Linkages Between Facial Expressions of Anger and Transient Myocardial Ischemia in Men With Coronary Artery Disease

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The authors examined whether facial expressions of emotion would predict changes in heart function. One hundred fifteen male patients with coronary artery disease underwent the Type A Structured Interview, during which time measures of transient myocardial ischemia (wall motion abnormality and left ventricular ejection fraction) were obtained. Facial behavior exhibited during the ischemia measurement period was videotaped and later coded by using the Facial Action Coding System (P. Ekman & W. V. Friesen, 1978). Those participants who exhibited ischemia showed significantly more anger expressions and nonenjoyment smiles than nonischemics. Cook–Medley Hostility scores did not vary with ischemic status. The findings have implications for understanding how anger and hostility differentially influence coronary heart disease risk.

If you do not have patience and hatred enters your mind, it is like a poisonous arrow piercing your heart.
—Gampopa, *The Jewel Ornament of Liberation*

Perhaps Gampopa was speaking metaphorically, but his statement not only withstands the test of time—it also holds up to the scrutiny of empirical investigation. Several lines of evidence suggest that anger is toxic to the heart. Hostility, an affective trait that reflects one's predisposition toward anger (Ekman, 1984; Rosenberg, 1998), predicts the incidence of coronary head disease (CHD); Barefoot, Dahlstrom, & Williams, 1983; J. E. Williams et al., 2000), the extent of coronary atherosclerosis (R. B. Williams et al., 1980), the occurrence of myocardial ischemia in daily life (Gullette et al., 1997), and blood pressure reactivity to anger-provocative situations (Suarez, Harlan, Peoples, & Williams, 1993; Suarez & Williams, 1989). Hostility scores are also associated with increases in plasma lipids and lipoproteins (Suarez, Bates, & Harralson, 1998).

Furthermore, the affective state of anger confers its own risks on CHD. In addition to its well-documented effects on blood pressure (Ax, 1958; Schuler & O'Brien, 1997; Siegman, Anderson, Herbst, Boyle, & Wilkinson, 1992), anger is linked with dangerous cardiovascular consequences, especially for people who already have coronary artery disease. Participation in an anger-recall task reduces left ventricular ejection in coronary patients relative to healthy controls (Ironson

et al., 1992). Self-reported anger in daily life is associated with myocardial ischemia in coronary patients (Gabbay et al., 1996). Also, anger outbursts precede a myocardial infarction in people with coronary artery disease more often than other kinds of stress (Mittleman et al., 1995). The 2-hr period before infarction is particularly hazardous (Verrier & Mittleman, 1996). Other research indicates that the risk of myocardial infarction may increase significantly for the 1 hr following an anger outburst (Moller et al., 1999).

These recent findings are important contributions to the understanding of the causal relationship between anger and CHD, but they raise questions as well. Some unresolved issues concern methodological limitations of the extant research. Most studies of how anger relates to coronary outcomes measure anger through self-reports, even in cases in which anger is elicited in the laboratory. At best, self-reports give us a glimpse of what is being experienced consciously at a particular point in time. At worst it suffers from memory bias (Thomas & Diener, 1990), demand characteristics, or beliefs about what people think they should be feeling (Nisbett & Wilson, 1977). Unless they are obtained on-line, self-reports do not reveal dynamic information about emotion (cf. Rosenberg & Ekman, 1994, 2000). One thing we do not know about anger and heart disease is this: If one observes people's emotional behavior *as it happens,* can one detect corresponding changes in coronary function? Indeed, Ironson et al. (1992) found that coronary patients who participated in an anger-recall task showed decreases in left ventricular ejection fraction (LVEF) compared with control participants, but there was no correlation between self-reports of anger and ejection fraction for the same period. Clearly anger can be implicated as a causal agent, but we need better measures of emotion to ascertain whether angry states actually cause coronary dysfunction.

Overview of Empirical Strategy

We examined whether spontaneous facial expressions of emotion could be linked with the incidence of transient myocardial ischemia in male coronary patients during the Type A Structured Interview (Rosenman et al., 1964). Transient myocardial ischemia (hereafter referred to as *ischemia*) is a clinical manifestation of CHD in which there is an insufficient blood supply to the muscle of the heart. Such ischemic events are often fleeting and painless, but they are predictive of serious and/or fatal coronary outcomes (Gottlieb, Gottlieb, & Achuff, 1988). Ischemia

can be triggered by laboratory and daily life stressors (Blumenthal et al., 1995; Gullette et al., 1997; Krantz et al., 1991; Rozanski et al., 1988) and is associated with increased hostility scores (Gullette et al., 1997; Helmers et al., 1993) and self-reports of anger (Gabbay et al., 1996). Taken together, these findings suggest that ischemia is an emotion-sensitive measure.

