

**An Investigation of  
Cognitive Processes in Chronic Pain**

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## ABSTRACT

This thesis examines information-processing in chronic pain. "Schematic" processing is investigated; selective memory for pain-related information is explored in depressed and non-depressed chronic pain patients, depressed non pain-patients and controls. A memory bias for sensory adjectives is found in the non depressed chronic pain group, while a tendency to over-recall both sensory and affective compared to neutral information is found in the depressed chronic pain group. No memory bias is observed in an acute pain group, and the implications of this are discussed. A possible cognitive avoidance mechanism is identified in depression.

A questionnaire assessing beliefs about pain ("conceptual" processing) is developed and validated, and shown to differentiate between chronic pain patients and controls. The impact of two interventions for chronic pain (surgery and cognitive-behavioural management) on schematic and conceptual processing is investigated prospectively. In general the endorsement of organic beliefs decreases while the emphasis on psychological beliefs increases post-intervention. Evidence is found to suggest that surgery, but not cognitive-behavioural treatment, reverses pain-related memory biases. This is discussed in relation to changes in pain intensity. Evidence is provided to suggest that beliefs are causally related to several pain-related measures including anxiety, depression, health locus of control, cognitive coping strategies and activity levels.

A word completion paradigm is employed to explore further the role of schematic processing in chronic pain, and finally, a lexical decision task is used to assess the role of word frequency effects in information-processing in chronic pain. These results suggest that memory biases in chronic pain cannot be explained by frequency effects, hence addressing the validity of the memory biases described earlier in the thesis.

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# *Chapter 1 Introduction*

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This introductory chapter aims first to provide an overview of the psychological factors identified as being involved in the experience of pain. Differences between acute and chronic pain, and the issue of organic versus psychogenic pain are outlined. This is followed by a brief description of the neurological foundations of pain, the Specificity and Gate Control theories of pain, and the extent to which each can account for clinical observations. A parallel processing model of pain-distress is then presented. The distinction between sensory and affective components of pain is discussed, with particular reference to their assessment. Next, the problem of depression in chronic pain patients is addressed, in relation to demographic, cognitive, coping and other psychological variables. Following this general introduction is a review of the literature concerning the role of affect in cognitive processes, including mood-related attentional biases, mood congruency and state dependent memory effects. The specific issue of the association between pain, affect and memory, with which much of this thesis is concerned is then presented, along with the empirical evidence. Associative network models, schemata theory and an integrated model for these findings are compared within an information-processing framework. Finally, the goals of the thesis are outlined.

## **The Puzzle of Pain**

In his seminal work "The Puzzle of Pain" (1973), Ronald Melzack eloquently describes one of the major problems facing all health care professionals working with

pain sufferers; namely, that there is no single consistent link between injury and pain. The commonly held belief that the greater the damage to the body, the more intense the pain, is not invariably the case. Frequently observed are instances where serious <sup>injury</sup> is sustained but no pain is felt, as in congenital analgesia, soldiers wounded in battle (Beecher, 1959), and more recently in a population of hospital accident and emergency department attenders (Melzack et al, 1982). Conversely, pain is sometimes present when no injury has occurred, no tissue-damaging disease process is present, or long after healing is completed, as in the case of tension headaches (Olesen, 1986), and low back pain where in 70% of cases no damage can be found responsible (Loeser, 1980). Clearly these examples provide the extremes of a continuum depicting the relationship between pain intensity and injury severity. Thus although pain has obvious survival value in certain circumstances, preventing further damage and promoting healing through enforced inactivity, it also serves no obvious useful purpose at many other times.

## **PSYCHOLOGICAL FACTORS IN PAIN**

The observation that there is no simple relationship between injury and pain implies that other factors are significantly influencing the experience of pain. Many factors, labelled "psychological" (as distinct from biological, physical or genetic etc.) have been identified as making an important contribution. Studies can broadly be grouped under the following headings, and will briefly be discussed in turn: culture, personality, learning, social modelling, control, cognition and attribution, placebos and hypnosis. Clearly, they are highly inter-related, with considerable overlap. The aim is not to be exhaustive, but to give a flavour of these areas of work.

