Components of the Anger-Hostility Complex and Symptom Reporting in Patients with Coronary Artery Disease: A Multi-Measure Study

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Abstract

Multiple anger and hostility variables were investigated for associations with coronary artery disease (CAD) symptoms and to examine if those relationships were different for disease severity. A two year follow-up study of 97 men with stenosed coronary arteries was undertaken. Questionnaires measured: nine forms of anger and hostility; Type A behaviour; anxiety; depression; social support; and nine symptom measures. CAD severity was derived from clinicians' ratings of coronary angiograms. Results are fourfold: anger-hostility variables are relatively unimportant predictors of symptoms compared with anxiety and depression; psychosocial measures (except for expressed anger) are uncorrelated with CAD severity, though correlate numerously with CAD symptoms; symptoms are not distinguishable empirically in terms of frequency, intensity and duration with regard to type ('angina pain', 'tiredness' and 'breathlessness and restricted mobility'); finally, CAD symptoms are unrelated to CAD severity. In conclusion, components of the anger-hostility complex are of limited use for predicting CAD symptoms. However, anger expression is of utility for differentiating between CAD symptoms and disease severity.

Keywords

anger, coronary artery disease, hostility, symptom reporting

OVER THE last two decades, social science and medical literatures have extolled the virtues of examining dimensions of key psychosocial variables in relation to the pathogenesis of coronary heart disease (Booth-Kewley & Friedman, 1987; Rozanski, Blumenthal, & Kaplan, 1999). In particular, there has been increasing interest in the potential role played by anger and hostility variables (Chesney & Rosenman, 1985; Dembroski & Costa, 1987; Miller, Smith, Turner, Guijarro, & Hallett, 1996; Siegman & Smith, 1994; Williams & Barefoot, 1988) and how their manifold forms may differentially contribute to the disease process (McDermott, Ramsay, & Bray, 2001; Siegman, Dembroski, & Ringel, 1987). While there is still some debate as to which component of the 'anger-hostility complex' (Williams & Barefoot, 1988) is the most coronary toxic, many investigators now believe that it is frequently and openly expressed anger that confers the highest risk for coronary artery disease (CAD) (Booth-Kewley & Friedman, 1987; Helmig, Houston, Vavak, & Mullin, 1991; Keinan, Ben-Zur, Zilka, & Carel, 1992; McDermott et al., 2001; Mendes de Leon, 1992; Miller et al., 1996). Less clear to date, however, is to what extent the different forms of anger and hostility are related to the symptoms experienced by CAD patients and whether the anger-hostility correlates of objective measures of CAD are different to those associated with its symptomatology.

One possibility is that the correlations as found between anger-hostility variables and CAD parallel those found between anger-hostility variables and symptoms. That this may be so is indicated by research conducted with other psychosocial CAD risk factors which suggests that some of these may serve a dual role and are involved in both the aetiology of CAD pathology and the experience of symptoms. Anxiety, for example, has been found to correlate positively both with the occurrence of heart disease and symptom reporting (Kubzansky & Kawachi, 2000; Russek, King, Russek, & Russek, 1990; Sullivan, LaCroix, Spertus, & Hecht, 2000). Other studies, however, indicate that the correlations found between anger-hostility variables and CAD symptoms may not reflect those found for disease.

In this vein, firstly, a number of studies show that there is no positive relationship between CAD severity and the experience of CAD-related symptomatology. This has been observed amongst patients with confirmed atherosclerosis (Jenkins, Stanton, Klein, Savageau, & Harkern, 1983; Krittayaphong, Light, Golden, Finkel, & Sheps, 1996; Marraccini, Levantesi, Michelassi, Dalle Vacche, & L'Abbate, 1989; Smith, Follick, & Korr, 1984) and amongst angiographic patients subsequently found to have no identifiable disease (Costa, Zonderman, Engel, Baile, Brimlow, & Brinker, 1985; Elias & Robbins, 1987; Elias, Robbins, Blow, Rice, & Edgecomb, 1982). Consequently, given this lack of relationship, it is possible that the anger-hostility correlates of symptoms and disease are dissimilar. Secondly, some psychosocial variables would appear to be differentially related to CAD and its symptoms. For example, whilst neuroticism is a positive correlate of self-reported angina pain (Elias, Robins, Blow, Rice, & Edgecomb, 1982; Razin, 1984), it is not associated with either heightened cardiovascular reactivity or with the development of CAD (Felsten & Leitten, 1993). Similarly, extraversion has been shown to co-vary with increased rates of 'complaint' behaviour, but it has not been found to be a risk factor for CAD or for heightened blood pressure (Bass, 1984; Bass & Wade, 1984; Burke, Beilin, German, Grosskopf, Ritchie, Puddey, & Rogers, 1992). From these studies, then, it is evident that the psychosocial correlates of CAD symptoms may not parallel those of disease and that specific constituents of the anger-hostility complex are likely to co-vary differentially with CAD and self-reported CAD symptoms. However, such issues have yet to be investigated systematically. To date, few investigators examining the psychosocial correlates of CAD symptoms have incorporated more than two or three measures of anger and hostility in one study.

In addition to the omission of manifold measures of anger and hostility, it has often been the case that multiple indices of CAD symptomatology likewise are not included—with the most clinically conspicuous symptom of CAD, angina pectoris, being a focus to the exclusion of others. Whilst chest pain is common in CAD and also associated with increased mortality (Lampe, Whincup, Wannamethee, Shaper, Walker, & Ebrahim, 2000; Rosengren, Wilhelmsen, Hagman, & Wedel, 1998), its significance as a hallmark symptom is overestimated in some

cases (Chambers & Bass, 1998). Categorizing patients on the basis of this symptom has overshadowed consideration of others such as respiratory difficulties, tiredness and problems with physical exertion. These latter symptoms are significant clinical indicators of functional ability and are also associated with an increased risk of suffering a major ischaemic event, such as a myocardial infarction (MI) or sudden cardiac death (Cook & Shaper, 1988; Fraser, 1978; Kop, Appels, Mendes de Leon, de Swart, & Bar, 1994). As such then, all of these symptoms should be incorporated into associated empirical research.