There were two main aims to this study. First, given the importance of determining whether dynamic indicators of emotion correlate with coronary changes, we examined linkages between spontaneous facial expressions of emotion and ischemia. Previous research has shown that facial expressions can be used to distinguish between Type A and B individuals (Chesney, Ekman, Friesen, Black, & Hecker, 1990; Contrada, Hilton, & Glass, 1991), but so far measures of coronary dysfunction have not been examined in relation to facial expressions. Our first hypothesis was that participants who showed evidence of ischemia during the Type A Structured Interview would show more anger expressions than nonischemics, an obvious prediction given the surmounting case against that emotion.

Second, this study offered the opportunity to examine the relative contributions of hostility and anger to a CHD-relevant outcome. The CHD risks of hostility may result directly from hostility's effects on the cardiovascular system (cf. Krantz & Durel, 1983) or indirectly from its facilitation of angry emotional states. Still others argue that hostility does not create CHD risk by enhancing anger-related cardiovascular reactivity but instead as a function of the relationship between hostility to anger inhibition, which is associated with low parasympathetic activity (low vagal tone; Brosschot & Thayer, 1998). The first step toward separating the state and trait influences on CHD is to look at how hostility and anger differentially relate to a coronary outcome. Thus, we examined the unique contributions of Cook–Medley Hostility (Ho) scores (Cook & Medley, 1954) and anger to variance in ischemia and hypothesized that facial expressions of anger would account for more variability in ischemia than Ho scores (Hypothesis 2). Our reasoning here was twofold: First, we were studying a transient coronary event, linkages with which would be more likely for a transient affective change than a stable affective trait. Second, as the Ho scale does not seem to measure dispositional anger but rather cynicism (Costa, Zonderman, McCrae, & Williams, 1986; Smith & Frohm, 1985), it was not predicted to vary as much with ischemia.

Method

Participants

The participants in this study were involved in a larger study of behavioral influences on ischemia conducted at the Duke University Medical Center, results of which have been reported elsewhere (Blumenthal et al., 1995). A total of 132 people originally participated in the study (117 men, 15 women), age 36 to 74 years ($M = 58.50$, $SD = 8.40$). All participants had documented coronary disease, as evidenced by prior myocardial infarction, coronary artery bypass graft, percutaneous transluminal coronary angiography, or significant coronary atherosclerosis determined by cardiac catheterization. All participants were withdrawn from β-blockers, calcium channel blockers, and long-acting nitrates at least 48 hr before testing.

As there were few women in the full sample, they were omitted from analyses. The final sample of participants who completed the Structured Interview consisted of 115 men. One participant did not complete the hostility questionnaire, so for analyses including hostility the total sample was 114. Demographic and clinical characteristics of the sample appear in Table 1.

Procedure

Although the larger study context involved several phases of assessment and intervention, the present study examined ischemia measurements and facial expressions of emotion from the preintervention Type A Videotaped Structured Interview (VSI). Measures of ischemia (described below) were obtained over a 2-min period of the VSI, which began with the first interview question relevant to anger. As in previous research (Blumenthal, O'Toole, & Haney, 1984), we used two alternate forms of the VSI, which contain different sets of questions that are very similar in semantic content. In Form A, the first question relevant to anger was "When you get angry or upset, do people around you know it? How do you show it?"; and in Form B, the initial anger question was "Most people have pet peeves. What sort of things aggravate you most?" Participants' faces were videotaped throughout the VSI, and they completed the Cook–Medley Hostility Scale at a separate session.