### *Cultural determinants of pain*

The way in which people respond to pain is known to vary enormously between cultures. Many instances have been documented where an individual is subjected to apparently appalling procedures, for example ritual insertion of hooks into the muscles of the back, then swinging from a cart for extended periods of time in parts of India (Kosambi, 1967), and trepanation (scraping of the skull) in East Africa. These individuals appear to experience no pain, and exhibit no signs of distress, indeed in the first example the procedure is associated with great honour and "exaltation".

Several studies have examined the influence of ethnicity on thresholds. Although evidence suggests that the majority of people share a common sensation threshold, pain perception and pain tolerance thresholds are culture-related. Sternbach and Tursky (1965) found that the level of electric shock needed for the stimulus to be detected did not differ between Italian, Jewish, Irish or Old American subjects. In contrast, Clark and Clark (1980) found that Nepalese climbing expedition porters require significantly higher levels of electric shock than their Occidental climbing visitors before they label them as painful. This is despite the fact that both groups were equally sensitive to changes in shock intensity.

Zborowski (1952) suggests that such findings reflect differences in attitudes towards pain and pain expression (ie. groaning, crying out), rather than intrinsic differences in the sensory experience between groups. These results highlight the importance of distinguishing between peoples' overt reaction to pain - their behaviour, which is readily observed - and their actual perception of pain, which can never truly be measured. Essentially, pain is a subjective experience which is

communicated to others solely through language and behaviour (issues related to the measurement of pain are discussed later).

### *Personality Variables*

A strong link exists between an individual's personality and their past experience and culture, to the extent that they are probably inextricable. However, other personality variables have been shown to be associated with certain pain conditions. Hanvik (1951) explored the relationship between low back pain and scales on the Minnesota Multiphasic Personality Inventory (MMPI). Thirty sufferers with clear pathology were compared with 30 with no clear pathology. The first group were observed to show increased scores on the depression scale, but lower hypochondriasis and hysteria scores. A second specific profile on the MMPI was demonstrated by Gentry *et al* (1974) in chronic low back pain patients. They found that this group had a strong need to appear socially acceptable, and they exhibited emotional conflicts somatically. They were characteristically extrovert and sociable, yet also self-centred, dependent and demanding of others. In a comparison of acute and chronic pain patients, Philips (1964) found that female low back pain patients had elevated hysteria, depression and hypochondriasis scores in relation to female fracture patients, whose scores in turn were only slightly raised compared to normal controls. Such personality variables have also been correlated with treatment outcome. Spinal fusions appear to be more successful in individuals scoring lower on the hysteria, hypochondriasis and depression scales of the MMPI compared to high scorers who fare less well (Wilfing *et al*, 1973). Although these studies provide a clue to the relationship between pain and personality, it is not clear whether such findings reflect

premorbid personality differences, or changes in personality attributes as a consequence of the pain, or, indeed a mixture of the two.

### *Learning*

The role of learning in pain is at one level plainly evident. From birth onwards we learn through experience to avoid hot or sharp objects, and that certain behaviours result in pain reduction. When dogs are raised in complete isolation, deprived of all environmental stimuli, they fail to develop normal behavioural and emotional arousal responses to noxious stimuli (Melzack and Scott, 1957). However, learning through experience is most effective in dealing with acute pain episodes. Fordyce provides a behavioural analysis of chronic pain based on Skinner's principles of "operants" - actions of the organism which can be either increased (reinforced) or decreased in frequency of occurrence by their consequences. Under this analysis pain behaviours (such as wincing, asking for help from others, taking medication, limping etc.), may, to a large extent, be under the control of contingent reinforcements. Such reinforcement may include attention and sympathy, pain medication, avoidance of household chores and other unpleasant responsibilities. Fordyce observes that if the relationship between pain behaviour and contingent reinforcers can be severed, marked improvements in the pain problem may occur, to the extent that the problem may be resolved (Fordyce, 1978).

### *Social Modelling*

The impact of witnessing the pain and distress of others is frequently great and long-lasting. Typically, watching others exposed to pain or injury provokes patterns



of physiological arousal suggesting empathic emotional arousal (Berger, 1962; Craig, 1968). A wealth of diverse evidence supports the claim that modelling processes influence cognitive and behavioural responses to noxious stimuli, the interpretation of painful events, and the degree of emotional arousal. Observational learning plays an important role in the acquisition of new and appropriate pain-related behaviours with the advantage that injury and pain can be avoided by the individual, but it also incurs the possibility that maladaptive responses, including sick-role behaviour, may be learned (Craig, 1978).