An issue allied to that of measuring CAD symptoms comprehensively is the question of whether such symptoms should be disaggregated into their constituent dimensions in terms of their frequency, intensity and duration. A few studies do make these distinctions at the operational level. Krittyaphong et al. (1996), for example, found a positive correlation between depression scores and duration of angina and a negative correlation between depression scores and time to the onset of angina pain. Depression was also found by Light, Herbst, Bragdon, Hinderliter, Koch, Davis, & Sheps (1991) to be negatively associated with time to onset of angina pain but positively correlated with greater pain severity and duration. From such studies, it is evident that a component approach to the investigation of CAD symptoms may be as warranted as taking such a stance with reference to the anger-hostility complex.

The lack of empirical investigations of the heterogeneous symptomatic presentation of CAD in relation to multiple indices of anger and hostility suggests that a study which incorporates all of these features is needed. In addition to this rationale as derived from an appraisal of the conceptual and methodological shortcomings of previous research, there are also sound clinical reasons why carrying out such work is important. Firstly, a systematic investigation is warranted because, as reported earlier, CAD symptoms are associated with increased mortality (Cook & Shaper, 1988; Fraser, 1978; Kop et al., 1994; Lampe et al., 2000; Rosengren et al., 1998). Further, the quality of life experienced by symptomatic CAD patients is often poor (Colin, Allaert, Cohen-Boulakia, Slama, & Bousquet, 1999; Mayou, Blackwood, Bryant, & Garnham, 1991). Consequently, it is essential that a full understanding of the precursors of CAD symptomatology is forthcoming. Secondly, building on research showing that emotional distress is related to symptom severity (Pennebaker, 1982; Bengston, Herlitz, Karlsson, & Hjalmarson, 1996; Verthein & Kohler, 1997), a number of psychosocial intervention studies have now been conducted with the aim of reducing symptoms by improving the emotional health of patients. Utilizing such techniques as stress management and relaxation exercises, these studies have demonstrated that the frequency of angina episodes can indeed be reduced, as can the risk of suffering a MI (Blumenthal, Jiang, Babyak, Krantz, Frid, Coleman, Waugh, Hanson, Appelbaum, O'Connor, & Morris, 1997; Lewin, Cay, Todd, Soryal, Goodfield, Bloomfield, & Elton, 1995; Ornish, Brown, Scherwitz, Billings, Armstrong, Ports, McLanahan, Kirkeeide, Brand, & Gould, 1990; Ornish, Scherwitz, Billings, Gould, Merrit, Sparler, Armstrong, Ports, Kirkeeide, Hogeboom, & Brand, 1998). It is important to establish, then, which specific psychosocial risk factors are associated with angina pain and other CAD symptoms, since such knowledge can be utilized in intervention programmes thereby leading to improved health outcomes and a better quality of life.

The purpose of the study reported here is to address these issues but with particular reference to the utility of components of the anger-hostility complex as predictors of self-reported CAD symptoms. Also examined are four additional psychosocial variables: anxiety, depression, Type A behaviour and social support. Past research has shown these variables to correlate with indices of CAD symptomatology (Channer & Rees, 1987; Haynes, Feinleib, & Eaker, 1982; Lichestein, Pedersen, Plomin, De Faire, & McClean, 1989; Wistow, Wakefield, & Goldsmith 1990). However, no previous research is identifiable within the literature that has investigated their relative roles in relation to multiple CAD symptoms and manifold forms of anger and hostility. By considering components of the anger-hostility complex and other psychosocial variables, a clearer understanding will emerge of the positive and negative, illusory and veridical relationships that exist between such psychosocial variables, CAD symptoms and disease severity. Thereby, the data reported here

investigates the differential associations of anger-hostility components, plus other traditional and psychosocial risk factors, with scores derived from a CAD-specific symptomatology and functional ability questionnaire (Devlen, Michaelson, & McGuire, 1989). The latter is composed of 23 items measuring CAD symptom types (angina, breathlessness, tiredness, and difficulties with physical exertion) and dimensions (frequency, intensity and duration). All symptoms and anger-hostility variables were measured twice, there being a 24 month lag between the two time-points. Also assessed was severity of CAD via review of coronary angiograms by cardiologists and as recorded at the first time point of the study. By employing this design, a multifactorial approach to both the anger-hostility complex and to CAD symptoms was undertaken.

Method

Participants

Patients were recruited utilizing outpatient information held at a hospital in south Manchester, north-west England. Initially, health records were inspected for all patients attending the hospital's Regional Cardiothoracic Unit for cardiac investigation within the previous four months. All male, 30–70 year old, Caucasian patients who had been diagnosed via angiography as having coronary artery disease and reporting symptoms of angina pain were selected for possible inclusion. 150 such patients were identified and subsequently approached by letter from their respective consultants.

At Time I, 121 of the patients responded (81%) and agreed to participate. At Time II, two years later, 97 of these patients (80%) also agreed to participate in the follow-up study. Of the 24 patients who did not take part at Time II, one was known to have died (although no data on cause of death was available), and three questionnaires were returned by the postal services unopened (the addressees no longer being at their Time I address). The remaining 20 patients either did not accept or respond to the postal invitation to take part in the follow-up study.

It is the 97 patients who participated at both Times I and II who are the focus of this paper. These patients resided in both urban and rural communities throughout the geographical area served by the Regional Cardiothoracic Unit. At the time of initial recruitment, their ages ranged from 30–67 years (mean 55 years, SD 7 years). Most patients had been or were employed in manual occupations (54%). Of the remainder, 34% had been or were engaged in non-manual skilled or intermediate occupations, and 12% had been or were employed in the professions. Ninety one percent of the sample were married or co-habiting, the remainder being either separated or divorced (6%), widowed (1%), or single (2%).

