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Anxiety in Patients Who Have Had a Myocardial Infarction: The Maintaining Role of Perceived Physical Sensations and Causal Attributions

Denise Ratcliffe

Chelsea and Westminster Hospital, London, UK

Andrew MacLeod

Royal Holloway, University of London, UK

Tom Sensky

West Middlesex University Hospital, Isleworth, UK

Abstract. This study investigated whether anxiety in patients who have had a myocardial infarction is maintained through similar processes to those proposed in the cognitive models of panic disorder (Clark, 1986) and health anxiety (Warwick and Salkovskis, 1990). Anxious ($n = 22$) and non-anxious ($n = 29$) patients, who all had an MI 3–12 months before testing, participated. The groups were compared on self-report measures of risk perception, bodily vigilance, illness perceptions the type of causal attributions (somatic, normalizing and psychological) generated for congruent and incongruent types of anxiety-related bodily sensations (cardiac, respiratory, gastro-intestinal and cognitive dyscontrol). Anxious, compared to non-anxious, MI participants perceived themselves to be at higher risk of a further MI, had higher levels of bodily vigilance and more negative emotional and cognitive representations of their MI. Anxious participants generated significantly more somatic attributions and fewer normalizing attributions, than non-anxious participants for cardiac sensations. There was also a trend for anxious participants to generate this pattern of attributions for respiratory items. The study provides evidence that the models of health anxiety (Warwick and Salkovskis, 1990) and panic disorder (Clark, 1986) are useful in understanding persistent anxiety following an MI.

Keywords: Persistent anxiety, myocardial infarction (MI), bodily sensations, causal attributions.

Reprint requests to Denise Ratcliffe, Psychological Medicine Unit, South Kensington & Chelsea Mental Health Unit, Chelsea & Westminster Hospital, 1 Nightingale Place, London SW10 9NG, UK. E-mail: d.ratcliffe@nhs.net

Introduction

Anxiety and depression are common psychological reactions following a myocardial infarction (MI) (e.g. Lane, Carroll, Ring, Beevers and Lip, 2002). Little attention has been paid to anxiety in MI patients although its prevalence tends to be higher than that of depression (e.g. Havik and Maeland, 1990; Mayou et al., 2000). Furthermore, there is evidence that a significant number of MI patients experience persistent and unremitting anxiety. Mayou et al. (2000) reported a decrease in mean anxiety and depression scores between hospital admission and 3 months but thereafter no changes were observed amongst the distressed patients. Lane et al. (2002) also reported higher correlations between anxiety scores at 4 months and 1 year ($r = .73$) than those between baseline and 4 months ($r = .48$). Approximately 40% of the MI patients were anxious at 4 months and one-year post-MI. These findings indicate that for a significant proportion of post-MI patients, anxiety is not just a transitory reaction and may become a persistent, chronic condition that requires detection, intervention and monitoring.

Whilst these studies provide important information on the proportion of MI patients affected, they do not explain *why* some individuals experience persistent anxiety. Do established models of anxiety provide a useful framework for understanding persistent anxiety in MI patients? Eifert (1992) suggested that heart-focused anxiety (a specific type of health anxiety) has features of health anxiety, panic disorder and phobia. Barsky, Barnett and Cleary (1994) suggested that health anxiety and panic disorder have similar presentation features and share a similar pathogenic mechanism: the cognitive misinterpretation and amplification of bodily sensations, which results in excessive alarm. The cognitive models of panic disorder (Clark, 1986) and health anxiety (Warwick and Salkovskis, 1990) propose that this mechanism is responsible for the maintenance of these disorders.

The cognitive models of panic disorder and health anxiety are based on the premise that an individual's anxiety is unfounded in terms of direct, personal experience of a feared illness or event. Clinical experience suggests that the maintaining processes outlined in these models might also be useful in understanding experience-based health anxiety (i.e. grounded in an objective experience). It has been suggested that if a patient's anxiety about an illness produces autonomic responses that overlap with the symptoms of this illness, then anxiety will escalate (Warwick and Salkovskis, 1990). A number of authors have noted the concordance between cardiac and anxiety symptoms (e.g. Fleet and Beitman, 1998a; Jeejeebhoy, Dorian and Newman, 2000). The significant overlap between cardiac symptoms and anxiety symptoms creates ample scope for anxious MI patients to generate catastrophic interpretations of benign bodily sensations. The present study aimed to investigate the model shown in Figure 1. This model, which is based on the maintaining cycle outlined in the health anxiety model (Warwick and Salkovskis, 1990), has been expanded in order to capture the experience-based nature of health anxiety in MI patients. The experience-based health anxiety model includes illness beliefs/representations and the role of domain-specific anxiety sensitivity.

Following an illness/adverse health event, it is suggested that individuals create internal representations of their illness and symptoms (Leventhal, Nerenz and Steele, 1984). Illness representations are stable knowledge bases that help us to evaluate health threats (Weinman, 1998) and they influence the type of cognitive and emotional processing (e.g. apprehension, vigilance) utilized by the individual. The individual's cognitive representation about their MI experience acts as a filter through which experiences (e.g. bodily sensations) that are perceived

