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Anger and Impatience/Irritability in Patients of Low Socioeconomic Status with Acute Coronary Heart Disease

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This case-control study examines the relationship between anger and impatience/irritability and acute coronary heart disease (CHD) in middle-aged men of low socioeconomic status (SES). Subjects included patients with myocardial infarction (MI) (N = 31) or unstable angina (AP) (N = 26), who were compared with hospital controls (N = 26). In separate multivariate analyses for each anger scale, MI was associated with Anger-Out and Impatience/Irritability, particularly in the subgroup of patients who did not have a previous MI. The same factors were associated with AP, but only when this acute ischemic event was not preceded by a MI. No relationship was found between Trait-Anger and Anger-In and either acute ischemic outcome. The results indicate that particularly overt behavioral expression of anger is related to CHD in lower SES patients and that there is similarity in the behavioral factors associated with acute CHD between low- and high-SES men.

KEY WORDS: anger; impatience; coronary heart disease; socioeconomic status.

INTRODUCTION

The last few decades have produced a wealth of research on psychosocial predictors of coronary heart disease (CHD). Recent studies have focused increasingly on hostility and anger, two related constructs that are often used interchangeably (Diamond, 1982) or considered one

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complex construct (Williams, 1988; Musante *et al.,* 1989). Despite the fact that individuals of lower socioeconomic status (SES) are at greater risk for CHD incidence and mortality (Pell and Fayerweather, 1985; Rogot and Hrubec, 1989), most of this research has been conducted in middle- to upper-class populations. Consequently, the role of psychosocial factors in the development of CHD in a population of lower SES remains largely unknown. The present study is an attempt to begin filling this gap by exploring the role of anger and its correlates in relation to CHD in patients of lower SES.

Research on the relationship between anger and CHD has produced somewhat ambiguous findings. The disposition to *experience* anger (trait-anger) has been found to be unrelated to various CHD end points (Smith *et al.,* 1984; Shocken *et al.,* 1985; Tennant *et al.,* 1987), with the exception of one report of a positive association with frequency of anginal complaints (Smith *et al.,* 1984). On the other hand, overt behavioral *expression* of anger (anger-out) appears significantly associated with CHD (Siegman *et al.,* 1987a, b; Dembroski *et al.,* 1989).

Another facet of anger is anger-in, which represents the inability or unwillingness to express feelings of anger. A self-report of the inability to express anger was predictive of CHD in the Framingham study (Haynes *et al.,* 1980). Behavioral ratings of anger-in, derived from the Structured Interview method for the assessment of the Type A pattern, also correlated positively with degree of coronary atherosclerosis (Dembroski *et al.,* 1985; MacDougall *et al.,* 1985) but failed to predict myocardial infarction (MI) and sudden death (Dembroski *et al.,* 1989).

A closely related aspect of anger is impatience and irritability, which has received much less attention in research on coronary-prone behaviors. One of the first studies to explore the predictive validity of various Type A components for CHD found that irritability was among the components that prospectively distinguished cases from noncases in the Western Collaborative Group Study (Matthews *et al.,* 1977). Irritability was also predictive of incidence of CHD in middle-aged hypertensive men with previous CHD but not in disease-free men (Koskenvuo *et al.,* 1988).

Thus, the evidence to date suggests that particularly overt behavioral expression of anger is related to coronary end points, while the findings with respect to other facets of anger remain inconclusive. No studies to date have examined the role of anger in CHD in low-SES men. The present case-control study describes the relationships of trait-anger, anger-out, anger-in, and impatience/irritability with clinical manifestation of acute CHD in patients of lower-SES background.

METHOD

Subjects

Cases in this study were white, male patients between 40 and 65 years old, admitted to a state-referral hospital in southeastern Texas, most of whom were treated there for lack of private health insurance. Controls consisted of patients with a variety of conditions using the same age inclusion criteria and sampled from the same hospital. No attempt was made to match cases with controls on age or any other factor.