Measures

An author-devised questionnaire booklet elicited the following information: (a) the patient's *recent medical history*—including screening questions about angina pain experienced over the preceding six months, whether the patient had undergone heart bypass surgery, any incidence of a heart attack or stroke, cholesterol status, personal history of diabetes, history of hypertension, and what medication (if any) was being taken for angina symptoms and/or high blood pressure; (b) *family history of coronary heart disease* (how many and who of their close relatives had died from CHD); (c) *cigarette smoking behaviour* (both past and present); (d) *alcohol consumption* (at Time II only); and (e) *demographic information*—including questions about age, occupational status, employment history, marital status, weight, height and, in addition at Time II, waist and hip measurements.

Participants also were asked to respond to the following standardized questionnaires at Times I and II. The *Quality of Life Questionnaire* (Devlen et al., 1989) examined for the preceding fortnight prior to test administration the *frequency*, *intensity* and *duration of angina pain*, the *frequency* and *intensity of shortness of breath*, *daytime sleeping* and the *intensity of tiredness*, and *difficulties with walking* and *climbing stairs*. A 42 item *Anger-Hostility Complex Questionnaire* authored by McDermott & Beech (1989) and comprising of two factors (anger-out, cynical hostility) was administered at each time point. Facets of the anger-hostility complex were also measured by other psychometrically valid and reliable questionnaires, namely: the *Anger Experience* (14 items) and the *Anger Expression*

(nine items) subscales of Costa, McCrae, & Dembroski (1989); the *Neurotic Hostility* (8 items) and *Neurotic Disagreeableness* (17 items) subscales of Costa & McCrae (1985); the 50 item '*Ho' Hostility Scale* as derived from the Minnesota Multiphasic Personality Inventory (Cook & Medley, 1954); and the *Resentment* (8 items) and *Suspiciousness* (10 items) subscales in the Buss-Durkee Hostility-Guilt Inventory (Buss & Durkee, 1957).

At Time II, additionally, four further psychosocial measures were administered: the 20 item *Trait Anxiety Scale* (Spielberger, Gorsuch, & Lushene, 1974); the 10 item *Framingham Type A Questionnaire* (Haynes, Levine, Scotch, Feinleib, & Kannel, 1978); the 21 item *Beck Depression Inventory* (Burns & Beck, 1978); and the six item *Social Support Scale* (Sarason, Sarason, Shearin, & Pierce, 1987) which examines the number of available social supports and perceived support satisfaction.

Objective angiographic information concerning each CAD patient's disease status was collected at Time I. Angiography is a diagnostic procedure where cardiac catheterisation is performed and an X-ray opaque liquid is injected into the heart chambers. A short X-ray film of the functioning coronary arteries is then recorded. Diseased arteries which cause ischaemia (lack of oxygenated blood to the heart muscle) show up on the film as narrowed (stenosed) and irregular in circumference along their length. The senior cardiologist working at the Cardiothoracic Unit reviewed all patients' coronary angiograms blind. After a scoring system developed by Ramsdale (1981), coronary artery maps were used to indicate the site, length and the extent of any coronary artery narrowing, severity being determined by the percentage of the artery occluded across its diameter. All angiograms were scored for degree of stenosis in both the left and right coronary arteries, thereby giving a 'total disease severity' score for each patient. A second cardiologist blindly and independently reviewed and re-rated a random subsample of the angiograms (n = 16). There was a high degree of inter-rater reliability between the two cardiologists (r = .79, p <.005 for a one-tailed test).

Follow-up angiographic data from patients at Time II was not collected. Angiography is used either as a diagnostic tool or as an essential component of a preoperative assessment. Thus, it was not justifiable for such an invasive procedure to be carried out on a second occasion. Moreover, it is known that the progression of arterial disease is gradual (Pearson, 1984; Shaper, 1988), so it was not expected that there would be significant differences in disease severity between Time I and Time II. In all following statistical analyses, then, disease severity measures are based on Time I scores only.

Procedure

All patients who participated at Time I and Time II were contacted by post. On both occasions an invitation letter from their consultant, a consent form, and a return stamped addressed envelope were enclosed. Patients were informed that the survey was examining heart disorder and personality in order to help further medical understanding of the causes of cardiovascular disease. They also were informed that their participation was entirely voluntary and that electing not to take part would not affect their future treatment or their relationship with their hospital consultant or other doctors involved in their treatment. All patients were assured that their answers would be treated confidentially.

Results

Non-respondents at Time II follow-up

Demographic comparisons between the 97 Time II participants and the 20 who either did not accept or respond to the postal invitation to take part at follow up, showed that the two groups did not differ in respect of age, marital status or socio-economic status. Further, patients declining to take part at follow-up did not differ significantly from Time II participants in terms of either disease severity or CHD symptomatology as measured at Time I. However, a between-groups difference was observed in relation to one of the risk factor measures. By comparison with patients who did take part at follow-up, non-respondents were found at Time I to have higher body mass (t = –2.52, p = .02).

Test-retest correlations

Test-retest correlations for all nine self-reported symptoms suggested that the patients' symptomatology remained relatively stable over the two

year time period. Seven of the nine correlations were highly statistically significant (p <.001), whilst those for the frequency and intensity of angina pain approached significance (p <.07). However, an inspection of the frequency analyses for these variables shows that some change was evident, particularly with respect to angina pain.

