

The Role of Psychological Factors in Chest
Pain with Normal Coronary Arteries : a
controlled study

Aileen S Thomson

Submitted for Doctor of Clinical Psychology
University of Edinburgh
2000



Declaration

The work presented in this thesis is my own, apart from the acknowledged assistance.

Acknowledgements

There are various people I would like to thank whose assistance has made this work possible. I am most grateful to Dr Stephen Potts. His guidance, advice, availability and encouragement at all stages of the study have helped me greatly and had made my task so much more enjoyable. I owe a great deal to Hugh Toner who not only allowed me the research time to carry out phase two of the study, but also encouraged and supported me throughout. I am indebted to Professor Mick Power for his valuable advice and whose clarity I much appreciated. I would like to thank Fife Primary Care Trust for their financial contribution and Professor Keith Fox not only for his financial help but also for his continuing interest and support.

This work would not have been possible without the cooperation of the Cardiology consultants at the Royal Infirmary. I would also like to extend my thanks to the administrative staff at the Cardiology dept and the staff of the PIU. Lastly, many patients gave time and effort in participating in the study. I am grateful to them all.

Abstract

One hundred and twenty three patients who were awaiting angiography for the investigation of chest pain were contacted by post and invited to participate in the study. Subjects were required to keep a chest pain diary for 14 days, and complete 5 self-report questionnaires examining physical and psychological aspects of their pain. Of the total sample of 123 patients who proceeded to angiogram, 72 (58.5%) were subsequently found to have Coronary Artery Disease (CAD) and 51 (41.5%) were found to have Normal Coronary Arteries (NCA). Seventy-two patients agreed to take part, 48 with CAD and 24 with NCA. This represents a return rate of 66.7% for CAD patients and 47% for NCA patients.

Comparison of the NCA and CAD cohorts using chi-squared and t-tests for independent samples revealed the main factors found to be significantly associated with a finding of NCA were : age (young), sex (female), non-elevated cholesterol, pain at rest , pain provoked by stress, wakening pain, relief by GTN after more than 5 minutes, and high levels of bodily awareness. Using these factors, a logistic regression was run. From this, factors which were found to be useful in discriminating between CAD and NCA patients were age, sex, somatic awareness and wakening pain. There was also found to be a lesser but consistent association with rest pain, anxiety and depression. These variables were found to correctly classify 85 % of cases. The classification of cases differed between groups with 64% of NCA cases correctly classified, and 91.5% of CAD cases correctly classified.

When the discriminatory power of this predictive equation was tested prospectively on a new sample of 74 patients (phase two) it was found to correctly predict 97.8% of the CAD cases and 58.3% of the NCA cases for an overall success rate of 89.5%. The implications of the results for the management of patients with chest pain and Normal Coronary Arteries are discussed. By inquiring routinely about psychological factors when taking a history, cardiologists would stand a better chance of anticipating which patients have an increased likelihood of having NCA. In addition to minimising unnecessary investigations, this could better prepare the patient psychologically for this finding from an early stage and allow more appropriate interventions to be more readily integrated.

Contents

Page numbers

Title		
Declaration		
Acknowledgements		
Abstract		
Contents		
<u>Chapter one</u>	Introduction	1
<u>Chapter two</u>	Methodology	26
<u>Chapter three</u>	Results	38
<u>Chapter four</u>	Discussion	74
References		
Appendix A	Letter of explanation to patient	
Appendix B	Consent form	
Appendix C	MSPQ	
Appendix D	Chest pain questionnaire	
Appendix E	Pain cognitions questionnaire	
Appendix F	Whitely index of hypochondriasis	
Appendix G	Hospital anxiety and depression scale	
Appendix H	Chest pain diary	
Appendix I	t-test tables - demographic data, phase one	
Appendix J	chi squared tables - demographic data, phase one	
Appendix K	chi squared tables - physical data, phase one	
Appendix L	t-test tables - physical data, phase one	
Appendix M	t-test tables - psychological data, phase one	
Appendix N	t-test tables - demographic data, phase two	
Appendix O	chi squared tables - demographic data, phase two	
Appendix P	chi squared tables - physical data, phase two	
Appendix Q	t-test tables - physical data, phase two	
Appendix R	t-test tables - psychological data, phase two	
Appendix S	chi squared tables for total data set traditional risk factors	
Appendix T	Test of model derived from phase two	

Chapter 1 - Introduction

The clinical features of angina have become well established since they were first described by Heberden in 1772, and are known to be associated with coronary artery disease. It has long been known however that not all chest pain can be attributed to coronary artery disease and recently more attention has been paid to the role of psychological abnormalities in chest pain without coronary artery disease.

The definitive investigation for coronary artery disease is coronary angiography. Normal coronary arteries (NCA) are now found in around 20% of all patients who undergo angiography for the investigation of chest pain (Chambers and Bass, 1990). By the time a patient reaches the stage of angiography, however, the belief in heart disease can be firmly entrenched. Angiography is also expensive and carries a low but definite morbidity and mortality. These factors underline a need for further research to identify clinical characteristics, physical and psychological, which can reliably predict a finding of normal coronary arteries prior to angiography. Prior to angiography, history taking is the first step when diagnosing chest pain. By including assessment of factors known to be associated with normal coronary arteries in the clinical interview, an estimate can be made as to the likelihood of such a finding. This can help prepare the patient, perhaps even minimising unnecessary investigations.

Previous attempts at differentiating between chest pain associated with coronary artery disease and with normal coronary arteries have emphasised organic and clinical features but there is increasing recognition of the importance of psychological factors. Much of the research to date has been done with patients who have already been through the process of angiography and are thus aware of their status. There is thus a need for a comprehensive study to further research the clinical and psychological factors associated with normal coronary arteries assessed at a pre-angiography stage.

The present study attempts to meet this need, and endeavors to provide and test a discriminatory model for the early detection of normal coronary arteries.

The study is introduced by setting the context of the investigation of chest pain with reference to its history, clinical features and aetiologies. Because the clinical features and possible aetiologies are important in the diagnosis of chest pain, considerable space is devoted to an overview of these before the role of psychological factors are detailed.

1.1 Historical Aspects

Early descriptions dating back to the middle of the nineteenth century use various terms to describe patients with cardiorespiratory symptoms which cannot be explained by ischaemic heart disease. In the American civil war, the term "muscular exhaustion of the heart" was used to describe such symptoms in soldiers (Hartshorne, 1864), a syndrome referred to as "irritable heart" by Da Costa shortly afterwards (Da Costa, 1871). These accounts predominantly focused on organic abnormalities in attempting to explain the symptoms. In 1894, however, Freud's description of anxiety neurosis emphasised the occurrence of cardiovascular symptoms. Interest in the condition seems to have been greatest at times of war, and during World War II it was concluded that a primary psychiatric diagnosis could usually be made in cases of Da Costa's syndrome (Wood, 1941). Further, Wood suggested a relationship with environmental stress. In recent years there has been an increasing recognition that psychological abnormalities are common in patients with such symptoms (eg Katon *et al*, 1988). These studies have demonstrated high levels of psychiatric morbidity in patients with chest pain but normal coronary arteries, but in essence the precise relationship and mechanisms remain unclear.

1.2 Clinical Presentation

When a patient presents to the cardiology clinic with a history of chest pain, efforts will be made to ascertain whether the presenting features indicate underlying coronary artery disease. Frequently, the patients presentation of pain is classified as 'typical' or 'atypical' of that suggesting coronary artery disease (CAD)(i.e. angina).

1.2.1 Features of angina

Anginal pain has five main characteristics : its location, its character, its relation to exercise, its duration, and its response to nitrates. Typically, anginal pain is felt in the central, retrosternal area radiating to the left arm. Although the pain may be greater elsewhere, the sternal region is usually involved to a greater or lesser extent. It is most commonly likened to a crushing, pressing feeling or a tight band. Pain, however, is not inevitable and patients may refer only to a feeling of discomfort. Angina is usually provoked by exertion, nearly always that of walking, particularly up a hill. It may be more likely after a big meal, in cold weather or windy conditions. Most attacks last 1 - 3 minutes and are usually relieved rapidly (0 - 3 minutes) by glyceryl trinitrate.

Diagnosis is largely based on this history along with evidence of inadequate coronary blood flow. The patient may be asked to exercise on a treadmill until chest discomfort is provoked. Electrocardiogram (ECG) traces are taken, and a positive exercise test is regarded as one in which the ECG shows signs (ST depression) characteristic of cardiac ischaemia (i.e. lack of blood to the heart). Often, however, history taking and exercise testing are insufficient to allow a diagnosis to be made and it is necessary to proceed to coronary angiography to establish the presence, extent, and surgical correctibility of coronary artery disease.

Angiography (also known as cardiac catheterisation) involves the introduction of a specially designed catheter into the coronary arteries (usually via the femoral artery). A radio-opaque medium is then injected and cine films are taken to observe the patterns of blood flow in the vessels. In reality angiography is usually done because the clinician expects CAD and wishes to determine if it is amenable to surgery. Although many patients undergo this investigation to allow a definitive diagnosis to be made, it is potentially hazardous and carries a mortality of 0.1 - 0.5% (Julian, 1988).

1.2.2 Features of NCA

Although clinical features vary widely, the chest pain associated with NCA is commonly:

- left-sided (often inframammary)
- not reproducibly related to exercise
- of variable duration and has been reported to last over 20 minutes in 50% of cases, sometimes persisting for days (Day & Sowton, 1976)
- associated with relief from glyceryl trinitrate, but often after more than 5 minutes, which suggests that the relief may be spurious (Chambers & Bass, 1990)
- described as sharp and stabbing in quality
- often occurring with stress or, more likely, after the completion of exercise
- coexistent with breathlessness.

The proportion of female patients is higher than those with coronary artery disease (Bass *et al*, 1983; Day & Sowton, 1976), they also tend to be younger and have fewer risk factors for CAD.

'Typicality' of chest pain (i.e. suggesting CAD) can be hard to determine as chest pain with NCA is associated with a variety of clinical findings, including :

- Atypical pain with no ST depression on exercise,
- Atypical pain with ST depression on exercise,
- Typical pain and no ST depression on exercise
- Pain at rest with ST elevation

(Chambers & Bass, 1990).

These groups may reflect differences in aetiology or outcome. "Syndrome X" is the term often used to describe patients with normal coronary arteries yet who show typical pain and ST depression on exercise.

It is evident, however, that the criteria used to classify pain as either typical or atypical are varied, and frequently not stated. Studies suggest that around 25% of patients with chest pain and NCA report pain typical of a cardiac origin, about 50% have resting ST wave changes, and 25% have significant ST-segment depression on exercise. Day and Sowton (1976) found 60% of NCA cases had pain that occasionally occurred during exercise, whilst in only 16% of cases did it occur reliably on exertion. It is therefore difficult to classify reliably the incidence of 'typical' pain.

1.3 Incidence and prevalence of NCA

Normal coronary arteries (NCA) are found in around 20% of patients undergoing angiography for the investigation of chest pain (range 6 - 31 %) (Bass *et al*, 1983).

Long-term follow up studies (Potts and Bass, 1993) show that approximately 75% of these patients continue to report pain, 50% remain unemployed or physically debilitated, and 50% continue to attend Accident and Emergency departments. Chest pain has also been found to occur commonly in the community (Hannay, 1978). Of

2717 healthy relatives of the Framingham cohort, 16% reported 'atypical' chest pain. Thus potentially up to 16% of the population might have chest pain with normal coronary arteries. Various aetiologies have been proposed, although there is little consensus about the possible mechanisms of pain production in these patients nor about how best to manage them.

1.4 Natural History

As stated earlier, about half of patients with NCA remain physically debilitated and continue to attend A & E departments. Only about one third to a half appear reassured that they do not have serious heart disease (Ockene *et al*, 1980). Most patients take cardiac medication but with little evidence of benefit (eg Potts and Bass, 1993). Persistence of pain has not been found to be related to its classification as 'typical' or 'atypical' (Kemp, 1973) or to the persistence of risk factors for ischaemic heart disease (Wielgosz, 1984). Faxon *et al* (1982) found that 'typical' pain was reported in 55% before, and in only 20% after catheterisation. This suggests that doctors were more inclined to record a non-cardiac history after the finding of NCA and that they do not take a detailed enough history first. Ockene *et al* (1980) showed a decrease in hospital admission rate by a factor of 10 in the 2 years following, compared with the 2 years preceding the finding of NCA. Attendance at Accident and Emergency departments (A&E) fell by only a factor of 3 however, which may mean that the apparent improvement can be accounted for in terms of physicians' confidence to discharge patients. The Potts and Bass (1993) study followed up 46 patients with NCA for a mean of 11.4 years. Continuing chest pain was reported by 74%, including frequent or severe pain in 38%. Fifty-eight percent had received further hospital treatment for chest pain, and 71% were taking cardiac medication. Belief in heart disease was stated in 44%. A poor outcome for chest pain was found to be associated with increased psychiatric morbidity, although there is no control group with which to draw comparison in this study.

Patients themselves are often confused by the finding of NCA. Ockene *et al* (1980) found that although 91% of patients found the diagnosis of NCA reassuring, 44% continued to believe that they had heart disease and 51% remained functionally disabled. This confusion is understandable given the mixed messages that patients often receive. As many as 50% of patients remain on cardiac medication (although sometimes this is for hypertension) which, unless an explanation is given, may negate the reassurance about the absence of coronary artery disease. As stated by Chambers and Bass (1990) "...the attitudes of both the patient and physician appear to interact and are probably more important than any other clinical characteristics in determining the persistence of pain in unselected cases with chest pain and NCA".

1.5 Cardiac morbidity and mortality

Patients with NCA have a low long-term morbidity and mortality. Myocardial infarction occurs in 1% of cases at most (Kemp *et al* 1973; Papanicolaou, 1986). The incidence of cardiac death is 0.6% after follow-up periods of up to 10 years (Proudfit, Brusckhe & Sones, 1980). By contrast, patients with coronary artery disease confined to a single vessel had a mortality of 15% at 48 months and 35% at 11 years (Veterans Administration Cooperative Study, Anonymous, 1984).

The cause of cardiac events in patients with NCA is not certain. As Chambers & Bass (1990) point out, despite the presence of risk factors for CAD, coronary stenoses almost never develop within 5 years unless there is minor pre-existing atheroma (Marchandise *et al*, 1978). Similarly, patients with NCA who die suddenly have no evidence of coronary stenoses or disease of the small arteries or arterioles (Kemp *et al*, 1973).

1.6 Aetiology

Various causes of NCA chest pain have been suggested. They can be broadly classified into cardiac and non-cardiac aetiologies.

1.6.1 Cardiac causes

Within the category of cardiac causes the basic assumption is that despite NCA, chest pain and ST depression reflects myocardial ischaemia.

These conditions include :

cardiomyopathy - damage to the heart muscle usually through inadequate blood supply which can be due to various causes including conduction defects.

small vessel disease – also known as “microvascular angina” - which is reduced coronary blood flow reserve in the arterioles.

coronary spasm – spasm of the epicardial coronary arteries.

mitral valve prolapse – a condition when the mitral valve (the valve between the right atrium and ventricle) goes “floppy”. This is often of no consequence but causes a characteristic heart murmur.

Estimates of prevalence vary and depend on the intensity with which investigation is pursued.

1.6.2 Non-cardiac causes

Non-cardiac aetiologies postulated include other medical conditions and psychological or behavioural causes :

oesophageal abnormalities
other gastrointestinal disorders
musculoskeletal pain
hyperventilation
psychological disorders i.e. anxiety, depression
psychological factors e.g. abnormal illness
behaviours, maladaptive beliefs

Associated breathlessness is reported in approximately 60% of cases (Kemp *et al*, 1973; Pasternak *et al*, 1980; Bass *et al*, 1983). The breathlessness may occur at rest when the patient might experience "air hunger" accompanied by frequent audible sighs. Hyperventilation provocation tests however, reproduce pain in only about 50% of patients who report symptoms suggestive of hyperventilation (Evans & Lum, 1977; Chambers *et al*, 1988). Hyperventilation may be an epiphenomenon in some circumstances with chest pain caused by another stress-related factor.

Although oesophageal abnormalities are commonly implicated in patients with chest pain and NCA, such abnormalities rarely coincide with pain episodes (Richter, Bradley & Castell, 1989). It has also been shown (Cooke *et al*, 1998) that the correlation of pH events with chest pain are as common in patients with angina as in patients with NCA.

More recently, attention has been paid to the relationship between hyperventilation and oesophageal motility, and the possible interaction between the two in the production of chest pain. Cooke *et al* (1996) showed that voluntary overbreathing could induce oesophageal spasm and nonspecific motility abnormalities in some patients, but that in none of the patients could the oesophagus be implicated as the source of pain because all patients remained asymptomatic. Furthermore effective treatment for an oesophageal abnormality does not always relieve chest pain. This suggests that psychological factors may play a part in the patients' experience of pain.

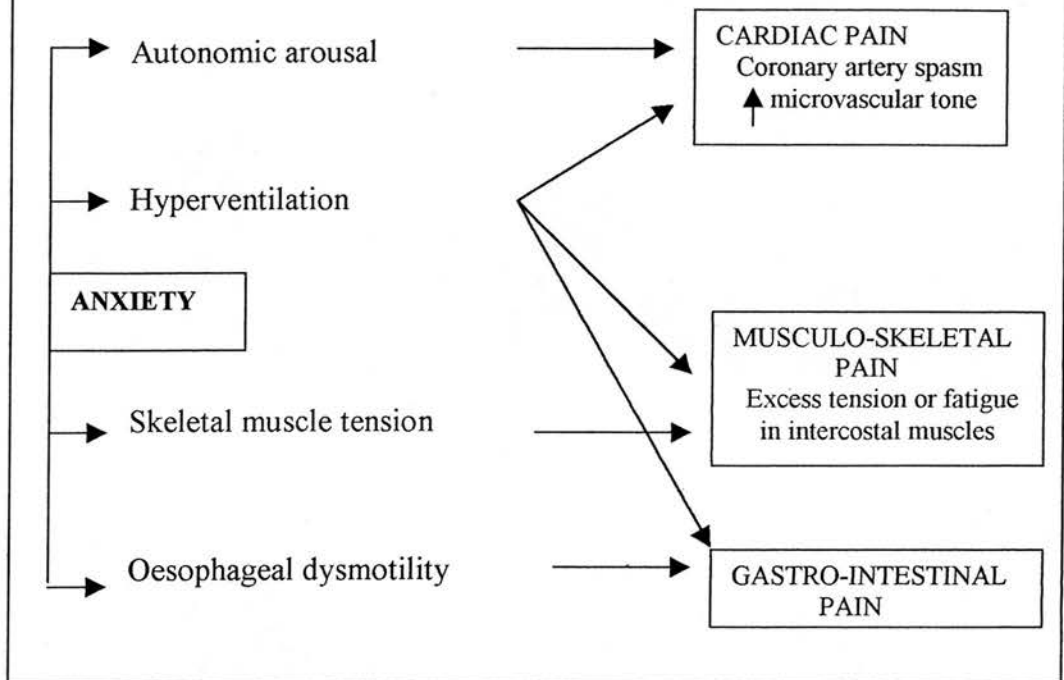
1.7 Psychological and behavioural factors

There are a number of ways in which psychological factors may be associated with chest pain.

1) A psychological disorder may indirectly be causative.

Anxiety → Physiological changes → Pain (see Figure 1).

Figure 1 : Mechanisms by which anxiety can cause chest pain



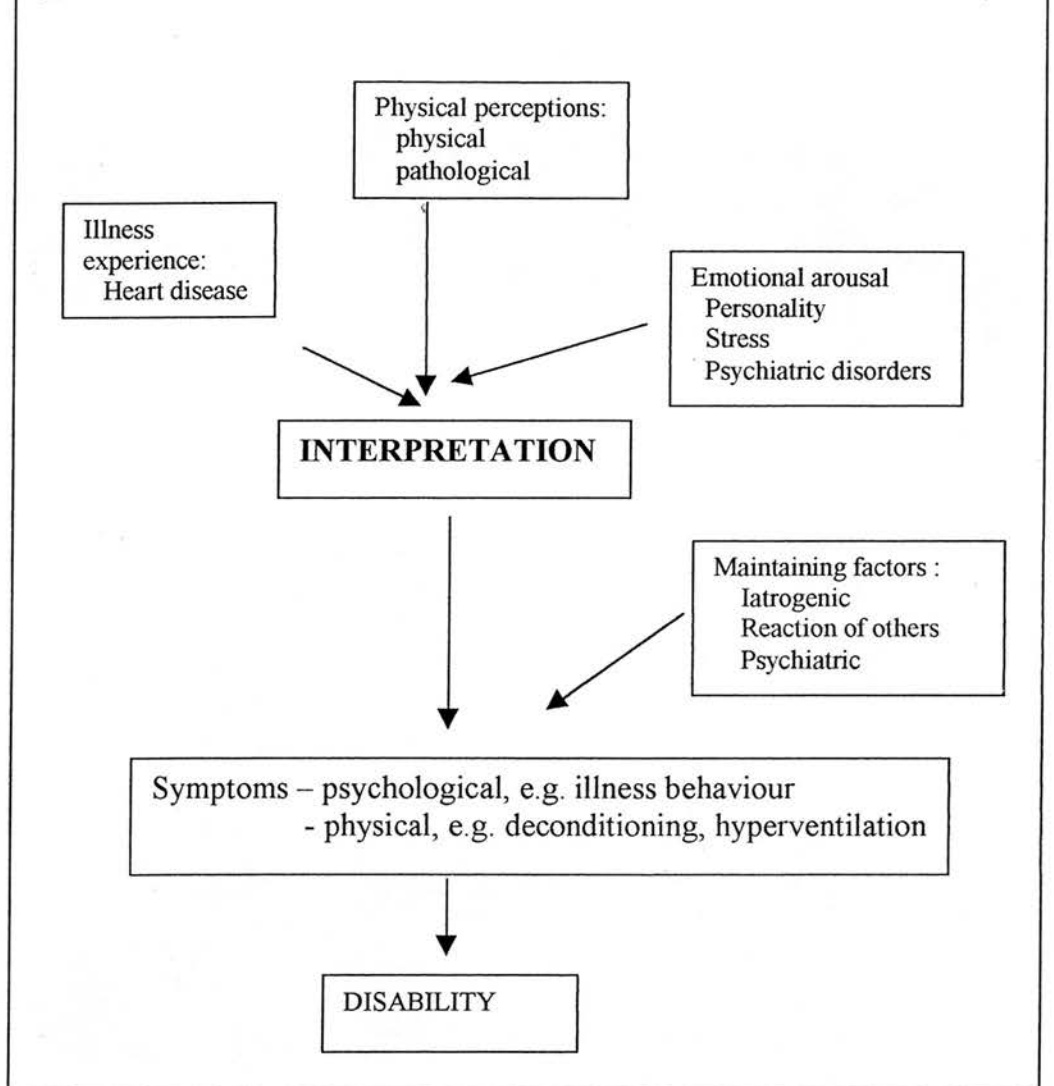
(from Potts and Bass, 1999)

Acute anxiety causes a release of catecholamines (Tyrrer, 1976), increased sympathetic activity, or both. During a panic attack large amounts of adrenaline and noradrenaline are released which leads to various physical effects including cardiovascular and respiratory changes. According to the cognitive model of panic disorder (eg. Gelder, Clark and Salkovskis 1993), the somatic accompaniments of panic are often attributed to a physical rather than a psychological cause. The fear that the perception of some serious physical pathology can bring then leads to further autonomic arousal, and so the symptoms can quickly escalate and feel uncontrollable. After this has occurred on one or more occasions, patients become sensitised to the onset of such symptoms and can become very somatically focussed. It can thus take comparatively minor symptoms, or indeed misinterpretation of entirely normal body sensations, to cause anxiety.

Because patients' subjective experience of chest pain plays such an important role in referral for cardiac catheterisation, variations in bodily sensitivity might partially explain variations in disease severity at the time of catheterisation. Perhaps people who are less sensitive to pain present later for catheterisation, and are therefore at a more advanced stage of disease progression and *vice versa*. This is supported by Frasure-Smith (1987) who found that patients with no coronary narrowing had extremely high levels of bodily awareness, and that those with very significant disease were apparently no more somatically aware than normal non-hospitalised individuals. In this study, whereas the General Health Questionnaire (GHQ) (Goldberg, 1972) did not clearly differentiate the groups, the Modified Somatic Perception Questionnaire (MSPQ) (Main, 1983) showed clear differences. Subsequent misinterpretation appears then to be perpetuated by what is seen as unclear or ambiguous medical advice (Pearce *et al*, 1990).