Cases were patients hospitalized for acute severe chest pain and/or other symptoms indicative of an acute or unstable coronary ischemic episode. Since after several days not all of these patients showed definite evidence of infarction, they were allocated to one of two case groups. Case group 1 consisted of 31 patients with acute myocardial infarction (MI), based on objective evidence of myocardial damage (as confirmed by enzyme levels and positive ECG changes). The remaining 26 patients were allocated to case group 2 and diagnosed with acute or unstable angina pectoris (AP). All diagnoses were provided by a cardiologist and verified in the discharge report. Patients without sufficient objective evidence that the chest pain was related to organic heart disease, as documented by coronary angiography and/or ECG (patients with "atypical chest pain"), were excluded from the second case group. Patients were also excluded from either case group if their ischemic event was complicated by another severe heart condition, such as congestive heart failure.

The control group consisted of 26 patients, the majority of whom (N = 23) were orthopedic patients, hospitalized for diseases of the musculoskeletal system and connective tissue, such as rheumatoid arthritis and osteoarthrosis (N = 14), or fractures of the upper or lower limbs (N = 9). The remaining three patients were obtained from the Departments of General Surgery and Urology. Patients with a history of heart disease were excluded from the control group. Patients with other chronic illnesses, in particular cancer, and patients with a history of psychiatric illness, were not included in any of the patient groups.

Participants were contacted in the hospital, usually several days after admission. The nursing staff indicated whether a patient had recovered well enough from the critical stage of his disease or from surgery to participate in the study. Each patient received a questionnaire which they were asked to complete before leaving the hospital. The questionnaire specifically instructed them to refer to the period *before* their current disease episode while evaluating themselves on the behavioral factors of interest. This in-

struction was repeated orally, after the patient was asked about background information.

Instruments

The various facets of anger were assessed by Spielberger's State-Trait Anger Scale (STAS) (Spielberger *et al.,* 1983) and Anger Expression Scale (Spielberger *et al.,* 1985). Trait-Anger was assessed by the 15-item Trait version of the STAS, with total scores ranging from 15 to 60. This is a measure of the tendency to experience anger and to be aware of experiences of anger. Internal consistency reliability of these items in the present sample was .91 (Cronbach's coefficient alpha). Anger-Out was assessed by the eight-item Anger-Out subscale of Spielberger's Anger Expression scale and reflects how often anger is expressed toward other people or objects in the environment. Total scores range from 8 to 32, and internal consistency reliability in the present sample was .82. Anger-In was assessed by the eight-item Anger-In subscale of Spielberger's Anger Expression scale. This scale assesses how frequent angry feelings are held in or are suppressed. Total scores range from 8 to 32, and internal consistency reliability was .68.

Impatience/Irritability. The impatience/irritability (Impat-Ir) scale consisted of five items identified through factor-analysis from the Jenkins Activity Survey (JAS) (Spence *et al.,* 1987). The scoring method of the items was adopted from Spence *et al.* (1987): for each item the responses indicating the least and the most irritability were coded as 1 and 5, respectively, with other responses coded at an equal distance between 1 and 5, depending upon the total number of possible responses. Cronbach's coefficient alpha for internal consistency was .70. This scale, although highly correlated with the original JAS Speed and Impatience subscale (Spence *et al.,* 1987), is a purer measure of impatience/irritability, because it eliminates items assessing speed, which has not been found to be related to coronary risk (Jenkins and Zyzanski, 1980).

Statistical Methods

Univariate differences were tested using unpaired t tests comparing each case group with the control group. Separate logistic regressions were computed for each of the anger and impatience scales in order to examine differences between cases and controls with adjustment for selected control variables, using the Catmod procedure in SAS (SAS Institute Inc., 1988). Patient group status (MI, AP, control) was used as the dependent variable,

Table I. Zero-Order Correlations Between Scales

	Trait anger	Anger-out	Anger-in
Anger-out	.71		
Anger-in	.37	.36	
Impatience-irritability	.58	.53	.36

with the control group serving as the referrent group with which the two case groups were compared in each model.

Control variables in the multivariate analyses included marital status (married vs. nonmarried), education (years of schooling completed), high blood pressure, smoking (in pack-years), and a dichotomous index (chronic vs. acute) for chronicity of the condition. Since age and employment differences between cases and controls were very small, these variables were omitted in view of the limited number of patients. The chronicity variable was added to adjust for the effects of previous and/or chronic symptoms preceding the current clinical episode. Cases with previous clinical manifestation of CHD were coded as chronic. Controls with arthritic conditions were coded as chronic, and those with fractures as acute.