Self-reported frequency, intensity and duration of angina pain decreased from Time I to II. At Time I the modal frequency of angina was 2–3 times weekly, the modal intensity was '*moderate*', and the modal duration of pain was two minutes. At Time II, two-thirds of CAD patients reported no pain within the last fortnight, so nil frequency, intensity and duration. Of those participants who did report angina pectoris at Time II, the modal frequency was 2–3 times per week, with pain of '*mild*' intensity and of two minute duration. For breathlessness, the proportion of participants not experiencing this symptom increased by 17% from Time I (40%) to Time II (57%), though the modal frequency (2–3 times weekly) and intensity ('*not too bad*') of this symptom in whom it was present remained the same at both time points. For tiredness, fewer patients reported feeling the need to sleep during the day (from 75% at Time I to 64% at Time II), although once again the modal intensity of tiredness for these patients remained the same at Times I and II, with patients reporting themselves to be '*moderately tired*'. For difficulties with physical exertion, more patients reported having no difficulties with climbing stairs (75%) at Time II than at Time I (54%), and likewise a greater number of patients reported at Time II (73%) being able to walk a mile at normal or brisk rate than at Time I (46%). These reductions in symptoms were not associated with increased usage of pharmacological treatments, such as vasodilators and beta-blockers. Over the two year period, usage of these treatments decreased by 50% and 61%, respectively.

All of the test-retest correlations for anger-hostility and traditional risk factor variables were statistically significant (p <.001). Some slight variation over time was observed, but means and standard deviations of the variables at Time I and Time II remained fairly consistent. Thus, no significant changes in participants' scores on risk factors were observed.

Symptom intercorrelations & factor analysis

With the exception of some correlations between '*daytime sleeping*' and other symptom items (see Table 1), most of the remaining eight symptoms at Time I and II intercorrelated at statistically significant levels of probability coefficients ranging from .19 to .89 (df = 95).

For the purposes of investigating the underlying dimensionality of symptoms, separate scree analyses (Cattell, 1966) for the Time I and Time II symptom data were carried out. Each suggested a two factor solution. An inspection of the residual correlation matrices for two and three factor solutions, however, indicated that the latter accounted for a greater amount of unexplained variance. A three factor solution reduced the percentage of residual correlation coefficients which were greater than .05 by 11% at Time I and by 22% at Time II. Additionally, a three factor solution was more readily interpretable, with a greater number of items loading discretely onto single factors at above a .3 threshold value. Accordingly, principal components analyses were computed for Time I and Time II symptoms, each specifying the extraction of three factors with varimax rotation.

These analyses were found to group items together in accordance with symptom type, not in accordance with the symptom dimensions of frequency, intensity and duration (see Table 2). Symptoms loading on Factor 1, labelled '*breathlessness and restricted mobility*', were breathlessness frequency and intensity, difficulty climbing stairs and distance walked outside the home. Angina frequency, intensity and duration all loaded on Factor 2 which therefore was labelled '*angina pain*'. Finally, tiredness intensity and daytime sleeping loaded on Factor 3 which was labelled '*tiredness*'. Coefficients of congruence for the three factors (which estimate the degree of similarity between Time I and Time II solutions) ranged from .8–.98, indicating an acceptable level of similarity (Tucker, 1951). Composite variables based on the pattern of substantive loadings (as underlined in Table 2) then were computed to form the three symptom dependent variables for all subsequent analyses.

Bivariate correlations between the three TI symptom factors (wherein the majority of CAD patients were reporting various degrees of angina pain, unlike at Time II), ranged between

Table 1. Bivariate correlations between nine CAD symptoms within Time I and Time II measures

	Angina intensity		*Angina duration*		*Breathlessness frequency*		*Breathlessness intensity*		*Tiredness intensity*		*Daytime sleeping*		*Distance walked*		*Difficulty climbing stairs*	
Symptoms	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*
Angina frequency	.73 (<.001)	.78 (<.001)	.54 (<.001)	.72 (<.001)	.37 (<.001)	.34 (<.001)	.39 (<.001)	.34 (<.001)	.42 (<.001)	.28 (= .003)	.20 (= .023)	-.02 (= .406)	.39 (<.001)	.41 (<.001)	.26 (= .005)	.31 (= .001)
Angina intensity			.66 (<.001)	.88 (<.001)	.42 (<.001)	.36 (<.001)	.48 (<.001)	42 (<.001)	.51 (<.001)	.28 (= .003)	.32 (= .001)	-.09 (= .198)	.54 (<.001)	.44 (<.001)	.43 (<.001)	.37 (<.001)
Angina duration					.31 (= .001)	.39 (<.001)	.32 (= .001)	.45 (<.001)	.48 (<.001)	.25 (= .006)	.19 (= .028)	-.03 (= .385)	.36 (<.001)	.41 (<.001)	.24 (= .009)	.36 (<.001)
Breathlessness frequency							.86 (<.001)	.89 (<.001)	.44 (<.001)	.50 (<.001)	.22 (= .016)	.27 (= .004)	.46 (<.001)	.55 (<.001)	.48 (<.001)	.59 (<.001)
Breathlessness intensity									.50 (<.001)	.51 (<.001)	.25 (= .007)	.24 (= .010)	.53 (<.001)	.55 (<.001)	.45 (<.001)	.59 (<.001)
Tiredness intensity											.34 (<.001)	.24 (= .008)	.53 (<.001)	.40 (<.001)	.38 (<.001)	.38 (<.001)
Daytime sleeping													.32 (= .001)	.10 (= .163)	.19 (= .030)	.16 (= .063)
Distance walked															.27 (<.001)	.35 (<.001)

All probability values (in parentheses) are for 1-tailed tests

Table 2. Rotated factor matrices for nine CAD symptoms at Time I and II

	Time I loadings			*Time II loadings*		
Symptoms	*Factor 1*	*Factor 2*	*Factor 3*	*Factor 1*	*Factor 2*	*Factor 3*
Angina frequency	.18	.85	.07	.24	.85	.07
Angina intensity	.32	.84	.20	.20	.93	.00
Angina duration	.10	.82	.14	.23	.90	.02
Breathlessness frequency	.88	.19	.01	.68	.39	.41
Breathlessness intensity	.86	.25	.07	.65	.39	.39
Tiredness intensity	.45	.44	.49	.34	.27	.60
Daytime sleeping	.04	.13	.89	.00	.15	.85
Distanced walked	.60	.17	.55	.85	.22	.07
Difficulty climbing stairs	.69	.12	.23	.89	.13	.01
Eigen values	4.35	1.30	.96	4.53	1.50	.91
Variance	48%	14.5%	11%	50%	17%	10%

Underlined loadings are interpreted for this factor

.51 (p <.001, n = 97) and .58 (p <.001, n = 97), indicating shared variance from 26%–34%. At Time II, a similar magnitude of correlation coefficients was observed, with the exception of that between angina pain and tiredness (r = .23, p = .01). Thus, while there is empirical overlap between these symptom factors, each also accounts for much unique variance in symptomatology.