Since no adequate unitary explanation emerges from the current evidence, it would seem expedient to consider such a multifactorial model in which physical factors interact with psychological and environmental/experiential factors to produce the perception of pain. Such a model can be seen in Figure 2.

Figure 2 : Multicausal interactive model of pain for non cardiac chest pain
(adapted from Mayou, 1991)



Thus, it is proposed that physical factors such as palpitations or hyperventilation may lead to increased tension in the intercostal muscles and poor exercise tolerance. These symptoms, or even normal body sensations such as rapid heartbeat, are misattributed to catastrophic causes such as heart attacks through enhanced awareness of, and

selective attention to, bodily sensations. Predisposing factors include enduring personality features such as neuroticism which is known to be associated with the tendency to report somatic complaints (Costa & McCrea, 1985), and previous exposure to cardiorespiratory disease in first-degree relatives or significant others. Maintaining factors include excessive health concern, abnormal illness beliefs and family adjustment.

Pain is a common symptom in depressed patients, and atypical chest pain may occur, as with other forms of chronic pain, as part of depression. In examining the prognostic importance of depression on chest pain perception 6 months after myocardial infarction (M.I.), Ladwig *et al* (1999) identified extracardiac sources as important in the perception of chest pain and found that the presence of depressed mood was associated with an almost 3-fold risk of reporting anginal symptoms. There is contradictory evidence, however, from a study indicating that psychological biases towards or against reporting perceived symptoms do not differentiate those who experience chest pain during exercise testing from those who do not (Freedland *et al*, 1996). The authors conclude from this that silent ischaemia is probably truly asymptomatic rather than due to stoic endurance or denial of symptoms. There have been other findings however that indicate that although they do not differ on depression or global alexithymia, patients with silent ischaemia rate higher than symptomatic patients on anger control, externally oriented thinking and somatosensory amplification (Torosian *et al*, 1997). This study suggests that affective and cognitive factors, but not biomedical factors, are associated with silent, as opposed to symptomatic, ischaemia.

Clearly there is little consensus on the role of psychological factors in the experience of silent ischaemia. These cases are interesting in what we can learn from them about the factors involved in the experience of pain. We could argue that such cases

represent the polar opposite to patients with chest pain yet normal coronary arteries. In an investigation of whether patients with Syndrome X have an abnormal perception of cardiac pain, Pasceri *et al* (1998) found that patients with Syndrome X were significantly more likely to report chest pain even in the absence of cardiac stimulation (atrial and ventricular pacing) than control subjects. In addition to this tendency to complain more, they also exhibited a selective enhancement of ventricular painful sensitivity to electrical stimulation. Other studies have found no differences between NCA patients and normal controls in pain thresholds but suggest NCA patients have altered processing of noiceptive (somatosensory and visceral) inputs (Frobert *et al*, 1995).

Iatrogenic factors may also be important in maintaining the belief in disease. This can occur through various components including the process of investigation, prescribed medication, sick notes and invalidity benefits.

1.8 Iatrogenic Factors

Once coronary arteries are found to be free of significant occlusion the patient can be reassured firmly about their good prognosis in terms of cardiac morbidity and mortality. Unfortunately, however, the patient may already have developed fixed ideas about his illness in the delay between initial presentation and investigation. Performing high-technology tests may entrench the idea of serious disease even if the results are normal (Warwick & Salkovskis, 1985) by which time reassurance is largely ineffective (Channer *et al*, 1987). The diagnosis of non-cardiac pain should therefore be made with the minimum of investigation while recognising the need not to miss cases of CAD. To facilitate this, it would be useful for clinicians to have a clearer idea about the factors which are likely to be predictive of NCA. This is exactly the point of the present study, detailed later. If we can clarify which clinical features point to normal coronary arteries, the possibility of this can be raised at an early stage

with the patient in a way which is integrated with their routine clinical care and normalised.

1.9 Psychological Features

When a patient's response to a finding of NCA is not one of relief, this is a clue to an underlying psychiatric problem. Psychiatric illness occurs in 58 - 70% of NCA patients compared with only 9 - 23% in those with coronary artery disease (Potts and Bass, 1994). Of those with psychiatric illnesses, anxiety and depressive disorders are the most common. Leibing *et al* (1998) found anxiety disorders (diagnosed according to ICD-10) in 60% of patients with NCA (compared with 25% in CAD patients).

Findings that NCA patients score higher on neuroticism, and that anxiety disorders and mitral valve prolapse (MVP) may be linked led McCroskery *et al* (1991) to examine prospectively the possibility that the greater incidence of MVP among NCA patients accounts for the higher neuroticism scores. They found, however, that there appeared to be an association between NCA and neuroticism independent of MVP.

A diagnosis of panic disorder is important to establish as its somatic accompaniments are often attributed to a physical rather than a psychological cause. Katon *et al* (1988) found panic disorder to be significantly more common in those without significant coronary artery disease (43% compared with 7%). The cognitions of patients during episodes of chest pain have been found to be helpful in differentiating between panic disorder and CAD (Fraenkel, Kindler and Melmed, 1996-7). Frightening cognitions dominated the physical symptoms in 4% of CAD patients compared to 83% of the panic disorder group and 48% of the NCA group. Ho *et al* (1998) found that patients with non-cardiac chest pain and no upper gastrointestinal disease had a higher proportion of panic disorder, obsessive compulsive disorder and major depressive

disorder than patients with gallstone disease. Forty-nine percent of such patients were considered to have non-psychotic psychiatric disturbance (as measured by the GHQ) compared with 14% of patients with gallstones.

A comparison of patients with chest pain but no ischaemia during treadmill testing, with patients with pain and ischaemia, and with patients with neither found that the noncardiac chest pain patients had the highest levels of parental divorce, personal psychiatric treatment, current depression, somatic awareness, anger control and negative attitudes towards the health care system (Lumley *et al*, 1997).

Small proportions of patients with NCA chest pain have somatoform disorders. In a study of 41 patients satisfying research criteria for somatization disorder, cardiovascular symptoms were among the most frequently reported (Smith, Monson & Ray, 1986). Atypical chest pain may also occur as part of a hypochondriacal illness, usually with the belief the heart is damaged. Often such people will engage in a number of ruminations and avoidance behaviours that appear to be similar to those of other people with health anxiety and morbid health preoccupations (Rachman, 1974; Warwick & Salkovskis, 1990). Hypochondriacal pain is defined by reference to the absence of organic causes of pain, or of preoccupation with bodily symptoms, persistent irrational beliefs in illness, and repeated seeking of medical reassurance of the absence of severe illness. As highlighted by Salkovskis and Warwick (1986) in their cognitive model of hypochondriasis, reassurance allays anxiety only in the short term and ultimately perpetuates fears.

1.10 I Illness behaviour

1.10.1 Sick role

The concept of 'illness behaviour' (Pilowsky, 1978) incorporates hypochondriasis in addition to disease conviction, affective inhibition, affective disturbance, somatic versus psychological perception of illness, denial, and irritability. 'Illness behaviour' as a concept was originally formulated by Mechanic in 1961, and refers to the way in which symptoms may be differentially perceived, evaluated and acted (or not acted) upon. Abnormal illness behaviour occurs when there is a fundamental discrepancy between the objective pathology present, and the patient's response to it. The original Illness Behaviour Questionnaire (Pilowsky, 1967) exists as 62 items, however a shorter 14 item version - the Whitely Index of Hypochondriasis can be used.

Normative data exist for several clinical populations, "hypochondriacs" and normals. More recently, a measure called the Illness Attitude Scales has been validated on chest pain patients and found to comprise 2 reliable subscales of health anxiety and illness behaviour (Dammen, Friis and Ekeberg, 1999). Eifert *et al* (1996) found NCA patients to exhibit more hypochondriacal beliefs and obsessive-compulsive concerns than all other groups including cardiac inpatients.

Individuals with chronic chest pain may rest and avoid activity, because they are initially advised to, due to worry about causing further 'damage', or for pain relief. Loss of cardiovascular fitness and respiratory function may ensue, providing further evidence for the patient's belief in heart trouble. The effect of avoidance will also undermine the patient's confidence and reinforce the association of anxiety reduction with abstinence from exertion. Inactivity, in itself, can lead to boredom and depression, which may contribute directly to the experience of pain, or indirectly through enhanced preoccupation with bodily symptoms. A patient may be more likely to remain in the sick role if anti-anginal medication is continued and if the patient is in receipt of disability payments.

1.10.2 Help-seeking behaviour

The health belief model (HBM) could be applied to the perception of chest pain with NCA. This model was developed by Becker (1974) to explain and predict health-related behaviour. Hence, internal cues (the perception and misattribution of chest pain) and external cues (such as family history of CAD or health education messages) will determine the behaviour of seeking medical help. The precise relationship between combinations of variables and behaviour is, however, unclear.

Health promotion strategies stress the need for rapid attendance at hospital for treatment of possible MI. Treatment effectiveness (e.g. thrombolytic therapy) is maximised with early intervention. This message may, however, lead to undue anxiety about chest pain and increase the likelihood of excessive symptom monitoring in individuals with those tendencies. This may suggest that there is an argument for more detail in such health promotion messages to educate people in recognising cardiac symptoms and excluding other symptoms. The benefits of such a development are debatable, however. There is emerging evidence (Foster and Malik, 1998) that women frequently have longer pre-hospital delays following a MI than men, suggesting that men are more ready to believe their chest pain is indicative of a heart attack. The majority of patients with chest pain subsequently found to be non-cardiac are women however. The implication of this is that men are better at correctly identifying the basis of their chest pain and that women are poor at this. Women with cardiac pain take longer to perceive (or act on) their symptoms whereas women with non-cardiac pain are more likely to misinterpret their symptoms as being cardiac in origin. Although improved health promotion information may improve female morbidity and mortality following genuine MI, unless effective in enabling people (targeting women in particular) to differentiate causes of chest pain it may also

increase the numbers of women attending chest pain clinics and A and E departments with non-cardiac pain.

A wider issue relates to experience of, and access to, specialist services. There is some evidence (e.g. Chaturvedi , Rai and Ben-Shlomo, 1997) that improvement in awareness of cardiac symptoms may not decrease delays in obtaining care.

Chaturvedi , Rai and Ben-Shlomo (1997) found that Hindus and Sikhs reported a greater likelihood of seeking immediate care for anginal symptoms than Europeans but they experience greater delays in the UK in obtaining specialist management for heart disease. Certainly in groups of ethnic origin, and possibly in other groups, this implies that delays in receiving care may relate to barriers in cardiology services which are unrelated to difficulties in interpretations of symptoms.

1.11 Differentiating cardiac and non-cardiac chest pain

Various attempts have been made to find valid instruments to assist in the discrimination of cardiac and non-cardiac chest pain. In a case control study, Bennett, Smith and Gallacher (1986) compared patients admitted to hospital with confirmed MI, those admitted with chest pain but no evidence of MI, and normal control subjects. They found that both a measure of 'vital exhaustion' (the Maastricht Questionnaire) and of physical symptoms (Pennebaker Inventory of Languid Languidness) failed to discriminate between cardiac and non-cardiac chest pain patients, only between patients and controls. Their results were however consistent with previous findings of strong associations between neuroticism and symptom reporting (Bass and Wade, 1984; Costa and McCrae, 1985).

An Italian study examined the relationship between psychological status and clinical symptoms in 22 patients with Syndrome X (angina and ST depression with

angiographically normal coronary arteries) compared with 30 patients with stable angina and CAD (Ruggeri *et al*, 1996). They found that patients with Syndrome X scored significantly higher on the Beck Depression Inventory, Hamilton Anxiety Rating Scale, State-Trait Anxiety Inventory, Sheehan Patient Rated Anxiety Scale, Brief Psychiatric Rating Scale, but not on the State-Trait Anger Expression Inventory. Evidence suggested that a high degree of anxiety correlated with increased transient myocardial ischaemia during daily life. The possibilities are therefore that neuroticism may itself cause changes in coronary microvascular function in Syndrome X, alternatively that it may modulate the threshold for ischaemia in the presence of underlying dysfunction.

Comparing 67 patients with NCA and 47 CAD patients, Serlie *et al* (1996) found the non-cardiac patients to be significantly younger, more often female, single and non-smokers. They also found this group to differ significantly on anxiety, somatization, obsessive compulsive behaviour, psychoneuroticism and hyperventilation. Age, gender, anxiety and hyperventilation were found to contribute significantly to the model for discriminating between the two groups. The model had an overall correct classification of 75.4%. One potential flaw of this study, however, is that it appears that patients had already undergone extensive cardiological testing in order to ascertain the nature of the chest pain prior to participating in the study, and were therefore aware of the outcome of their angiogram.

A similar criticism could be leveled at Cooke, Smeeton and Chambers (1997) who compared chest pain characteristics in 65 patients found to have NCA with 65 sex matched CAD patients. A standardised questionnaire modified from Master was used to record demographic details and chest pain characteristics. There were no important differences between the groups in the site, radiation or quality of pain. Only three symptom variables were of statistical value in separating the groups: the

reproducibility of pain with physical activity; the occurrence of unprovoked pain at rest; and the usual duration of pain episodes. Patients with NCA were less likely to report a consistent relationship of pain provocation with exercise, more likely to experience unprovoked pain at rest and report pain of longer duration. They found that these 3 symptoms had discriminatory value when expressed in a binary fashion. They classed the symptoms as either “typical” or “atypical” with the criterion of typicality of exertional pain being a reproducibility index of 10/10 (i.e. pain was reproduced by exercise on 10 out of 10 occasions). Pain duration was classed as typical for 5 minutes or less. Unprovoked rest pain was considered typical if it was reported in 0-10% of pain episodes. They found all three symptoms to be atypical in 21 (32%) of patients with NCA but only in one patient with CAD. There was a 2% chance of CAD if patients had no typical features and were under 55 years old, and a 12% if over 55 years old.

Ladwig, Hoberg and Busch (1998) collected data on depression, anxiety and somatic complaints in 77 subjects awaiting angiography. They also found that the prevalence of emotional disorders was markedly more pronounced in both groups found to have NCA and CAD in comparison to the normal population, but that those features did not discriminate between the groups. This study also found that “long acting” chest pain was predictive for high degrees of emotional disability and that chest pain at rest was a major source of anxiety, depression and subsequent somatic preoccupation irrespective of its ischaemic or functional origin. By contrast, in a prospective study, Leibing *et al* (1998) found anxiety symptoms were predictive of coronary status, with the Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983) to be a particularly useful predictive tool. They suggest that a screening measure for anxiety and a standardised interview would be both practicable and beneficial within the medical clinic.

It would therefore appear that there is a body of evidence supporting the role of psychological factors in chest pain with normal coronary arteries. There is not, however, a comprehensive study which examines these factors together, before the angiography procedure, and at a point and location out of hospital (and thus more distant from the investigation and management of the patients chest pain). The present study aims to fill this gap.

1.12 Management implications

Treatment for individuals with chest pain and NCA is often difficult. The heterogeneous nature of this group can mean a variety of further investigations and medical treatments are undertaken. It has been noted, however, that effective treatment for an observed abnormality does not always lead to relief of the chest pain.

Several authors have suggested that psychological treatments for those patients with persistent pain is appropriate (Bass *et al*, 1983; Ockene *et al*, 1980; Lantinga *et al*, 1988). Indeed, the interactive model proposed above would seem to indicate various points for psychological intervention. Until recently, there have been few systematic trials of such treatments, however Klimes *et al* (1990) describe a cognitive-behavioural treatment and show it to be effective in the management and relief of chronic non-cardiac chest pain. Relief of chest pain in this study was associated with improvements of mood, reductions of limitations of daily life, and change in beliefs about the cause of symptoms. The treatment was effective for patients both with and without overt anxiety disorders. In a randomised control trial, group CBT was also found to be effective for the management of NCA chest pain (Potts *et al*, 1999). Patients who underwent group treatment showed improvements in frequency and severity of chest pain, less psychological distress, reduced functional disability and improved exercise tolerance.

Before such treatment can occur however, it is important to prepare the patient at an early stage (pre-angiogram if possible) for the finding of normal coronary arteries, and the possible role of psychological factors. Mayou, Bass and Bryant (1999), recognising this, and that psychological treatment is not always feasible or acceptable in routine clinical settings, studied the effects of reassurance by cardiologists with and without objective evidence of normal coronary arteries (i.e. an angiogram). Patients who were offered reassurance following angiography were more likely to have a longer history of chest pain, report exertional breathlessness, have had their pain previously labeled as angina, prescribed anti-anginal medication and been admitted to hospital as an emergency. They found that outcome was good at 6-week follow-up, although most patients had persistent, clinically significant symptoms and distress. Those that had been offered (group) psychological treatment found it helpful, although 15% appeared to need more intensive individual therapy. They suggest that a “stepped” model of aftercare would be useful with management tailored according to clinical need. Chambers and Bass (1998) suggest patients found to have NCA should be reassessed 4-6 weeks later when management decisions should be made according to this stepped approach. They propose a multidisciplinary chest pain clinic run jointly by a cardiologist, psychiatrist (and possibly a gastroenterologist). The presence of a psychiatrist seems to be more acceptable to patients at such a combined clinic than having to be referred to another part of the hospital. In a study assessing to what extent patients with unexplained chest pain are interested in a “medical psychological treatment”, van Peski-Oosterbaan *et al* (1998) found that younger patients and males were most interested in treatment and that limitations in activities rather than frequency or intensity of chest pain was the most important predictor of interest in treatment.

1.13 Aims of the study

The study has been carried out in 2 phases. The first phase aims to characterise prospectively (i.e. advance of angiography) physiological and psychological features of those patients referred to angiography with chest pain suggestive of CAD and subsequently found to have normal coronary arteries. These features will be assessed outwith the cardiology setting. The study will also establish a set of variables indicative of increased probability of normal coronary arteries. This is important in order to identify which factors are indicative of such a finding so that they can be raised in advance with the patient, and so that medical investigations which carry a small but definite risk can be minimised. Clarification of the factors characteristic of NCA in patients prior to angiography may also help to suggest the role of various psychological factors in the experience of chest pain with NCA. The question this phase of the study sets out to answer is then : what factors are associated with patients with NCA?

Once a set of variables has been established as differentiating normal coronary arteries from coronary artery disease in phase one, this set will be applied to a new data sample. The objective of phase two was therefore to test the predictive power of the statistical model based on demographic and psychological variables that can, in an early stage, quantitatively discriminate between chest pain patients with diseased coronary arteries and NCA patients.

Chapter 2 - Methodology

2.1 Phase 1

Approval for this study was sought and obtained from the Lothian Area Ethics of Medical Research Committee.

2.1.1 Objectives

The purpose of this phase of study was to identify physiological and psychological factors which significantly discriminate between patients with chest pain undergoing angiography who are found to have NCA, and those who are found to have Coronary Artery Disease (CAD). Those factors were then used to derive a predictive model.

2.1.2 Design

The design was a prospective cohort case control study of patients with chest pain referred to cardiology clinics who subsequently undergo coronary angiography. The result of angiography then indicated which subjects were cases with Normal Coronary Arteries, or controls with Coronary Artery Disease. Cases of insignificantly narrowed coronary arteries were classed as “normal” for the purposes of this research. Because of the low numbers in this group it was not feasible to examine this as a separate group, and made more sense to class this with the entirely normal coronary arteries since all were experienced symptoms unexplained by a purely cardiac aetiology.

2.1.3 Setting

Subjects were recruited from two cardiology units in Edinburgh teaching hospitals - the Royal Infirmary and the Western General Hospital. Both units run a 'diary' system of booking in patients for coronary angiography, and it was from this that potential subjects were identified.

2.1.4 Pilot study

Before recruitment for the main study was commenced, a one month pilot study was undertaken at the Programmed Investigation Unit of the Royal Infirmary. This is the unit patients are admitted to when they come in for scheduled procedures including coronary angiography. The purpose of this developmental phase was to ascertain whether the content and quantity of the questionnaire measures to be used was suitable for the subject group, and whether their responses on those measures was sufficiently reliable. This was achieved by the researcher approaching patients during their admission and conducting a structured interview, comprising the various questionnaires intended for use. This was particularly important for the Chest Pain Questionnaire (see section 2.9), which was being developed in self-report format in this study. Over the pilot study period, 20 subjects were recruited. In addition to answering the questionnaires part of the interview, each subject was asked to comment on the following aspects : overall ease of completion of each measure, specific ambiguities in any question or response format, total time required to complete the questionnaires, and any objections they would have if this was sent to their home. Information gained from this part of the study was used to inform and modify measures to be used. In particular, some questions in the Chest Pain Questionnaire were found to be difficult to understand and were altered accordingly.

2.1.5 Subjects

The study recruited 144 consecutive patients referred to two Edinburgh hospitals between 8 December 1994 and 1 June 1995. Each patient's symptoms were considered sufficiently consistent with CAD by their cardiologist to warrant angiography. Patients attending the cardiology clinics who had chest pain but who were not considered for angiography were not included in the study.

2.1.6 Inclusion criteria

Patients recruited in the study were aged between 18 and 80 years old. They were required to be proceeding to coronary angiography for the first time.

2.1.7 Exclusion criteria

Patients with previous evidence of myocardial infarction, valvular heart disease, cardiomyopathy, or clinical or x-ray evidence of congestive heart failure were excluded from the study. Such patients would be expected to differ in their experience of symptoms or differ in pain attributions due to previous experience. Also excluded were those with previously demonstrated evidence of occlusions, patients who had undergone bypass surgery or coronary angioplasty, and those with insufficient knowledge of English to understand the questionnaires. Patients referred from Coronary care were not included due to both practical reasons and because of possible attributional bias.

2.1.8 Procedure

Subjects were contacted by post **at their home** 2 - 3 weeks **before** they were due to be admitted to hospital for their angiogram. The prospective nature of the assessment, and the assessment being outwith the cardiology clinics are probably key features of the study. At this stage, information on age and cardiac history was obtained from clinical records to allow assessment of eligibility for entry into the study. An explanation of the study was sent, along with a number of standardised questionnaires and a pain recording diary. The explanation of the study was signed by the researcher, supervisor and the patient's cardiologist (Appendix A). Patients were also required to sign and return a brief consent form (Appendix B).

Where possible, patients were contacted by telephone 2 - 5 days prior to their admission to answer any questions they may have and (if appropriate) to prompt them to return the questionnaires.

Patients were asked to complete the questionnaires before their admission and hand them in to the ward on their arrival.

Angiogram data were obtained from discharge summaries. Patients were classified

by independent cardiologists as having either : significant coronary artery disease (defined as having greater than 50% blockage in one or more vessels) ; or insignificantly narrowed or normal coronary arteries.

Information pertaining to chest pain characteristics, CAD risk factors (smoking hypertension, diabetes, obesity, hyperlipidaemia and family history of IHD), relevant medical history, investigations such as ECG, exercise tolerance and thallium scan, and psychiatric history (if noted) was also collected from discharge summaries.

2.1.9 Measures

Data were obtained using the following self-rating scales : the Modified Somatic Perception Questionnaire (MSPQ; Main, 1983), the Chest Pain Questionnaire, the Pain Cognitions Questionnaire (PC; Boston et al, 1990), the Whitely Index of Hypochondriasis (WI; Pilowsky, 1967), the Hospital Anxiety and Depression Scale (HAD; Zigmond and Snaith, 1983), and chest pain diaries.

Modified Somatic Perception Questionnaire (Main, 1983) (Appendix C)

This questionnaire was devised originally as a measure of awareness of bodily functioning for patients with chronic back pain. It has since been used as a measure of somatic awareness in patients prior to cardiac catheterisation (Frasure-Smith, 1987).

The MSPQ consists of 13 somatic and autonomic symptoms which are not directly cardiac related. Subjects are asked to rate each symptom on a 4 point scale (not at all, a little, quite a bit, extremely) according to how they have felt over the past week.

Chest Pain Questionnaire (Appendix D)

This questionnaire was derived from that devised by Master (1964). This questionnaire has previously been used and found to be useful by Bass (1984) in a similar context and was recently used by Cooke, Smeeton and Chambers (1997). It was adapted to self-report format and piloted during interviews with a separate sample of 20 patients during their admission for coronary angiography (see section

2.4).