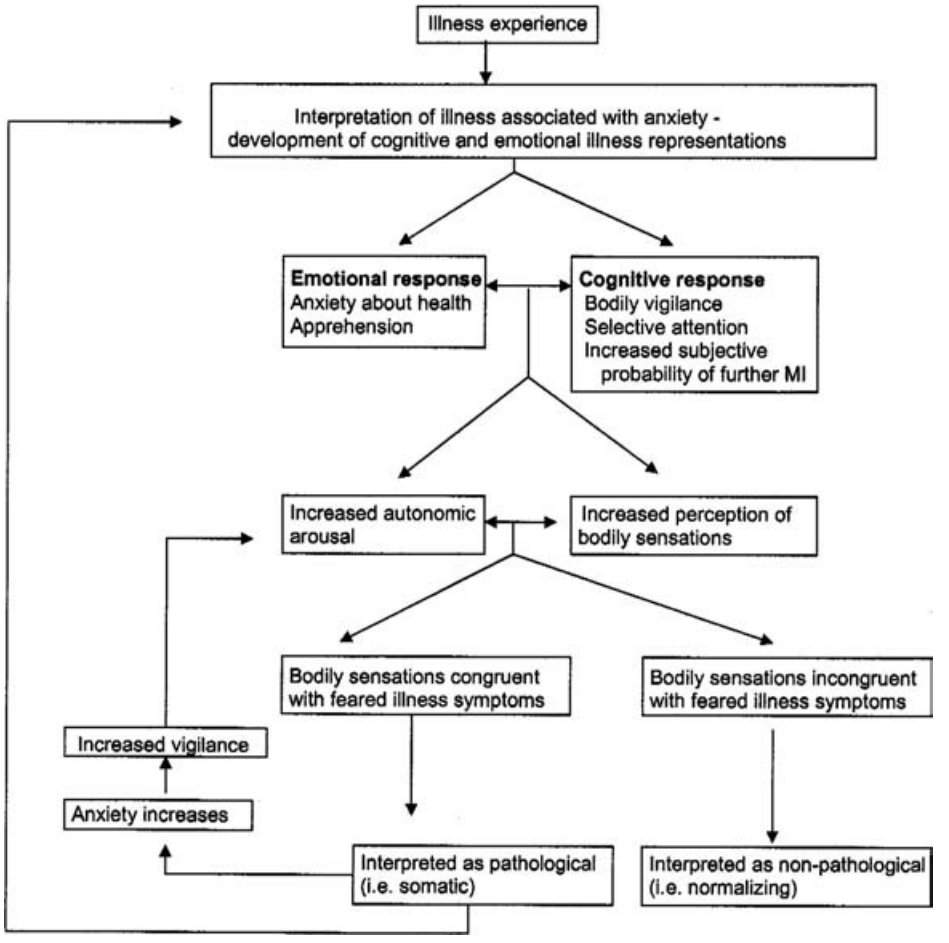

Figure 1 Hypothesized mechanism for the persistence of anxiety that develops after a myocardial infarction

to be similar to the MI experience are appraised. This model suggests that bodily vigilance is one of the cognitive responses associated with anxiety-focused illness representations. Vigilance involves scanning for threat-relevant cues (Miller, Brody and Summerton, 1988) and can be conceptualized as a cognitive strategy to reduce uncertainty (Kohlmann, Ring, Carroll, Mohiyeddini and Bennett, 2001). Illness representations, perceived consequences and importance assigned to the somatic sensation influence the individual's level of vigilance (Cioffi, 1991). Kohlmann et al. (2001) reported that individuals who are vigilant to cardiac sensations demonstrate a tendency to interpret these sensations in a threat-related manner rather than an increased sensitivity to detecting these sensations. Cioffi (1991) found that individuals who focus on negative, threatening interpretations of somatic sensations tend to bias the processing of incoming sensory information negatively. Thus, a self-fulfilling

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prophecy is established between anxiety, vigilance, perception of bodily sensations and negative interpretation of these.

It is widely recognized that increased anxiety is associated with a stronger attentional focus on internal bodily states as well as higher levels of symptom reporting (Watson and Pennebaker, 1989). Research on whether anxious individuals are more accurate than non-anxious individuals at detecting their heartbeat has yielded conflicting results. Van der Does, Antony, Ehlers and Barsky (2000) reported that panic disorder patients are more accurate at heart beat detection tasks than non-anxious patient groups but that accuracy is still uncommon. They suggested that for anxious patients, bodily sensations or symptoms might trigger an "anxiety schema" that influences attentional focus, perception and arousal. The authors suggested that the perception of symptoms is determined by the individual's coping schema. On the other hand, Steptoe and Vogele (1992) also reported that the tendency for anxious individuals to report more bodily sensations reflects a cognitive-perceptual bias rather than an enhanced sensitivity to accurately detect bodily changes. Similarly, Pauli et al. (1991) reported that panic disorder patients and healthy controls recorded a similar frequency of cardiac perceptions when they were given a 24-hour ECG. However, the ECGs revealed that healthy controls had a heart rate deceleration following cardiac perception whereas the panic patients had a further acceleration. This provides evidence of the vicious cycle between cardiac perception and intensified anxiety.

The model outlined in Figure 1 suggests that anxious post-MI patients will not generate pathological interpretations for all bodily sensations. Recent work highlighting the multidimensional nature of anxiety sensitivity was used to understand the specific relationships between bodily sensations and anxiety. The model was refined using the concept of anxiety sensitivity (AS), defined by Taylor and Cox (1998) as the fear of "anxiety-related bodily sensations, based on beliefs that the sensations have harmful somatic, social or psychological consequences" (p. 38). AS is viewed as a general amplification factor for interoceptive events and bodily sensations (Eifert, Zvolensky and Lejeuz, 2001). Cox (1996) proposed that in anxiety disorders (particularly panic disorder), AS is multidimensional and identified four factors (fear of cardiac symptoms, respiratory symptoms, gastrointestinal symptoms and cognitive dyscontrol). It is suggested that AS interacts with triggers congruent with the fear to produce catastrophic cognitions and anxiety, but not with incongruent triggers. Indeed, Eifert et al. (2001) reported that predictions about anxiety levels improved with increasing correspondence between specific domains of anxiety sensitivity and events that are congruent with the fear. Extrapolating from this work on anxiety and panic, the model proposes that anxiety in MI patients may be maintained through perception and catastrophic interpretation of bodily sensations that the individual judges to be illness-congruent.