Association of psychosocial and traditional cardiac risk factors with symptom factors and disease

In total, for the three symptom factors and nine measures of anger-hostility (see Table 3), 18 (67%) of the 27 correlations at Time I attained statistical significance, whilst at Time II 19 (70%) were found to do so. Moreover, there were 13 instances (48%) wherein anger-hostility variables correlated significantly with the same symptom factors at both time points. At a 5 percent probability level, at each time point only one of the correlations might have been expected by chance to attain statistical significance. The probability of Type I errors occurring, then, arguably was very low and of an acceptable order, particularly wherein replicated relationships were evident.

Firstly, considering Time I relationships only, a number of patterns are observable in the matrix of bivariate correlations. Anger experience, neurotic disagreeableness, 'Ho' hostility and suspiciousness each correlated positively with all three symptom factors. Conversely, anger expression was the only component not to correlate with any symptom factor at all. Lastly, it was only for *breathlessness and restricted mobility* that eight of the nine anger-hostility components were significant correlates, coefficients ranging from .19–.33.

A slightly different pattern of results emerged at Time II. Again, suspiciousness was positively associated with all three symptom factors, but on this occasion neurotic hostility, resentment and anger-out also were significantly correlated with all three. Anger expression still was not associated with any symptom factor. Similarly, once again, *breathlessness and restricted mobility* correlated with the highest number of anger-hostility components. There were eight significant correlations ranging from .27–.43, correlations with suspiciousness and resentment being the largest coefficients obtained for any symptom factor at either time point.

In terms of similarities and differences in the patterning of correlations across the two time points, three features of the symptom data were notable. Firstly, for all three symptom categories, there was replication across the two time points for two anger-hostility components. Suspiciousness correlated significantly with all symptom categories, whilst anger expression was not associated with any. Secondly, those anger-hostility measures which correlated significantly with *angina pain* varied as a function of time. Only suspiciousness continued to have a significant association with *angina pain* at Time I and Time II. Other anger-hostility components

Table 3. Bivariate correlations: three CAD symptom factors and disease with psychosocial cardiac risk factors

	CAD measures						
	Symptom Factor 1 'breathlessness/ restricted mobility'		*Symptom Factor 2 'angina pain'*		*Symptom Factor 3 'tiredness'*		*Disease severity*
Psychosocial risk factors	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*
Anger experience	.33 (p = .001)	.34 (p<.001)	.18 (p = .04)	.12 (p = .13)	.19 (p = .03)	.29 (p = .002)	–.20 (p = .03)
Anger expression	.12 (p = .13)	.05 (p = .32)	–.02 (p = .41)	–.13 (p = .10)	.14 (p = .09)	.04 (p = .35)	.17 (p<.05)
Neurotic hostility	.22 (p = .02)	.31 (p = .001)	.10 (p = .16)	.19 (p = .03)	.12 (p = .13)	.24 (p = .01)	–.01 (p = .48)
Neurotic disagreeableness	.24 (p = .01)	.30 (p = .002)	.20 (p = .02)	.06 (p = .27)	.23 (p = .01)	.02 (p = .43)	.15 (p = .07)
'Ho' hostility	.33 (p<.001)	.27 (p = .004)	.26 (p = .005)	.04 (p = .36)	.23 (p = .01)	.18 (p = .04)	.03 (p = .37)
Resentment	.19 (p = .03)	.43 (p<.001)	.08 (p = .21)	.25 (p = .006)	.26 (p = .004)	.27 (p = .004)	–.04 (p = .34)
Suspiciousness	.33 (p = .001)	.43 (p<.001)	.28 (p = .003)	.21 (p = .02)	.33 (p = .001)	.36 (p<.001)	.03 (p = .37)
Anger-out	.25 (p = .006)	.37 (p<.001)	.16 (p = .06)	.22 (p = .02)	.06 (p = .29)	.31 (p = .001)	–.10 (p = .17)
Cynical hostility	.22 (p = .02)	.38 (p<.001)	.18 (p = .04)	.12 (p = .12)	.13 (p = .10)	.25 (p = .006)	–.03 (p = .40)
Anxiety	N/A	.40 (p<.001)	N/A	.32 (p = .001)	N/A	.41 (p<.001)	N/A
Depression	N/A	.59 (p<.001)	N/A	.23 (p = .01)	N/A	.57 (p<.001)	N/A
Type A	N/A	.03 (p = .37)	N/A	.05 (p = .30)	N/A	.21 (p = .02)	N/A
Social support	N/A	–.26 (p = .005)	N/A	–.14 (p = .09)	N/A	–.14 (p = .09)	N/A
Disease severity	–.01 (p = .46)	N/A	–.02 (p = .43)	N/A	.00 (p = .49)	N/A	N/A

All probability values are for 1-tailed tests; NA, not applicable

correlating with this factor did so only at one of the two time points. Thirdly, it is notable that *breathlessness and restricted mobility* correlated with all but one of the anger-hostility components at both time points. Finally, for CAD severity (measured at Time I only), the sole anger-hostility component to correlate in the predicted direction with this disease measure was anger expression (r = .17).