RESULTS

There were no substantial sociodemographic differences between the two case groups and the control group. The MI case group, AP case group, and control group were, on the average, 52.4, 51.9, and 54.0 years old, respectively, and had completed, on the average, 10.3, 11.5, and 12.0 years of formal schooling, while, respectively, 78, 85, and 69% were married and 53, 62, and 54% were employed at the time of hospitalization. Overall, the refusal rate was 10% and was slightly higher in the control group (12%) than in the combined case groups (9%).

As shown in previous research (Smith and Frohm, 1985; Spielberger *et al.,* 1983) intercorrelations between measures of facets of hostility and anger are modest to high. The highest correlation in the present sample was found between Spielberger's Trait-STAS and his Anger-Out scales (r = .71), while the Anger-In scale was least, although still modestly, correlated with the other scales (see Table I).

The results of the univariate analyses indicate that MI patients had significantly higher mean Anger-Out (16.7 vs. 14.4; $p < .05$) and Impat-Ir (17.4 vs. 15.2; $p < .05$) scores than the control group (see columns 2–4 in

Table II. *T* Tests Comparing MI and AP Patient Groups with Controls on Each of the Anger and Impatience Variables

	Any-event sample			First-event sample		
	MI	AP	Controls	MI	AP	Controls
Trait anger	30.7[a]	30.8	28.7	30.3	32.1	28.7
	(9.9)	(7.0)	(7.3)	(10.4)	(7.0)	(7.3)
Anger-out	16.7*	15.2	14.4	16.8*	16.1*	14.4
	(6.0)	(3.3)	(4.1)	(6.1)	(3.8)	(4.1)
Anger-in	16.3	16.1	15.6	16.2	16.1	15.6
	(4.1)	(2.6)	(5.4)	(4.4)	(3.2)	(5.4)
Impatience/ Irritability	17.4*	16.6	15.2	17.5*	17.7*	15.2
	(3.3)	(3.0)	(3.8)	(3.5)	(3.2)	(3.8)

[a]Mean scores (standard deviations).
*$p < .05$, two-tailed.

Table II). On the other hand, Trait-Anger and Anger-In levels were very similar for these two patient groups. The AP group did not exhibit significantly different mean scores on the anger and impatience/irritability scales compared to the control group.

It should be noted that some of the patients in the case groups had suffered a MI prior to the current disease episode. This may have affected some of the relationships, since predictors of a first MI may differ from those of a recurrent event. Prior MI may lead to behavioral change and thus could result in an attenuation between behavior and disease status. In addition, cardiac risk factors are known to be more important in predicting recurrent events, and predictive validity of behavioral risk factors for recurrent CHD may be limited to patients with the lowest cardiac risk profiles after MI (Powell and Thoresen, 1988). Therefore, comparisons were repeated after excluding patients with a previous MI from the case groups, resulting in a reduction of 5 patients in the MI group ("first-event" MI group; $n = 26$), and of 12 patients in the AP case group ("first-event" AP group; $n = 14$).

Exclusion of patients with previous MI affected mainly the results for the AP group. The first-event AP group had significantly higher average Anger-Out (16.1 vs. 1447; $p < .05$) and Impat-Ir (17.7 vs. 15.2; $p < .05$) scores than the control group, despite the substantial loss in number of patients (see columns 5–7 in Table II). This finding indicates that previous MI weakened the relationship between anger-out and impatience/irritability and unstable AP in this study.

Table III. Logistic Regression Models[a] for Each of the Anger and Impatience Variables Separately

	Any-event sample, b (SE)[a]		First-event sample, b (SE)[a]	
	MI	AP	MI	AP
Trait-anger	.022 (.037)	.038 (.039)	.019 (.040)	.053 (.044)
Anger-out	.157* (.072)	.125 (.075)	.183* (.081)	.193* (.090)
Anger-in	.052 (.068)	.067 (.073)	.060 (.069)	.074 (.079)
Impatience/ irritability	.206* (.095)	.118 (.098)	.294** (.109)	.318** (.126)

[a]All models are adjusted for age, marital status, education, smoking, high blood pressure, and chronicity of condition.
[b]Logistic regression coefficients and standard errors.
*$p < .05$, two-tailed.
**$p < .01$, two-tailed.