The perception of bodily sensations is responsible for setting interpretative processes in motion. Generating an illness-related hypothesis about a bodily sensation increases selective attention to confirmatory evidence (Snyder and Gangestad, 1981). The type of causal attributions generated about bodily sensations are linked to anxiety levels. MacLeod, Haynes and Sensky (1998) compared the attributions for common bodily sensations generated by patients with general anxiety, hypochondriasis and healthy controls. They found that both anxiety groups were more likely to generate psychological explanations, and less likely to generate normalizing explanations than controls. However, only those with hypochondriacal anxiety were significantly more likely to generate somatic attributions for the sensations. There is also evidence to suggest that individuals show biased perseverance (Koehler, 1991)

in the type of attributions they generate. It is likely that the habitual generation of somatic explanations for bodily sensations may play a significant role in maintaining health anxiety.

To summarize, the aim of this study was to investigate whether the same feedback loop that maintains panic disorder and health anxiety is useful in explaining persistent anxiety in MI patients. It was hypothesized that anxious, compared to non-anxious, MI patients would perceive themselves to be at increased risk of having a further MI, have higher levels of body vigilance, and more negative emotional and cognitive representations of their heart condition. It was predicted that anxious participants would generate more somatic, and fewer normalizing, attributions for bodily sensations congruent with their illness experience (i.e. cardiac and respiratory) but that there would be no group differences in the attributions generated for incongruent sensations (i.e. gastrointestinal and cognitive dyscontrol). Finally, it was hypothesized that anxious participants would report that they experienced the bodily sensations more frequently and rate them as more anxiety-provoking.

Method

Participants

Seventy-nine patients were identified who were consecutive admissions of the Coronary Care Unit (CCU) at West Middlesex University Hospital and who had had an MI between 3–12 months before testing. Patients were excluded from the study if their MI occurred as a result of coronary artery bypass surgery, angiography, if they were cognitively impaired or unable to speak English.

Overall, 64.5% ($n = 51$) of those eligible provided informed consent and completed the study. The mean age of the participants was 60.5 years (*SD* 13.2), 75% (38) of the sample were male, 71% (36) were married, 45% (23) were employed and 37% (19) were retired. Eighty-six percent (44) were Caucasian. Participants and non-participants did not differ in age ($t(77) = 1.92$, $p = .06$), gender ($\chi^2(1) = .39$, $p = .60$) or time elapsed since their MI ($t(77) = 1.30$, $p = .19$).

Two groups ("anxious" and "non-anxious") were formed on the basis of anxiety scores obtained on the Hospital Anxiety and Depression Scale (HADS-A; Zigmond and Snaith, 1983). Twenty-nine participants (57%), who scored below 8 on the HADS-A, formed the non-anxious group. The remaining 22 (43%) participants formed the anxious group.

Measures

Medical and demographic information was collected from participant's medical records and by interview. The HADS (Zigmond and Snaith, 1983) is a self-assessment tool that consists of two 7-item subscales that measure anxiety (HADS-A) and depression (HADS-D). The HADS was developed for use with non-psychiatric populations and its validity with patients from different medical settings has been documented (Herrmann, 1997). It has been used with MI patients (e.g. Martin and Thompson, 2000; Mayou et al., 2000). Snaith and Zigmond (1994) interpret HADS-A or HADS-D scores of 8–10 as borderline and a threshold score of 8 is commonly used in studies (MacLeod et al., 1998; Martin and Thompson, 2000). This threshold score was used in the current study in order to facilitate comparison with other studies. Perceived risk

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Table 1 Somatic Interpretation Task

Symptom type	SIT item	Order of presentation
Cardiac	If I felt my heart beating rapidly, I would think . . .	4
	If I felt my heart beating erratically, I would think . . .	8
	If I felt my heart pounding, I would think . . .	12
Respiratory	If I felt like I couldn't breathe properly, I would think	1
	If I felt numb, I would think . . .	5
	If I felt pain in my chest, I would think. . .	9
Gastro-intestinal	If I felt sick in my stomach (nauseous), I would think. . ..	3
	If I was feeling bloated, I would think . . .	7
	If I felt like I was about to vomit, I would think . . .	11
Cognitive dyscontrol	If I was finding it difficult to concentrate, I would think . . .	2
	If I was having trouble remembering things, I would think. . .	6
	If my thoughts seem to race, I would think . . .	10

of having a further MI was assessed on a 5-point scale (1 = not at all likely and 5 = extremely likely).

The Body Vigilance Scale (BVS; Schmidt, Lerew and Trakowski, 1997) measures attentional focus to internal bodily sensations. Three items assess the degree of attentional focus, perceived sensitivity to changes in bodily sensations and the average amount of time spent attending to bodily sensations. The fourth item on the scale (ratings of the amount of attention paid to 15 sensations associated with panic disorder) was not used to avoid replication with the Somatic Interpretation Task. Whilst the BVS was developed for patients with panic disorder, it is likely to be suitable for individuals who have health anxiety as both disorders involve bodily vigilance (Barsky et al., 1994).