The resulting questionnaire contains a total of 23 questions covering the frequency, duration, site, quality and radiation of pain, as well as other details such as precipitating and relieving factors, medication, associated symptoms, relationship of pain to exertion, and whether the pain woke the patient from sleep. Subjects were required to select the most appropriate answer for each question, and to indicate site and spread on a simple diagram. Cooke, Smeeton and Chambers (personal communication, 1994 and subsequently published in 1997) carried out a study comparing chest pain characteristics in patients with NCA with CAD controls. They found simple questions about the consistency of with which pain was reproduced by exercise ("typical" 10/10), the duration of pain episodes ("typical" 5 minutes), and the frequency of unprovoked rest pain ("typical" 10% of pain episodes) to provide the most important diagnostic information. Two additional questions were therefore included in the Chest Pain Questionnaire. These were tested and modified in the pilot phase, resulting ultimately in the following questions:

"Would you expect to get pain every time you climb a steep hill?"

and

"If no, on how many occasions out of ten would you expect to get chest pain?"

to assess the consistency with which pain was reproduced by exercise; and

"Of your last ten episodes of pain, how many occurred at rest?"

to assess frequency of spontaneous rest pain.

Pain Cognitions Questionnaire (Boston et al, 1990) (Appendix E)

This is a standardised 30 item questionnaire assessing thoughts which patients have had at the time of their pain over the past week. Subjects respond on a 4 point scale (not at all, some_times, often, most of the time). Analyses of the questionnaire has yielded 4 factors, forming the following scales :

- distraction/reassurance
- hopelessness
- helplessness

- support/trust

Whitely Index of Hypochondriasis (Pilowsky, 1967) (Appendix F)

This is the precursor to the 62 item Illness Behaviour Questionnaire. It comprises 14 items, yielding a total score and the following factors :

- bodily preoccupation : increased awareness of symptoms of somatic concern
- disease phobia : a fear response in which a person may ask for reassurance about conditions which he will often admit he does not really believe he is suffering from.
- disease conviction : a belief which is characterised by firm conviction of the presence of serious pathology accompanied by a paranoid attitude to relatives and medical staff.

Subjects are asked to answer each question on a simple yes/no basis.

The Hospital Anxiety and Depression Scale (Zigmond and Snaith, 1983) (Appendix G)

This well established scale has been used as a screening instrument for anxiety and depression in non-psychiatric populations. It's validity and reliability have been demonstrated (eg Mooney et al ,1991). It consists of two 7 item scales in which subjects answer 12 questions indicating their response on a 4 point scale. A score of 10 or above is commonly taken as indicative of "caseness". It has been used in many clinical populations and minimises the effects of concurrent physical illness.

Chest Pain Diary (Appendix H)

Subjects were asked to complete pain recording diaries for the last 14 full days preceding their angiogram admission. This gave measures of average duration, severity and frequency of pain episodes, along with total GTN use and pain free days.

2.1.10 Analysis

The characteristics of the patient groups were examined using descriptive statistics

including measures of central tendency, dispersion, distribution and shape. For parametric data, t-tests for independent samples were used to compare group means, and establish the significance of any differences. One-tailed tests were used where the direction of the difference was being predicted; two-tailed tests were used for the remainder. Chi-squared analyses were done on non-parametric data. The Statistical Package for Social Scientists (SPSS) version 4 for Mac was used for all analyses. To allow comparison with the results of Cooke, Smeeton and Chambers (1997) the variables of exertional pain, pain duration and rest pain were also examined according to their classification as “typical” (i.e. of angina). Data from phase I variables of pain duration, pain at rest, and exertional pain were therefore recoded into new variables to create comparable indices.

Variables were then entered into a logistic regression to explore predictors of group membership (angiogram result). Any significant differences on extraneous variables were controlled for. Variables were entered into the equation in both a forward step wise, and a backward step wise manner. Variables were included in the final equation only if they made an independent contribution to predicting group (NCA vs CAD) membership.

A logistic regression was also used to explore predictors of participation in the research. The patients were classified as responders or non-responders and variables predictive of this were modelled.

2.2 Phase 2

The results from phase 1 of the study signaled the differences between the 2 groups of chest pain patients but more work was needed to quantitatively test the model in which the variables with differential potential are entered, thereby allowing for a quicker diagnosis and more tailored treatment. The objective of this phase was therefore to test the predictive power of the statistical model on a new patient sample. If validated the model based on demographic and psychological variables can, at an early stage, be used to quantitatively discriminate between chest pain patients with diseased coronary arteries and NCA patients.

Approval for this phase of the study was again sought and obtained from the Lothian Area Ethics of Medical Research Committee.

2.2.1 Objectives

In addition to the objective of testing the predictive model derived in phase 1 on a new patient group, it was also thought useful to combine the data from phases one and two to allow the exploration of a model useful for discriminating NCA from CAD patients in this larger sample.

In addition, less central aims would be to do the following :

- derive a predictive equation from phase 2 data
- test the phase 2 model on phase 1 data

The aim was also to assess the accuracy of cardiologists predictions of angiogram result, and, for those found to have NCA, to explore cardiologists explanations for chest pain. The hypothesis would be that for NCA patients whom cardiologists had

predicted to have CAD, they would be more likely to attribute pain to a cardiac (e.g. microvascular spasm) than to a non-cardiac cause.

2.2.2 Design

The design was a prospective cohort case control study of patients with chest pain referred to cardiology clinics who subsequently undergo coronary angiography. The result of angiography then indicated which subjects were cases with Normal Coronary Arteries, or controls with Coronary Artery Disease.

2.2.3 Setting

Subjects were recruited from the cardiology unit in Edinburgh's Royal Infirmary.

2.2.4 Subjects

The study recruited 110 consecutive patients referred to Edinburgh Royal Infirmary 17 April 1997 and 27 November 1998. Each patient's symptoms were considered sufficiently consistent with CAD by their cardiologist to warrant angiography. Patients attending the cardiology clinics whom had chest pain but who were not considered for angiography were not included in the study. Five (from a potential 6) consultant cardiologists agreed that patients from their list could be approached.

2.2.5 Inclusion criteria

Patients recruited in the study were aged between 18 and 70 years old. They were required to be proceeding to coronary angiography for the first time.

2.2.6 Exclusion criteria

As for phase 1.

2.2.7 Procedure

As before, subjects were contacted by post at their home 2 - 3 weeks before they were due to be admitted to hospital for their angiogram. At this stage, information on age and cardiac history was obtained from clinical records to allow assessment of eligibility for entry into the study. An explanation of the study was sent, along with a number of standardised questionnaires. The explanation of the study was signed by the researcher and the patient's cardiologist (Appendix A). Patients were also required to sign and return a brief consent form (Appendix B). Patients were asked to complete the questionnaires before their admission and hand them in to the ward on their arrival.

Because of problems encountered attempting to telephone 2 - 5 days prior to their admission to prompt them to return the questionnaires, and because it did not appear to improve compliance, this was not attempted in phase 2.

Angiogram data and information on CAD risk factors were obtained from discharge summaries, as described for phase one.

To assess cardiologists predictions of the angiogram result a pro forma (Appendix) was attached to the patients notes, which the cardiologist was asked to complete prior to carrying out the angiogram. The sheet was then detached from the notes and retained in the Cardiology department for collection by the researcher.

2.2.8 Measures

Data were obtained using the following self-rating scales (as detailed in section 2.1.9): the Modified Somatic Perception Questionnaire (MSPQ; Main, 1983), the Chest Pain Questionnaire, the Whitely Index of Hypochondriasis (WI; Pilowsky, 1967) and the Hospital Anxiety and Depression Scale (HAD; Zigmond and Snaith, 1983). The Pain Cognitions Questionnaire (PC; Boston et al, 1990) had not been found to be useful in

phase 1 of the study, apparently yielding no association with angiogram outcome (see Chapter 3), and its use was therefore discontinued in phase 2. The results from phase 1 also indicated that the chest pain diaries were neither reliably nor consistently completed by patients, such as to render their use ineffective. It was also considered possible that the daily completion of such diaries was sufficiently taxing for patients that it was deterring them from taking part in the study. The omission of the diaries in phase 2 was therefore also in the hope of improving response rates.

2.10 Analysis

The characteristics of the patient groups were examined using descriptive statistics including measures of central tendency, dispersion, distribution and shape. For parametric data, t-tests for independent samples were used to compare group means, and establish the significance of any differences. One-tailed tests were used where the direction of the difference was being predicted; two-tailed tests were used for the remainder. Chi-squared analyses were done on non-parametric data. The Statistical Package for Social Scientists (SPSS) version 7.5 for windows was used for all analyses.

To allow comparison with the results of Cooke, Smeeton and Chambers (1997) the variables of exertional pain, pain duration and rest pain were also examined according to their classification as “typical” (i.e. of angina). The criterion these authors used to define typicality is described previously. Data from phase 2 variables of pain duration, pain at rest, and exertional pain were therefore recoded into new variables to create comparable indices.

Variables were then entered into a logistic regression to explore predictors of group membership for phase 2 data. Any significant differences on extraneous variables were controlled for. Variables were entered into the equation in both a forward step-wise manner. Variables were included in the final equation only if they made an independent contribution to predicting group (NCA vs CAD) membership.

The logistic regression model derived from phase 1 data was tested with phase 2 data and classification rate determined for this new patient sample. Likewise, the model derived from phase 2 was rerun with the phase 1 data.

The samples were then combined to give one big data set. A logistic regression was run using the variables age and sex alone. This was done both for the total data set of responders and non-responders, and for the data on the patients who responded only. To this core model, different variables which had previously been found to be associated with angiogram result were added and removed according to the significance of their added predictive power.

Chapter 3 - Results

Phase one

3.1.1 Description of data

During the recruitment period of phase one, 147 patients were invited by letter to take part in the study. 21 were however, subsequently found to have had previous M.I.s therefore were excluded from the study. Of the remaining 126, 105 patients were recruited from the Royal Infirmary, and 21 from the Western General Hospital. Overall, 51 patients did not reply and 3 did not attend for angiography. The final sample size of 72 (from a possible 123 – 126 minus the 3 whose angiograms did not proceed) therefore represents a compliance of 58.5%.

Response rates differed between the groups. 66.7% of the CAD cohort agreed to participate in the research compared to 47.0% of the group subsequently found to have NCA. Using a chi-squared test this was found to be a significant difference (Pearson chi-square 4.73, df 1, $p < 0.05$). It should be noted that at the time of participating in the study that both the subjects and the researchers were unaware of which group the patient belonged to.

Missing data were coded as such and excluded from the analyses. Exploratory data analyses were performed using SPSS Release 4.0 for Macintosh (as detailed in Chapter 2). The data were summarised and examined with frequency distributions and histograms. For quantitative variables, the distribution appeared to be acceptably normally distributed to allow the use of parametric statistics. Descriptive statistics, including means, medians, standard deviations, and range were calculated for each quantitative variable.

3.1.2 Demographic data

Basic demographic information such as age and sex was obtained for all subjects including those who did not reply. Data on angiogram result, CAD risk factors and investigations were also obtained for all patients.

The mean age for the total sample (including non-responders) was 57.2 years, with a range of 35 to 70 years. 69 patients (56.1%) were male; 54 (43.9%) were female. There were no significant differences between the responders and the non-responders on sex (Pearson chi-square 0.78, df 1, p=.38). There was, however, a significant difference on mean age (responders mean age 59.6 years; non-responders mean age 54.8 years, $t=3.04$, df 121, $p=.003$, two-tailed).

Of the total sample of 123 patients who proceeded to angiogram, 72 (58.5%) were found to have Coronary Artery Disease and 51 (41.5%) were found to have Normal Coronary Arteries. 63.9% of the CAD group were male (36.1% female), compared with 45% of the NCA group (55% female). The mean age of patients was 58.9 years for those who were found to have CAD, and 53.9 years for the NCA group. Characteristics of the two groups are displayed in Table 1. Using a t-test for independent samples, age was also found to be significantly different between the groups ($t= 3.25$, df 121, $p<0.001$ one-tailed) (see Appendix I).

Table 1 : Demographic characteristics of the NCA and CAD groups.

Variable	<u>NCA group</u>		<u>CAD group</u>	
	Mean	SD	Mean	SD
Age	53.88	7.87	58.94	8.92

A chi-squared test indicated that sex significantly differentiated the CAD and NCA groups, at a significance level of $p<0.05$ as can be seen in Table 2 .

Table 2 : Chi Square results

Summary Information				
Demographic variable	NCA group	CAD group	Pearson Value	Sign.
	Percentage	Percentage		
Sex - Male	44	67	4.28	.007
Marital status - single	8	6	N/A *	N/A
- married	76	76	N/A	N/A
Divorced/separated	16	6	N/A	N/A
Widowed	0	11	N/A	N/A
Living status –alone	8	19.4	N/A	N/A
- spouse/partner	68	73	N/A	N/A
- spouse + kids	16	5	N/A	N/A
- parents	4	2	N/A	N/A
- kids	4	2	N/A	N/A
Employment status - full-time	24	26	N/A	N/A
- part-time	4	0	N/A	N/A
-sick leave	8	11	N/A	N/A
-longterm sick	8	10	N/A	N/A
- retired	28	42	N/A	N/A
- housework	20	2	N/A	N/A
- unemployed	8	10	N/A	N/A
Employment change – given up work	21	26	N/A	N/A
- lighter duties	17	12	N/A	N/A
- reduced hours	8	10	N/A	N/A
- no change	54	51	N/A	N/A

* Where Pearson values are not available this is due to chi-squared analyses not carried out because coding of the variable render this inappropriate

76% of both the NCA and the CAD groups were married. 24% of the NCA group were in full-time employment compared with 26% of the CAD group. Of the NCA group, a further 16% were on sick leave and 28% retired. The corresponding figures for the CAD group are 21% on sick leave and 42% retired. With the exception of employment status, these variables were not found to significantly differentiate the groups, as can be seen in Table 2. Full chi-squared matrices can be found in Appendix J.

3.1.3 Physical data

Summary information for non-parametric data on physical measures (pain characteristics etc) are shown in Table 3. Chi-squared analyses were performed comparing this data for the NCA group with the CAD group. Full details of the results of this procedure can be found in Appendix K.

Table 3 : Chi squared results

Summary Information				
Demographic variable	NCA group	CAD group	Pearson	Sign.
Smoking	Percentage	Percentage	Value	
- smoked in lifetime	60	71	.98	.32
- current smoker (reported by physician)	26	21	N/A	N/A
- previous smoker (reported by physician)	10	30	N/A	N/A
- current smoker (self report)	32	14	3.46	.06

Summary Information				
Demographic Information	NCA group	CAD group	Pearson value	Sign.
	Percentage	Percentage		
Obesity- risk	22	18	.46	.50
Lipid status- risk	18	46	10.56	.01
Diabetes- risk	2	5	.62	.43
Hypertension- risk	29	31	.06	.81
Family history- risk	41	38	.13	.72
Positive exercise test	44	85	14.60	.01
Abnormal ECG	50	57	2.55	.28
Effective GTN relief	73	91	3.95	.05
Pain when alert	96	59	10.90	.01
Wakening pain	67	35	6.82	.01
Stress provoked	68	42	3.98	.05
Cold provoked	59	60	.01	.94
Food provoked	14	13	.01	.97
Sex provoked	14	13	.01	.97
Stooping provoked	55	42	.90	.34

Lipid status was found to be a significant differentiator, with the CAD group being more likely to have a clinically high lipid level ($p < 0.01$). Pain when alert was found to be significantly higher ($p < 0.01$) in the NCA group than the CAD group. Wakening pain was also significantly more associated with NCA than CAD ($p < 0.01$).

The NCA group reported their chest pain to be more likely to be stress provoked ($p < 0.05$) than those with CAD. The CAD group were significantly more likely to have a positive exercise tolerance test ($p < 0.01$). The CAD found more pain relief derived from GTN use ($p < 0.05$). No other significant differences on physical

variables were found between the groups. Crosstabulations for other pain characteristics including pain frequency, suddenness of pain onset, pain duration, pain site, pain type, pain aggravators and pain spread can be found in Appendix K. These did not show marked differences between the groups. 35% of the CAD group reported their pain as lasting less than 5 minutes compared with 22 % of the NCA group. Pain onset was sudden in 63% of the CAD group and in 50% of the NCA group. There were no clear differences in pain site or spread.

For interval-level variables, 1-tailed t-tests for independent samples were carried out to compare group means, the summary results of which are reported in table 4. (See Appendix L for detailed tables on each variable.)

Table 4 : Means and t values for NCA and CAD groups

Variable	NCA group		CAD group		t value	Sign
	Mean	SD	Mean	SD		
Number smoked	4.75	7.39	2.98	8.24	0.89	.38
Exertional pain	7.90	3.25	9.25	1.71	2.19	.01
Pain at rest	5.09	2.96	2.29	2.97	3.57	.00
Pain episodes	12.91	18.80	14.29	21.14	0.23	.82
GTN use	16.34	30.36	23.68	44.71	0.60	.55
Pain free days	2.09	1.86	2.13	2.19	0.05	.96

The variables of pain episodes, GTN use and pain free days listed in Table 4 all came from the chest pain diaries that patients were asked to complete on a daily basis. The inclusion of these diaries, however, was not particularly successful as many patients either did not complete them at all, or appeared to fill them in sporadically. The data

derived from this measure is therefore likely to be unreliable and based on a small sample of patients.

When asked to think back to their last 10 episodes of pain, the NCA group reported a mean of 5.1 occurring at rest compared with 2.3 in the CAD group. This is statistically significant ($p < 0.001$). The question designed to determine exertional pain ('If you were to climb 10 hills, on how many would you expect to experience chest pain?') however, yielded no significant difference, although the trend was for the CAD group to report more exertional pain as predicted.

To allow comparison with the results of Cooke, Smeeton and Chambers (1997) the variables of exertional pain, pain duration and rest pain were also examined according to their these authors classification as "typical".

Table 5 : Comparison of pain index scores

Variable	NCA group	CAD group	Pearson Value	Signif.
Exertion index (10/10)	25.5%	81.8%	3.04	.08
Pain duration (5 mins)	21.7%	34.8%	1.23	.27
Rest pain index (0-10%)	9.1%	53.7%	12.06	.001

As Table 5 shows, the CAD group report more typical symptoms for all three criteria. The reproducibility of pain with exertion index was approaching a significant difference with 81.8% of CAD patients, compared with 25.5% of NCA patients, reporting pain on 10/10 occasions of exertion. There was a higher incidence of unprovoked rest pain in NCA, with 53.7% of CAD patients reporting unprovoked pain at rest occurring in only 0-10% of pain episodes compared with 9.1% of NCA cases

(i.e. 90.9% of NCA experienced unprovoked pain at rest in over 10% of pain episodes). This was a highly significant difference ($p=.001$).

3.1.4 Psychological data

All psychological variables were measured on ordinal scales and were suitable for inter-group comparisons using t-tests, as summarised in Table 6. Appendix M contains fuller information.

Table 6 : Means and t values for NCA and CAD groups

<u>Variable</u>	<u>NCA group</u>		<u>CAD group</u>		<u>t value</u>	<u>Sign</u>
	<u>Mean</u>	<u>SD</u>	<u>Mean</u>	<u>SD</u>		
MSPQ	10.30	7.40	7.49	5.34	1.75	.04
HAD anxiety	8.87	6.10	7.23	3.53	1.42	.08
HAD depression	6.96	4.95	5.45	3.71	1.43	.08
WI total	3.73	3.71	4.31	4.24	0.56	.29
WI disease conviction	0.55	0.98	0.50	0.72	0.22	.41
WI illness phobia	0.82	1.10	0.92	1.01	0.37	.36
WI bodily preoccupation	1.27	1.12	1.12	1.04	0.54	.30
PC distractibility	22.64	4.28	22.56	5.12	0.06	.47
PC hopelessness	7.41	2.34	7.12	1.99	0.53	.30
PC helplessness	5.32	2.10	4.95	1.45	0.82	.21
PC support	14.32	2.26	14.53	1.50	0.46	.32

Only one psychological variable was found to significantly distinguish between the NCA and CAD groups. The mean MSPQ scores (a measure of somatic awareness) show a significant difference ($p<0.05$) with the NCA cohort reporting higher levels of

somatic awareness. No other psychological variables showed significant differentiation although the HAD anxiety and the HAD depression in particular show differences in the mean scores in the predicted direction. The small sample size may have prevented these differences from reaching significance (see Chapter 4).

3.1.5 Logistic regression for phase one predicting angiogram result

The demographic, physical and psychological variables which had been found to significantly differentiate between the groups on the chi-squared and t-tests on the less stringent $p < 0.1$ level were put forward for a logistic regression. This level of significance was used to allow more variables to be considered. This meant the following variables were available : age, sex, employment status, lipid status, lifetime smoking risk, time of GTN relief, exercise tolerance test, pain on hills, rest pain, alert pain, wakening pain, stress provoked pain, and MSPQ.

The variables were entered in a forward step-wise manner into the regression, with angiogram result being the dichotomous dependent variable. The results were produced and the variables, which were contained in the equation, are listed in Table 7.

Table 7 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Age	.07	.05	2.11	1	.15	.04	1.07
MSPQ	.02	.06	.13	1	.72	.00	1.02
Rest pain	-.27	.11	6.60	2	.01	-.26	.76
Stress provoked (1)	-.32	.70	.21	1	.65	.00	.73
Constant	-2.25	3.05	.54	1	.46		

Table 8 : Classification Table for angiogram result

<u>Angiogram outcome</u>			
<u>Predicted</u>			
<u>Observed</u>	NCA	CAD	% correct
NCA	8	9	47.1%
CAD	5	34	87.2%
			Overall 75.0%

A test of the full model with all four predictors against a constant only model was statistically reliable (chi-square 11.93, df 4, $p < .05$) indicating that the predictors as a set reliably distinguished between NCA and CAD patients. As can be seen in Table 8 above, the equation correctly predicts 87.2% of the CAD cases and 47.1% of the NCA cases - overall 75.0 % of cases correctly classified. However a lot of cases ($n=67$) have had to be excluded because of missing data on key variables. Even with dropping variables that contain most missing data, the data set was unable to be maximised any more than this (remembering that 51 patients did not respond by returning questionnaires). The equation contains only 1 variable which is a significant differentiator - rest-provoked pain. The remaining variables do not approach significance although do appear to add to the predictive power of the equation.

In an attempt to address the problems with the above equation, an exploratory process of entering different combinations of variables, guided by clinical rather than statistical criteria, was then done. Variables were entered in a forward step-wise manner and were retained only if they significantly added to the discriminating power of the equation. The best equation derived via this process is shown in Table 9 with values listed for the final equation.

Table 9 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Sex (1)	1.89	.91	4.29	1	.04	.18	6.59
HAD depression	-.21	.12	3.16	1	.08	-.13	.81
WI bodily preoccup.	1.10	.63	3.06	1	.08	.12	3.01
Age	.13	.06	4.53	1	.03	.17	1.14
Exertional pain	.38	.19	4.00	1	.05	.17	1.46
Stress provoked (1)	-1.96	1.04	3.58	1	.06	-.15	.14
Pain while alert (1)	-3.20	1.40	5.23	1	.02	-.21	.04
Constant	-7.24	4.25	2.90	1	.09		

Table 10 : Classification Table for angiogram result

<u>Angiogram outcome</u>			
<u>Predicted</u>			
<u>Observed</u>	NCA	CAD	% correct
NCA	14	4	77.8%
CAD	4	38	90.5%
			Overall 86.7%

As can be seen in Table 10, this equation correctly predicts 86.7% of angiogram results. It is slightly superior in classifying CAD cases however, with 90.5% of CAD results correctly predicted. 77.8% of NCA cases were correctly predicted. This is reasonably impressive - based on prevalence of NCA cases only approximately 25% would be predicted by chance. Table 9 shows that all variables are significant ($p < .05$) or approaching significance. Substituting the reproducibility of pain with exertion index for the 'raw' exertional pain score gives the same result. Variables (such as MSPQ score) which may have been assumed to have a place in the equation based on Chi-

squared and t-test results are not included as they are too inter-correlated with other variables in the equation and therefore do not add predictive power moreover.