The logistic regression models with adjustment for control variables for each of the anger scales separately produced generally the same pattern of results as the univariate findings (see Table III). In the any-event case groups (the original case groups), only Anger-Out ($b = .157$, $p < .05$) and Impat-Ir ($b = .206$, $p < .05$) were significantly associated with MI. The associations were somewhat more pronounced in the first-event case groups, particularly for the Impat-Ir scale, which was associated with MI and AP at the $p < .01$ level (see columns 4 and 5 in Table III). Anger-Out was now also significantly associated with AP ($b = .193$, $p < .05$). None of the associations for the Trait-Anger or Anger-In were statistically significant.

The previous analyses examined the relationship between each of the anger and impatience scales and the end points separately. Given the moderate to high intercorrelations between the various anger variables, it was important to test the *independent* relationships of each of these variables to the end points. To that end, logistic regressions were computed, using the BMDP-LR procedure (Dixon, 1988), with stepwise selection of the independent variables after forced entry of the control variables. Given the small sample size, the significance level of the approximate chi-squares for entering or removing terms from the equations was set at $p = .15$.

Impat-Ir was the only variable to enter the models comparing MI patients and controls, which it did at a marginally significant level ($p <$

.10) for the any-event case group but at a significant level ($p < .05$) for the first event MI case group. Anger-Out was the only variable associated with AP, again at a marginally significant level ($p < .10$) for the any-event patient group but a significant level ($p < .05$) in the first-event case group (data not shown).

DISCUSSION

Despite the uneven share in the burden of this chronic and often debilitating disease, low-SES populations are understudied with respect to behavioral risk factors for CHD. In this study of patients of lower socioeconomic background, anger-out and impatience/irritability were associated with acute MI and with unstable angina. The experience of anger and the suppression of anger were not related to these manifestations of CHD. Since these findings were based on cross-sectional observations, they do not permit causal inferences. However, they can provide the basis for more detailed investigation in prospective research.

The finding that anger-out was related to acute CHD is consistent with increasing evidence that only overt expression of anger is related to coronary risk. In a study of coronary patients of comparable educational background, overt verbal and other behavioral anger expressions were found to be related to severity of coronary artery disease (Siegman *et al.,* 1987a, 1987b) and to hard CHD end points such as myocardial infarction and sudden death (Dembroski *et al.,* 1989). Anger expression and expressive vocal behavior have also been shown to increase cardiovascular reactivity in anger-arousing situations (Suarez and Williams, 1990; Siegman *et al.,* 1990). Overt expression of anger is thought to represent a style of interaction characteristic of antagonistic hostility. It is this type of hostility, as opposed to neurotic hostility, that is presumed to be truly coronary prone (Siegman *et al.,* 1987a; Musante *et al.,* 1989; Costa *et al.,* 1989).

The present study shows that both anger-out and impatience/irritability were significantly associated with acute CHD, with impatience/irritability showing the strongest relationship with MI and anger-out with unstable AP. It should be noted that, although the differences in anger-out and impatience/irritability between cases and controls are significant in a statistical sense, they are fairly small in magnitude, generally ranging between about a half and a full standard deviation. In addition, these characteristics are highly correlated ($r = .53$), indicating that they frequently occur together in the same person. This finding should therefore not be regarded as an indication of any specificity regarding the relationship between anger-out and impatience/irritability and these two clinical manifes-

tations of acute CHD. Rather, both appear to be important as factors associated with acute CHD. Previous studies reporting specificity in the relationship between psychological factors and various manifestations of CHD have typically contrasted MI and diagnosis of angina based on symptoms (Ostfeld *et al.,* 1964; Jenkins *et al.,* 1978). The latter two CHD end points may very well differ more in terms of their psychological precursors than the two types of acute ischemic events evaluated in this study.