Social modelling also provides an effective means of controlling pain and distress in a clinical setting, especially in children (Melamed *et al*, 1975; Melamed and Siegel, 1975). However, the model needs to provide a realistic, credible message, which must not differ too widely from the individual's expectations, for the process to be effective. Several laboratory experiments have shown that tolerant and intolerant models (people who are not subjected to pain, but behave either stoically or demonstratively in front of the subject) have a significant impact on the intensity of electric shock accepted by subjects (eg. Craig and Weiss, 1971; Craig and Best, 1977). Indeed, where models describe as painful a level of electrical stimulation usually described as tingling, subjects also described the stimulation as painful on 77% of trials. This is compared to 3% of trials when no model was present (Craig, Best and Ward 1975; Craig and Weiss, 1972). Psychophysical measures suggest that exposure to a tolerant model is associated with reductions in autonomic reactivity below that observed in a control (no model) group (Craig and Prkachin, 1978). All these results point to the importance of social context in the experience of pain.

### *Control, and the meaning of the situation*

Thompson (1981) provides a definition of control which recognises that control does not need to <sup>be</sup> exercised, or even real, for it to be effective - simply the perception of control is sufficient: control is "the belief that one has at one's disposal a response that can influence the aversiveness of an event". Thompson categorizes control into behavioural, cognitive, information and retrospective, and emphasizes the importance of the meaning of the situation for the individual, in terms of the endurability, desirability and predictability of the aversive event. In a clinical setting it has been demonstrated that patients who are pre-operatively given accurate information about the type, intensity and duration of pain to expect, and provided with relaxation coping strategies, report less pain and require less medication than patients who receive no instructions (Egbert *et al*, 1964). However, information alone may magnify pain and anxiety (Langer *et al*, 1975), and inadequate control may be worse than no control at all (Weisenberg *et al*, 1985).

In a study relating locus of control to pain coping strategies and psychological distress, Crisson and Keefe (1988) found that chronic pain patients who viewed health outcomes as controlled by chance tended to rely on maladaptive pain coping strategies and showed greater psychological distress, including depression, anxiety and obsessive-compulsive symptoms, and feelings of helplessness, compared to patients with other locus of control orientations.

The belief that pain and its effects on life are under personal control has also recently been examined in chronic pain patients by Jensen and Karoly (1991). They demonstrated that the belief that it is within one's ability and resources to manage pain, is positively related to well-being and activity levels.

### *Cognitive factors in pain*

Ross, Gil and Keefe (1988) have pointed out that the cognitions commonly associated with the onset of acute pain, such as the idea that pain is a warning signal, and that some form of remedial action is indicated, can be seen to be adaptive. However, as the pain persists, and patients pass into the sub-chronic and chronic phases, cognitions frequently become irrational and maladaptive, including anxiety, depression, guilt, anger and fear, and are typically resistant to change.

Cognitive distortions in chronic low back pain (CLBP) patients have been shown to be correlated with general distress but not somatizations, and this correlation is not due to the association of these factors with pain severity (Smith *et al*, 1986). Cognitive distortions have additionally been shown to be closely related to levels of disability (Smith *et al* 1986). Also, Lefebvre (1981) demonstrated that depressed LBP patients make cognitive errors in interpreting experiences related to low back pain compared to depressed non-pain subjects. Negative pain-related cognitions, particularly those relating to feelings of helplessness and hopelessness have been significantly associated with measures of pain intensity (during the past week), pain distress, behavioural disruption and anxiety, and in addition cognitions concerning feelings of hopelessness have, not altogether surprisingly, been found to be related to depression (Boston *et al*, 1990). These results were, however, complicated by the confounding of measures of pain and anxiety; anxiety was found to correlate significantly with the cognitive coping strategies and measures of pain. This could be interpreted as suggesting that these coping strategies are more closely associated with affective distress than pain intensity.