The cardiac version of the Revised Illness Perception Questionnaire (IPQ-R (Heart); Moss-Morris et al., 2002) was used to provide a measure of the key components of patient's illness representations. It consists of seven subscales that measure the patient's perception of the illness. The subscales measure the perceived timeline of the illness, perceived consequences, degree of personal and treatment control, illness coherence, predictability of the symptoms and emotional representation of the illness.

The Somatic Interpretation Task (SIT) was adapted from a task employed by MacLeod et al. (1998) who presented participants with 10 common bodily sensations from the Symptom Interpretation Questionnaire (Robbins and Kirmayer, 1991) and coded the participant's causal attributions as normalizing, psychological or somatic. A similar approach was adopted in the SIT although the bodily sensations were taken from the Anxiety Sensitivity Index (ASI; Taylor and Cox, 1998). This questionnaire consists of four lower order factors of anxiety sensitivity (fear of cardiac symptoms, respiratory symptoms, gastrointestinal symptoms and cognitive dyscontrol). The 12 symptoms (three from each factor) shown in Table 1 were presented to participants and they were given 60 seconds to generate as many causal explanations as they could for why they themselves might experience that sensation. Participants were not made aware that the sensations were taken from an anxiety questionnaire.

The causal explanations generated by patients were coded using the same basic categories (i.e. normalizing, psychological and somatic) as Robbins and Kirmayer (1991) and MacLeod et al. (1998). However, both of these studies focused on healthy individuals who experienced health anxiety, whereas the current study focused on those whose anxiety was based on a physical antecedent (MI). This meant that the coding system required some amendment to take into account the range of somatic responses that people who have experienced a health event may generate. The categories of normalizing and psychological attributions remained the same. The somatic category was expanded to allow more detailed analysis and because many of the participants had significant co-morbid illnesses (e.g. diabetes).

An independent rater, blind to group membership, categorized all the responses of 10% ($n = 5$) of the participant's causal explanations. The level of agreement between the two raters (calculated using Kappa's coefficient) was 0.93.

For each SIT item, participants were asked to indicate on a 5-point scale how afraid they were of the sensation (1 = not frightened and 5 = extremely frightened). They were also asked to indicate on a 5-point scale how often they had experienced the sensation during the last month (1 = never and 5 = daily).

Procedure

Patients who had their MI within the relevant timeframe were identified through the admissions book in CCU and were sent a letter and information sheet about the study. They were contacted by telephone one week later to answer any queries and to see if they would be willing to participate. Forty-nine (96%) participants were interviewed at home and the remaining two participants were interviewed on hospital premises.

Results

Prevalence of anxiety

Of the 51 participants who completed the study, 29 (57%) scored below 8 on the HADS-A and 22 (43%) scored 8 or above. The mean HADS-A score for all participants was 6.35 (*SD* 4.46).

Characteristics of anxious and non-anxious participants

There were significant differences between anxious and non-anxious participants in gender, age and employment status. Table 2 shows that anxious participants were younger, more likely to be female, more likely to be unemployed or on long term sick leave and less likely to be retired than the non-anxious group. Anxious, compared to non-anxious, participants had significantly longer hospital admissions for their MI and more of them reported previous psychological difficulties.

Questionnaire measures

Table 3 shows mean scores on the questionnaire measures for anxious and non-anxious participants. Anxious participants perceived themselves to be at greater risk of having a further heart attack and had higher levels of body vigilance than non-anxious participants.

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Table 2 Demographic and clinical characteristics of anxious and non-anxious MI patients

	Non-anxious ($n = 29$)	Anxious ($n = 22$)	Significance
Mean age (*SD*, range)	64.6 (12.1, 34–82)	55.2 (12.9, 34–87)	$t(49) = 2.65, p = .011$
Gender			
Male	89.7% (26)	54.5% (12)	
Female	10.3% (3)	45.5% (10)	$\chi^2(1) = 8.11, p = .006$
Marital status			
Married	79.4% (23)	59.1% (13)	
Divorced	0 (0%)	27.2% (6)	
Single	10.3% (3)	9.1% (2)	n.s.
Widowed	10.3% (3)	4.5% (1)	$\chi^2(5) = 9.54, p = .089$
Living circumstances			
Living alone	20.7% (6)	36.4% (8)	n.s.
With partner	79.3% (23)	63.7% (14)	$\chi^2(2) = 4.01, p = .134$
Ethnicity			
White	89.7% (26)	81.8% (18)	n.s.
Asian	10.3% (3)	18.2% (4)	$\chi^2(2) = 2.20, p = .333$
Employment status			
Employed	41.4% (12)	50% (11)	
Unemployed	3.4% (1)	13.6% (3)	
Sick Leave	0% (0)	22.7% (5)	
Retired	55.2% (16)	13.6% (3)	$\chi^2(3) = 14.25, p = .003$
Mean (*SD*) CK-total	905 (628)	1003 (658)	n.s. $t(45) = .53, p = .600$
Mean (*SD*) number of days in hospital	11.03 (8.5)	16.86 (10.3)	$t(49) = 2.22, p = .037$
Mean (*SD*) number of days since MI	205.6 (98.8)	191.3 (91.1)	n.s. $t(49) = .53, p = .598$
Had post-MI surgery	38.5% (10)	45.5% (10)	n.s. $\chi^2(1) = .24, p = .770$
Waiting for surgery	15.4% (4)	18.2% (4)	n.s. $\chi^2 = (2) = .92, p = .633$
First MI	89.7% (26)	90.9% (20)	n.s. $\chi^2 = (1) = .02, p = .881$
Has current angina	20.7% (6)	31.8% (7)	n.s. $\chi^2(2) = .82, p = .665$
Had previous history of angina	17.2% (5)	18.2% (4)	n.s. $\chi^2(2) = .10, p = .951$
% with comorbid illness (e.g., diabetes, asthma)	83% (24)	68.7% (15)	n.s. $\chi^2 = (10) = 8.87, p = .544$
Self-reported history of psychological difficulties	13.8% (4)	68.2% (15)	$\chi^2(2) = 17.98, p < .001$

IPQ-R (Heart). Anxious participants experienced significantly more of the listed symptoms than non-anxious participants. Anxious participants considered a greater proportion (mean = .74) of the listed symptoms they experienced to be cardiac-related than non-anxious participants (.52).