The results suggest that in a low-SES population, impatience/irritability may be an important, and thus far largely overlooked, element of the coronary-prone aspects of the anger/hostility complex. It could be speculated that anger-out and impatience/irritability constitute different, although related, facets of anger expression. Inspection of the scale items suggests that anger-out represent more explosive and openly aggressive behavior (e.g., "slamming doors," "saying nasty things"), while impatience/irritability may well reflect the less aggressive, but milder and more commonly occurring behavioral manifestations of anger. This raises the possibility that over long periods of time, frequent, but less severe behavioral expressions of anger may be as harmful to the cardiovascular system as more aggressive forms of anger expression are.

Differences in self-reports of impatience/irritability and anger-out between CHD patients and controls may evidently have been due to recall bias, which, on the basis of the design of this study, cannot be ruled out. There is no obvious reason, however, why impatience/irritability and anger-out are more likely to be subject to recall-bias than, for example, trait-anger, for which no differences were found between cases and controls (unless in reality, trait-anger is a protective factor, which should have been lower among cases). Second, anger-out and impatience/irritability may have increased as a result of the disease, rather than precede onset of the disease. The retrospective design does not permit to address this issue adequately. However, adjustment for previous anginal symptoms and manifestations of CHD did not alter the results substantially. On the contrary, excluding patients with previous MI strengthened rather than attenuated the observed associations.

Although trait-anger and anger-in correlated considerably with anger-out and impatience/irritability, they were not related to either CHD outcome in the present study. Despite the reports about a positive relationship between anger-in and severity of CAD (Dembroski *et al.,* 1985; MacDougall *et al.,* 1985), this finding is consistent with the lack of a prospective relationship with CHD (Dembroski *et al.,* 1989). This inconsistency has been explained by the suggestion that anger-in may increase as a consequence of the disease rather than being elevated prior to onset of symptoms. This could be the result of the recommendation to CHD patients to avoid situa-

tions that may provoke anginal attacks, which would lead them to keep angry feelings inside rather than to express them. Therefore, anger-in may show a positive correlation with coronary atherosclerosis, but is not elevated at baseline of a prospective study (Dembroski and Costa, 1987). The present finding, however, is not congruent with that explanation, since anger-in was assessed when the disease had already progressed to an advanced stage, and manifested itself in an acute ischemic event. It seems hardly plausible to suggest that anger-in is related only to the underlying atherosclerotic disease process, and not to the acute clinical manifestation of the disease that results from that process.

Trait-anger was not related to acute CHD, which corroborates previous findings about the lack of an association between the disposition to experience anger and CHD outcomes (Smith *et al.,* 1984; Shocken *et al.,* 1985; Tennant *et al.,* 1987; Siegman *et al.,* 1987). In addition, trait-anger and anger-in are considered main components of neurotic hostility, which is the type of hostility presumed to be unrelated to CHD (Siegman *et al.,* 1987a; Musante *et al.,* 1989; Costa *et al.,* 1989).

It should be noted that these results are limited to white, low-SES, male patients. The study also included some Black and Hispanic patients, but the number was too small for doing separate analyses on these groups. Inspection of their responses, however, revealed that Black patients reported much *lower* levels of trait anger, anger expression, and impatience/irritability, but not for anger-in, which was similar to the other patients. If Blacks do experience anger, they seem more likely to keep their anger inside, rather than express it overtly. Hispanic patients, on the other hand, were similar to the Whites, except that those with MI had extremely low scores on the anger scales, while those with unstable angina had extremely high trait-anger levels. These results are based on very few patients and, therefore, should be interpreted with caution. However, they demonstrate the importance of examining the influence of ethnic background on the expression of anger and of studying its relationship with CHD for each ethnic group separately.

The findings suggest that there is similarity in the behavioral factors that are associated with acute manifestations of CHD between low- and high-SES while men: the recently advocated difference in coronary-prone relevance between anger expression and anger experience, or, alternatively, antagonistic versus neurotic hostility, also seems to hold up in low-SES populations. Whether anger-out and impatience/irritability have validity as risk factors in these SES populations can, of course, be established only in prospective studies. Such studies, which should include sufficient numbers of persons from various ethnic background to permit stratified analysis, are

clearly needed to elucidate further the role of these behavioral risk factors for CHD in low-SES populations.

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