Further evidence that cognitions can have significant impact on the experience of

pain and distress is through the use of cognitive coping strategies, in the management of pain. Cognitive methods aimed at altering the subjective component of pain include imaginative inattention, transformation of context, imaginative transformation, attention diversion and somatisation. Although not successful in increasing pain tolerance in every individual, on every occasion, all these methods have therapeutic value for some people, on some occasions (Turk, Meichenbaum and Genest, 1983).

### *Suggestion, placebos and attributions*

The power of suggestion over the experience of pain is undisputed. Placebos, which can be defined as chemically inert substances or procedures administered with the suggestion that they will relieve pain, can exert considerable influence on pain, to the extent that around 35% of patients gain relief from placebo compared to only 70% of patients treated with even high doses of morphine (Beecher, 1972). Many curious properties of the placebo effect have been documented, including the finding that their efficacy is always around 50% of the drug with which they are being compared (in double blind trials) (Evans, 1985); they are more effective for severe than mild pain (Evans, 1985); a dose-response relationship exists, injections are more effective than oral preparations, and even the colour of the tablet appears to be of importance (Blackwell *et al*).<sup>1972</sup> All these observations imply that both the implicit and explicit suggestions made by the person supplying the drug are influential. However, as Richardson (1989) points out, qualities such as the size and appearance of the medication can have no influence on pain independently of the recipient's perception of them, and as such, conclusions about the efficacy of placebos may be meaningless. Although researchers have attempted to identify the "placebo

responder", no personality, intelligence or suggestibility variables have consistently been associated with response to placebo (see Shapiro and Morris, 1978). Several theories have been advanced to account for the placebo effect, including reporting error, cognitive dissonance, conditioning processes and anxiety reduction; each is likely to play a role in the effect, and as yet the exact mechanisms are poorly understood.

Considerable evidence exists to suggest that under hypnosis, where attention is focused intensely on the hypnotist, and away from other stimuli, and with appropriate suggestion, subjects can be subjected to severe pain, even undergoing major surgery, and yet report that they felt no pain, only sensations (Hilgard and Hilgard, 1986). McGlashan *et al* (1969) provide evidence that such findings are more than merely special examples of the placebo effect, by demonstrating that pain perception and pain tolerance levels are markedly increased during hypnosis, but only the pain perception threshold is increased after administration of a placebo.

## **ACUTE vs CHRONIC PAIN**

The distinction between acute and chronic pain is made primarily on the basis of duration. Acute pain, which is of recent onset or short duration is typically associated with autonomic changes, anxiety and behavioural responses directed at seeking relief from the pain. On the other hand, chronic pain, which is traditionally defined as pain which has persisted for 6 months or longer (France, Krishnan and Houpt, 1988), is often associated with a pattern of vegetative signs, helplessness, hopelessness and depression. The pain is no longer biologically functional and frequently no adequate explanation can be provided by doctors for its existence. Psychological factors play

an increasingly important role as the duration of pain increases, often with severe impact on family and lifestyle, and the emergence of "abnormal illness behaviour" (Pilowsky, 1969). Zarkowska and Philips (1986) found that although measures of pain behaviour (complaint, avoidance and help-seeking), and subjective indices of pain (sensory and affective) did not distinguish between acute and chronic pain sufferers, the relationship between the subjective and behavioural components of pain was different in these two groups, and became stronger with increased duration. An understanding of the process of change between acute and chronic pain states is of obvious relevance to health professionals, in both aiding prevention of chronicity and in the prediction of which patients with acute pain conditions are likely to become chronic.

#### **ORGANIC vs PSYCHOGENIC PAIN**

Where no clear physical cause for pain can be identified, the pain is often labelled "psychogenic" in origin. The patient is assumed to have some emotional, motivational or personality problem as the primary cause of their pain behaviours (Fordyce, 1978). For example, Engel (1959) identified a subgroup of patients without detectable lesions who were characterized by excessive guilt feelings, intolerance of personal success, and family histories where pain and aggression were prominent. However, there is compelling evidence to suggest that psychological disturbance is the result, rather than the precipitant of chronic pain. For example, although chronic pain patients show elevated scores on the hysteria, depression and hypochondriasis scales of the MMPI, these scores decrease significantly after successful treatment of the pain (Sternbach, 1974; Sternbach and Timmermans, 1975).

The distinction between organic and psychogenic pain is therefore of little value, since psychological factors play a major role in all cases of chronic pain, and as yet medical science is unable to confidently claim that where organic causes exist they are able to invariably detect or identify them.