3.1.6 Logistic regression predicting participation in the study

To determine the factors involved in patients' participation in the study (i.e. returning the questionnaires), a logistic regression was carried out. This time, return category (yes or no) was the dependent variable, and the basic information available on all (respondents and non-respondents) was entered as possible discriminators. A forward step-wise analysis was done from which the following variable was found to be significantly predictive of return (Table 11).

Table 11 : Variables in the Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Age	-0.07	0.02	8.37	1	.004	-0.19	0.94
Constant	3.38	1.30	6.80	1	.009		

Table 12 : Classification Table for return rate

<u>Returned questionnaires</u>			
<u>Predicted</u>			
<u>Observed</u>	<u>Yes</u>	<u>No</u>	<u>% correct</u>
<u>Yes</u>	60	12	83.3%
<u>No</u>	30	21	41.2%
			Overall 65.8%

As can be seen in Table 12 above, the equation correctly predicts 83.3% of those returning the questionnaires and 41.2% of the non-respondents - overall 65.8% of cases correctly classified. The factor which seems to discriminate those who

responded from those that did not is age, with younger subjects significantly less likely to participate.

Phase two

3.2.1 Description of phase two data

During the recruitment period of phase two, 110 patients were invited by letter to take part in the study. All patients were recruited from the Royal Infirmary. 1 patient was subsequently found to have had a previous M.I. and was therefore excluded from the study. Of the remaining 110 cases, 36 patients did not reply. The final sample size of 74 therefore represents a compliance of 66.7% (better than the 58.5% compliance for phase one).

Response rates differed between the groups. 67.9% of the CAD cohort agreed to participate in the research, compared to 65.4% of the group subsequently found to have NCA. This was not a significant difference.

Missing data were coded as such and excluded from the analyses.

Exploratory data analyses were performed using SPSS 7.5 for Windows (as detailed in Chapter 2). The data were summarised and examined with frequency distributions and histograms. For quantitative variables, the distribution appeared to be acceptably normally distributed to allow the use of parametric statistics. Descriptive statistics, including means, medians, standard deviations, and range were calculated for each quantitative variable.

3.2.2 Demographic data

Basic demographic information such as age and sex was obtained for all subjects including those who did not reply. Data on angiogram result, CAD risk factors, and investigations were also obtained for all patients.



The mean age for the total sample (including non-responders) was 57.7 years, with a range of 32 to 76 years. 70 patients (63.6%) were male; 40 (36.4%) were female. There were no significant differences between the responders and the non-responders on mean age (responders mean age 58.4; non-responders mean age 56.2, df 108, $t=1.12$, $p=.27$) or on sex (Pearson chi-square 2.73, $p=.10$).

Of the total sample of 110 patients who proceeded to angiogram, 84 (76.4%) were found to have Coronary Artery Disease and 26 (23.6%) were found to have Normal Coronary Arteries. 72.6% of the CAD group were male (27.4% female), compared with 34.6% of the NCA group (65.4% female). The mean age of patients was 59.2 years for those who were found to have CAD, and 52.6 years for the NCA group. Characteristics of the two groups are displayed in Table 13. Using a t-test for independent samples, age was also found to be significantly different between the groups ($t= 3.15$, $df 108$, $p<0.001$ one-tailed) (see Appendix N).

Table 13 : Demographic characteristics of the NCA and CAD groups.

Variable	<u>NCA</u>	<u>group</u>	<u>CAD</u>	<u>group</u>
	Mean	SD	Mean	SD
Age	52.62	9.81	59.25	9.26

A chi-squared test indicated that sex significantly differentiated the CAD and NCA groups, at a significance level of $p<0.001$ as can be seen in Table 14.

Table 14 : Chi Square results

Summary Information				
Demographic variable	NCA group	CAD group	Pearson Value	Sign.
	Percentage	Percentage		
Sex – male	35	73	12.39	.00
Marital status - single	12	0	N/A *	N/A
- married	69	71	N/A	N/A
-divorced/separated	12	16	N/A	N/A
- widowed	6	12	N/A	N/A
Living status –alone	25	16	N/A	N/A
- spouse/partner	38	77	N/A	N/A
- spouse + kids	31	4	N/A	N/A
- parents	0	0	N/A	N/A
- kids	6	4	N/A	N/A
Employment status - full-time	12	28	N/A	N/A
- part-time	12	9	N/A	N/A
-sick leave	12	7	N/A	N/A
-longterm sick	31	5	N/A	N/A
- retired	25	44	N/A	N/A
- housework	0	7	N/A	N/A

- unemployed	6	0	N/A	N/A
Employment change – given up work	40	28	N/A	N/A
- changed job	0	1	N/A	N/A
- lighter duties	0	8	N/A	N/A
- reduced hours	7	6	N/A	N/A
- no change	47	55	N/A	N/A

* Where Pearson values are not available this is due to chi-squared analyses not carried out because coding of the variable render this inappropriate

68.8% of the NCA group were married; 71.4% of the CAD group were married. 12.5% of the NCA group were in full-time employment compared with 28.1% of the CAD group. Of the NCA group, a further 43.8% were on sick leave and 25% retired. The corresponding figures for the CAD group are 12.3% on sick leave and 43.9% retired. As can be seen in Table 14, living status was found to differentiate significantly between the groups with the NCA group more likely to be living with spouse and children rather than spouse alone as in the majority of the CAD group. This is probably a function of the NCA group being younger however and therefore more likely to have dependent children still. Employment status was also found to be a significant differentiator, with NCA patients more likely to be off on sick leave and less likely to be retired or in full-time work than CAD patients. Full chi-squared matrices can be found in Appendix O.

3.2.3 Physical data

Summary information for non-parametric data on physical measures (pain characteristics etc) are shown in Table 15. Chi-squared analyses were performed comparing these data for the NCA group with the CAD group. Full details of the results of this procedure can be found in Appendix P.

Table 15 : Chi squared results

Summary Information				
Demographic variable Smoking	NCA group	CAD group	Pearson Value	Sign.
	Percentage	Percentage		
- smoked in lifetime	62	74	1.34	.51
- current smoker (reported by physician)	36	42	N/A	N/A
- previous smoker (reported by physician)	21	28	N/A	N/A
- current smoker (self report)	12	22	0.76	.38

(continued below)

Summary Information				
Clinical Information	NCA group	CAD group	Pearson value	Sign.
	Percentage	Percentage		
Obesity- risk	88	86	0.01	.91
Lipid status- risk	79	76	0.05	.83
Diabetes- risk	50	74	0.87	.35
Hypertension- risk	47	58	0.68	.41
Family history- risk	64	74	0.44	.51
Positive exercise test	33	91	30.69	.01
Abnormal ECG	11	37	1.98	.16
GTN relief effective	85	88	0.09	.76
Pain while alert	64	53	0.57	.45
Wakening pain	50	17	6.86	.01
Stress provoked pain	56	40	1.38	.24
Cold provoked pain	50	57	0.27	.60
Food provoked pain	6	15	.85	.36
Sex provoked pain	19	15	0.12	.73
Stooping provoked pain	44	28	1.35	.24

None of the traditional risk factors for CAD were found to differentiate significantly between the groups. The CAD group were however significantly more likely to have a positive exercise tolerance test ($p < 0.01$). 91% of the phase two sample found to have CAD had had a positive exercise tolerance test (i.e. ECG changes on exertion consistent with CAD) compared with only 33% of the NCA patients. Pain sufficient to cause nocturnal waking was found to be significantly more common ($p < 0.01$) in the NCA group than the CAD group (50% c.f. 16.7%).

The majority of patients in both groups reported central retrosternal pain (see Appendix P). More NCA patients (80%) reported pain of sudden onset than CAD patients (44%). The NCA group reported their chest pain to be more likely to be aggravated by coughing or breathing deeply than those with CAD who were more likely to experience problems with bending or stooping. No other clear differences on physical variables were found between the groups.

For interval level variables, t-tests for independent samples were carried out to compare group means, the summary results of which are reported in table 16. (See Appendix Q for detailed tables on each variable.)

Table 16 : Means and t values for NCA and CAD groups

Variable	NCA group		CAD group		t value	Sign.
	Mean	SD	Mean	SD		
Number smoked	3.00	8.41	2.99	5.71	0.38	.70
Exertional pain	9.31	2.02	7.92	3.17	1.65	.10
Pain at rest	2.64	3.08	1.80	2.58	1.02	.31

There were no significant differences between the groups on number of cigarettes smoked per day, nor on the amount of pain episodes occurring at rest. The NCA group

reported a mean of 2.64 episodes of pain at rest (from their last 10 pain episodes) compared with an average of 1.80 in the CAD group. The question designed to determine exertional pain ('If you were to climb 10 hills, on how many would you expect to experience chest pain?') yielded no significant difference. Puzzlingly the trend was for the NCA group to report more exertional pain, which is the opposite of what was predicted.

Again, the data on pain duration, exertional pain and pain at rest were recoded according to their classification as "typical". Table 17 shows the comparative percentages.

Table 17 : Comparison of pain index scores

Variable	NCA group	CAD group	Pearson Value	Significance
Exertion index (10/10)	87.5%	67.3%	2.48	.12
Pain duration (5 mins)	20.0%	34.6%	1.16	.28
Rest pain index (0-10%)	50.0%	63.0%	.76	.38

As Table 17 shows, the CAD group reports more typical symptoms for pain duration and (lack of) rest pain. The reproducibility of pain with exertion index was, surprisingly, in the opposite direction to that predicted and runs contrary to the findings in phase one. 67.3% of CAD patients, compared with 87.5% of NCA patients, report pain on 10/10 occasions of exertion. This was not a significant difference. There was a higher incidence of unprovoked rest pain in NCA, with 63.0% of CAD patients reporting unprovoked pain at rest occurring in only 0-10% of pain episodes compared with 50.0% of NCA cases (i.e. 50% of NCA experienced unprovoked pain at rest in over 10% of pain episodes). Again, this was not a

significant difference . The typicality percentages for pain duration of 5 minutes or under is very similar to that found in phase one. 20% of NCA cases report typical pain duration compared with 34.6% of CAD cases (not significant).

3.2.4 Psychological data

All psychological variables were measured on ordinal scales and were suitable for inter-group comparisons using t-tests, as summarised in Table 18. Appendix R contains fuller information.

Table 18 : Means and t values for NCA and CAD groups

<u>Variable</u>	<u>NCA group</u>		<u>CAD group</u>		<u>t value</u>	<u>Sign</u>
	<u>Mean</u>	<u>SD</u>	<u>Mean</u>	<u>SD</u>		
MSPQ	11.81	7.64	6.84	6.10	2.73	.01
HAD anxiety	10.12	3.69	6.30	3.67	3.76	.00
HAD depression	8.12	4.44	5.12	3.28	3.03	.00
WI total	4.71	4.30	3.05	3.19	1.63	.11
WI disease conviction	1.00	0.96	0.37	0.67	2.89	.01
WI illness phobia	0.93	1.33	0.56	0.73	1.40	.16
WI bodily preoccupation	1.57	1.22	0.98	1.03	1.84	.07

MSPQ, HAD anxiety, HAD depression and WI disease conviction were all found to distinguish significantly between the NCA and CAD groups. The mean MSPQ scores show a significant difference ($p < 0.01$) with the NCA cohort reporting higher levels of somatic awareness. The mean HAD anxiety score was 10.12 for the NCA group and 6.30 for the CAD group (a score of 10 or above is generally taken to represent “caseness”) – this was a significant difference ($p < 0.001$). The HAD depression score

also showed differences in the mean scores in the predicted direction ($p < 0.005$) as did the Whitely Index disease conviction measure (NCA mean 1.00; CAD mean 0.37, $p < 0.005$).

3.2.5 Subject response

In phase one of the study there was found to be a differential response rate, with NCA patients apparently significantly less likely to respond. It was found, however, that response of subjects in phase one (i.e. participation in the research) could be predicted by age, and that the NCA patients were generally younger. To determine if the same pattern was evident in the phase two cohort a crosstabulation was done (Table 19). Response rates were not found to differ significantly between the groups. 67.9% of the CAD cohort agreed to participate in the research, compared to 65.4% of the group subsequently found to have NCA ($p = .82$).

Table 19 : Subject response by angiogram result

	<u>Angiogram Outcome</u>		<u>Total</u>
	<u>NCA</u>	<u>CAD</u>	
<u>Responded</u>	17 (65.4%)	57 (67.9%)	74 (67.3%)
<u>Did not respond</u>	9 (34.6%)	27 (32.1%)	36 (32.7%)
<u>Total</u>	26 (100%)	84 (100%)	110 (100%)

3.2.6 Logistic regression for phase two predicting angiogram result

A logistic regression analysis was performed on angiogram result as outcome with the psychological variables MSPQ, HAD anxiety, HAD depression, WI total, WI disease conviction, WI illness phobia and WI bodily preoccupation as possible predictors as well as sex and age, in which a significant difference had been shown between the groups. Also offered as possible predictors were clinical variables which had been hypothesized as discriminators. Analysis was performed using SPSS 7.5 for Windows. After deletion of 44 cases with missing values, data from 66 patients who underwent angiography were available for analysis: 13 who were subsequently found to have NCA and 53 who were found to have CAD. Missing data appeared to be scattered randomly across categories of outcome.

Table 20 shows the regression coefficient, standard error score, Wald statistics, degrees of freedom, significance, partial correlation and odds ratio for each of the predictors.

Table 20 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Sex (1)	3.41	1.29	6.99	1	.01	.28	30.28
Age	.21	.07	7.96	1	.00	.30	1.23
Wakening pain	2.27	1.06	4.63	1	.03	.20	9.72
WI disease conviction	-2.07	.72	8.29	1	.00	-.31	.13
Constant	-13.98	4.78	8.57	1	.00		

Table 21 : Classification Table for angiogram result

<u>Observed</u>	<u>Predicted</u>		<u>% correct</u>
	<u>NCA</u>	<u>CAD</u>	
<u>NCA</u>	9	4	69.2%
<u>CAD</u>	2	51	96.2%
			Overall 90.9%

A test of the full model with all four predictors against a constant only model was statistically reliable (chi-square 35.18, df 4, $p < .0001$) indicating that the predictors as a set reliably distinguished between NCA and CAD patients. As can be seen in Table 21 above, the set correctly predicts 96.2 % of the CAD cases and 69.2% of the NCA cases for an overall success rate of 90.9%. Because there was a high level of inter-correlation between the psychological variables, it was found that Whitely Index disease conviction score could be substituted (less impressively) for other variables. For example, added to age and sex the Whitely Index bodily preoccupation score predicts 50% of NCA cases, 94% of CAD cases – overall 86%. HAD anxiety and HAD depression (with age and sex) both predict equally effectively separately (47% of NCA, 93% of CAD – overall 82%) but actually diminish in predictive power when combined (41% of NCA, 93% of CAD – overall 81%). Added to sex and age, MSPQ score and WI bodily preoccupation predicts 50% of NCA cases, 95% of CAD cases – overall 86%. It should be noted however, that numbers in each cell (the NCA categories in particular) are small. To summarise, all different combinations involving adding various of MSPQ, wakening pain, WI bodily preoccupation, WI disease conviction, HAD anxiety, HAD depression, to age and sex were modeled however none were as powerful as the set in Table 20. These issues will be covered further in chapter 4.

Out of interest, this logistic regression was tested against the data from phase one of the study. The details of this can be found in Appendix T.

3.3 Cardiologists predictions

As described in chapter 2, for subjects in phase two, prior to carrying out each patient's angiogram, Cardiologists were asked to make a prediction of whether the outcome of the procedure would be one of CAD, NCA or if they were unsure. Although anonymous, the Cardiologist was also asked to indicate their grade (e.g. "Consultant"). Unfortunately out of a possible 110, there were only 12 responses (11% compliance). Clearly it is difficult to do much with such a small (and possibly biased) sample other to describe the data.

Of the 12 responses, 5 were from Consultants and 7 were from Senior Registrars. They predicted 10 CAD outcomes and 2 NCA outcomes. They were correct in all but one case. The case predicted wrongly was by a Consultant who predicted CAD in a patient who turned out to have NCA.

3.4 Testing predictive power of models

3.4.1 Model derived from phase one

To test out the reliability of the best logistic regression model derived from phase one data (see Table 9), this equation was applied to a new set of data (the phase two sample). After deletion of 53 cases with missing values, data from 57 patients who underwent angiography were available for analysis: 8 who were subsequently found to have NCA and 49 who were found to have CAD. A test of the full model with all seven predictors against a constant only model was statistically reliable (chi-square 25.46, df 7, $p < .001$) indicating that the predictors as a set reliably distinguished between NCA and CAD patients. As can be seen in Table 23 below, the set correctly predicts 97.8% of the CAD cases and 58.3% of the NCA cases for an overall success rate of 89.5%. The prediction rate for CAD cases in this new sample is particularly good.

Table 22 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Sex (1)	2.82	1.18	5.70	1	.02	.25	16.69
HAD depression	-.25	.15	2.76	1	.09	-.11	.78
WI bodily preoccup.	-.61	.53	1.33	1	.25	.00	.54
Age	.16	.06	7.47	1	.01	.31	1.17
Exertional pain	-.21	.21	1.09	1	.30	.00	.81
Stress provoked(1)	.18	.94	.04	1	.85	.00	1.20
Pain when alert (1)	.37	1.23	.09	1	.77	.00	1.44
Constant	-4.65	3.59	1.68	1	.19		

Table 23 : Classification Table for angiogram result

<u>Angiogram outcome</u>			
<u>Predicted</u>			
<u>Observed</u>	<u>NCA</u>	<u>CAD</u>	<u>% correct</u>
<u>NCA</u>	7	5	<u>58.3%</u>
<u>CAD</u>	1	44	<u>97.8%</u>
			Overall 89.5%

The only variables which achieve significance in the logistic regression are sex and age. The other variables are unimpressive in adding much predictive power, indeed the variables of WI bodily preoccupation, exertional pain and stress provoked pain are associated with angiogram outcome in the opposite direction to that found in the phase one data. On the total data set (n=110), age and sex alone, however, predict only 34.6% of NCA cases, 92.9% of CAD cases – overall 79.1%. On the data set of subjects who responded (n=77) – a fairer comparison with the sample comprising the fuller model - age and sex predict only 35.3% of NCA cases, 93.0% of CAD cases – overall 79.7%. This supports the conclusion that the responders and the non-responders do not differ significantly on age or sex. It also suggests that, despite their apparent insignificance, the variables of HAD depression, WI bodily preoccupation, exertional pain, stress provoked pain and pain while alert do improve the classification rate – particularly of the NCA cases.

So, the set of predictors derived from phase one correctly predict 97.8% of the CAD cases and 58.3% of the NCA cases for an overall success rate of 89.5% in a new

sample. This is particularly convincing given the percentage of NCA cases expected by chance would be approximately 25%.

3.5 Analysis of the total data set

3.5.1 Typicality of symptoms

To allow direct comparison with the findings of Cooke, Smeeton and Chambers (1997), described in Chapter 1, the total data set of phase one and phase two combined were analysed according to typicality (as defined by these authors criteria) of the symptoms of pain duration, reproducibility of pain with exercise and unprovoked rest pain. Chi-squared crosstabulations were done for coexistence of the three symptom indexes for NCA cases (Table 24) and CAD cases (Table 25).

Table 24 : Typicality of symptoms in all NCA patients

<u>Rest pain index</u>	<u>Pain duration index</u>	<u>Exertion</u>	<u>Index</u>	<u>Total</u>
		Typical	Atypical	
Typical	Typical	1	0	1
	Atypical	6	0	6
	Total	7	0	7
Atypical	Typical	3	3	6
	Atypical	15	7	22
	Total	18	10	28

Table 25 : Typicality of symptoms in all CAD patients

<u>Rest pain index</u>	<u>Pain duration index</u>	<u>Exertion</u>	<u>Index</u>	<u>Total</u>
		Typical	Atypical	
Typical	Typical	16	8	24
	Atypical	25	4	29
	Total	41	12	53
Atypical	Typical	6	3	9
	Atypical	19	9	28
	Total	25	12	37

As Tables 24 and 25 show, all three symptoms are atypical in 20.0% of NCA patients and in 10.0% of CAD cases. Interestingly no NCA patients report pain at rest in 0-10% of episodes, report having pain on less than 10/10 occasions of exercise. There is a significant correlation between pain duration index and exertion index in NCA cases (chi-square Pearson value 4.15, $p < .05$), but not between the other variables. For CAD cases, however, there is a significant correlation between pain duration index and rest pain index (chi-square Pearson value 5.94, $p < .05$).

3.5.2 Analysis of traditional risk factors

Given the relatively small sample sizes of each of the phases individually, it was thought worthwhile examining both data sets together to see if larger numbers gave any further indication of the role of traditional risk factors for CAD. These factors (unlike the other factors examined in this study) have been demonstrated in large studies as associated with increased risk of CAD. As detailed in Appendix S, the variables of diabetes, lipid status, and smoking were all found to differentiate significantly between the NCA and CAD groups. Of the CAD patients, 21.4% had diabetes compared with 5.8% of the NCA group ($p < .05$). Sixty one point nine percent of the CAD group had clinically high lipid levels compared with 32.3% of NCA patients ($p < .001$). Current or previous smokers represented 60.3% of the CAD group and only 41.9% of the NCA group (p unable to be calculate due to coding differences in this variable). No significant differences were shown between the groups on family history risk, hypertension or obesity.

3.5.3 Logistic regression for the total data set (phases 1 and 2 data)

Analysis of the combined data from phase one and phase two gives a larger sample (n=233) from which to explore a model that applies to both patient sets. Accordingly, the same process of logistic regression analysis was performed on angiogram result. Initially the 'core' variables of age and sex were offered as predictors. Applied to the total patient group (responders and non-responders) these predictors correctly predict 87.2 % of the CAD cases and 41.6% of the NCA cases - overall 72.1%. Applied to the set of patients who responded to the questionnaires (n=146; 72 from phase one and 74 from phase two) these predictors correctly predict 92.4 % of the CAD cases and 34.2% of the NCA cases - overall 76.0%. Table 26 shows the regression coefficient, standard error score, Wald statistics, degrees of freedom, significance, partial correlation and odds ratio for both of the predictors as applied to the subjects who responded.

Table 26 : Variables in the Logistic Regression Equation (total data set n=146)

Variable	B	SE	Wald	Df	Sign.	R	Exp(B)
Age	.08	.02	12.68	1	.00	.25	1.09
Sex(1)	1.31	.41	10.20	1	.00	.22	3.70
Constant	-4.50	1.37	10.71	1	.00		

Table 27 : Classification Table for angiogram result

Observed	Predicted		% correct
	NCA	CAD	
NCA	14	27	34.2%
CAD	8	97	92.4%
			Overall 76.0%

As is evident in Table 27, these predictors are good at correctly classifying CAD cases but poor for NCA cases (not much better than chance). Accordingly, based on variables found to be reasonable predictors in phase one and in phase two and using variables which were significantly associated with angiogram outcome and those which were considered to be relevant and which may reach significance with this larger data set, various logistic regression analyses were considered.

For example, added to age and sex the MSPQ score predicts 36.1% of NCA cases, 92.3% of CAD cases – overall 79.4% (based on 141 valid cases). Although nearly significant as a predictor in this set ($p=.06$), this variable does not really improve classification much. HAD anxiety does slightly better in correctly predicting 42.5% of NCA cases (92.3% of CAD cases – overall 78.5%). The other psychological variables, including HAD depression and the WI sub-scores, predict angiogram result significantly but less well. There was nothing to be gained by adding combinations of psychological predictors because of high levels of inter-correlation. Wakening pain (which was significant as a predictor for the phase two data) – with age and sex - does appear to assist overall classification by improving classification of NCA cases. This is not entirely surprising as wakening pain was found to be significantly correlated with NCA cases in both phases. A test of the full model with all three predictors against a constant only model was statistically reliable (chi-square 36.41, df 3, $p<.0001$) indicating that the predictors as a set reliably distinguished between NCA and CAD patients ($n=136$). As can be seen in Table 28 below, the set correctly predicts 90.9% of the CAD cases and 45.9% of the NCA cases for an overall success rate of 78.7%.