Ischemia Measurement

Ischemia can be reflected in disturbances in the contraction of the left ventricular wall (wall motion abnormality) and/or changes in the amount of blood pumped out of the left ventricle (LVEF). Measures of wall motion abnormality and LVEF were obtained by

Table 1
Demographic and Clinical Characteristics of Sample

Characteristic	Ischemia status during VSI	
	Yes ($n = 42$)	No ($n = 73$)
Age (years)	58.67 (8.18)	59.05 (8.28)
Ethnicity (frequency)		
Black	0	2
White	42	69
Other	0	1
Unknown	0	1
Education (frequency)		
High school	16	19
Some college	13	23
4-year degree	8	11
Graduate work	5	13
Unknown	0	7
HR (bpm)	75.33 (12.68)†	71.39 (10.30)
SBP (mmHg)	147.49 (19.34)	141.02 (17.98)
DBP (mmHg)	86.99 (8.40)	84.57 (10.28)
Chol.	214.50 (40.74)	204.52 (37.08)
BMI	28.35 (3.24)	27.58 (3.33)

Note. All physiological variables are baseline measurements; all quantitative measures are means with standard deviations in parentheses. VSI = Videotaped Structured Interview; HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; bpm = beats per minute; Chol. = total cholesterol; BMI = body mass index.
† $p < .10$ (marginally significant), two-tailed. No other tests reached significance.

using radionuclide ventriculography (RNV). The RNV method (for details, see Blumenthal et al., 1995) produced images of the left ventricle. From these images, segmental wall motion disturbance was rated on a severity scale from 1 (*least severe*) to 7 (*most severe*) by a consensus of experienced physicians, all of whom were unaware of the experimental conditions.

Participants were classified as ischemic or nonischemic on the basis of wall motion change *or* LVEF change, as per the following criteria: (a) at least a 1-point deviation from baseline rating on the 1–7 rating scale in one or more of the four wall segments (this procedure has been shown to be reliable and valid in previous research [Brady et al., 1980]) and (b) an LVEF decrease from baseline of more than 5%.

Facial Measurement

We focused our analyses on the aforementioned 2-min portion of the interview when the ischemia measurements were obtained. All observable facial movement during this period was coded from the videotape by using Ekman and Friesen's (1978) Facial Action Coding System (FACS). FACS is a comprehensive, anatomically based coding scheme that de-

scribes all visually distinguishable facial activity in terms of 44 unique action units (AUs), as well as several categories of head and eye positions and movements. In using FACS, coders do not make emotion interpretations; instead they describe all the muscular movement visible in the face in terms of AUs and combinations of AUs.

Intercoder agreement. Two experienced FACS coders completed the scoring for this study. Both coders were unaware of the health status of the patients, and all coding was conducted with the audiotrack turned off. The coders' reliability levels had been previously established against a standard criterion (Ekman & Friesen's own scoring). For this sample, intercoder agreement was calculated by obtaining a ratio of the number of agreements on the AUs for each expression divided by the total number of agreements and disagreements. The mean agreement ratio between coders across a third of the sample was .80 (*SD* = .07).

Interpretation of facial codes. The FACS codes for each expression were submitted to a computer program called the *FACS/EMFACS Emotion dictionary*,[1] which determines whether each facial expression includes core movements that characterize certain facial expressions of emotion. The program's interpretations draw on an empirically and theoretically derived database from Ekman's laboratory and others', and it has been used for the classification of spontaneous facial behavior in previous studies (e.g., Ekman, Davidson, & Friesen, 1990; Rosenberg & Ekman, 1994). The AU-based facial codes were submitted to the dictionary program, which then determined whether each facial expression was emotional or not, and if so, which emotions the codes reflected. The dictionary has algorithms for numerous expressions, including anger, fear, disgust, surprise, happiness, sadness, blends among these emotions, as well as nonemotion categories. Emotion interpretations are done very conservatively. For example, the program checks that a configuration contains all of the core components of anger (the lowering and bringing together of the eyebrows, raising of the eyelid, and the tightening of the muscles in the lips) before labeling it "anger."

Results

Forty-two patients showed evidence of ischemia during the 2-min RNV measurement period of the VSI by either wall motion, LVEF, or both, and were classified as ischemic. Those who showed no evidence of ischemia from either measure were classified

as nonischemic. All analyses below rely on these dichotomous designations of ischemic status.

Standard Risk Factors for CHD

There were no differences between ischemics and nonischemics on standard risk factors such as age, resting blood pressure, total cholesterol, or body mass index. There was a nonsignificant trend for ischemics to have a higher resting heart rate than nonischemics, $t(113) = 1.81, p = .073$, two-tailed. Table 1 provides the descriptive statistics for each of these variables for each ischemic group.