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Table 3 Mean questionnaire scores for anxious and non-anxious participants

	Anxious Mean (*SD*)	Non-anxious Mean (*SD*)	Significance
Risk perception	3.0 (1.1)	2.1 (1.3)	$Z = -2.9, p = .004$
Body vigilance	16.3 (5.7)	6.7 (7.0)	$t(47) = 5.1, p < .001$
IPQ symptoms experienced	6.8 (3.1)	3.7 (2.6)	$t(49) = 3.9, p < .001$
IPQ symptoms – cardiac related	4.9 (2.9)	1.9 (1.4)	$t(43) = 2.7, p < .011$
IPQ – timeline	23.1 (4.7)	20.8 (5.1)	$t(49) = 4.8, p < .001$
IPQ – consequences	22.2 (4.0)	16.0 (4.7)	$t(44) = 4.8, p < .001$
IPQ – control	21.6 (2.5)	24.3 (2.9)	$t(43) = 3.4, p < .001$
IPQ – treatment	17.5 (1.8)	19.5 (2.4)	$t(44) = 3.2, p = .002$
IPQ – coherence	15.7 (4.5)	11.1 (3.9)	$Z = -3.3, p = .001$
IPQ – cyclical timeline	12.8 (3.8)	8.3 (3.4)	$Z = -3.9, p < .001$
IPQ – emotional representation	22.0 (3.5)	14.1 (4.0)	$t(41) = 6.9, p < .001$

Significant differences between anxious and non-anxious participants were found on all subscales of the IPQ-R (Heart). Compared to non-anxious participants, anxious participants perceived their illness to have a chronic, rather than acute, timeline, perceived their heart problem to have more serious consequences, considered themselves to have less control over their heart condition and thought that their treatment was less effective. They had a more negative emotional representation of their heart problem, more difficulties in making sense of their condition, and perceived their cardiac symptoms to be more unpredictable and cyclical. All of these results remained significant following adjustment for Bonferroni correction for the number of tests.

Somatic Interpretation Task

A series of 2 × 3 ANOVAs (anxiety group × attribution type) were used to investigate group differences in the type of attributions generated for the four SIT symptom types. Post-hoc independent *t*-tests were used to investigate significant interactions. As there were significant differences in the demographic and clinical characteristics of the two groups, significant post-hoc independent *t*-tests were repeated as univariate ANCOVAs.

Cardiac SIT items. The 2 × 3 ANOVA revealed a significant main effect of attribution type ($F(1.4, 66.2) = 21.23, p < .001$) but not anxiety group ($F(1,49) = 3.88, p = .054$). There was a significant interaction between anxiety and attribution type ($F(1.4,66.2) = 10.61, p = .001$). As shown in Figure 2a, anxious participants generated significantly fewer normalizing attributions ($t(49) = 2.71,\ p = .009$) than anxious participants. Conversely, anxious participants made significantly more somatic attributions ($t(49) = 3.73,\ p < .001$). There was no significant difference between the two groups in the number of psychological attributions generated ($t(36.5) = 0.92,\ p = .365$). When ANCOVA's were performed, the group differences in normalizing ($F(1,44) = 5.05,\ p = .030$) and somatic attributions ($F(1,44) = 5.95,\ p = .019$) remained significant.

Respiratory SIT items. The 2 × 3 ANOVA revealed a significant main effect of attribution type ($F(1.6,\ 76.9) = 82.5,\ p < .001$) but no main effect of anxiety group ($F(1,49) = 3.84,$

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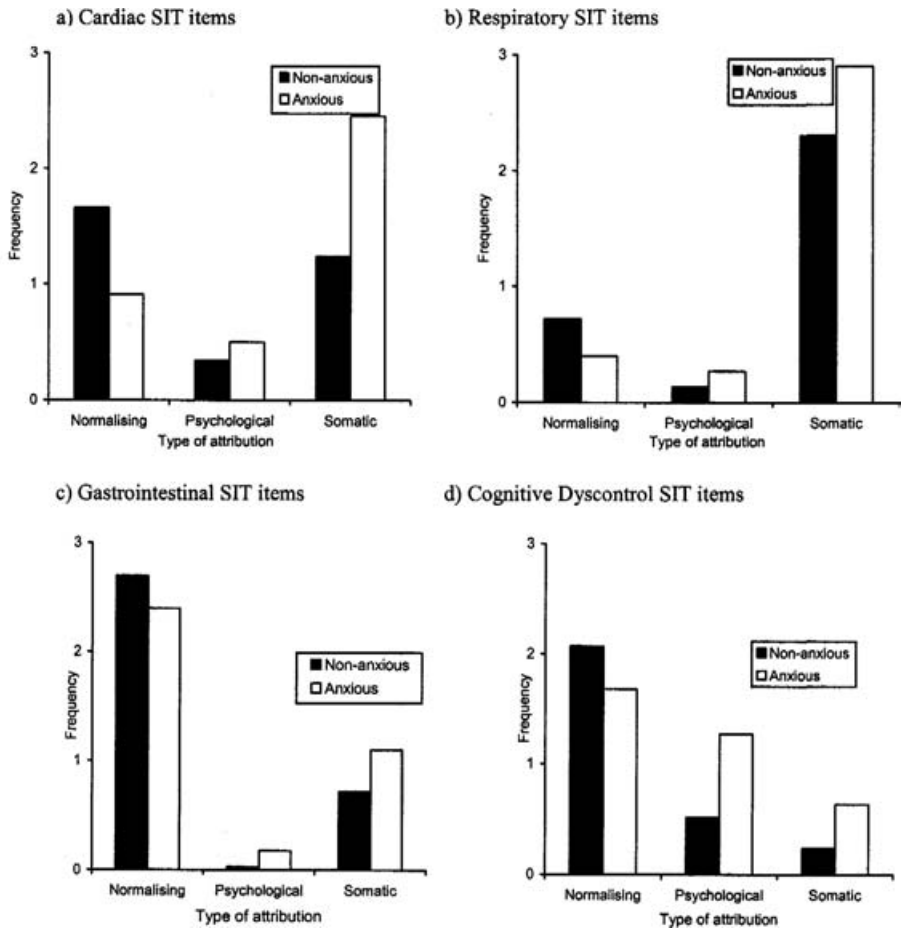