For the additional psychosocial variables measured at Time II, anxiety and depression scores correlated positively with all of the symptom categories, coefficients for anxiety ranging from .32–.41 and for depression from .23–.59. For both of these psychosocial variables, the smallest coefficient was attained with the symptom factor *angina pain*. Notably, scores on the Framingham Type A scale correlated with only one of the three symptom factors, this being a positive association with *tiredness*, whilst social support likewise correlated with only one factor, this being a negative correlation with *breathlessness and restricted mobility*.

For the traditional cardiac risk factors at Time I (see Table 4), no one measure related to all three symptom categories, though the number of years of diabetes was positively associated with two factors, *angina pain* and *tiredness*. Three other measures each correlated positively with one symptom factor: the number of cigarettes smoked per week correlated with *breathlessness and restricted mobility*, body mass index

Table 4. Bivariate correlations: three CAD symptom factors and disease with traditional cardiac risk factors

	CAD measures						
	Symptom Factor 1 'breathlessness/ restricted mobility'		*Symptom Factor 2 'angina pain'*		*Symptom Factor 3 'tiredness'*		*Disease severity*
Traditional risk factors	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*	*Time II*	*Time I*
Age	.01 (p = .45)	–.03 (p = .38)	.12 (p = .12)	–.12 (p = .11)	–.00 (p = .49)	–.03 (p = .38)	.15 (p = .07)
Body mass index	.12 (p = .11)	.10 (p = .17)	.17 (p<.05)	.04 (p = .33)	.14 (p = .09)	.02 (p = .42)	.11 (p = .15)
Number of years of smoking	.15 (p = .07)	.17 (p = .05)	.03 (p = .38)	.14 (p = .08)	.18 (p = .04)	.17 (p<.05)	.27 (p = .003)
Number of cigarettes smoked per week	.19 (p = .03)	.08 (p = .21)	–.01 (p = .44)	.16 (p = .06)	.03 (p = .37)	.09 (p = .20)	.13 (p = .11)
Number of years of hypertension	.01 (p = .48)	–.03 (p = .38)	.10 (p = .17)	–.03 (p = .34)	.02 (p = .44)	.12 (p = .07)	.03 (p = .40)
Number of family members with CHD	–.02 (p = .43)	–.02 (p = .42)	.07 (p = .25)	.07 (p = .26)	.03 (p = .39)	.01 (p = .45)	.15 (p = .07)
Number of years of diabetes	.13 (p = .09)	–.02 (p = .42)	.22 (p = .02)	–.14 (p = .09)	.20 (p = .02)	.12 (p = .13)	.04 (p = .36)
Number of units of alcohol per week	N/A	–.20 (p = .02)	N/A	–.03 (p = .39)	N/A	–.11 (p = .14)	N/A
Waist to hip ratio	N/A	.09 (p = .19)	N/A	.05 (p = .30)	N/A	.14 (p = .08)	N/A
Disease severity	–.01 (p = .46)	N/A	–.02 (p = .43)	N/A	–.00 (p = .49)	N/A	N/A

All probability values are for 1-tailed tests; NA, not applicable

with *angina pain*, and the number of years of smoking with *tiredness*. At Time II, only one statistically significant correlation in the predicted direction was found: the association between the number of years of smoking and *tiredness*. This latter correlation was the only one replicated over time for any of the traditional risk factor measures. Finally, for CAD severity, only the number of years as a smoker was associated positively with this Time I outcome variable.

Notably, none of the three symptom categories correlated to statistically significant degrees with CAD severity, emphasising that symptoms and disease are not good indicators of each other and should be examined separately as health outcomes.

Regression analyses: psychosocial and traditional cardiac risk factor correlates of symptom categories

Six regression analyses were carried out (see Table 5) to test the uniqueness and strength of the psychosocial and traditional risk factors' associations with *breathlessness and restricted mobility*, *angina pain* and *tiredness* at Time I and Time II. All statistically significant bivariate correlates of the symptom factors were considered eligible for inclusion in the analyses. However, 'redundant' anger-hostility measures were dropped where high multicollinearity (r >.7) among these risk factor measures was identified. Where measures shared high multicollinearity, those having the lowest zero-order correlation with the given symptom factor were judged to be redundant and omitted from that analysis. All analyses were conducted using the standard regression method.

Overall, at Time I, only one risk factor was found to account for statistically significant levels of unique variance, this being the number of years of diabetes. This traditional risk factor explained 4.7% of the unique variance for *angina pain* (p = .03) and 4.1% of the unique variance for *tiredness* (p = .04). None of the

Table 5. Regression analyses of psychosocial and traditional cardiac risk factors onto three composite symptom measures at two time points