## **NEUROPHYSIOLOGICAL FOUNDATIONS OF PAIN**

The preceding sections clearly highlight the need for any neurophysiological model of pain to be able to account for the many and diverse psychological influences on pain. In the following two sections, 2 major models of pain will be presented. The first, the traditional Specificity Theory will only be briefly outlined, since it has been superseded as a result of a scientific revolution, by the second, the Gate Control Theory.

### *The Specificity Theory*

The traditional theory of pain, in its simplest form, was first described by Descartes in 1664. It proposes that messages from pain receptors in the skin are carried to a pain centre in the brain. Numerous experiments (reviewed by Rose and Mountcastle 1959; Sinclair, 1982) have shown that there exists a one-to-one relationship between receptor type, fibre size and quality of sensory experience - cold, warmth, touch and pain. Thus proponents of the specificity theory talk of each type of fibre as having a distinct pathway to a specific centre in the brain. In 1957 Keele identified a "pain pathway" in the spinal cord - the spinothalamic tract - which is essential for pain sensation.

The physiological assumption inherent in this theory, that receptors are

specialized, remains undisputed. However, the assumption that there exists a direct, invariant relationship between the physical stimulus and the psychological sensory dimension has been vociferously challenged. The implication that there is a direct connection from a receptor to a brain centre where pain is "felt", which when stimulated *always* produces pain, and only pain, is clearly untrue.

### *The Gate Control Theory*

Detailed descriptions of this theory, first published in 1965, are readily found in for example Melzack and Wall (1988) and therefore many of the complexities and neurological details will be omitted here. The basic tenet of the theory is that the flow of nerve impulses from peripheral fibres to the spinal cells which project to the brain can be increased or decreased by the action of a neural mechanism - the "gate" - located in the dorsal horns of the spinal cord. Thus the modulating influence of the gate is able to act *before* pain is perceived or responded to. Modulation is proposed to occur at any of the synapses between the spinal cord and brain. Large fibre inputs tend to close the gate, inhibiting transmission, while small A-delta and C fibre inputs typically open it, thereby facilitating transmission. Extensive work by Wall and his colleagues has revealed that the cells comprising the substantia gelatinosa in particular seem to be responsible for modulating the input (Wall 1964) from peripheral fibres to spinal cord transmission (T) cells. It is proposed that the gating mechanism is influenced by the Central Control Trigger, a system of large diameter, fast-conducting fibres which activate selective cognitive processes. The theory also proposes a central Action System, comprising the neural areas underlying the behaviours and experience that makes pain what it is. This system is activated when



the output of the T cells exceeds a critical level. Powerful control over the sensory input can also be exerted via efferent fibre conduction relating to memories of past pain experiences, attention, emotions and other cognitive influences.

### *A Parallel Processing Model of Pain Distress*

In 1979 Howard Leventhal and Deborah Everhart published a model of the relationship between pain and emotion. They proposed two separated but interacting parallel pathways for the processing of pain, from the point of sensory input onwards. The first is a sensory-perceptual or informational pathway which deals with the location, intensity, duration and other physical attributes of the stimulus. The other, the emotion pathway generates the distress component of the pain experience. Elaboration of the input from these pathways is the result of a hierarchical system of three processing mechanisms, termed perceptual-motor, schematic and conceptual processing. Perceptual-motor processing is the earliest stage of processing which is considered automatic and to a large extent innate. It generates outputs that are the perception of the sensory attributes of the noxious stimulus (Johnson, 1973; Johnson and Leventhal, 1974), along with a perceptual signal identifiable as an emotional response. Schematic processing concerns the integration of pain stimuli and responses in memory systems, providing representations of pain which influence the perception of, and response to, future episodes of pain. Schemata are also thought to play the role of "attention selectors", dictating which aspects of the pain-distress experience enter focal awareness - the material attended to at any given time. Thus, under this model, the vast bulk of processing occurs preconsciously, with only a small proportion of what is perceived and processed entering focal awareness. In support

of schematic processing the authors provide evidence from phantom limb pain research, hypnosis, and research on the effects of sensation information and attention on pain-distress experience. Finally, the model proposes a conceptual level of processing which involves the individual's conceptualization of pain-distress, including beliefs about the experience of pain-distress. These are of enormous importance, since they will have impact on factors including adjustment to, and coping with chronic pain, and compliance with treatment regimens. This level of processing is assumed to have a modifying effect on both perceptual-motor and schematic processing.