Figure 2 Type of attributions generated for SIT symptoms by anxious and non-anxious participants

$p = .056$). As for the cardiac items, there was a significant interaction between anxiety and attribution type ($F(1.6.76.9) = 4.08$, $p = .029$). Post-hoc t-tests revealed that anxious patients generated significantly fewer normalizing than non-anxious participants attributions ($t(48.5) = 2.14$, $p = .038$). There were no significant differences between the groups in number of somatic ($t(49) = 1.85, p = .070$) or psychological attributions ($t(49) = .15, p = .884$) generated (see Figure 2b). The group difference in normalizing attributions became non-significant when an ANCOVA was performed ($F(1,44) = 2.32, p = .135$).

Gastrointestinal SIT items. The 2×3 revealed a significant main effect of attribution type ($F(1.3, 64.5) = 92.88, p < .001$) but no main effect of anxiety group ($F(1,49) = .755, p = .389$). Figure 2c shows that, as predicted, there was no significant interaction between anxiety group and type of attribution ($F(1.3,64.5) = 2.41, p = .117$) generated.

Cognitive Dyscontrol SIT items. The 2×3 ANOVA revealed a significant main effect of attribution type ($F(1.6, 76.2) = 22.63, p < .001$) and anxiety group ($F(1,49) = 5.94, p = .018$).

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There was also a significant interaction between anxiety level and type of attribution generated ($F(1.6, 76.2) = 3.65$, $p = .041$). Anxious participants, compared to non-anxious participants, generated significantly more psychological attributions for cognitive dyscontrol items ($t(33.6) = 2.87$, $p = .007$). As shown in Figure 2d, there was no significant difference in the number of somatic ($t(49) = 1.65$, $p = .108$) or normalizing attributions ($t(49) = 1.16$, $p = .253$) made by the two groups. An ANCOVA was performed on the group difference in psychological attributions and the difference was reduced to non-significance ($F(1,44) = 2.49$, $p = .121$).

Tendency to generate somatic attributions

It was hypothesized that anxious participants would show a greater tendency to generate somatic attributions for cardiac and respiratory items than gastrointestinal or cognitive dyscontrol. In order to test this hypothesis, a new variable reflecting the tendency to make somatic attributions was created by subtracting the total number of an individual's normalizing and psychological attributions from the total number of somatic attributions they generated across the four SIT symptom types.

A 2 × 4 (anxiety group × symptom type) ANOVA revealed a main effect of symptom type ($F(2.75, 134.8) = 84.3$, $p < .001$) and anxiety group ($F(1,49) = 7.67$ $p = .008$). There was a significant interaction between symptom type and anxiety group ($F(2.75, 134.8) = 4.85$, $p = .004$). Post-hoc independent *t*-tests revealed that anxious participants, compared to non-anxious participants, generated significantly more somatic attributions for cardiac ($t(49) = 3.33, p = .002$) and respiratory items ($t(49) = 1.98, p = .05$) but not for gastrointestinal ($t(49) = 1.06$, $p = .293$) or cognitive dyscontrol symptoms ($t(49) = .42$, $p = .674$). The group differences in generating somatic versus other attributions for cardiac items remained significant when the effects of the covariates had been removed ($F(1,44) = 4.78, p = .034$).

Subcategories of somatic attributions

The subcategories of somatic attributions were a) cardiac related; b) other diagnosed illness (e.g. diabetes, asthma); c) other undiagnosed illness; d) medication/side-effects. A 2 × 4 (anxiety group × somatic subcategory) ANOVA revealed a main effect for subcategory of somatic attribution ($F(1.8, 89.7) = 78.46$, $p < .001$) and anxiety group ($F(1,49) = 10.54$, $p = .002$), as well as a significant interaction between the two ($F(1.8, 89.7) = 10.00, p < .001$). Anxious participants were significantly more likely to generate cardiac-related somatic attributions ($t(49) = 3.82, p < .001$). There were no significant group differences in the number of attributions relating to other diagnosed illnesses ($t(49) = .78$, $p = .44$), undiagnosed illnesses ($t(41.2) = .49$, $p = .62$[1]) or medication/side-effects ($t(49) = .81$, $p = .43$). When an ANCOVA on the number of cardiac-related somatic attributions generated by anxious and non-anxious participants was performed, the group difference still remained significant ($F(1,44)$ 4.84, $p = .033$).