Facial Expression Differences as a Function of Ischemic Status

These analyses examined whether there were differences between ischemics and nonischemics on the types of facial expressions of emotion that they showed during the RNV sampling period of the VSI. A multivariate analysis of variance (MANOVA) was used to test differences between ischemic and nonischemic groups on each of the following facial expression variables, wherein the dependent variable for each category of emotion was the number of expressions of each type: total positive emotion (summation of all positive emotion expressions), total negative emotion (summation of all negative emotion expressions), anger, contempt, disgust, fear, enjoyment smiles, nonenjoyment smiles, and sadness. The overall MANOVA was significant (Hotelling's $T^2 = 2.64, p = .004$). Univariate F tests were then conducted to examine the effects for each of the expression variables that did not violate homogeneity of variance assumptions; for the remainder, unequal variance t tests were used (Algina & Oshima, 1990). As hypothesized, (Hypothesis 1) ischemics showed more anger expressions than nonischemics, $F(1, 113) = 4.27, p = .04$, (see Figure 1). Figure 1 illustrates that ischemics also showed more nonenjoyment smiles (when not speaking) than nonischemics, $t(58.76) = 2.07, p = .04$, which was not predicted. The meaning of this category of smiling behavior is explained in more detail in the Discussion section. The other facial expression categories listed above did not differ significantly between ischemics and

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[1] The FACS/EMFACS Emotion dictionary has evolved into the FACS Affect Interpretation Database (FACSAID). For information contact Paul Ekman, Human Interaction Laboratory-UCSF, 401 Parnassus Avenue, San Francisco, California 94143-0984.
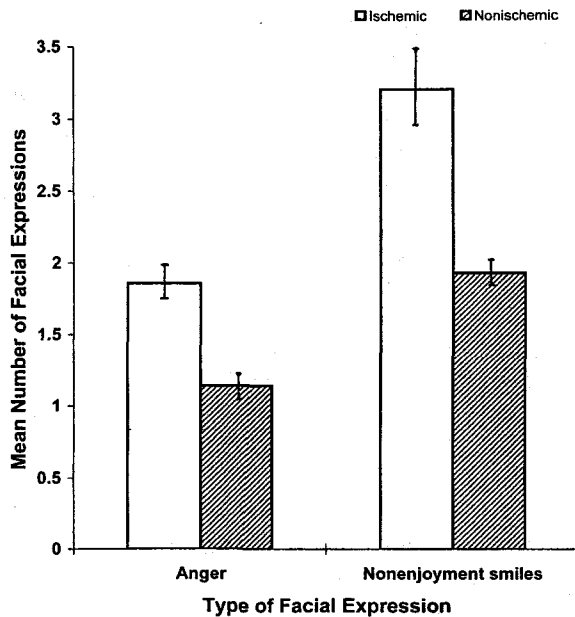
Figure 1. Anger expression and nonenjoyment smiling as a function of ischemic status. Error bars depict ±*SEM*.

nonischemics. Table 2 provides descriptive statistics of emotion expressions that did not vary by ischemic status. It is important to note that ischemics and nonischemics did not differ on overall expressivity, $F(1, 113) = .04$, *ns*, or total number of emotion expressions, $F(1, 113) = .32$, *ns*, which indicates that the specific emotion effects mentioned previously are not merely a function of greater expressivity in one of the patient groups.

The MANOVAs suggest that anger and nonenjoyment smiling were the primary variables that differed between ischemics and nonischemics, but they do not tell us how many people in each group showed those

Table 2

Descriptive Statistics on Facial Expressions That Did Not Vary by Ischemic Status (n = 115)

Variable	M	SD	Mdn	Mode
Total expression	52.10	18.85	51.00	57.00
Total emotion	28.40	13.87	28.00	29.00
Total negative	18.86	11.23	16.00	14.00
Total positive	8.40	7.18	7.00	1.00
Contempt	2.08	3.07	1.00	0.00
Disgust	7.58	7.27	6.00	0.00
Enjoyment smiles	4.59	4.05	4.00	1.00
Fear	0.67	1.27	0.00	0.00
Sadness	4.56	3.95	4.00	0.00

Note. Values reported are based on the number of expressions shown during the 2-min RNV period.

expressions. Of the 42 ischemics, 23 (or 55%) showed at least one anger expression and at least 1 nonenjoyment smile during the RNV period compared with 28 out of 73 (or 38%) of the nonischemics who showed the same behavior. The 17% difference between the number of participants in each group who showed this behavior is marginally significant ($z = 1.71, p = .08$, two-tailed).