This model is not incompatible with Melzack and Wall's (1965) gate theory of pain. Indeed, Leventhal and Everhart (1979) suggest that the interaction between informational and emotional pathways is, in effect, the gate mechanism.

## **THE ASSESSMENT OF SENSORY AND AFFECTIVE COMPONENTS OF PAIN**

There is considerable evidence to corroborate Leventhal and Everhart's (1979) claim that the sensory and affective/distress components are distinct aspects of the pain experience. Hilgard, Morgan and MacDonald (1975) demonstrated that anaesthesia instructions given while under hypnosis result in a dissociation between the 'informational' (sensory) and 'emotional' (distress) components of pain. Similarly, Leventhal *et al* (1979) showed that pain intensity and pleasantness are differentially affected by instructions on how to monitor pain.

These findings indicate the value of not limiting the assessment of pain to the

sensory component. Indeed, multidimensional pain assessment involves measurement of the following areas: physiological, sensory, affective, behavioural, cognitive and lifestyle impact. Physiological changes considered important in the experience of pain include muscle tension, measured by electromyographic (EMG) changes, and vascular changes, thought to cause tension and migraine headaches respectively. Studies investigating the prediction that tension headache sufferers show elevated EMG activity have provided mixed results: Haynes *et al* (1975) found evidence for a reduction in EMG levels, while Martin and Mathews (1978) and Pearce and Morley (1981) found no clear relationship between pain intensity and EMG activity. Pearce and Richardson (1987) therefore suggest that physiological variables such as EMG may not be diagnostically helpful, even though traditionally they may be thought to play a causal role.

The subjective component of pain is commonly assessed using rating scales, in a variety of forms including numerical or verbal categories (1,2,3,4,5; none, mild, moderate, severe, unbearable), Visual Analogue Scales (VAS; 10 cm line anchored at two extremes of pain intensity) and the 101-point numerical rating scale. The relative reliability and validity of these scales is a matter of some debate, with some authors claiming the VAS to be more sensitive to change than the Verbal Rating Scale (VRS) (eg. Joyce *et al*, 1975), but more vulnerable to response bias (Gracely, 1979). A combination of the VAS and VRS has been devised (Heft and Parker, 1984), with verbal anchors spaced at intervals along a 10cm line at distances which reflect magnitude of differences in intensity ie. 'faint', 'weak' and 'mild' are more closely spaced than 'moderate', 'strong' and 'intense'. However this measure is not often reported as being used in the literature. Jensen, Karoly and Braver (1986)

compared six different methods, concluding that on indices of utility and validity the methods are comparable, but the 101-point numerical scale may be the most practical.

Perhaps the most widely used measure of the qualitative aspects of pain is the McGill Pain Questionnaire (MPQ; Melzack, 1975) which comprises groups of pain descriptors increasing in 'intensity', reflecting the sensory (eg. stabbing, sharp), affective (eg. fearful, cruel) and evaluative (eg. miserable, troublesome) aspects of pain. Subjects indicate which words accurately describe their pain, providing either a score of the number of words chosen in each category, or an index of pain intensity. The latter measure has provoked some criticism, since the assumption that adjectives in each of the groups (particularly the affective and evaluative) form a single intensity dimension may be unjustified.

Following this line of argument Morley (1989) found that the affective adjectives of Tursky's Pain Perception Profile required a 3-dimensional model. Using a task where patients rated the similarity of all combinations of 12 pairs of affective descriptors, Morley and Pallin (1992) again found evidence for three dimensions, 'tolerability/emotional reaction', 'distraction/distress', and a third, more ambiguous and as yet, unnamed dimension.

In addition to measures of the affective qualities of pain, several instruments are typically used in the assessment of general emotional state in chronic pain patients. These include the Spielberger State-Trait Inventory (Spielberger *et al*, 1970) and Beck Depression Inventory (BDI; Beck *et al*, 1961). The BDI has been found to be a useful index of depression (Turner and Romano, 1984), despite the overlap of some of the symptoms of depression and pain itself (eg. sleep disturbance, inability to

work).

