according to serum cholesterol level, level of physical activity, or body mass index, al-

though numbers were sparse for stratified analyses. Hence, we found only partial support for effect modification by lifestyle factors, though there was limited variability in such factors within this relatively healthy population.

There were several methodological limitations to this study. A plausible explanation for our unexpected findings is the relatively short follow-up period. Previous reports of adverse effects have been based on longer periods of follow-up (1–3, 23–25). Underlying mechanisms such as atherogenesis may be relatively slow-acting and require more time before adverse effects become manifest. Although not statistically significant, the adverse association with angina, often indicative of early stage CHD, weighs in favor of this latter explanation. In addition, the nonresponse rate was moderately high (28%). However, although angrier men may have been less likely to respond, it seems unlikely that nonresponse was related to anger *and* subsequent CVD given the prospective design of the study. Average age and risk of CHD were similar for respondents and nonrespondents; based on limited available data, social network index levels were also comparable. Underreporting due to social desirability or lack of self-awareness could have occurred (63). Underreporting would lead to nondifferential misclassification of anger levels and bias to the null since men with higher levels would report themselves as less angry independently of future disease status. Alternatively, single measurements of anger in mid to late life may not have captured the etiologically relevant period of exposure. Although we intended to measure anger as a stable trait, anger levels may decline with age. In normative samples, mean Spielberger anger-out scores range from 15.7 for men aged 18 to 30 years to 14.3 for those aged 41 years and more (26). Any resulting nondifferential misclassification may have biased estimates to the null but could not have produced the protective effects we observed. Finally, our findings were detected among older men with high SES and may not be generalizable to younger or lower-status individuals.

In conclusion, we found that moderate levels of anger expression were protective against stroke and nonfatal MI among older male health professionals. To clarify the relation between anger and CVD further, future studies should simultaneously assess different aspects of anger, including frequency, intensity, duration, and coping styles (both negative and positive). In addition, the potential for effect modification by SES should be considered; stratifying analyses by SES may reveal important differences in the relationship between anger and CVD according to status.

This study was supported by Research Grants HL 35464 and CA 55075 from the National Institutes of Health.

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APPENDIX

The Spielberger Anger-Out Expression Scale (26)

The following statements describe how people act when they feel angry or furious. Please indicate how often you generally react or behave in the manner described.

1. I express my anger.
2. I make sarcastic remarks to others.
3. I do things like slam doors.
4. I argue with others.
5. I strike out at whatever infuriates me.
6. I say nasty things.
7. I lose my temper.
8. If someone annoys me, I'm apt to tell him how I feel.

Response categories: 1 = almost never; 2 = sometimes; 3 = often; 4 = almost always.