Hostility, Expression, and Ischemia

Our second hypothesis predicted that anger would account for more variability in ischemia than Ho scores. Given the dichotomous nature of the ischemia variable, we could not look at proportion of variance accounted for by each type of affective variable (i.e., hostility vs. anger). We could, however, determine whether each variable differed as a function of ischemic status. The significant differences in anger expression between ischemic and nonischemic patients, reported previously, attest to the fact that anger differentiated between ischemics and nonischemics. But what about Ho? Cook–Medley Hostility scores for ischemics ($M = 18.86$, $SD = 8.46$) did not differ from nonischemics ($M = 19.03$, $SD = 7.70$, $t(112) = 0.11$, *ns*, two-tailed, indicating that Ho scores offered no utility in differentiating between groups.

The relationships between Ho scores and facial expression variables have been reported elsewhere (cf. Rosenberg, Ekman, & Blumenthal, 1998). Briefly, the authors' 1998 article reported that only facial expressions of contempt were related to Ho scores ($r = 20$, $p = .03, N = 116$). Facial expressions of anger were not ($r = -.07, N = 116$), nor were any other facial expressions of emotion.

Discussion

Facial expressions of emotion differentiated between those male coronary patients who showed ischemia and those who did not. As hypothesized, ischemic men were more likely than nonischemic men to show anger. To our knowledge this study is the first to show dynamic linkages between facial expressions of anger and a clinically significant measure of coronary dysfunction.

Implications for the Study of CHD

Our results on spontaneous emotional behavior complement recent findings on how self-reported anger episodes trigger ischemia and myocardial infarction (Gabbay et al., 1996; Ironson et al., 1992; Mittleman et al., 1995; Moller et al., 1999; Verrier &

Mittleman, 1996). Given that it is difficult to determine whether retrospectively reported anger indeed triggers coronary events, the temporal linkages between affective behavior and ischemia demonstrated here contribute an important element to the story of anger and heart disease. Although our data only demonstrated *linkages* between anger and ischemia, it seems most likely that anger caused the ischemia rather than vice versa. Ischemia is often silent (i.e., unfelt); thus, it is likely that the ischemic event would cause an emotion or a facial expression of emotion. Indeed, retrospective subjective ratings of pain taken at the end of the VSI did not differ between ischemics and nonischemics, $t(110) = .33$, *ns*, but given the limitations on retrospective reporting one cannot be certain that momentary pain did not relate to ischemic events during the 2-min measurement period.

Our findings also contribute to the understanding of the differential influences of hostility and anger on CHD outcomes. As predicted, hostility did not account for variance in ischemia, even though anger expression did. How can these null findings be reconciled with the numerous positive findings on hostility and CHD outcomes? First, one must keep in mind that these null findings on hostility may be due to problems with the Ho scale. Several studies suggest that the Cook–Medley measures a cynical form of hostility, one that is more characterized by distrust, resentment, and paranoia than by anger (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Blumenthal, Barefoot, Burg, & Williams, 1987; Smith & Frohm, 1985). In fact, contempt expression appears to predict Cook–Medley Hostility scores better than anger expression (Rosenberg et al., 1998). Perhaps a more anger-relevant hostility measure would have shown a relationship with ischemia.

Another possible explanation for the failure of Ho to relate to ischemic status is that it is problematic to relate a trait measure of affect to a transient change in cardiac function. It would make sense that state-level affect is more likely to reveal a transient physiological change than is trait-level affect. Aggregating several ischemia measurement intervals over a longer period may reveal a relationship between hostility and ischemia, as indeed other research has indicated (Helmers et al., 1993).

An important conceptual question mentioned earlier is whether hostility acts on CHD by increasing susceptibility to anger elicitation, or more directly, without anger mediation. Certainly our findings bring something to bear on this issue—in that anger expression related to ischemia and hostility did not—but this study was not the most powerful test of the question. Ideally, we would have tested the differential contributions of hostility and anger to ischemia through a mediational model, wherein the contribution of hostility, independently of anger (a direct effect), could be examined with respect to the indirect effect of hostility through the mediating variable of anger. The statistical approach to testing such mediational effects laid out by Baron and Kenny (1986), however, requires that the independent variable (in this case hostility) be related to the dependent variable (ischemia) and the proposed mediator (in this case, anger). Because there was no relationship between anger and hostility, we could not test this model. Elsewhere we have discussed the implications of a lack of a relationship between Ho scores and anger expression for the construct validity of Ho (Rosenberg et al., 1998). A more valid measure of hostility may have been related to both anger and ischemia, allowing for a more precise test of direct versus indirect paths linking hostility to ischemia.