Discussion

The current study differs from most previous studies of health anxiety in that it investigated experience-based anxiety; the patients in this study had all had an illness experience that

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could form the basis for realistic health concerns. It was hypothesized that post-MI anxiety is maintained through the same mechanism central to the cognitive models of panic disorder (Clark, 1986) and health anxiety (Warwick and Salkovskis, 1990). The central hypothesis of these models is that anxiety is maintained via a negative feedback loop between anxiety, hypervigilance, increased perception and catastrophic interpretation of benign symptoms. The same psychological concepts and processes were hypothesized to be at the core of experience-based health anxiety. In fact, we considered that this maintaining process may be more apparent because of the increased possibility of overlap between physiological symptoms of anxiety and symptoms that the individual perceives to be associated with the illness they fear (leading to an increased chance of pathological interpretation). The experience-based model of health anxiety proposes that the perceived congruence between specific bodily sensations and feared illness symptoms is an important maintaining factor. The model also incorporates information about the individual's interpretation of their illness experience. The results of this preliminary study support the proposed model and it is likely that the model could be extended to other illnesses where symptoms and anxiety symptoms overlap and where there is a possibility of recurrence (e.g. cancer, epilepsy).

This study revealed high rates of anxiety (43% HADS-A >8) amongst MI patients. As predicted, anxious participants, compared to non-anxious participants, perceived themselves to be at increased risk of having a further MI. Anxious participants also had significantly higher levels of bodily vigilance, which were comparable to those found amongst patients with panic disorder (Schmidt et al., 1997). Anxious participants had more negative cognitive and emotional representations of their heart condition than non-anxious participants. Illness representations influence the individual's psychological adjustment and their cognitive and behavioural responses, and these may maintain their anxiety. In terms of symptom-reporting, anxious participants reported experiencing significantly more symptoms when presented with a general list (e.g. not just cardiac related) of somatic symptoms on the IPQ-R (Heart). Furthermore, they considered a greater proportion of the symptoms they experienced to be cardiac-related. This probably means that they were less discriminating than the non-anxious patients in attributing cardiac causes to symptoms. The relationship between anxiety and increased symptom-reporting is well established (Watson and Pennebaker, 1989) and reflects increased vigilance to physical sensations as well as the narrowing focus of causal explanations generated by anxious individuals.

The current study incorporated recent findings on the multidimensional nature of anxiety sensitivity (Cox, 1996; Taylor and Cox, 1998) and investigated the effects on attributions about bodily sensations of congruence (or otherwise) of these sensations with the illness experience. Participants generated causal attributions for MI-congruent (cardiac and respiratory) and MI-incongruent (gastrointestinal and cognitive dyscontrol) bodily sensations. The experience-based health anxiety model proposes that anxiety is maintained where there is perceived congruency between the bodily sensations associated with anxiety and symptoms of a feared illness because of the pathological attribution the individual will generate in response to the bodily sensation. As predicted, anxious participants generated significantly more somatic attributions, and fewer normalizing attributions, for cardiac SIT items. Whilst there was a trend for anxious participants to generate fewer normalizing, and more somatic attributions for respiratory items, this failed to reach statistical significance. Both anxious and non-anxious participants generated more somatic attributions than normalizing attributions for respiratory SIT items. This may reflect the fact that of all the SIT items, these were most similar to

the symptoms experienced during a heart attack (see Table 1). The fact that 80% ($n = 40$) of patients reported that chest pain was their primary MI symptom supports this.

The finding that anxious MI participants generated fewer normalizing attributions for MI-congruent bodily sensations is similar to recent work on anxiety and future-oriented thinking. MacLeod, Tata, Kentish, Carroll and Hunter (1997) reported that anxious individuals found it harder than non-anxious individuals to generate reasons why future negative events would be unlikely to occur, and also described difficulties among anxious individuals in generating alternative, anxiety-reducing explanations. Similar findings have been reported previously among frequent attenders in primary care (Sensky, MacLeod and Rigby, 1996) and among patients with hypochondriacal anxiety (MacLeod et al., 1998). These findings demonstrate the tendency for anxious individuals to generate evidence that confirms their catastrophic cognitions. However, it is currently unclear whether it is the presence of somatic attributions or the absence of normalizing attributions that has the most potent effect on anxiety.

According to the proposed model, anxious and non-anxious individuals will tend to generate normalizing attributions for bodily sensations that are incongruent with their feared illness. In terms of MI-incongruent sensations (i.e. gastrointestinal and cognitive dyscontrol), it was predicted that there would be no group differences in the type of attributions generated. This hypothesis was supported for gastrointestinal items. Whilst there were no group differences in the number of somatic or normalizing attributions generated for cognitive dyscontrol items, anxious participants generated significantly more psychological attributions than non-anxious participants. It is likely that a different pattern of attributions was generated for cognitive dyscontrol items compared to other SIT items because they relate more directly to mental/psychological processes and anxious participants may have recognized cognitive dyscontrol symptoms from their recent or previous experiences of anxiety. The fact that Taylor (1996) found that fear of cognitive dyscontrol loaded onto a separate factor to fear of somatic sensations also explains why a different pattern of attributions emerged for these items.