Table 28 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Age	.10	.03	13.57	1	.00	.27	1.10
Sex(1)	1.16	.45	6.51	1	.01	.17	3.19
Wakening pain(1)	-1.58	.46	11.90	1	.00	-.25	.21
Constant	-4.51	1.52	8.76	1	.00		

Table 29 : Classification Table for angiogram result

<u>Observed</u>	<u>Predicted</u>		
	<u>NCA</u>	<u>CAD</u>	<u>% correct</u>
<u>NCA</u>	17	20	45.9%
<u>CAD</u>	9	90	90.9%
			Overall 78.7%

Although, in overall terms, this model is slightly less impressive than the set including MSPQ, it is considerably better at NCA prediction. Added to age and sex, wakening pain plus HAD anxiety improved classification to 48.6% of NCA cases correctly predicted, 92.9% of CAD cases – overall 81.2% although the HAD anxiety predictor became non-significant ($p=.41$) (wakening pain $p=.009$).

Also with the aim of improving NCA prediction the variables of exertional and rest pain were examined. In phase one exertional pain was significantly associated with CAD, however this was not replicated in phase two (indeed the trend was for NCA patients to report more exertional pain) and, unsurprisingly, this variable was found to add nothing in predictive terms. Pain at rest, however was found to associated with NCA cases in phase one ($p=.001$) and not significantly in phase two ($p=.31$). Added to age and sex, rest pain was found to improve classification, predicting correctly 47.2% of NCA cases, 87.4% of CAD cases – overall 75.6% ($n=123$). A logistic regression was then run with predictors of age, sex, wakening pain and rest pain ($n=121$). Table 30 shows the regression coefficient, standard error score, Wald

statistics, degree of freedom, significance, partial correlation and odds ratio for the predictors.

Table 30 : Variables in the Logistic Regression Equation

Variable	B	SE	Wald	df	Sign.	R	Exp(B)
Age	.09	.03	9.65	1	.00	.23	1.09
Sex(1)	1.31	.45	7.05	1	.01	.19	3.70
Wakening pain(1)	-1.40	.52	7.21	1	.01	-.19	.24
Rest pain	-.13	.08	2.42	1	.12	-.05	.88
Constant	-3.68	1.61	5.20	1	.02		

Table 31 : Classification Table for angiogram result

Observed	Predicted		% correct
	NCA	CAD	
NCA	19	16	54.3%
CAD	10	76	88.4%
			Overall 78.5%

Added to age and sex, wakening pain plus rest pain improved classification to 54.3% of NCA cases correctly predicted, 88.4% of CAD cases – overall 78.5%. Substituting the raw rest pain score for the unprovoked pain at rest index reduces the classification to 48.6% of NCA cases, 87.2% of CAD cases – overall 76.0%.

The age, sex, wakening pain, rest pain set was then supplemented with each of the predictive psychological variables – HAD anxiety and MSPQ – separately. The addition of HAD anxiety removes a further 3 cases due to missing data, leaving 118 valid cases. Although the classification is, on the surface, improved to 54.6% of NCA cases, 88.2% of CAD cases – overall 78.8%, the reality is that this is achieved by the removal of a case each cell (with the exception of the predicted NCA /CAD observed

cell). MSPQ added to age, sex, wakening pain and rest pain further restricts the data sample to 115 cases. This variable improves the classification of CAD cases at the slight expense of NCA cases as can be seen in Table 32.

Table 32 : Variables in the Logistic Regression Equation

<u>Variable</u>	<u>B</u>	<u>SE</u>	<u>Wald</u>	<u>df</u>	<u>Sign.</u>	<u>R</u>	<u>Exp(B)</u>
Age	.09	.03	9.57	1	.00	.24	1.10
Sex(1)	1.20	.52	5.38	1	.02	.16	3.33
Wakening pain(1)	-1.60	.61	6.75	1	.01	-.19	.20
Rest pain	-.14	.09	2.62	1	.10	-.07	.87
MSPQ	.04	.05	.83	1	.36	.00	1.04
Constant	-4.25	1.87	5.15	1	.02		

Table 33 : Classification Table for angiogram result

<u>Observed</u>	<u>Predicted</u>		
	<u>NCA</u>	<u>CAD</u>	<u>% correct</u>
<u>NCA</u>	16	14	53.3%
<u>CAD</u>	6	79	92.9%
			Overall 82.6%

The models are not directly comparable due to different numbers of valid cases for each, and often what may appear to be a notable improvement in classification can be attributed to movement of only several cases between cells because of the relatively low numbers in the NCA cells. However, with these reservations, possibly the best model on a number of criteria appears to be the set detailed in Table 32. A test of the

full model with all five predictors against a constant only model was statistically reliable (chi-square 32.84, df 5, $p < .0001$) indicating that the predictors as a set reliably distinguished between NCA and CAD patients. As can be seen in Table 35 above, the set correctly predicts 92.9 % of the CAD cases and 53.3% of the NCA cases for an overall success rate of 82.6%.

To summarise, all different combinations involving adding various predictors to age and sex were modeled for the total data set of phases 1 and 2 data combined. This gives a slightly different model than was found for each phase alone although the variables of age and sex appear robust throughout different data samples. Wakening pain also appears consistent in its association with NCA. Of the psychological variables, many are interchangeable although MSPQ is possibly superior in its predictive capacity.

Chapter 4 - Discussion

4.1 Main Findings

The main findings will be discussed in relation to their demographic, physical and psychological characteristics.

4.1.1 Demographic data

The results of this study show a significant difference between the NCA group and the CAD group on sex. As predicted, females were more prevalent in the NCA cohort (55%, compared with 36% CAD in phase one and 65% NCA compared with 27% CAD in phase two). This is in line with previous research (e.g. Day and Sowton, 1976).

The results of this study indicate that the patients found to have NCA were significantly younger (mean age 53.9 years for phase one and 52.6 for phase two) than those found to have CAD (mean age 58.9 years for phase one and 59.2 for phase two). Taken together, the findings that NCA were more likely to be comparatively young and female as has been consistently found in other research studies may indicate that the NCA cohort in the present study can be considered representative of this population.

4.1.2 Physical data

It was predicted that CAD patients would report more exertional pain in response to being asked about pain provoked by walking up a hill given that one of the characteristics of angina is that it is usually provoked by exertion. No significant difference on this variable was found between the NCA and CAD groups, although in the CAD group chest pain did appear to be more reliably associated with exertion (but

not significantly so) in the phase one sample. This may be explained by screening by GPs and Cardiologists, so that mainly patients reporting typical pain make it through to this stage of assessment and those not reporting exertional pain are screened out earlier. It is also conceivable that patients learn to respond to doctors questions in a way which elicits more interest or action. In this way patients may be more likely to concur with suggestions of exertional pain. Puzzlingly, in the phase two cohort, the trend was for the NCA group to report more exertional pain, which is the opposite of what was predicted. Interestingly though, in phase one a significant difference was found between the groups on chest pain experienced at rest with the NCA patients reporting more pain episodes at rest. This difference did not attain significance in phase two although the trend was in the same direction. We have then, in effect, a measure of exertion that does not differentiate between the groups, and a measure of pain at rest which discriminates between those with NCA and CAD. This suggests that the concept of effort related (or exertional) pain needs to be expanded into two elements in an endeavor to clarify the confusion surrounding typicality of pain. In the interim between phase one and phase two of this study being carried out, attempts were made to do this by Cooke, Smeeton and Chambers (1997). Using, as in this study, a standardised questionnaire modified from that used by Master (1964), no important differences were found between the groups in the site, radiation or quality of pain. This study also showed no clear differences in these pain characteristics. Cooke, Smeeton and Chambers found the only three symptom variables which were of statistical value in separating the groups (as described previously) were the reproducibility of pain with physical activity; the occurrence of unprovoked pain at rest; and the usual duration of pain episodes, classed according to their classification as "typical". Patients with NCA were less likely to report a consistent relationship of pain provocation with exercise, more likely to experience unprovoked pain at rest and report pain of longer duration. In phase one the CAD group reported more typical symptoms for all three criteria. The reproducibility of pain with exertion index was

approaching a significant difference and there was a significantly higher incidence of unprovoked rest pain in NCA. The differential characteristics for pain duration of 5 minutes or under were very similar in phase one and phase two with more CAD patients experiencing pain of shorter duration (22% and 20% NCA compared with 35% and 35% CAD) although this is not a significant finding. In phase two the CAD group also reported more typical symptoms for (lack of) rest pain. The reproducibility of pain with exertion index was, surprisingly, in the opposite direction to that predicted and runs contrary to the findings in phase one. There was a higher incidence (but not significant) of unprovoked rest pain in NCA. Putting all this together, the findings of Cooke, Smeeton and Chambers are borne out to some extent. Although the reproducibility of pain with physical activity was not found to be useful in separating the groups, the occurrence of unprovoked pain at rest and (to a lesser extent) the usual duration of pain episodes were found to discriminate between the NCA and CAD groups.

Experiencing rest pain while conscious and alert and nocturnal wakening pain seemed to be associated with NCA chest pain. Cooke et al (1995) found nocturnal pain to be similar in both groups, however the results of the present study appear to be consistent with other research (Frasure-Smith, 1987), which found that events triggering cardiac symptoms while resting or sleeping were most common among patients with no significant blockage.

Chest pain in NCA patients has been found to often occur with stress (Potts and Bass, 1999). This was supported by the results of phase one of this study which indicated that 56% of NCA (compared with 40% of CAD patients) reported their pain being provoked by stress. Such patients are also known to perceive benefit from GTN but after more than 5 minutes. Patients with CAD on the other hand usually gain relief

rapidly (0-3 minutes) by GTN, which is what is expected given the pharmacology of GTN effect. This is substantiated by the results of this study, which supports the view (Chambers and Bass, 1990) that such relief may be spurious in NCA patients.

Coughing or breathing deeply was found to be more likely to aggravate NCA than CAD pain, which may add support to the role of hyperventilation and musculoskeletal factors in the pain origin. Both coughing and hyperventilation can create strain on the intercostal muscles (Evans, 1977). Results of the present study indicate that these factors did exacerbate chest pain in NCA patients suggesting that the pain aetiology may involve musculoskeletal strain of the chest wall muscles either through hyperventilation or some other factor.

The following risk factors for CAD were assessed in this study : smoking, obesity, hypertension, diabetes and lipid status. No significant difference (in either phase separately) was found between the groups on measures of smoking (self-report and assessed by the cardiologist). The data of both phases taken together, however, show that current or previous smoking is more likely in the CAD group. Similarly the results of both phases individually are consistent in that family history of CAD failed to show any difference and hypertension was no more common in the CAD group. These factors still failed to differentiate between the groups when the total data set was analysed. This largely supports the findings of the Cooke *et al* (1995) study in which there were no significant differences in smoking, family history of CAD, or history of hypertension, but is contrary to those of Dhawan (1993) who found smoking, family history and hypertension to be useful predictors of CAD. Lipid status and diabetes were found to be significantly more likely to be associated with the CAD group when both phases were taken together. It is likely, however, that these risk factors may have been involved in the selection of patients for angiography so may be explained by a work up bias. Essentially what this means is that patients who

have several of the known risk factors for CAD are more likely to be extensively assessed and investigated for the presence of CAD than those who do not (eg. Davis *et al*, 1996). The more traditional risk factors a person has, the more likely they are to go forward for angiography (Davis *et al*, 1996).

The present study found no significant differences in obesity, cholesterol levels or diabetes. Overall, it appears therefore that the NCA patients did not report significantly fewer risk factors. It should be noted, however, that many of these risk factors have been originally established as a result of studies on a very large population (eg. Haynes and Feinleib, 1982), which have highlighted a small but definite risk attached to the particular factor. Because of the small numbers of subjects involved in this study, it would have been surprising if such risk factors were shown to be significant. It is also possible that age is a confounding variable. The NCA group were significantly younger than the CAD group therefore possibly less likely to be overweight or hypertensive.

4.1.3 Psychological data

No significant differences were found in anxiety and depression scores between the NCA and CAD groups in phase one although significant differences were found in phase two, with both anxiety and depression being higher in NCA patients. Other research indicates that psychiatric illnesses are approximately three to six times more common in NCA than CAD patients, and of those, anxiety and depressive disorders are the most common (Potts and Bass, 1994). This is not borne out in phase one and such disorders appear to be approximately twice as common in NCA than CAD in the phase two sample. In both samples the mean scores for both anxiety and depression were below the threshold for psychiatric caseness. It could be speculated that the overall prevalence of anxiety and depression is underrepresented as the more anxious

or depressed NCA patients would be less likely to participate in the study, or that the heterogeneous nature of the NCA group may be masking such differences. It is also possible that the HAD is not the best instrument to assess caseness in this population, although previous work in this area does not indicate anything more appropriate.

On a global measure of hypochondriasis, as well as sub-scales of bodily preoccupation and illness phobia, no differences were found between the NCA and CAD cohorts. This does not add support to the view (eg. Eifert *et al*, 1996) that NCA patients engage in maladaptive illness behaviours. Eifert *et al* (1996) found that compared to cardiac and surgical inpatients, and nonpatient controls, NCA patients reported more hypochondriacal beliefs, anxiety disorders, negative affect and physical symptoms. *However*, phase two data shows the disease conviction sub-scale does distinguish significantly between the NCA and CAD groups which may suggest that although no different on global hypochondriasis, NCA patients have a more firmly entrenched belief in the presence of disease. For the total hypochondriasis score, the mean values for this measure are 3.7 for NCA patients, and 4.3 for CAD patients. These scores are most similar to the norms (Pilowsky, 1978) for a sample of non-psychiatric hospital patients (3.20 for males; 4.47 for females), and lie somewhere in-between the norms for hypochondriacal inpatients (8.92 for males; 7.90 for females) and those of normal controls (1.67).

Results from both phases of the study do separately show that NCA patients have significantly higher levels of bodily awareness. Frasure-Smith (1987) also found that groups of patients with varying degrees of coronary artery occlusion (categorised in terms of range of disease severity from NCA to CAD) differed significantly in this respect with the NCA patients having the highest scores. She found the greater the occlusion, the lower the level of bodily awareness. The limitations of the present study in categorising severity of blockage into two extreme groups does not allow

extrapolation of the findings to this extent, however it does show that NCA patients have a higher level of bodily awareness than those with CAD.

NCA patients reported pain cognitions which did not differ from those reported by CAD patients. Further, they appear dissimilar from those commonly associated with chronic pain patients (Boston et al, 1990). A possible reason for this may be that the instrument used (Pain Cognitions Questionnaire) is not applicable to this group. Alternatively, the instruments may not be useful at the time it was used (i.e. pre-angiogram), but may become more useful both once a diagnosis of CAD is eliminated and the unexplained chest pain continues, and with chronicity of pain. Whatever, the inclusion of the Pain Cognitions Questionnaire was not found to be useful and was not used in phase two.

In relation to the models discussed in chapter one, the findings as a whole lend some limited support to the multicausal interactive model of pain detailed on page 13. Certainly, there appears to be differences in NCA patients in physical characteristics of their pain, relationship to stress, elevated levels of anxiety and depression, and higher levels of bodily awareness. According to this model these factors would interact to influence NCA patients interpretation of sensations, leading to increased disability. However, the additional contribution of psychological factors in NCA patients is not huge and it could be argued that support for this model is limited. There are also a number of psychological variables on which no significant differences between the groups were shown. Possibly this model could apply to any pain, particularly chronic pain, condition.

4.2 Main predictor variables

In phase one the main factors found to be significantly associated with a finding of NCA were: age (young), sex (female), pain at rest, pain provoked by stress, wakening pain, relief by GTN after more than 5 minutes and high levels of bodily awareness. None of the other questionnaire measures or diary variables were found to be significantly different between the groups. In the phase two sample, NCA were associated with being young, female, having a negative exercise tolerance test, wakening pain, high levels of bodily awareness, disease conviction, and high levels of anxiety and depression. There appears to be some consistency then in the importance of age, sex, wakening pain and bodily awareness in separating the groups, and a clear but lesser association with rest pain, anxiety and depression.

Using the factors found to be associated with NCA, a logistic regression was run. This was one of the main objectives of the study. From this, factors which were found to be useful in predicting the likelihood of a patient having NCA in phase one of the study were age, stress provoked pain, bodily awareness, and rest pain. These variables were found to correctly classify 85 % of cases. The other variables found to be associated with NCA were not found to be independently predictive as they were considerably inter correlated with the existing variables. The classification of cases differed between groups with 64% of NCA cases correctly classified, and 91.5% of CAD cases correctly classified. The differential classification rate is good as it is safer not to misclassify CADs as NCAs, even at the expense of missing NCA cases. If these factors are to be of any clinical use, it is, understandably, of the utmost importance to cardiologists not to miss someone with genuine CAD. When the discriminatory power of this predictive equation was tested on a new patient sample (phase two) it was found to correctly predict 97.8% of the CAD cases and 58.3% of the NCA cases for an overall success rate of 89.5%. **This is actually superior in**

overall classification than for the sample the equation was derived from. This appears to be because the equation is excellent at predicting cases of CAD of which there are a higher proportion in phase two. The predictive power of the model compares favourably with other such attempts cited in the literature. Serlie *et al* (1996), for example, achieved an overall classification of 75.4% in using an empirical psychological model to differentiate cardiac and non-cardiac chest pain. This study would appear, therefore, to have produced a model which, when tested on a new patient sample, is impressive at differentiating NCA cases from CAD cases.

A high proportion (overall 33%) of the patients in this study were found to have NCA when compared with other studies which find approximately 20% of patients to have NCA. Rather than the prevalence of NCA being higher in this sample, it is likely than the exclusion criteria for entry to the study such as having had a myocardial infarction, previous angiography, angioplasty or bypass grafting increases the likelihood of finding a patient with NCA. The higher proportion of NCA patients found in phase one compared with phase two (41.5% compare with 23.6%) could be explained by the possibility that Cardiologists are now better at screening out NCA patients at an earlier stage and thus less were scheduled for angiography by phase two (4 years later).

In phase one, responding was found to differ between the groups with the CAD cohort showing a significantly higher response rate (66.7% compared with 47%). It could have been speculated that the higher proportion of NCA non-responders indicated something different about these patients, for example that, like chronic pain patients, they are less amenable to considering psychological approaches. Because only basic demographic data and result of angiogram were available for non-responders it is difficult to comment on this. A logistic regression was done on all the non-responders (NCA and CAD) in an attempt to clarify this point. The basic information available

for these patients was entered, and age was found to be the most powerful predictor ($p < 0.005$), with younger patients less likely to respond. Because NCA patients are generally younger, more non-responders are found to have NCA. It seems that this is partly a function of age, but the influence of some other factor, possibly psychological, specific to NCA patients remains unclear. In examining the reasons for poor uptake of and adherence to psychological treatment for non-cardiac chest pain, Sanders *et al* (1997) found that many patients declined to participate in cognitive-behavioural treatment for non-cardiac chest pain because they found the approach “too psychological”. This may indicate that many NCA patients are averse to participating (be it treatment or assessment) in anything they perceive as psychological. Although in the present study neither patients or doctors knew the angiogram outcome at the point of assessment, it may be that NCA patients have picked up on the absence of any positive findings this far as a message that their chest pain is all in “in their mind”. This would certainly be the kind of picture seen with other chronic pain patients.

However, the low NCA participation was not borne out in phase two with no significant difference in response rates between the CAD and NCA groups (67.9% compared with 65.4%). Given that the recruitment procedure and inclusion criteria were the same for both phases of the study, and there seems to be no discernable reason why the patient group scheduled for angiography should have changed in the intervening years, it is unclear why this may be.

4.3 Physicians' Predictions

Although the response rate of patients in the study could be considered poor (particularly that of NCA patients in phase one), participation in the research was considerably worse for Cardiologists. Despite agreeing to participating in principle,

predictions of angiogram outcome were made for only 11% of angiograms. The majority of these were from senior registrars (one in particular), despite the vast majority of angiograms being carried out by Consultants. It is difficult to know the reasons behind this poor response rate. It could be speculated that Consultants are too busy to fill out the form – although the form was only a matter of ticking a box and stating grade of doctor. The form was stapled to the front of patients notes to allow ease of access so it could be that many forms became detached when pulled out of crammed filing cabinets. Unfortunately no further information is available to illuminate this point. Of those that did respond, predictions were correct in nearly 92% of cases. Clearly it is unwise to make too much of this figure as it is based on such a small and probably unrepresentative sample.

4.4 Implications

It is important to identify as early as possible the likelihood of finding NCA. This can reduce the necessity for an intensive cardiological work-up which, if prolonged, can help convince patients that something is wrong with their heart. Medication could also be reviewed in the light of such findings. As has been discussed in Chapter 1, continued prescriptions of anti-anginal medication can lead to confusion for the patient, and encourage chronic invalidism.

By introducing the possibility of a finding of NCA to the patient early in their cardiology career, it is likely that the eventual outcome will be more easily integrated. Patients can then be provided with possible alternative aetiologies and reassured about their favourable long-term cardiac prognosis. Clear, unambiguous advice about exercise and work capacity should be given to the patient (and their General Practitioner).

Results from this study add support to the view (e.g. of Cooke *et al*, 1997) that the emphasis needs to be given to the relationship of pain to rest (and exercise possibly) rather than the traditionally stressed site, radiation and quality of pain in the differentiation of NCA from CAD pain. Psychological factors – particularly somatic awareness and anxiety – also need to be assessed more routinely. By clarifying factors associated with NCA, other treatments, such as cognitive-behavioural therapies can be implemented more quickly, and before the condition is chronic. Such information will also provide indicators which can be used to further refine these therapies. It could be speculated that cognitive-behavioural treatments would be even more effective with less chronic patients, and would be associated with fewer relapses than drug treatment. The results of this study, taken together with other findings (eg Mayou, Bass and Bryant, 1997) suggest that psychological issues should be integrated into the cardiology clinics routinely from an early stage. This would help normalise the role of psychological factors in the experience of pain and it's management, and facilitate a more easily integrated psychological management approach .

This study goes some way to achieving the above, providing a quick screening tool comprising only several factors, which could be easily integrated in busy cardiology clinics.

4.5 Flaws of the study

One of the major limitations of the study is the subject numbers involved in each phase, although putting phases one and two data together gives a respectable sample. Despite approaching every consecutive patient suitable for inclusion over a 6-month period, and best attempts at contacting patients by letter (and by telephone in phase one), response rates were disappointingly low. This was particularly the case for

NCA patients, the very group of which it was important to maximise the numbers. As a result of this, it was difficult to know whether some of the variables showing trends in the predicted direction would be significant with larger numbers of cases. The high non-participation rate also led to the study being limited in its inability to determine whether the non-responders in phase one were different in some, possibly psychological respect, from the responders.

Information about risk behaviours on each patient was gained by reference to medical notes. Factors such as hypertension, obesity, and lipid status were coded as being either clinically significant or not as determined by the patient's cardiologist. These variables however are not really dichotomous but linear, and can exist to varying degrees of risk. Given this, it would have been better, both methodologically and clinically, for the absolute values (eg. cholesterol level, body mass index) to be noted for this research. Similarly, only two categories of coronary artery status were coded - NCA or CAD. It would have been interesting, although impractical in terms of already low numbers in each group, to also look at a cohort with insignificant CAD.

The NCA group studied was likely to be heterogeneous with regard to pain aetiology, which may have muddied the results somewhat. It may have been more informative (but again limited in terms of low patient numbers) to examine those patients fulfilling the criteria for Syndrome X (for definition see page 5). It could be speculated that the factors associated with NCA found in this study may be attributable to ischaemia through say coronary artery spasm. It would be interesting to look at clusters of symptoms in this way through factor analysis to clarify this.

It is also worth noting that the results of this study apply only to patients whose chest pain is usually fairly chronic and may be less applicable to those with recent onset pain and those who have pain considered less typical.

4.6 Directions for future research

Although in this study family history was not found to differ between the groups, it was only genetic family history which was assessed. It would be interesting in a future study to look at exposure to IHD in relatives, friends, neighbours, the media etc. as it could be hypothesised that experience of IHD, rather than only direct family history of IHD is particularly salient in NCA patients.

The present study involved collecting information via self-report questionnaires. Contact with patients was therefore minimal. A study examining similar factors but using structured interviews would provide an exciting, and possibly more flexible and informative comparison.

As noted above, it would also be useful to look at various categories of disease severity including insignificantly narrowed coronary arteries, instead of the dichotomy used in this study. Three categories of CAD, insignificant CAD and NCA might be viable.