DV symptom categories/ IV cardiac risk factors	*Time I* *r*	*Beta*	*t*	*DV symptom categories/ IV cardiac risk factors*	*Time II* *r*	*Beta*	*t*
Breathlessness/restricted mobility (DV)				**Breathlessness/restricted mobility (DV)**			
Anger experience	.33	.26	1.79	Depression	.59	.51	4.23***
Suspiciousness	.33	.23	1.57	Social support	–.26	–.18	–2.04*
'Ho' hostility	.33	.18	1.19	Neurotic disagreeableness	.30	.14	1.08
Neurotic hostility	.22	–.16	–1.05	'Ho' hostility	.27	–.11	–.84
Number of cigarettes smoked weekly	.19	.13	1.33	Cynical hostility	.38	.06	.51
Resentment	.19	–.11	–.80	Suspiciousness	.43	.04	.25
Cynical hostility	.22	–.03	–.26	Anger-out	.37	.02	.17
				Resentment	.43	–.00	–.01
(Redundant variables: anger-out; neurotic disagreeableness)		Adjusted r^2 = .12 f = 2.87**		(Redundant variables: neurotic hostility; anger experience; anxiety)		Adjusted r^2 = .35 f = 7.51***	
Angina pain (DV)				**Angina pain (DV)**			
Number of years of diabetes	.22	.22	2.23*	Anxiety	.32	.30	2.21*
Body mass index	.17	.18	1.72	Anger-out	.22	.04	.27
Suspiciousness	.28	.15	1.15	Suspiciousness	.21	.00	.01
Cynical hostility	.18	.09	.75				
'Ho' hostility	.26	.10	.67				
Anger experience	.18	–.01	–.11				
(Redundant variables: neurotic disagreeableness)		Adjusted r^2 = .10 f = 2.84*		(Redundant variables: neurotic hostility; resentment; depression)		Adjusted r^2 = .08 f = 3.62*	
Tiredness (DV)	.20	.21	2.11*	**Tiredness (DV)**			
Number of years of diabetes	.33	.24	1.62	Depression	.57	.60	5.27***
Suspiciousness	.18	.15	1.49	Resentment	.27	–.25	–1.91
Number of years of smoking	.26	.09	0.69	'Ho' hostility	.18	–.22	–1.65
Resentment	.19	–.06	–0.44	Type A	.21	.15	1.59
Anger experience	.23	.04	0.29	Suspiciousness	.36	.21	1.54
'Ho' hostility				Number of years of smoking	.17	.10	1.11
				Cynical hostility	.25	.08	.69
				Anger-out	.31	.03	.26
(Redundant variables: neurotic disagreeableness)		Adjusted r^2 = .11 f = 3.04**		(Redundant variables: neurotic hostility; anger experience; anxiety)		Adjusted r^2 = .33 f = 7.01***	

*** p<.001 ** p<.01 * p<.05

anger and hostility variables emerged as independent predictors of the three symptom types. Nonetheless, it was noted that suspiciousness was the pre-eminent anger-hostility measure in terms of predicting *angina pain* and *tiredness*. Further, for *breathlessness and restricted mobility*, although the highest percentage of unique variance was explained by anger experience, this was followed by suspiciousness.

For the Time II analyses, once again none of the anger-hostility variables were found to be independent predictors of symptom types. Rather, it was found that the additional psychosocial variables of depression, anxiety and poor social support had independent predictive utility with respect to various symptom factors and that these superseded those for components of the anger-hostility complex. For *breathlessness and restricted mobility*, both depression and social support were independent correlates, with depression explaining 12.1% of the unique variance (p <.001) and social support accounting for 2.8% (p = .04). Depression was also the only independent correlate of *tiredness* and explained 19.3% of the variability in this symptom (p <.001). For *angina pain*, the sole independent correlate was anxiety, with this measure accounting for 4.7% (p = .03) of the unique variance. Notably, on this occasion, suspiciousness was no longer a pre-eminent anger-hostility predictor of any of the three symptom factors.

Discussion

The primary aim of this study was to discriminate and identify those forms of anger and hostility that are associated with the symptoms of CAD. The results show that eight of the nine anger-hostility variables correlated bivariately with scores on at least one CAD symptom factor either at Time I or Time II, the exception being expressed anger. The most notable of the eight anger-hostility variables to correlate with symptoms was suspiciousness. Suspiciousness was the only variable to correlate with all three symptom factors at both Time I and Time II. Moreover, these correlations tended to be more substantive than those observed between the other anger-hostility variables and symptom factors. In multivariate analyses, however, the importance of suspiciousness and other anger-hostility correlates was not confirmed. Multiple regression analyses of the data, wherein components of the anger-hostility complex and additional psychosocial and traditional measures were included, showed that none of the anger-hostility variables independently predicted scores on any of the three symptom factors. For the Time I data, only one statistically significant independent predictor variable emerged, this being the number of years of diabetes. A history of diabetes predicted scores on both *angina pain* and *tiredness*. For the Time II variables, when scores for additional psychosocial measures were available for the first time, three such variables independently predicted symptom scores: depression and an absence of social support predicted *breathlessness and restricted mobility*, depression predicted *tiredness*, and anxiety predicted *angina pain*.

Thus, in comparative terms, the components of the anger-hostility complex are of little direct utility in the prediction of symptoms. Rather, any observed relationships with self-reported CAD symptomatology can be seen as reflecting the degree to which these anger-hostility variables are indirect indices of negative emotional states—specifically, as measured here, indirect indices of depression and anxiety. All eight of the anger-hostility variables that correlated with symptoms also were associated with anxiety and depression. Conversely, expressed anger, the only anger-hostility variable not associated with any of the symptom types, was not a correlate of either anxiety or depression.

The present results, then, support previous research which also shows that heightened emotional distress is associated with the experiencing and reporting of symptoms. For example, in the wider health literature, neuroticism or 'negative affectivity' has been shown to be positively correlated with health complaints (Pennebaker, 1982; Suls & Marco, 1990; Watson & Pennebaker, 1989). Likewise and as reported earlier, in studies specific to ischaemic heart disease, researchers have demonstrated that negative emotions are associated with reported angina pain. For example, Bengston et al. (1996) found that anxiety and depression correlated with the frequency and intensity of chest pain in patients awaiting coronary angiography. Similarly, Verthein & Kohler (1997) demonstrated a relationship between stress and angina pain in CAD patients who were asked to complete weekly diaries for a period of one year.

A second aim of this study has been to investigate whether the psychosocial correlates of objective coronary artery disease are different from those associated with CAD symptoms. From our results, it is evident that there is such a difference and that the patterning of correlations does indeed change as a function of whether clinicians' ratings of angiographically identified CAD severity or self-reported symptom data are considered. Thus, it was found here that measures which correlated with disease severity did not correlate with symptomatology, and vice versa. The only psychosocial variable to correlate bivariately with disease severity was anger expression. Further, in a multiple regression analysis reported separately (McDermott et al., 2001), this variable was found also to be one of only three independent predictors of disease severity scores, the other two predictors each being a 'traditional' CAD risk factor (the number of years as a cigarette smoker and age). Such findings suggest that individuals who frequently are uninhibited about responding angrily to events perceived as threatening, provoking or frustrating are likely to be at increased risk for coronary artery disease, a conclusion that concurs with previous research (Mendes de Leon, 1992; Miller et al., 1996; Siegman et al., 1987). However, expressed anger clearly is not associated with symptom reporting. In the present study, anger expression did not correlate with any of the three symptom types, a finding which is of particular note given that all eight of the other anger-hostility measures correlated with at least one of the symptom factors. This differential association with symptomatology and disease severity is important since it indicates that anger expression may be of value as a psychological discriminator between the two.