There are a number of caveats and limitations to the current study. In order to facilitate comparison with relevant studies (e.g. Mayou et al., 2000), patients who had HADS-A scores that were 8 or above formed the anxious group. However, 10 of the 22 participants in the anxious group had scores that fell into the borderline range (8–10). There has been a long-running debate about the over-inclusiveness of the HADS-A threshold. Group membership was determined on the basis of participant's HADS-A scores and it was an implicit assumption of the current study that those who obtained high HADS-A scores were anxious because of their MI. This may not be the case given that a high proportion of anxious participants reported previous psychological difficulties (although this was self-report and possibly subject to individual interpretation and reporting bias). It could be argued that the results obtained are an artefact of general anxiety, not specific cardiac anxiety. However, there are a number of reasons to suggest that this is not the case. The profile of attributions generated by anxious MI patients is identical to the profile of individuals with health anxiety, rather than general anxiety (MacLeod et al., 1998). In addition, these data suggest that the somatic attributions of anxious MI participants are specifically focused on congruent bodily sensations (cardiac and possibly respiratory) and the content of these attributions tends to be cardiac focused. It is possible that for MI patients who were previously vulnerable to generalized anxiety, the experience of a traumatic health event may provide a specific focus for their anxiety. The fact that there is a greater proportion of women in the anxious group is a potential limitation of this study

as it could be argued that differences in SIT attributions between anxious and non-anxious individuals simply reflect a gender difference in the tendency to generate certain types of attributions. However, when we compared the attribution profiles of non-anxious male and female participants there were no significant differences and this suggests that the differences in type of attributions are related to anxiety, not gender.

The findings of the current study suggest that the same processes which maintain health anxiety that is focused on a feared, imagined future event is replicated in individuals whose anxiety is related to direct experience of an adverse health event. The role of selective attention and catastrophic interpretation of bodily sensations is central to health anxiety and it is likely that for individuals who have illnesses that may recur, the conditions are ripe for these processes to become persistent.

In order to minimize the risk of an individual developing health anxiety, a key intervention during hospital admission is psychoeducation about the potential overlap between anxiety symptoms and cardiac symptoms. The role of hypervigilance and causal explanations should also be covered in detail on cardiac rehabilitation programmes, although attendance on cardiac rehabilitation programmes tends to be poor (Cooper, Jackson, Weinman and Horne, 2002). Further work is needed to clarify the risk factors for the development of persistent health anxiety. However, the current study suggests that women and younger patients with a history of previous psychological difficulties may be at particular risk.

The clinical presentation of experience-based health anxiety and traditional health anxiety may appear similar, but there are some specific issues to consider. It is important to fully assess how accurate the individual was at detecting their original symptoms, their initial beliefs regarding their symptoms, and their subsequent diagnosis. These experiences are likely to have been influential in the formation of specific beliefs and assumptions about their health (e.g. "If I am vigilant then I can detect early signs of MI").

There is scope for intervention at various points of the feedback loop between anxiety, hypervigilance and somatic interpretation of benign bodily sensations. Schmidt et al. (1997) reported that a brief cognitive-behavioural treatment programme that specifically targeted bodily vigilance was successful in reducing anxiety and panic. Changes in bodily vigilance were highly associated with changes in anxiety sensitivity. This is one example of how treatments developed for use with panic disorder patients could be adapted for use with anxious, hypervigilant MI patients.

Clark et al. (1998) reported on the short-term and long-term efficacy of CBT for hypochondriasis and it is likely that this approach would also be helpful for MI patients. An important aspect of treatment for hypochondriacal anxiety is cognitive modification, which involves challenging and disputing beliefs and thoughts about illness (Furer, Walker and Freeston, 2001). However, where health anxiety is based upon direct experience of an adverse health event, discounting somatic attributions is likely to be less helpful than broadening the range and type of attributions that are generated. An important intervention would focus on helping MI patients to generate more normalizing attributions for their bodily sensations. Challenging the validity of illness beliefs and causal explanations for experience-based health anxiety is potentially inappropriate and may alienate the individual from their therapist. The focus should be on helping individuals to break the pattern of habitual, confirmatory attributions by increasing their cognitive flexibility to generate alternative attributions. However, one obviously needs to be mindful of the dangers of encouraging MI patients to ignore or normalize important symptoms that should be acted upon promptly.

The findings of the current study suggest that a significant number of individuals remain anxious 3–12 months post MI and the model of experience-based health anxiety is a useful framework for understanding the processes involved in its maintenance. In terms of future directions, this study has raised a number of questions about the type of causal attributions generated – for example, is it the presence of somatic or absence of normalising attributions that is crucial? Is it the process of increasing cognitive flexibility (i.e. the number of alternatives) or belief in an alternative that is crucial in reducing anxiety?

Safety-seeking and help-seeking behaviours are important aspects of health anxiety (Salkovskis, 1996). They have been noted in anxious patients who have non-cardiac chest pain (Aikens, Michael, Levin and Lowry, 1999) or have had a heart attack (Mayou et al., 2000). It would be useful to incorporate behavioural indices of help-seeking and safety-seeking and relate this to anxiety and tendency to generate somatic attributions.

The effect of anxiety on lifestyle change following an MI is not clear-cut. Some researchers have suggested it can have positive, motivating effects (e.g. Herrmann, Brand-Driehorst, Buss and Ruger, 2000) whilst others claim that it produces avoidant and restrictive behaviour (e.g. Aikens et al., 1999). It is likely that there is an optimum level of anxiety following an MI and it would be useful to identify the point at which anxiety becomes a debilitating, rather than motivating, factor.

To conclude, anxious MI participants differed from non-anxious participants on the self-report measures and attribution task that tapped into different aspects of the cognitive model of health anxiety. The results of this study provide preliminary evidence that the model of experience-based health anxiety is useful in understanding persistent anxiety in MI patients. However, it is a theoretical and clinical challenge to delineate the boundaries between traditional health anxiety and experience-based health anxiety. Whilst this study supports the idea that the same theoretical maintaining processes are operational, therapeutic interventions are likely to require modification for experience-based health anxiety.

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