The question can be raised as to whether the anger expression shown in a 2-min measurement period might reflect an angry state, an angry disposition (i.e., hostility), or both.[2] One would expect, however, that if men who showed more anger were characteristically more angry people, then they would have had higher Ho scores. They did not. Maybe this is a function of problems with the construct validity of Ho. That said, frequency of anger expression can be used as a proxy for hostility or anger on trait level, but one really must examine behavior in multiple contexts to test such trait-level generalizations. A 2-min sampling period in one context is simply too short to support such a conclusion.

The findings on nonenjoyment smiling were not predicted and require explanation. All smiles involve the contraction of *zygomatic major*, the muscle that raises the lip corners. Smiles that accompany self-reports of enjoyment also include the contraction of *orbicularis oculi*, a band of muscles that encircles the eye, which creates crow's feet and lifts the cheeks when contracted (Ekman et al., 1990). Nonenjoyment smiles do not involve the contraction of *orbicularis oculi*, nor do they relate to measures of felt positive emotion (Ekman et al., 1990). Nonenjoyment smiles have several possible functions, including but not lim-

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[2] This interesting point was raised by an anonymous reviewer.

ited to efforts to be polite, to mislead, to modulate an expression of another feeling, or to falsify a message already sent (Ekman & Friesen, 1975, 1982). It is very important to note that the nonenjoyment smiles exhibited here involved only *zygomatic major*—they showed no additional facial actions that might be indicative of sarcasm, derision, or masked negative affect.

So, what is a possible explanation for the smiling results? Most of the ischemics showed both anger and nonenjoyment smiling within the 2-min ischemia measurement period. Thus, this facial expression pattern may reveal anger as well as attempts at managing or inhibiting anger. This interpretation would be consistent with findings that "anger-in," or the inability or unwillingness to verbally express anger, may be a risk factor for CHD (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; MacDougall, Dembroski, Dimsdale, & Hackett, 1985). These interpretations are speculative, however, and further research may determine the meaning of anger and nonenjoyment smiling in people who show ischemia during the VSI.

Implications for the Study of Emotions

Our findings inform controversial issues in emotion research as well. For one, the literature on emotion is replete with examples of how facial expression of anger are linked with cardiovascular physiology, primarily in terms of sympathetic nervous system changes (e.g., Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, & Friesen, 1990). There is very little research on the direct implications of angry facial expressions for clinically significant coronary changes, which this report provides. Also, there is the debate regarding specificity versus generality of emotion-related physiology (cf. Ekman et al., 1983; Lang, 1994; Prkachin, Williams-Avery, Zwaal, & Mills, 1999). By examining specific categories of emotion, we were able to uncover differences between ischemics and nonischemics that would not have been apparent if we had examined expression on simple positive versus negative dimensions. There were no differences between groups on global positive versus negative emotion expression categories; the differences emerged only with respect to a specific negative emotion (anger) and a specific type of smile. It is not that other negative or positive emotions were not elicited; indeed, contempt and disgust occurred at higher frequencies than anger, and enjoyment smiles occurred frequently (see Table 2). These other types of emotion expressions simply did not relate to ischemia.

Limitations and Issues for Further Research

Although a range of emotional expressions were elicited in this brief sample of the VSI, it would be interesting to see what would happen in samples of behavior that were not only longer in duration but were also elicited from more emotionally evocative and more direct anger-provocative contexts (e.g., Suarez & Williams [1989] harassment paradigm). Examining expression and ischemia data obtained from multiple contexts would greatly inform our understanding of the role of anger in coronary disease and would allow for a more valid test of the role of state versus trait affect in CHD. A dynamic measure of ischemia—rather than a dichotomous grouping variable—would also allow for a more powerful test of anger–CHD relations. Practical limitations of this study also precluded more extensive subjective measures of emotion, which would have undoubtedly strengthened the research by providing a more complete picture of the emotional responses.

The fact that our sample included only men, most of whom were White, also limits the generalizability of our findings. Future research should examine the relationship between emotion and ischemia in women and minorities, populations that until recently have not received as much attention in the study of CHD risk.

Clearly, much more work is needed that carefully documents the sequential relationships between anger—as measured by behavioral as well as subjective indicators—and coronary outcomes. We hope that a primary contribution of our research will be that it helps demonstrate the utility of bringing methods of emotion research into the domain of health psychology, where emotions have begun to figure prominently in the study of many diseases.

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