4.7 Conclusions

It seems then, that there are clear factors which are associated with patients found to have normal coronary arteries. These factors are age, sex, somatic awareness and wakening pain. They have been reliably found to significantly discriminate between patients subsequently found to have CAD and those found to have NCA. There is also

a lesser but consistent association with rest pain, anxiety and depression. Those variables may be useful clinically. By inquiring routinely about such factors when taking a history, cardiologists would stand a better chance of anticipating which patients have an increased likelihood of having NCA. In addition to minimising unnecessary investigations, this could better prepare the patient psychologically for this finding from an early stage and allow more appropriate treatments to be instigated.

This study adds to the existing body of research on the importance of psychological characteristics and predictors of normal coronary arteries. It has provided an indicator of which clinical and psychological features are relevant, assessed **prior** to the patients angiogram.

REFERENCES

Alexander PJ, Prabhu SG, Krishnamoorthy ES, Halkatti PC (1994). Mental disorders in patients with noncardiac chest pain. Acta Psychiatrica Scandinavica 89(5) : 291-3.

Anonymous. (1984). Eleven year survival in the Veterans Administration randomised trial of coronary bypass surgery for stable angina. New Engl Journal of Medicine 311 : 1333-9.

Bass C *et al.* (1983). Patients with angina with normal and near normal coronary arteries : clinical and psychosocial state 12 months after angiography. British Medical Journal 287 : 1505-8.

Bass C and Wade C (1984). Chest pain with normal coronary arteries: a comparative study of psychiatric and social morbidity. Psychological Medicine 14, 51-61.

Bass C (1992). Chest pain and breathlessness : relationship to psychiatric illness. American Journal of Medicine 92(1A) : 12-17S.

Bass C (1994). Unexplained chest pain : clinical and psychosocial studies in patients with presumptive angina. Unpublished MD thesis, Institute of Psychiatry, University of London.

Becker MH (Ed) (1974). The health belief model and personal health behaviour. Health Education Monographs 2 : 324-508.

Bennett P, Smith P, Gallacher JEJ (1996). Vital exhaustion, neuroticism and symptom reporting in patients with cardiac and non-cardiac chest pain. British Journal of Health Psychology 1, 309-313.

Boston K, Pearce SA, Richardson PH (1990). The Pain Cognitions Questionnaire. Journal of Psychosomatic Research 34 : 103-9.

Bradley LA *et al* (1992). Psychosocial and psychophysical assessments of patients with unexplained chest pain. American Journal of Medicine 92(5A) : 65S-73S.

Chambers JB *et al.* (1988). Measurement of end-tidal partial pressure of carbon dioxide increases the diagnostic power of treadmill exercise testing. British Medical Journal 296 : 1281-5.

Chambers J, Bass C (1990). Chest pain with normal coronary anatomy. In: Jackson G (ed). Difficult Cardiology : Practical Management and Decision Making. Martin Dunitz.

Chambers J, Bass C (1991). Towards a confident diagnosis of non-cardiac chest pain. In : Creed F, Mayou R, Hopkins A (eds). Patients with medically unexplained physical symptoms. London : Royal College of Physicians and Royal College of Psychiatrists.

Chambers J, Bass C (1998). Atypical chest pain: looking beyond the heart. Quarterly Journal of Medicine; 91;239-244.

Channer RP *et al.* (1987). Failure of a negative exercise test to reassure patients with chest pain. Quarterly Journal of Medicine , 63 : 315-22.

Chaturverdi N, Rai H, Ben-Shlomo Y (1997). Lay diagnosis and health-care-seeking behaviour for chest pain in south Asians and Europeans. Lancet. 350 (9091): 1578-83.

Cooke RA, Chambers JB, Anggiansah A, Henderson RA, Sowton E, Owen W (1991). Chest pain and normal coronary arteries : a clinical evaluation with oesophageal function tests, exercise ECG, end-tidal CO₂ measurement and psychiatric scores. European Heart Journal 12 (suppl) : 103.

Cooke RA, Anggiansah A, Wang J, Chambers JB, Owen W (1996). Hyperventilation and esophageal dysmotility in patients with noncardiac chest pain. The American Journal of Gastroenterology. 91(3), 480-84.

Cooke RA, Smeeton N, Chambers JB (1997). Comparative study of chest pain characteristics in patients with normal and abnormal coronary angiograms. Heart 78(2): 142-146.

Costa PT, McCrea RR (1985). Hypochondriasis, neuroticism, and ageing. When are somatic complaints unfounded? American Psychologist 40 : 19-28.

Da Costa JM (1871). On irritable heart: a clinical study of a form of functional cardiac disorder and its consequences. American Journal of Medical Science 61 : 17-52.

Dammen T, Friis S, Ekeberg O (1999). The Illness Attitude Scales in chest pain patients : a study of psychometric properties. Journal of Psychosomatic Research. 46(4) : 335-42.

Davis MA, Keerbs A, Hoffman JR, Baraff LJ (1996). Admission decisions in emergency department chest pain patients at low risk for myocardial infarction: patient versus physician preferences. Annals of Emergency Medicine. 28(6): 606-11.

Day LJ, Sowton E (1976). Clinical features and follow-up of patients with angina and normal coronary arteries. Lancet ii : 334-7.

Eifert GH (1992). Cardiophobia : a pragmatic behavioural model of heart-focused anxiety and non-anginal chest pain. Behavior Research and Therapy 30(4) : 329-45.

Eifert GH, Hodson SE, Tracey DR, Seville JL, Gunawardane K (1996). Heart-focussed anxiety, illness beliefs, and behavioral impairment: comparing healthy heart-anxious patients with cardiac and surgical inpatients. Journal of Behavioral Medicine. 19(4): 385-99.

Evans DW, Lum LC (1977). Hyperventilation : an important cause of pseudoangina. Lancet i : 155-7.

Faxon DP *et al.* (1982). Therapeutic and economic value of a normal coronary angiogram. American Journal of Medicine 73 : 500-5.

Foster S, Malik M (1998). A comparative study of differences in the referral behaviour patterns of men and women who have experienced cardiac-related chest pain. Intensive and Critical Care Nursing. 14(4); 192-202.

Fraenkel YM, Kindler S, Melmed RN (1996-1997). Differences in cognitions during chest pain of patients with panic disorder and ischaemic heart disease. Depression & Anxiety. 4(5) : 217-22.

Frasure-Smith N (1987). Levels of somatic awareness in relation to angiographic findings. Journal of Psychosomatic Research 31 : 545-54.

Freedland KE *et al* (1996). Psychological determinants of anginal pain perception during exercise testing of stable patients after recovery from acute myocardial infarction or unstable angina pectoris. American Journal of Cardiology. 77(1): 1-4.

Freud S (1894). On the grounds for detaching a particular syndrome from neurasthenia under the description "anxiety neurosis". In : The 2nd Edition of the Complete Psychological Works of Sigmund Freud. Vol III Early Psychoanalytical Publications . London : Hogarth Press, 1962; 90-115.

Gelder MG, Clark DM, Salkovskis P (1993). Cognitive treatment for panic disorder. Journal of Psychiatric Research. 27 Suppl 1:171-8.

Goldberg DP (1972). The detection of psychiatric illness by questionnaire. London : Oxford University Press.

Hannay DR (1978). Symptom prevalence in the community. Journal of the College of General Practitioners 28 : 492-9.

Hartshorne H (1864). On heart disease in the army. American Journal of Medicine Sci 48: 889-92.

Haynes S and Feinleib M (1982). Type A behaviour and the incidence of coronary heart disease in the Framingham Heart Study. Advances in Cardiology, 29: 85-95.

Ho KY, Kang JY, Yeo B, Ng WL (1998). Non-cardiac, non-oesophageal chest pain: the relevance of psychological factors. Gut. 43(1) : 105-10.

Julian DG (1988). Cardiology. Balliere Tindall.

Kaski JC (ed) (1999). Chest pain with normal coronary angiograms : pathogenesis, diagnosis and management. Kluwer Academic Publishers.

Katon W, Hall ML, Russo J, Cormier L, Hollifield M, Vitaliano PP *et al.* (1988). Chest pain: relationship of psychiatric illness to coronary arteriographic results. American Journal of Medicine 84 : 1-9.

Kemp HG *et al.* (1973). The anginal syndromes associated with normal coronary arteriograms : report of a six year experience. American Journal of Medicine 54 : 735-42.

Klimes I *et al* (1990). Psychological treatment for atypical non-cardiac chest pain : a controlled evaluation. Psychological Medicine 20 : 605-11.

Ladwig KH, Hoberg E, Busch R (1998). Psychological comorbidity in patients with alarming chest pain symptoms. Psychotherapie, Psychosomatik, Medizinische Psychologie. 48(2): 46-54.

Ladwig KH, Roll G, Breithardt G, Borggreffe M (1999). Extracardiac contributions to chest pain perception in patients 6 months after acute myocardial infarction. American Heart Journal. 137(3): 528-35.

Lantinga LJ *et al* (1988). One-year psychosocial follow-up of patients with chest pain and angiographically normal coronary arteries. American Journal of Cardiology 62 : 209-13.

Leibing E, Schunemann I, Herrmann C, Ruger U (1998). Psychiatric disorder or coronary heart disease? Psychological test data and ICD-10 diagnoses in patients undergoing coronary angiography. [German]. Psychotherapie, Psychosomatik, Medizinische Psychologie. 48(1) : 30-6.

Lumley MA, Torosian T, Ketterer MW, Pickard SD (1997). Psychosocial factors related to noncardiac chest pain during treadmill testing. Psychosomatics. 38(3) : 230-8.

Main CJ (1983). The Modified Somatic Perception Questionnaire. Journal of Psychosomatic Research 27: 503-14.

Marchandise B *et al* (1978). Angiographic evaluation of the natural history of normal coronary arteries and mild coronary atherosclerosis. American Journal of Cardiology 41 : 216-20.

Master AM (1964). The spectrum of anginal and noncardiac chest pain. Journal of the American Medical Association 187: 894-99.

Mayou R (1973). The patient with angina : symptoms and disability. Postgraduate Medical Journal 42 : 539-49.

Mayou RA (1991). Patients' fears of illness : chest pain and palpitations. In : Creed F, Mayou R, Hopkins A (eds). Patients with medically unexplained physical symptoms. London : Royal College of Physicians and Royal College of Psychiatrists.

Mayou RA, Bryant BM, Sanders D, Bass C, Klimes I, Forfar C (1997). A controlled trial of cognitive behavioural therapy for non-cardiac chest pain. Psychological Medicine 27: 1021-1031.

Mayou RA, Bass CM, Bryant BM (1999). Management of non-cardiac chest pain : from research to clinical practice. Heart. 81(4): 387-92.

McCroskery *et al* (1991). Mitral valve prolapse and neuroticism in chest pain patients with normal and diseased coronary arteries. International Journal of Psychiatry in Medicine 21(3): 233-44.

Mechanic D (1961). The concept of illness behaviour. Journal of Chronic Disability 15 : 189-94.

Ockene IS *et al* (1980). Unexplained chest pain in patients with normal coronary arteriograms. New England Journal of Medicine 303 : 1249-52.

Papanicolaou MN *et al* (1986). Prognostic implication of angiographically normal and insignificantly narrowed coronary arteries. American Journal of Cardiology 58 : 1181-7.

Pasceri V *et al* (1998). Role of abnormal pain sensitivity and behavioural factors in determining chest pain in syndrome X. Journal of the American College of Cardiology. 31(1) : 62-6.

Pasternak RC *et al* (1980). Chest pain with angiographically insignificant coronary arterial obstruction. Clinical presentation and long-term follow-up. American Journal of Medicine 68 : 813-17

Pearce MJ, Mayou RA, Klimes I (1990). The management of atypical non-cardiac chest pain. Quarterly Journal of Medicine 281 : 991-96.

Pilowsky I (1967). Dimensions of hypochondriasis. British Journal of Psychiatry 113 : 89-93.

Pilowsky I (1978). A general classification of abnormal illness behaviours. British Journal of Medical Psychology 51 : 131-37.

Pilowsky I (1978). Pain as abnormal illness behaviour. Journal of Human Stress 4 : 22-27.

Potts SG, Bass CM (1993). Psychosocial outcome and use of medical resources in patients with chest pain and normal or near-normal coronary arteries : a long-term follow-up study. Quarterly Journal of Medicine 86 : 583-93.

Potts SG, Bass CM (1994). Chest pain with normal coronary arteries : psychological aspects. In Kaski JC (Ed). Angina pectoris with normal coronary arteries : syndrome X. 65-89. Kluwer Academic Publishers.

Potts SG, Bass CM (1995). Psychological morbidity in patients with chest pain and normal or near-normal coronary arteries : a long-term follow-up study. Psychological Medicine 25 : 339-47.

Potts SG, Bass C (1999). Chest pain with normal coronary arteries : psychological aspects. In Kaski JC (ed) (1999). Chest pain with normal coronary angiograms : pathogenesis, diagnosis and management. Kluwer Academic Publishers.

Potts SG, Lewin R, Fox KAA, Johnstone EC, Cay EL. Group psychological treatment for chest pain and normal coronary arteries : a controlled trial. Submitted.

Proudfit WL, Brusckhe AVG, Sones FM (1980). Clinical course of patients with normal or slightly or moderately abnormal coronary arteriograms : 10 year follow-up of 521 patients. Circulation 62: 712-17.

Rachman S (1974). Some similarities and differences between obsessional ruminations and morbid preoccupations. Canadian Psychiatric Association Journal 18 : 71-3.

Richter JE, Bradley LA & Castell DO (1989). Esophageal chest pain : current controversies in pathogenesis, diagnosis and therapy. Annals of Internal Medicine 110 : 66-78.

Ruggeri A *et al* (1996). The correlation between the clinical characteristics and psychological status in syndrome X patients [Italian]. Cardiologia. 41(6): 551-7.

Savage DD *et al.* (1983). Mitral valve prolapse in the general population. 2. Clinical Features : The Framingham Study. American Heart Journal 106 : 577-81.

Salkovskis P M (1992). Psychological treatment of noncardiac chest pain: the cognitive approach. American Journal of Medicine 92(5A): 114-121.

Sanders D, Bass C, Mayou RA, Goodwin S, Bryant BM, Tyndel S (1997). Non-cardiac chest pain: why was a brief intervention apparently ineffective? Psychological Medicine 27: 1933-1040.

Serlie AW *et al* (1996). Empirical psychological modeling of chest pain: a comparative study. Journal of Psychosomatic Research. 40 (6), 625-35.

Smith GR, Monson RA & Ray DC (1986). Patients with multiple unexplained symptoms : their characteristics, functional health, and health care utilisation. Archives of Internal Medicine 146 : 69-72.

Torosian T, Lumley MA, Pickard SD, Ketterer MW (1997). Silent versus symptomatic myocardial ischemia: the role of psychological and medical factors. Health Psychology. 16(2):123-30.

Tyrer P (1976). The role of bodily feelings in anxiety. Oxford University Press : London.

van Peski-Oosterbaan AS, Spinhoven P, van der Does AJJ, Bruschke AV (1998). Noncardiac chest pain : interset in medical psychological treatment. Journal of Psychosomatic Research. 45(5): 471-6.

Warwick HMC & Salkovskis PM (1985). Reassurance. British Medical Journal 290 : 1028.

Wielsgosz AT *et al*. (1984). Unimproved chest pain in patients with minimal or no coronary disease : a behavioral problem. American Heart Journal 108 : 67-72.

Wood P (1941). Da Costa's syndrome (or effort syndrome). British Medical Journal i : 767-851.

Zigmond AS , Snaith P (1983). The hospital and anxiety and depression scale. Acta Psychiatrica Scandinavia 67: 361-70.

Appendix A – Letter of explanation

INFORMATION SHEET FOR PATIENTS

An investigation of chest pain in patients prior to coronary angiography

We have contacted you because you are due to come into hospital soon for a coronary angiogram (also called cardiac catheter) to find out the cause of your chest pain. We are running a research project on people with different types of chest pain, and hope you will consider taking part.

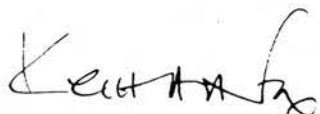
What we would like you to do is fill in some questionnaires which are enclosed. These cover aspects of your chest pain, it's effect on what you do and how you feel. The questionnaires can be filled in any time between now and your hospital admission.

This information will be stored anonymously on computer, and kept in the strictest confidence. You are free to decline to participate in the study, or can withdraw your input at any time. Whether or not you fill in the questionnaires will in no way affect any treatment you are receiving. If you do decide to take part, the answers you give will have no influence on your medical treatment. We simply hope to use the information to help doctors to decide the best way to find out the causes of chest pain in other people in the future.

If you agree to take part please sign the consent form, and put this with the completed questionnaires into the envelope provided. **Bring this with you when you come into hospital for your angiogram, and hand it in when you arrive on the ward.** If you would like to discuss any aspects of the study, or have any questions about the questionnaires, please contact Aileen Thomson on 01592-643785.

The research project has the approval of Lothian Health Board ethics committee. If you would like to refer to someone independent of the study to discuss the research, contact Dr Ronan O'Carroll on 01786 466369.

Thank you for your cooperation



Professor K A A Fox
Consultant Cardiologist



Aileen S Thomson
Chartered Clinical Psychologist

Appendix B – Consent form for patients

NAME : _____

I agree to participate in this study.

I have read the Information Sheet for Patients.

I understand that I am under no obligation to take part in the study, and that a decision not to participate will not alter the treatment I would normally receive.

I understand that I have the right to withdraw from the study at any stage, and that to do so will not affect my treatment.

Signature _____

Date _____

Appendix C – Modified Somatic Perception Questionnaire (MSPQ)

The Modified Somatic Perception Questionnaire

Please describe how you have felt during the PAST WEEK by making a check mark (✓) in the appropriate box. Please answer ALL questions. Do not think too long before answering.

	Not at all	A little/ slightly	A great deal/ quite a bit	Extremely/ could not have been worse
Mouth becoming dry				
Blurring of vision				
Breathing becomes faster				
Sweating all over				
Stomach churning				
Muscles twitching & jumping				
Feeling hot all over				
Dizziness				
Tense feeling across forehead				
Legs feel weak				
Nausea				
Pain in stomach				
Neck muscles aching				

Appendix D – Chest Pain Questionnaire

CHEST PAIN QUESTIONNAIRE

Marital status : _____

Who do you live with ? _____

What is your current employment status ? Full-time work
Part-time work
Sick leave
Long term sick
Retired
Housework
Unemployed

Has chest pain caused you to : Give up work completely
Change to lighter duties at work
Reduce hours of work
Change jobs
No change
Change your work in another
way (please say how _____
_____)

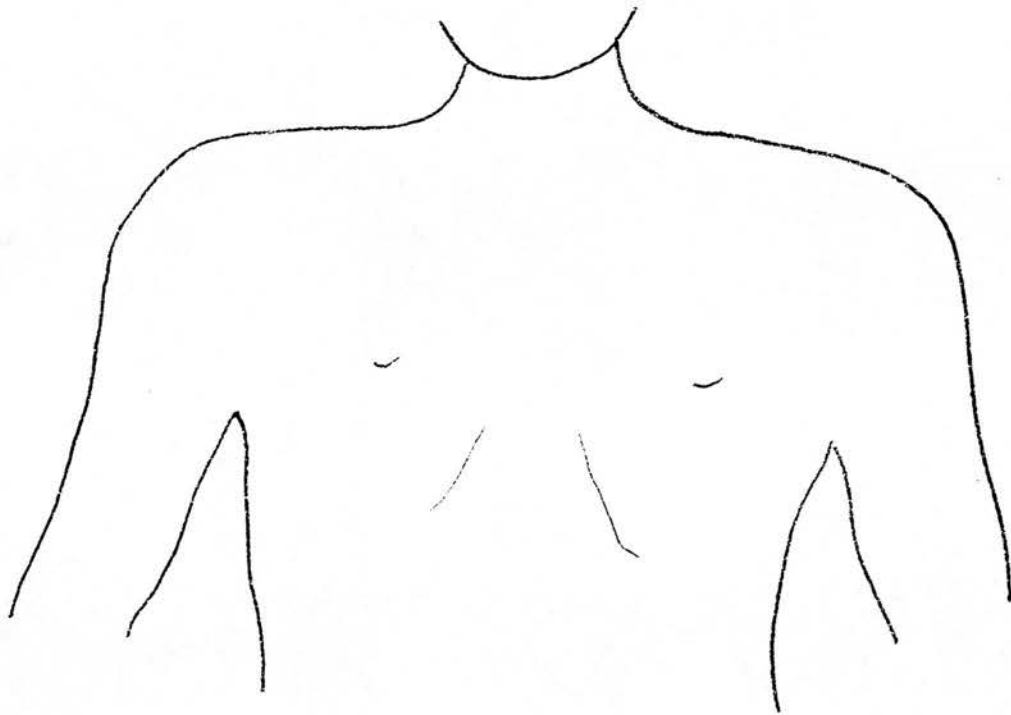
Have you ever smoked ? Yes/No

Do you smoke now ? Yes/No

How many per day do you smoke ? _____

For how long have you been getting chest pain ?
less than 6 months
1 - 6 months
more than 6 months (say how
long _____)

Please mark on the diagram the main site(s) of pain by marking an "X",



Once the pain begins, does it move anywhere else? Please use an arrow to indicate pain spread on the above diagram.

Where else is the pain? (e.g. arm, jaw, back) _____

How long does your pain usually last? less than 5 minutes
5 - 10 minutes
20 minutes - 2 hours
more than 2 hours
(say how long _____)
_____)

Does the pain usually begin gradually (minutes)
suddenly (seconds)

How often does it occur? more than once a day
every day
more than once a week
less than once a week
(say how long _____)
_____)

Appendix E – Pain Cognitions Questionnaire

10

PAIN COGNITIONS QUESTIONNAIRE

There follows a list of thoughts which patients have reported thinking when in pain. Please indicate how often YOU have had these thoughts AT THE TIME OF YOUR PAIN over the last WEEK.

Please circle one answer for each thought.

1	Find yourself thinking you have given up all hope.	not at all	sometimes	often	most of the time
2	Think of something pleasant rather than concentrate on the pain.	not at all	sometimes	often	most of the time
3	Trust the doctors and believe they can do something.	not at all	sometimes	often	most of the time
4	Want not to wake up in the morning.	not at all	sometimes	often	most of the time
5	Take a hopeful view of things.	not at all	sometimes	often	most of the time
6	Think that further treatment will cause more pain.	not at all	sometimes	often	most of the time
7	Think it is unfair that you can't do the things you used to do.	not at all	sometimes	often	most of the time
8	Reassure yourself that you can get used to being in pain.	not at all	sometimes	often	most of the time
9	Remind yourself about the support and encouragement you get from other people	not at all	sometimes	often	most of the time
10	Think that you might become a burden to your family and friends.	not at all	sometimes	often	most of the time
11	Think that others pressurise you to do things you can't.	not at all	sometimes	often	most of the time
12	Think that even your close friends are no help.	not at all	sometimes	often	most of the time
13	Think that there is no-one there to care about you.	not at all	sometimes	often	most of the time
14	Think that the doctors might start to dislike you.	not at all	sometimes	often	most of the time
15	Remind yourself that you have to be positive about the pain.	not at all	sometimes	often	most of the time
16	Reassure yourself that you can cope now because you have coped in the past.	not at all	sometimes	often	most of the time
17	Make a conscious effort to think the pain away.	not at all	sometimes	often	most of the time

18	Think anxiously about the things that might bring on the pain.	not at all	sometimes	often	most of the time
19	Think that you won't let the pain get the better of you.	not at all	sometimes	often	most of the time
20	Ask what you have done to deserve this pain.	not at all	sometimes	often	most of the time
21	Blame the doctor (or hospital, or operation) for your condition.	not at all	sometimes	often	most of the time
22	Tell yourself that you must be optimistic.	not at all	sometimes	often	most of the time
23	Tell yourself that there is no point in sitting around crying.	not at all	sometimes	often	most of the time
24	Think that people patronise you because of your condition.	not at all	sometimes	often	most of the time
25	Wish the pain would go away.	not at all	sometimes	often	most of the time
26	Accept the pain to an extent.	not at all	sometimes	often	most of the time
27	Expect there to be no relief at all.	not at all	sometimes	often	most of the time
28	Think of things to do to help distract yourself from the pain.	not at all	sometimes	often	most of the time
29	Think about not being able to go on putting up with the pain.	not at all	sometimes	often	most of the time
30	Reassure yourself that you are not generally unhappy	not at all	sometimes	often	most of the time

Appendix F – Whitely Index of Hypochondriasis

Appendix G – Hospital Anxiety and Depression Scale

HAD Scale

Name: _____

Date: _____

Doctors are aware that emotions play an important part in most illnesses.