A further aim of this study has been to ascertain if the self-reported symptoms of CAD should be investigated separately or whether they should be aggregated empirically. Patients provided data on nine primary symptomatology measures. Factor analyses of these showed a three-factor solution that co-varied with three main *types* of CAD symptoms: *angina*, *breathlessness and restricted mobility*, and *tiredness*. Such results suggest that it is not tenable to sustain an empirical or conceptual distinction between the sub-dimensions of frequency, intensity and duration of CAD symptoms within these symptom types. This finding has implications for future research into CAD symptomatology and for clinical practice, as it would suggest that single or aggregated indices of these symptom types are adequate as research measures or as clinical indicators of CAD-related distress. It is apparent that there is little to be gained by further distinguishing between the frequency, intensity and duration of symptoms.

A final result to note is that no significant correlations were observed between symptom scores and CAD severity. A similar lack of relationship between objective disease measures and subjective illness behaviour has been reported elsewhere (Jenkins et al., 1983; Katon, 1990; Smith et al., 1984). As such then, it is evident that self-reported symptomatology is not a reliable indicant of disease severity. This does not mean, however, that the symptoms experienced by patients should be discounted since they are of clinical importance—not only in relation to patients' increased risk for future major cardiac events, but also with reference to of their subjective well-being and the psychological treatments now shown to reduce the frequency and severity of symptoms.

One limitation of the present study is that no measures of emotional denial were investigated here. Denial is measured by comparing patients' self-reports of emotionality with the ratings of spouses or friends. Recently, a growing body of research has illustrated the importance of the denial of emotional distress as a predictor of CAD symptoms and outcomes. Coronary disease status, for example, has been found to be predicted by denial of hostility and outwardly expressed anger (Kneip, Delamater, Ismond, Milford, Salvia, & Schwartz, 1993; Siegman, Townsend, Blumenthal, Sorkin, & Civelak, 1998). Similarly, current angina frequency and future cardiac events (including MI, revascularisation and death) have been associated with the denial of resentment and anger, as well as denial of anxiety, depression, exhaustion and dependency (Ketterer, Huffman, Lumley, Wassef, Gray, Kenyon, Kraft, Brymer, Rhoads, Lovallo, & Goldgberg, 1998; Ketterer, Kenyon, Foley, Brymer, Rhoads, Kraft, & Lovallo, 1996; Trijsburg, Erdman, Duivenvoorden, Thiel, & Verhage, 1987). Given the present study has not

measured emotional denial, it is acknowledged that the indices of dysphoric emotions used here have not taken into account the possibility that some respondents may have under-reported their negative emotions. If more objective measures of emotionality had been incorporated (such as the ratings of significant others), then additional and more substantive relationships between psychosocial variables and CAD symptoms and coronary artery disease may have been found. Such speculations constitute directions for future research.

A further limitation of the findings presented here relates to the cross-sectional design of the study and, in particular, the problems of establishing cause and effect and sampling bias. In health research, the most reliable method for determining causal relationships is to conduct prospective research on initially disease-free populations. For present purposes, this preferred method was not an available option. Consequently, it is not possible to conclude that being depressed and anxious caused the CAD patients in this study to experience more symptoms, since it is equally possible that these patients became depressed and anxious as a result of experiencing angina pain and other debilitating symptoms. Nonetheless, as noted earlier, other research findings do suggest that emotionality has a causal role in the experience of symptoms. Thus, intervention studies have shown that CAD symptomatology can be decreased following participation on psychologically-orientated cardiac rehabilitation programmes (Lewin, 1997; Lewin et al., 1995). An additional problem of conducting a cross-sectional study of arteriographically defined CAD patients is the issue of sampling bias. As Pearson (1984) points out, patients with moderate to severe angina and those who have suffered non-fatal MIs are likely to be over-represented in angiographic studies since patients suffering sudden fatal MIs are missed and those with mild angina or 'silent' MIs are not as likely to be referred for angiographic investigation. Moreover, some individuals who are relatively free of disease may over-report their symptoms and thus are referred early, whilst others with substantial CAD may under-report their disease and so only find themselves being referred to such a clinical service in the later stages of their illness. The sample incorporated here then, should not be regarded as representative of all those with CAD in the community at large, many of whom may never find themselves referred for assessment by a cardiologist.

In summary and conclusion, the results of this cross-sectional study suggest that the components of the anger-hostility complex are of limited and indirect use as psychosocial predictors of CAD-related symptomatology. Rather, they can be regarded as proxies for other psychosocial variables that constitute negative affectivity, notably the dysphoric emotional states of depression and anxiety. Also, our results show psychosocial measures (with the exception of expressed anger) are uncorrelated with clinician's ratings of angiographically identified CAD severity, despite often being correlates of self-reported CAD symptoms. Notably, however, measures of anger expression may be useful when differentiating between symptomatic CAD sufferers and symptomatic disease-free referrals. Thirdly, this study has shown that CAD symptoms are reducible in terms of three types: *angina, breathlessness and restricted mobility*, and *tiredness*. Thus, it has been shown that attempting to distinguish between the frequency, intensity and duration of a CAD symptom is not empirically tenable. Finally, it has been found, as elsewhere, that self-reported CAD symptoms are not related to or reliable indicants of coronary artery disease severity.

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