This questionnaire is designed to help your doctor to know how you feel. Read each item and place a firm tick in the box opposite the reply which comes closest to how you have been feeling in the past week.

Don't take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

Tick only one box in each section

I feel tense or 'wound up':

- Most of the time
- A lot of the time
- Time to time, Occasionally
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel as if I am slowed down:

- Nearly all the time
- Very often
- Sometimes
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I still enjoy the things I used to enjoy:

- Definitely as much
- Not quite so much
- Only a little
- Hardly at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get a sort of frightened feeling like 'butterflies' in the stomach:

- Not at all
- Occasionally
- Quite often
- Very often

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get a sort of frightened feeling as if something awful is about to happen:

- Very definitely and quite badly
- Yes, but not too badly
- A little, but it doesn't worry me
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I have lost interest in my appearance:

- Definitely
- I don't take so much care as I should.....
- I may not take quite as much care
- I take just as much care as ever

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can laugh and see the funny side of things:

- As much as I always could
- Not quite so much now
- Definitely not so much now
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel restless as if I have to be on the move:

- Very much indeed
- Quite a lot
- Not very much
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

Worrying thoughts go through my mind:

- A great deal of the time
- A lot of the time
- From time to time but not too often
- Only occasionally

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I look forward with enjoyment to things:

- As much as ever I did
- Rather less than I used to
- Definitely less than I used to
- Hardly at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I feel cheerful:

- Not at all
- Not often
- Sometimes
- Most of the time

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I get sudden feelings of panic:

- Very often indeed
- Quite often
- Not very often
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can sit at ease and feel relaxed:

- Definitely
- Usually
- Not often
- Not at all

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

I can enjoy a good book or radio or TV programme:

- Often
- Sometimes
- Not often
- Very seldom

<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

Do not write below this line

Appendix H – Chest Pain Diary

Chest Pain Diary

Name: Date started:

Please fill in this diary every day for the next 7 days. It would be best if you could carry it with you and fill it in every time you get any of the symptoms of chest pain, (pain or tightness in the chest, pain in the arms or throat etc).. If that is not possible please try and fill it in at the same time each day, convenient times might be, on getting up - at lunch time - around mid-day - at tea time - after six o'clock - and on going to bed.

Please put the time the chest pain started and finished.

Please give a score out of 100 as to how bad the pain or tightness was, 100 would be "worst possible pain" and 1 would be "so slight hardly noticed it".

Please put down the number of pills or puffs of spray you needed to take. If you sometimes take a pill or a spray before you get the pain or tightness, please put that down as well. Don't put down the other pills you may have to take each day on a regular basis. If you need extra space please use more paper.

This record is very important, thanks for your help.

1. Day

Midnight to 7 O'clock in morning
 Time How bad 100 = worst

Example
 Time 6.30-6.45
 How bad 60

Number of pills taken
 How bad 100 = worst

7 o'clock to 12 o'clock in morning
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

12 Midday to 6 O'clock in afternoon
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

6 pm to 12 o'clock at night
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

2. Day

Midnight to 7 O'clock in morning
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

7 o'clock to 12 o'clock in morning
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

6 pm to 12 o'clock at night
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

3. Day

Midnight to 7 O'clock in morning
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

7 o'clock to 12 o'clock in morning
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

12 Midday to 6 O'clock in afternoon
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

6 pm to 12 o'clock at night
 Time How bad 100 = worst

Number of pills taken
 How bad 100 = worst

Appendix I – Phase I demographic data t-tests

Table I.1: Age by angiogram result

T-Test

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
AGE	NCA	51	54.6667	7.8247	1.0957
	CAD	72	59.6944	8.8789	1.0464

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means								
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean	
									Lower	Upper
AGE	Equal variances assumed Equal variances not assumed	.634	.427	-3.247	121	.002	-5.0278	1.5482	-8.0929	-1.9627
				-3.319	115.271	.001	-5.0278	1.5151	-8.0288	-2.0268

Appendix J: Chi squared tables and crosstabulations for phase one demographic data

Table J.1: Sex by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Sex	Male	Count	23	62	85
		% within ANGIORES	44.2%	67.4%	59.0%
	Female	Count	29	30	59
		% within ANGIORES	55.8%	32.6%	41.0%
Total		Count	52	92	144
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	7.369 ^b	1	.007		
Continuity ^a Correction	6.442	1	.011		
Likelihood Ratio	7.340	1	.007		
Fisher's Exact Test				.008	.006
Linear-by-Linear Association	7.317	1	.007		
N of Valid Cases	144				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 21.31.

Table J.2: Marital status by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
MARITAL	married	Count	19	47	66
		% within ANGIORES	76.0%	75.8%	75.9%
	single	Count	2	4	6
		% within ANGIORES	8.0%	6.5%	6.9%
	divorced/separated	Count	4	4	8
		% within ANGIORES	16.0%	6.5%	9.2%
	widowed	Count		7	7
		% within ANGIORES		11.3%	8.0%
Total		Count	25	62	87
		% within ANGIORES	100.0%	100.0%	100.0%

Table J.3: Living status by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
LIVING	spouse/partner	Count	17	45	62
		% within ANGIORES	68.0%	72.6%	71.3%
	alone	Count	2	12	14
		% within ANGIORES	8.0%	19.4%	16.1%
	spouse+kids	Count	4	3	7
		% within ANGIORES	16.0%	4.8%	8.0%
	parents	Count	1	1	2
		% within ANGIORES	4.0%	1.6%	2.3%
	kids	Count	1	1	2
		% within ANGIORES	4.0%	1.6%	2.3%
Total		Count	25	62	87
		% within ANGIORES	100.0%	100.0%	100.0%

Table J.4: *Employment status by angiogram result*

Crosstab

			ANGIORES		Total
			NCA	CAD	
employ	full-time	Count	6	16	22
		% within ANGIORES	24.0%	25.8%	25.3%
	part-time	Count	1		1
		% within ANGIORES	4.0%		1.1%
	sick leave	Count	2	7	9
		% within ANGIORES	8.0%	11.3%	10.3%
	long-term sick	Count	2	6	8
		% within ANGIORES	8.0%	9.7%	9.2%
	retired	Count	7	26	33
		% within ANGIORES	28.0%	41.9%	37.9%
	housework	Count	5	1	6
		% within ANGIORES	20.0%	1.6%	6.9%
	unemployed	Count	2	6	8
		% within ANGIORES	8.0%	9.7%	9.2%
Total		Count	25	62	87
		% within ANGIORES	100.0%	100.0%	100.0%

Table J.5: Change in employment by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
JOBCHAN	given up	Count	5	15	20
		% within ANGIORES	20.8%	26.3%	24.7%
	lighter duties	Count	4	7	11
		% within ANGIORES	16.7%	12.3%	13.6%
	reduced hours	Count	2	6	8
		% within ANGIORES	8.3%	10.5%	9.9%
	no change	Count	13	29	42
		% within ANGIORES	54.2%	50.9%	51.9%
Total		Count	24	57	81
		% within ANGIORES	100.0%	100.0%	100.0%

Appendix K – Chi squared tables and crosstabulations for phase one physical data

Table K.1: Lifetime smoking risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
EVERSMOK	yes	Count	15	44	59
		% within ANGIORES	60.0%	71.0%	67.8%
	no	Count	10	18	28
		% within ANGIORES	40.0%	29.0%	32.2%
Total		Count	25	62	87
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.982 ^b	1	.322		
Continuity Correction ^a	.544	1	.461		
Likelihood Ratio	.962	1	.327		
Fisher's Exact Test				.325	.229
Linear-by-Linear Association	.971	1	.325		
N of Valid Cases	87				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 8.05.

Table K.2: Current smoker (self-report) by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Risk smoking	current smoker	Count	13	18	31
		% within ANGIORES	26.5%	20.9%	23.0%
	non-smoker	Count	31	42	73
		% within ANGIORES	63.3%	48.8%	54.1%
	previous smoker	Count	5	26	31
		% within ANGIORES	10.2%	30.2%	23.0%
Total		Count	49	86	135
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.081 ^a	2	.029
Likelihood Ratio	7.785	2	.020
Linear-by-Linear Association	4.431	1	.035
N of Valid Cases	135		

a. 0 cells (.0%) have expected count less than 5.
The minimum expected count is 11.25.

Table K.3: Smoking risk (as assessed by cardiologist) by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
CURRSMOK	yes	Count	8	9	17
		% within ANGIORES	32.0%	14.5%	19.5%
	no	Count	17	53	70
		% within ANGIORES	68.0%	85.5%	80.5%
Total		Count	25	62	87
		% within ANGIORES	100.0%	100.0%	100.0%

Table K.5: Lipid risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
RISKLIP	yes	Count	9	39	48
		% within ANGIORES	18.4%	46.4%	36.1%
	no	Count	40	45	85
		% within ANGIORES	81.6%	53.6%	63.9%
Total		Count	49	84	133
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.565 ^b	1	.001		
Continuity Correction ^a	9.384	1	.002		
Likelihood Ratio	11.189	1	.001		
Fisher's Exact Test				.001	.001
Linear-by-Linear Association	10.486	1	.001		
N of Valid Cases	133				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 17.68.

Table K.6: Diabetes risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Riskdiabetes	Yes	Count	1	4	5
		% within ANGIORES	2.0%	4.7%	3.7%
	No	Count	48	81	129
		% within ANGIORES	98.0%	95.3%	96.3%
Total		Count	49	85	134
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.615 ^b	1	.433		
Continuity Correction ^a	.097	1	.756		
Likelihood Ratio	.672	1	.412		
Fisher's Exact Test				.652	.395
Linear-by-Linear Association	.610	1	.435		
N of Valid Cases	134				

a. Computed only for a 2x2 table

b. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.83.

Table K.7: Hypertension risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Riskhyper	yes	Count	14	26	40
		% within ANGIORES	28.6%	30.6%	29.9%
	No	Count	35	59	94
		% within ANGIORES	71.4%	69.4%	70.1%
Total		Count	49	85	134
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.060 ^b	1	.806		
Continuity Correction ^a	.002	1	.960		
Likelihood Ratio	.061	1	.806		
Fisher's Exact Test				.847	.483
Linear-by-Linear Association	.060	1	.807		
N of Valid Cases	134				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 14.63.

Table K8.: Family history risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
riskfh	yes	Count	20	32	52
		% within ANGIORES	40.8%	37.6%	38.8%
	no	Count	29	53	82
		% within ANGIORES	59.2%	62.4%	61.2%
Total		Count	49	85	134
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.131 ^b	1	.717		
Continuity Correction ^a	.032	1	.858		
Likelihood Ratio	.131	1	.717		
Fisher's Exact Test				.717	.428
Linear-by-Linear Association	.130	1	.718		
N of Valid Cases	134				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 19.01.

Table K.9: Lifetime smoking risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
POSETT	yes	Count	11	35	46
		% within ANGIORES	44.0%	85.4%	69.7%
	no	Count	13	4	17
		% within ANGIORES	52.0%	9.8%	25.8%
	Inconclusive	Count	1	2	3
		% within ANGIORES	4.0%	4.9%	4.5%
Total		Count	25	41	66
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	14.599 ^a	2	.001
Likelihood Ratio	14.601	2	.001
Linear-by-Linear Association	4.692	1	.030
N of Valid Cases	66		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 1.14.

Table K.10: ECG result by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
ECG positive	Count	6	8	14	
	% within ANGIORES	50.0%	57.1%	53.8%	
normal	Count	6	4	10	
	% within ANGIORES	50.0%	28.6%	38.5%	
equivocal	Count		2	2	
	% within ANGIORES		14.3%	7.7%	
Total	Count	12	14	26	
	% within ANGIORES	100.0%	100.0%	100.0%	

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.547 ^a	2	.280
Likelihood Ratio	3.308	2	.191
Linear-by-Linear Association	.079	1	.779
N of Valid Cases	26		

a. 3 cells (50.0%) have expected count less than 5. The minimum expected count is .92.

Table K.11: *GTN relief by angiogram result*

Crosstab

			ANGIORES		Total
			NCA	CAD	
GTNREL	yes	Count	16	48	64
		% within ANGIORES	72.7%	90.6%	85.3%
	no	Count	6	5	11
		% within ANGIORES	27.3%	9.4%	14.7%
Total		Count	22	53	75
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.953 ^b	1	.047		
Continuity Correction ^a	2.656	1	.103		
Likelihood Ratio	3.629	1	.057		
Fisher's Exact Test				.071	.056
Linear-by-Linear Association	3.900	1	.048		
N of Valid Cases	75				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.23.

Table K.12: *Other relief by angiogram result*

Crosstab

			ANGIORES		Total
			NCA	CAD	
OTHREL	rest	Count	19	47	66
		% within ANGIORES	86.4%	90.4%	89.2%
	other drugs	Count	3	3	6
		% within ANGIORES	13.6%	5.8%	8.1%
	positions	Count		2	2
		% within ANGIORES		3.8%	2.7%
Total		Count	22	52	74
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.054 ^a	2	.358
Likelihood Ratio	2.517	2	.284
Linear-by-Linear Association	.000	1	.987
N of Valid Cases	74		

a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .59.

Table K.13: Alert pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
ALERTPAI	yes	Count	23	33	56
		% within ANGIORES	95.8%	58.9%	70.0%
	no	Count	1	23	24
		% within ANGIORES	4.2%	41.1%	30.0%
Total		Count	24	56	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.896 ^b	1	.001		
Continuity ^a Correction	9.209	1	.002		
Likelihood Ratio	13.587	1	.000		
Fisher's Exact Test				.001	.000
Linear-by-Linear Association	10.759	1	.001		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.20.

Table K.14: Wakening pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
WAKEPAIN	yes	Count	16	20	36
		% within ANGIORES	66.7%	35.1%	44.4%
	no	Count	8	37	45
		% within ANGIORES	33.3%	64.9%	55.6%
Total		Count	24	57	81
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	6.821 ^b	1	.009		
Continuity Correction ^a	5.602	1	.018		
Likelihood Ratio	6.864	1	.009		
Fisher's Exact Test				.014	.009
Linear-by-Linear Association	6.737	1	.009		
N of Valid Cases	81				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 10.67.

Table K.15: Pain site by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINSITE	central retrosternal	Count	14	23	37
		% within ANGIORES	56.0%	41.1%	45.7%
	left sternal edge	Count	2	6	8
		% within ANGIORES	8.0%	10.7%	9.9%
	inframammary	Count	3	8	11
		% within ANGIORES	12.0%	14.3%	13.6%
	right side of chest	Count		3	3
		% within ANGIORES		5.4%	3.7%
	epigastric	Count		5	5
		% within ANGIORES		8.9%	6.2%
	left shoulder + arm	Count	1	1	2
	% within ANGIORES	4.0%	1.8%	2.5%	
neck	Count		5	5	
	% within ANGIORES		8.9%	6.2%	
multiple L + R sites	Count	3	4	7	
	% within ANGIORES	12.0%	7.1%	8.6%	
jaw	Count	2	1	3	
	% within ANGIORES	8.0%	1.8%	3.7%	
Total	Count	25	56	81	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table K.16: Pain spread by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINSPRE	to L arm/shoulder	Count % within ANGIORES	6 24.0%	7 12.3%	13 15.9%
	to L sternal edge	Count % within ANGIORES		2 3.5%	2 2.4%
	to R arm/shoulder	Count % within ANGIORES	1 4.0%	2 3.5%	3 3.7%
	to both arms	Count % within ANGIORES	2 8.0%	9 15.8%	11 13.4%
	to back	Count % within ANGIORES	1 4.0%	1 1.8%	2 2.4%
	to neck	Count % within ANGIORES	2 8.0%	6 10.5%	8 9.8%
	radiates L + R	Count % within ANGIORES	2 8.0%	1 1.8%	3 3.7%
	to L arm + neck	Count % within ANGIORES	4 16.0%	3 5.3%	7 8.5%
	no spread indicated	Count % within ANGIORES	7 28.0%	25 43.9%	32 39.0%
	across chest	Count % within ANGIORES		1 1.8%	1 1.2%
	Total	Count % within ANGIORES	25 100.0%	57 100.0%	82 100.0%

Table K.17: *Other pain sites by angiogram result*

Crosstab

			ANGIORES		Total
			NCA	CAD	
OTHERSIT	jaw	Count	2	6	8
		% within ANGIORES	8.0%	10.3%	9.6%
	arm	Count	4	22	26
		% within ANGIORES	16.0%	37.9%	31.3%
	back	Count	6	9	15
		% within ANGIORES	24.0%	15.5%	18.1%
	other	Count	1		1
	% within ANGIORES	4.0%		1.2%	
	none	Count	5	16	21
		% within ANGIORES	20.0%	27.6%	25.3%
	neck/throat	Count	7	5	12
		% within ANGIORES	28.0%	8.6%	14.5%
Total		Count	25	58	83
		% within ANGIORES	100.0%	100.0%	100.0%

Table K.18: Pain aggravators by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINAGG	coughing	Count	1	4	5
		% within ANGIORES	4.3%	7.0%	6.3%
	breathing deeply	Count	4	4	8
		% within ANGIORES	17.4%	7.0%	10.0%
	swallowing	Count		2	2
		% within ANGIORES		3.5%	2.5%
bending or stooping	Count	10	24	34	
	% within ANGIORES	43.5%	42.1%	42.5%	
other	Count	1	3	4	
	% within ANGIORES	4.3%	5.3%	5.0%	
nothing	Count	7	20	27	
	% within ANGIORES	30.4%	35.1%	33.8%	
Total	Count	23	57	80	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table K.19: Pain duration by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINDUR	< 5 mins	Count	5	21	26
		% within ANGIORES	20.8%	36.8%	32.1%
	5 - 10 mins	Count	2	2	4
		% within ANGIORES	8.3%	3.5%	4.9%
20 mins - 2 hrs	Count	7	23	30	
	% within ANGIORES	29.2%	40.4%	37.0%	
> 2 hrs	Count	10	11	21	
	% within ANGIORES	41.7%	19.3%	25.9%	
Total	Count	24	57	81	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table K.20: Pain onset by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINON	gradually	Count	12	38	50
		% within ANGIORES	48.0%	66.7%	61.0%
	suddenly	Count	13	18	31
		% within ANGIORES	52.0%	31.6%	37.8%
	DK	Count		1	1
		% within ANGIORES		1.8%	1.2%
Total		Count	25	57	82
		% within ANGIORES	100.0%	100.0%	100.0%

Table K.21: Pain frequency by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINFREQ	> once a day	Count	8	17	25
		% within ANGIORES	36.4%	31.5%	32.9%
	every day	Count	3	16	19
		% within ANGIORES	13.6%	29.6%	25.0%
	> once a week	Count	10	16	26
		% within ANGIORES	45.5%	29.6%	34.2%
	< once a week	Count	1	4	5
		% within ANGIORES	4.5%	7.4%	6.6%
	< once a month	Count		1	1
		% within ANGIORES		1.9%	1.3%
Total		Count	22	54	76
		% within ANGIORES	100.0%	100.0%	100.0%

Table K.21: Pain stress-provoked by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
STRESS	yes	Count	16	24	40
		% within ANGIORES	69.6%	42.1%	50.0%
	no	Count	7	33	40
		% within ANGIORES	30.4%	57.9%	50.0%
Total		Count	23	57	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.943 ^b	1	.026	.047	.023
Continuity Correction ^a	3.905	1	.048		
Likelihood Ratio	5.045	1	.025		
Fisher's Exact Test					
Linear-by-Linear Association	4.881	1	.027		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 11.50.

Table K.22: Pain cold-provoked by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
COLD	yes	Count	14	36	50
		% within ANGIORES	60.9%	63.2%	62.5%
	no	Count	9	21	30
		% within ANGIORES	39.1%	36.8%	37.5%
Total		Count	23	57	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.037 ^b	1	.848		
Continuity Correction ^a	.000	1	1.000		
Likelihood Ratio	.036	1	.849		
Fisher's Exact Test				1.000	.522
Linear-by-Linear Association	.036	1	.849		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 8.63.

Table K.23: Pain food-provoked by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
FOOD	yes	Count	3	6	9
		% within ANGIORES	13.0%	10.5%	11.3%
	no	Count	20	51	71
		% within ANGIORES	87.0%	89.5%	88.8%
Total		Count	23	57	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.104 ^b	1	.747		
Continuity Correction ^a	.000	1	1.000		
Likelihood Ratio	.101	1	.750		
Fisher's Exact Test				.712	.509
Linear-by-Linear Association	.103	1	.749		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 2.59.

Table K.24: Pain sex-provoked by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
SEXPROV	yes	Count	3	9	12
		% within ANGIORES	13.0%	15.8%	15.0%
	no	Count	20	48	68
		% within ANGIORES	87.0%	84.2%	85.0%
Total		Count	23	57	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.097 ^b	1	.756		
Continuity ^a Correction	.000	1	1.000		
Likelihood Ratio	.099	1	.753		
Fisher's Exact Test				1.000	.528
Linear-by-Linear Association	.096	1	.757		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.45.

Table K.25: Pain stooping-provoked by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
STOOP	yes	Count	13	22	35
		% within ANGIORES	56.5%	38.6%	43.8%
	no	Count	10	35	45
		% within ANGIORES	43.5%	61.4%	56.3%
Total		Count	23	57	80
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	2.140 ^b	1	.144		
Continuity Correction ^a	1.473	1	.225		
Likelihood Ratio	2.130	1	.144		
Fisher's Exact Test				.213	.113
Linear-by-Linear Association	2.113	1	.146		
N of Valid Cases	80				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 10.06.

Table K.26: Questionnaire return by angiogram result

responded * ANGIORES Crosstabulation

Count		ANGIORES		Total
		NCA	CAD	
responded	yes	24	48	72
	no	27	24	51
Total		51	72	123

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.729 ^b	1	.030		
Continuity Correction ^a	3.956	1	.047		
Likelihood Ratio	4.729	1	.030		
Fisher's Exact Test				.041	.023
Linear-by-Linear Association	4.691	1	.030		
N of Valid Cases	123				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 21.15.

Appendix I – t-tests phase one physical data

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
NOSMOKE	NCA	24	4.7500	7.3854	1.5075
	CAD	48	2.9792	8.2423	1.1897
episodes pain on hill /10	NCA	21	7.9048	3.2543	.7101
	CAD	44	9.2500	1.7135	.2583
num episodes pain on rest / 10	NCA	22	5.0909	2.9586	.6308
	CAD	41	2.2927	2.9685	.4636
GTNREL	NCA	21	1.2381	.4364	9.524E-02
	CAD	43	1.0930	.2939	4.482E-02

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means								
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean	
									Lower	Upper
NOSMOKE Equal variances assumed Equal variances not assumed	.911	.343	.889	70	.377	1.7708	1.9927	-2.2035	5.7452	
episodes pain on hill /10 Equal variances assumed Equal variances not assumed	16.305	.000	-2.190	63	.032	-1.3452	.6144	-2.5730	-.1175	
num episodes pain on rest / 10 Equal variances assumed Equal variances not assumed	.065	.799	3.571	61	.001	2.7982	.7836	1.2313	4.3652	
GTNREL Equal variances assumed Equal variances not assumed	9.553	.003	1.573	62	.121	.1451	9.221E-02	-3.92E-02	.3294	
			1.378	29.159	.179	.1451	.1053	-7.02E-02	.3603	

Appendix M – t-tests for phase one psychological data

T-Test

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
HADanx	NCA	23	8.8696	6.1000	1.2719
	CAD	47	7.2340	3.5338	.5155
had depression	NCA	23	6.9565	4.9495	1.0321
	CAD	47	5.4468	3.7114	.5414
MSPQ	NCA	20	10.3000	7.3991	1.6545
	CAD	47	7.4894	5.3401	.7789
WIBP	NCA	22	1.2727	1.1205	.2389
	CAD	48	1.1250	1.0442	.1507
WIDC	NCA	22	.5455	.9625	.2052
	CAD	48	.5000	.7146	.1031
WIIP	NCA	22	.8182	1.0970	.2339
	CAD	48	.9167	1.0071	.1454
WITOTAL	NCA	22	3.7273	3.7056	.7900
	CAD	48	4.3125	4.2434	.6125

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means									
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean		
									Lower	Upper	
HADanx Equal variances assumed	10.882	.002	1.420	68	.160	1.6355	1.1518	-1.1695	3.9338		
Equal variances not assumed			1.192	29.440	.243	1.6355	1.3724	-1.1695	4.4406		
had depression Equal variances assumed	3.907	.052	1.429	68	.158	1.5097	1.0567	-.5989	3.6183		
Equal variances not assumed			1.295	34.522	.204	1.5097	1.1654	-.8574	3.8768		
MSPQ Equal variances assumed	1.514	.223	1.750	65	.085	2.8106	1.6059	-.3967	6.0179		
Equal variances not assumed			1.537	27.792	.136	2.8106	1.8287	-.9365	6.5578		
WIBP Equal variances assumed	.901	.346	.537	68	.593	.1477	.2751	-.4012	.6966		
Equal variances not assumed			.523	38.334	.604	.1477	.2825	-.4239	.7194		
WIDC Equal variances assumed	2.679	.106	.221	68	.826	4.545E-02	.2058	-.3652	.4562		
Equal variances not assumed			.198	32.037	.844	4.545E-02	.2297	-.4222	.4122		

T-Test

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
PCDISTR	NCA	22	22.6364	4.2822	.9130
	CAD	43	22.5581	5.1237	.7814
PCHHELP	NCA	22	5.3182	2.1018	.4481
	CAD	43	4.9535	1.4467	.2206
PCHOPE	NCA	22	7.4091	2.3434	.4996
	CAD	43	7.1163	1.9906	.3036
PCSUPP	NCA	22	14.3182	2.2549	.4807
	CAD	43	14.5349	1.5016	.2290

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means									
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean		
									Lower	Upper	
PCHHELP	Equal variances assumed Equal variances not assumed	1.433	.236	.822	63	.414	.3647	.4439	-.5224	1.2518	
PCHOPE	Equal variances assumed Equal variances not assumed	.471	.495	.528	63	.599	.2928	.5543	-.8149	1.4005	
PCDISTR	Equal variances assumed Equal variances not assumed	.354	.554	.061	63	.951	7.822E-02	1.2738	-2.4672	2.6237	
PCSUPP	Equal variances assumed Equal variances not assumed	4.613	.036	-.462	63	.645	-.2167	.4688	-1.1534	.7200	
				-.407	30.817	.687	-.2167	.5325	-1.3030	.8696	

Appendix N –t-test for phase two demographic data

Table N.1: age by angiogram result

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
AGE	NCA	26	52.6154	9.8105	1.9240
	CAD	84	59.2500	9.2566	1.0100

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means								
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean	
									Lower	Upper
AGE	Equal variances assumed	.069	.794	-3.149	108	.002	-6.6346	2.1068	-10.8107	-2.4585
	Equal variances not assumed			-3.053	39.767	.004	-6.6346	2.1730	-11.0272	-2.2421

Appendix O – Chi squared tables and crosstabulations for phase two demographic data

Table O.1 : Questionnaire return by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
responded	yes	Count	17	57	74
		% within ANGIORES	65.4%	67.9%	67.3%
	no	Count	9	27	36
		% within ANGIORES	34.6%	32.1%	32.7%
Total		Count	26	84	110
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.055 ^b	1	.814		
Continuity ^a Correction	.000	1	1.000		
Likelihood Ratio	.055	1	.815		
Fisher's Exact Test				.815	.496
Linear-by-Linear Association	.055	1	.815		
N of Valid Cases	110				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 8.51.

Table N.2 : Sex by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Sex	Male	Count	9	61	70
		% within ANGIORES	34.6%	72.6%	63.6%
	Female	Count	17	23	40
		% within ANGIORES	65.4%	27.4%	36.4%
Total		Count	26	84	110
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	12.392 ^b	1	.000		
Continuity Correction ^a	10.804	1	.001		
Likelihood Ratio	12.046	1	.001		
Fisher's Exact Test				.001	.001
Linear-by-Linear Association	12.279	1	.000		
N of Valid Cases	110				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 9.45.

Table O.3 : Marital status by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
MARITAL	married	Count	11	40	51
		% within ANGIORES	68.8%	71.4%	70.8%
	single	Count	2		2
		% within ANGIORES	12.5%		2.8%
	divorced/separated	Count	2	9	11
		% within ANGIORES	12.5%	16.1%	15.3%
	widowed	Count	1	7	8
		% within ANGIORES	6.3%	12.5%	11.1%
Total		Count	16	56	72
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	7.554 ^a	3	.056
Likelihood Ratio	6.636	3	.084
Linear-by-Linear Association	.184	1	.668
N of Valid Cases	72		

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .44.

Table O.4 : Living status by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
LIVING	spouse/partner	Count	6	43	49
		% within ANGIORES	37.5%	76.8%	68.1%
	alone	Count	4	9	13
		% within ANGIORES	25.0%	16.1%	18.1%
	spouse+kids	Count	5	2	7
		% within ANGIORES	31.3%	3.6%	9.7%
	kids	Count	1	2	3
		% within ANGIORES	6.3%	3.6%	4.2%
Total		Count	16	56	72
		% within ANGIORES	100.0%	100.0%	100.0%

Table O.5 : Employment status by angiogram result

Appendix P – Chi squared tables and crosstabulations for phase two physical data

Table P.1 : Lifetime smoking risk by angiogram result

EVERSMOK * ANGIORES Crosstabulation

			ANGIORES		Total
			NCA	CAD	
EVERSMOK	yes	Count	10	44	54
		% within ANGIORES	62.5%	75.9%	73.0%
	no	Count	6	14	20
		% within ANGIORES	37.5%	24.1%	27.0%
Total		Count	16	58	74
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.135 ^b	1	.287		
Continuity Correction ^a	.559	1	.455		
Likelihood Ratio	1.083	1	.298		
Fisher's Exact Test				.344	.224
Linear-by-Linear Association	1.120	1	.290		
N of Valid Cases	74				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 4.32.

Table P.2 : Current smoker (self-report) by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
CURRSMOK	yes	Count	2	13	15
		% within ANGIORES	12.5%	22.4%	20.3%
	no	Count	14	45	59
		% within ANGIORES	87.5%	77.6%	79.7%
Total		Count	16	58	74
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.763 ^b	1	.383		
Continuity Correction ^a	.273	1	.602		
Likelihood Ratio	.831	1	.362		
Fisher's Exact Test				.499	.313
Linear-by-Linear Association	.752	1	.386		
N of Valid Cases	74				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.24.

Table P.3 : Smoking risk (as assessed by cardiologist) by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Risk smoking	current smoker	Count	5	25	30
		% within ANGIORES	35.7%	41.7%	40.5%
	non-smoker	Count	6	18	24
		% within ANGIORES	42.9%	30.0%	32.4%
	previous smoker	Count	3	17	20
		% within ANGIORES	21.4%	28.3%	27.0%
Total	Count	14	60	74	
	% within ANGIORES	100.0%	100.0%	100.0%	

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.878 ^a	2	.645
Likelihood Ratio	.853	2	.653
Linear-by-Linear Association	.002	1	.969
N of Valid Cases	74		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 3.78.

Table P.4 Obesity risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
riskobes1	yes	Count	7	12	19
		% within ANGIORES	87.5%	85.7%	86.4%
	no	Count	1	2	3
		% within ANGIORES	12.5%	14.3%	13.6%
Total	Count	8	14	22	
	% within ANGIORES	100.0%	100.0%	100.0%	

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.014 ^b	1	.907		
Continuity ^a Correction	.000	1	1.000		
Likelihood Ratio	.014	1	.906		
Fisher's Exact Test				1.000	.709
Linear-by-Linear Association	.013	1	.909		
N of Valid Cases	22				

a. Computed only for a 2x2 table

b. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.09.

Table P.5 : Hypertension risk by angiogram result

Crosstab

		ANGIORES		Total
		NCA	CAD	
Riskhyper	yes	Count 8	31	39
		% within ANGIORES 47.1%	58.5%	55.7%
	No	Count 9	22	31
		% within ANGIORES 52.9%	41.5%	44.3%
Total	Count	17	53	70
	% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.682 ^b	1	.409		
Continuity Correction ^a	.297	1	.586		
Likelihood Ratio	.678	1	.410		
Fisher's Exact Test				.576	.292
Linear-by-Linear Association	.672	1	.412		
N of Valid Cases	70				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.53.

Table P.6 : Lipid risk by angiogram result

Crosstab

		ANGIORES		Total	
		NCA	CAD		
RISKLIP	yes	Count	11	47	58
		% within ANGIORES	78.6%	75.8%	76.3%
	no	Count	3	15	18
		% within ANGIORES	21.4%	24.2%	23.7%
Total		Count	14	62	76
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.048 ^b	1	.826		
Continuity ^a Correction	.000	1	1.000		
Likelihood Ratio	.049	1	.825		
Fisher's Exact Test				1.000	.566
Linear-by-Linear Association	.048	1	.827		
N of Valid Cases	76				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.32.

Table P.7 : Diabetes risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
Riskdiabetes	Yes	Count	2	14	16
		% within ANGIORES	50.0%	73.7%	69.6%
	No	Count	2	5	7
		% within ANGIORES	50.0%	26.3%	30.4%
Total		Count	4	19	23
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.875 ^b	1	.349		
Continuity ^a Correction	.114	1	.735		
Likelihood Ratio	.821	1	.365		
Fisher's Exact Test				.557	.352
Linear-by-Linear Association	.837	1	.360		
N of Valid Cases	23				

a. Computed only for a 2x2 table

b. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.22.

Table P.8 Family history risk by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
riskfh	yes	Count	9	28	37
		% within ANGIORES	64.3%	73.7%	71.2%
	no	Count	5	10	15
		% within ANGIORES	35.7%	26.3%	28.8%
Total		Count	14	38	52
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.440 ^b	1	.507		
Continuity ^a Correction	.101	1	.750		
Likelihood Ratio	.429	1	.512		
Fisher's Exact Test				.511	.368
Linear-by-Linear Association	.432	1	.511		
N of Valid Cases	52				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 4.04.

Table P.0 : Exercise tolerance test by angiogram result

Crosstab

		ANGIORES		Total
		NCA	CAD	
POSETT	yes	Count 7	61	68
		% within ANGIORES 33.3%	91.0%	77.3%
no	Count 11	4	15	
	% within ANGIORES 52.4%	6.0%	17.0%	
Inconclusive	Count 3	2	5	
	% within ANGIORES 14.3%	3.0%	5.7%	
Total	Count 21	67	88	
	% within ANGIORES 100.0%	100.0%	100.0%	

Table P.10 : ECG by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
ECG	positive	Count	1	7	8
		% within ANGIORES	11.1%	36.8%	28.6%
	normal	Count	8	12	20
		% within ANGIORES	88.9%	63.2%	71.4%
Total		Count	9	19	28
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.981 ^b	1	.159		
Continuity Correction ^a	.921	1	.337		
Likelihood Ratio	2.216	1	.137		
Fisher's Exact Test				.214	.170
Linear-by-Linear Association	1.911	1	.167		
N of Valid Cases	28				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 2.57.

Table P.11 : GTN relief by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
GTNREL	yes	Count	11	43	54
		% within ANGIORES	84.6%	87.8%	87.1%
	no	Count	2	6	8
		% within ANGIORES	15.4%	12.2%	12.9%
Total		Count	13	49	62
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.090 ^b	1	.764		
Continuity Correction ^a	.000	1	1.000		
Likelihood Ratio	.087	1	.768		
Fisher's Exact Test				.670	.536
Linear-by-Linear Association	.089	1	.766		
N of Valid Cases	62				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 1.68.

Table P.12 : Other relief by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
OTHREL	rest	Count	12	44	56
		% within ANGIORES	85.7%	88.0%	87.5%
	other drugs	Count	2	2	4
% within ANGIORES		14.3%	4.0%	6.3%	
	positions	Count		4	4
		% within ANGIORES		8.0%	6.3%
Total		Count	14	50	64
		% within ANGIORES	100.0%	100.0%	100.0%

Table P.13 : Wakening pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
WAKEPAIN	yes	Count	7	9	16
		% within ANGIORES	50.0%	16.7%	23.5%
	no	Count	7	45	52
		% within ANGIORES	50.0%	83.3%	76.5%
Total		Count	14	54	68
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	6.865 ^b	1	.009		
Continuity ^a Correction	5.138	1	.023		
Likelihood Ratio	6.132	1	.013		
Fisher's Exact Test				.015	.015
Linear-by-Linear Association	6.764	1	.009		
N of Valid Cases	68				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 3.29.

Table P.14 : Pain site by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINSITE	central retrosternal	Count	7	32	39
		% within ANGIORES	43.8%	58.2%	54.9%
	left sternal edge	Count	4	1	5
		% within ANGIORES	25.0%	1.8%	7.0%
	inframammary	Count	2	2	4
		% within ANGIORES	12.5%	3.6%	5.6%
	right side of chest	Count		5	5
		% within ANGIORES		9.1%	7.0%
	epigastric	Count		3	3
		% within ANGIORES		5.5%	4.2%
left shoulder + arm	Count		2	2	
	% within ANGIORES		3.6%	2.8%	
neck	Count	1	6	7	
	% within ANGIORES	6.3%	10.9%	9.9%	
multiple L + R sites	Count	2	4	6	
	% within ANGIORES	12.5%	7.3%	8.5%	
Total	Count	16	55	71	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table P.15 : Pain spread by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINSPRE	to L arm/shoulder	Count % within ANGIORES		5 9.3%	5 7.2%
	to L sternal edge	Count % within ANGIORES	1 6.7%	2 3.7%	3 4.3%
	to R arm/shoulder	Count % within ANGIORES		3 5.6%	3 4.3%
	to both arms	Count % within ANGIORES	1 6.7%	5 9.3%	6 8.7%
	to neck	Count % within ANGIORES	1 6.7%	7 13.0%	8 11.6%
	radiates L + R	Count % within ANGIORES	1 6.7%	4 7.4%	5 7.2%
	to L arm + neck	Count % within ANGIORES		1 1.9%	1 1.4%
	no spread indicated	Count % within ANGIORES	8 53.3%	24 44.4%	32 46.4%
	across chest	Count % within ANGIORES	3 20.0%	3 5.6%	6 8.7%
	Total	Count % within ANGIORES	15 100.0%	54 100.0%	69 100.0%

Table P.16 : Other pain sites by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
OTHERSIT	jaw	Count		2	2
		% within ANGIORES		3.6%	2.9%
	arm	Count	3	9	12
		% within ANGIORES	21.4%	16.4%	17.4%
	back	Count	3	10	13
		% within ANGIORES	21.4%	18.2%	18.8%
	other	Count	3	3	6
		% within ANGIORES	21.4%	5.5%	8.7%
	none	Count	3	23	26
		% within ANGIORES	21.4%	41.8%	37.7%
	neck/throat	Count	2	8	10
		% within ANGIORES	14.3%	14.5%	14.5%
Total		Count	14	55	69
		% within ANGIORES	100.0%	100.0%	100.0%

Table P.17 : Pain characteristics by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINCHAR	pressing,gripping,tight	Count	8	31	39
		% within ANGIORES	50.0%	59.6%	57.4%
	sharp,stabbing	Count	2	6	8
		% within ANGIORES	12.5%	11.5%	11.8%
	mixed	Count	5	10	15
		% within ANGIORES	31.3%	19.2%	22.1%
	other	Count	1	5	6
		% within ANGIORES	6.3%	9.6%	8.8%
Total		Count	16	52	68
		% within ANGIORES	100.0%	100.0%	100.0%

Table P.18 : Pain aggravators by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINAGG	coughing	Count	3	2	5
		% within ANGIORES	20.0%	4.1%	7.8%
	breathing deeply	Count	3	1	4
		% within ANGIORES	20.0%	2.0%	6.3%
	swallowing	Count	1	2	3
		% within ANGIORES	6.7%	4.1%	4.7%
	bending or stooping	Count	2	15	17
% within ANGIORES		13.3%	30.6%	26.6%	
other	Count	1	4	5	
	% within ANGIORES	6.7%	8.2%	7.8%	
nothing	Count	5	25	30	
	% within ANGIORES	33.3%	51.0%	46.9%	
Total	Count	15	49	64	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table P.21 : Pain duration by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINLEN	1 - 6 months	Count	3	27	30
		% within ANGIORES	18.8%	49.1%	42.3%
	6 - 12 months	Count	4	6	10
		% within ANGIORES	25.0%	10.9%	14.1%
	1 - 2 years	Count	5	5	10
% within ANGIORES		31.3%	9.1%	14.1%	
2 - 5 years	Count	2	9	11	
	% within ANGIORES	12.5%	16.4%	15.5%	
> 5 years	Count	2	8	10	
	% within ANGIORES	12.5%	14.5%	14.1%	
Total		Count	16	55	71
		% within ANGIORES	100.0%	100.0%	100.0%

Table P.20: Pain onset by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINON	gradually	Count	3	28	31
		% within ANGIORES	20.0%	56.0%	47.7%
	suddenly	Count	12	22	34
		% within ANGIORES	80.0%	44.0%	52.3%
Total		Count	15	50	65
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5.994 ^b	1	.014		
Continuity ^a Correction	4.638	1	.031		
Likelihood Ratio	6.366	1	.012		
Fisher's Exact Test				.019	.014
Linear-by-Linear Association	5.902	1	.015		
N of Valid Cases	65				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.15.

Table P.21 Pain frequency by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
PAINFREQ	> once a day	Count	4	11	15
		% within ANGIORES	26.7%	21.6%	22.7%
	every day	Count	4	10	14
		% within ANGIORES	26.7%	19.6%	21.2%
	> once a week	Count	7	14	21
% within ANGIORES		46.7%	27.5%	31.8%	
< once a week	Count		13	13	
	% within ANGIORES		25.5%	19.7%	
< once a month	Count		3	3	
	% within ANGIORES		5.9%	4.5%	
Total	Count	15	51	66	
	% within ANGIORES	100.0%	100.0%	100.0%	

Table P.22 : Stress-provoked pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
STRESS	yes	Count	9	21	30
		% within ANGIORES	56.3%	39.6%	43.5%
	no	Count	7	32	39
		% within ANGIORES	43.8%	60.4%	56.5%
Total		Count	16	53	69
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.383 ^b	1	.240		
Continuity ^a Correction	.789	1	.374		
Likelihood Ratio	1.373	1	.241		
Fisher's Exact Test				.264	.187
Linear-by-Linear Association	1.363	1	.243		
N of Valid Cases	69				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.96.

Table P.23 : Cold-provoked pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
COLD	yes	Count	8	31	39
		% within ANGIORES	50.0%	57.4%	55.7%
	no	Count	8	23	31
		% within ANGIORES	50.0%	42.6%	44.3%
Total		Count	16	54	70
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.274 ^b	1	.600		
Continuity ^a Correction	.056	1	.812		
Likelihood Ratio	.273	1	.601		
Fisher's Exact Test				.775	.404
Linear-by-Linear Association	.271	1	.603		
N of Valid Cases	70				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.09.

Table P.24 : Food-provoked pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
FOOD	yes	Count	1	8	9
		% within ANGIORES	6.3%	15.1%	13.0%
	no	Count	15	45	60
		% within ANGIORES	93.8%	84.9%	87.0%
Total		Count	16	53	69
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.848 ^b	1	.357		
Continuity ^a Correction	.247	1	.619		
Likelihood Ratio	.974	1	.324		
Fisher's Exact Test				.674	.328
Linear-by-Linear Association	.835	1	.361		
N of Valid Cases	69				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 2.09.

Table P.25 : Sex-provoked pain by angiogram result

Crosstab

			ANGIORES		Total
			NCA	CAD	
SEXPROV	yes	Count	3	8	11
		% within ANGIORES	18.8%	15.1%	15.9%
	no	Count	13	45	58
		% within ANGIORES	81.3%	84.9%	84.1%
Total		Count	16	53	69
		% within ANGIORES	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.123 ^b	1	.726		
Continuity Correction ^a	.000	1	1.000		
Likelihood Ratio	.119	1	.730		
Fisher's Exact Test				.708	.496
Linear-by-Linear Association	.121	1	.728		
N of Valid Cases	69				

a. Computed only for a 2x2 table

b. 1 cells (25.0%) have expected count less than 5. The minimum expected count is 2.55.

Table P.26 : Stooping-provoked pain by angiogram result

Crosstab

		ANGIORES		Total	
		NCA	CAD		
STOOP	yes	Count	7	15	22
		% within ANGIORES	43.8%	28.3%	31.9%
no	Count	9	38	47	
	% within ANGIORES	56.3%	71.7%	68.1%	
Total	Count	16	53	69	
	% within ANGIORES	100.0%	100.0%	100.0%	

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.350 ^b	1	.245		
Continuity ^a Correction	.733	1	.392		
Likelihood Ratio	1.304	1	.253		
Fisher's Exact Test				.359	.195
Linear-by-Linear Association	1.331	1	.249		
N of Valid Cases	69				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.10.

Appendix Q – t-tests for phase two physical data

T-Test

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
NOSMOKE	NCA	15	3.0000	8.4092	2.1712
	CAD	55	2.2909	5.7055	.7693
episodes pain on hill /10	NCA	16	9.3125	2.0156	.5039
	CAD	52	7.9231	3.1722	.4399
num episodes pain on rest / 10	NCA	14	2.6429	3.0786	.8228
	CAD	46	1.8043	2.5809	.3805
GTNREL	NCA	13	1.1538	.3755	.1042
	CAD	49	1.1224	.3312	4.731E-02

Independent Samples Test

	Levene's Test for Equality of Variances		t-test for Equality of Means							
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean		
								Lower	Upper	
NOSMOKE	Equal variances assumed Equal variances not assumed	1.255 .267	.383 .308	68 17.664	.703 .762	.7091 .7091	1.8517 2.3035	-2.9859 -4.1370	4.4040 5.5552	
episodes pain on hill /10	Equal variances assumed Equal variances not assumed	14.177 .000	1.648 2.077	66 39.781	.104 .044	1.3894 1.3894	.8432 .6689	-.2941 3.731E-02	3.0729 2.7415	
num episodes pain on rest / 10	Equal variances assumed Equal variances not assumed	.661 .420	1.017 .925	58 18.906	.313 .367	.8385 .8385	.8243 .9065	-.8114 -1.0595	2.4884 2.7365	
GTNREL	Equal variances assumed Equal variances not assumed	.334 .566	.296 .274	60 17.280	.769 .787	3.140E-02 3.140E-02	.1062 .1144	-.1811 -2.097	.2439 .2725	

Appendix R – t-tests for phase two psychological data

T-Test

Group Statistics

	ANGIORES	N	Mean	Std. Deviation	Std. Error Mean
HADanx	NCA	17	10.1176	3.6892	.8948
	CAD	57	6.2982	3.6692	.4860
had depression	NCA	17	8.1176	4.4424	1.0775
	CAD	57	5.1228	3.2791	.4343
MSPQ	NCA	16	11.8125	7.6352	1.9088
	CAD	58	6.8448	6.1009	.8011
WIBP	NCA	14	1.5714	1.2225	.3267
	CAD	58	.9828	1.0343	.1358
WIDC	NCA	14	1.0000	.9608	.2568
	CAD	57	.3684	.6717	8.896E-02
WIIP	NCA	14	.9286	1.3281	.3549
	CAD	57	.5614	.7324	9.700E-02
WITOTAL	NCA	14	4.7143	4.3044	1.1504
	CAD	57	3.0526	3.1871	.4221

Independent Samples Test

	Levene's Test for Equality of Variances	t-test for Equality of Means									
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Mean		
									Lower	Upper	
HADanx	Equal variances assumed Equal variances not assumed	.133	.716	3.762	72	.000	3.8194	1.0152	1.7956	5.8432	
had depression	Equal variances assumed Equal variances not assumed	.269	.605	3.035	72	.003	2.9948	.9867	1.0279	4.9618	
MSPQ	Equal variances assumed Equal variances not assumed	2.035	.158	2.727	72	.008	4.9677	1.8216	1.3364	8.5989	
WIBP	Equal variances assumed Equal variances not assumed	1.846	.179	1.844	70	.069	.5887	.3192	-4.79E-02	1.2252	
WIDC	Equal variances assumed Equal variances not assumed	1.867	.176	2.881	69	.005	.6316	.2192	.1943	1.0689	
				2.324	16.254	.033	.6316	.2718	5.622E-02	1.2069	