A wide variety of methods and instruments are available for the assessment of behavioural and cognitive aspects of pain, along with the impact of chronic pain on lifestyle and family (details of which can be found in Pearce and Wardle, 1989 and Pearce and Richardson, 1987).

## **CHRONIC PAIN AND DEPRESSION**

It is generally accepted, then, that pain is not merely a sensory experience, but incorporates a distinct emotional/distress component which can be separately evaluated. In addition to this affective component of the pain, which typically accompanies all pain experience, many patients with a long-term painful condition also suffer symptoms of depression. The proportion of chronic pain patients reported to be depressed ranges from around 10% to 100% (Romano and Turner, 1985). This large amount of variation can be attributed to the lack of consistency in diagnostic criteria for depression (RDC, DSM-III, major depression, minor depression etc.), and method of assessment (clinical interview, self-report measure) used in different studies. The problem also arises that some symptoms of depression such as fatigue, insomnia and changes in appetite, are also frequently associated with the physical aspects of the condition itself, in the absence of depression.

Further controversy exists as to the direction of causality between chronic pain and depression. Three general models have been proposed to account for the relationship. The first is a biological model which suggests that common substrates or neurochemical mechanisms may underlie both disorders (eg Ward *et al*, 1982). The second model, which applies to pain patients where there is no demonstrable

organic pathology (and hence is not generalizable to all chronic pain patients and weak in consequence), proposes that chronic pain is a variant of depressive disease, ie. a masked depression (Engel, 1959; Blumer and Heilbronn, 1982). The third model integrates cognitive and behavioural formulations based on ideas of reduced ability to engage in activities, perception of personal control and social rewards (Fordyce, 1976; Turk and Rudy, 1986). Strong support for the last model has been provided by Rudy, Kerns and Turk (1988), who provide evidence for a cognitive-behavioural mediation model in which measures of perceived life interference and self-control were found to be significant intervening variables between pain and depression. Interestingly, they found the direct link between pain and depression to be non-significant. It is therefore perhaps not surprising that Brown (1990) was unable to find a strong, consistent causal relationship between the two, using a two latent-variable, cross-lagged design, although he did find some evidence that pain predicted depression during the last 12 months of his 3 year study.

Considerable effort has been expended in differentiating depressed and non-depressed chronic pain patients on a variety of pain-related variables. In chronic pain patients depression has been significantly associated with greater pain intensity, interference due to pain and pain behaviours (Haythornthwaite, Sieber and Kerns, 1991), catastrophizing (Sullivan and D'Eon, 1990), passive coping style (Brown, Nicassio and Wallston, 1989) and reported loss of ability for social and recreational activities (Doan and Wadden, 1989). Haley, Romano and Turner (1985) found sex differences in patterns of the relationship between depression activity and pain, although no demographic or medical history differences were observed. In women pain intensity was associated with depression, whereas in men impairment in activity

was a better predictor of depression.

Given the strong association between depression and chronic pain, and the considerable impact of depression on both cognitive and behavioural aspects of long-term pain, it is clearly of importance that negative affect is not neglected in the study of chronic pain.

## **AFFECT AND COGNITION**

The relationship between affect cognition has received an enormous amount of attention in recent years. Interest has focused almost exclusively on anxiety and depression, and their effect on the processes involved in attentive mechanisms and memory. The relevant experimental evidence will now be presented, leaving theoretical interpretations of the results to subsequent sections, however general cognitive deficits associated with affective states will not be considered.

### **Anxiety**

#### ***Attention processes***

In exploring the influence of affect on attention - the capture of processing resources for a particular task or stimulus - a variety of experimental paradigms have been used. Attentional biases towards anxiety arousing stimuli (as distinct from attention to an anxiety response) have been shown to occur using a modified Stroop (Stroop, 1935) colour naming task. In the classic experiment, subjects are required to name the colour ink in which words in a list are written, ignoring the word content. If the word is a colour name, written in a different colour ink to the one named, for example the word "blue" written in green ink, this causes interference,

significantly decreasing the speed with which words are read.

In an initial study, Mathews and MacLeod (1985) presented generally anxious patients and controls separate lists of physical threat, social threat and positive (control) words. They found that, compared to the normal controls, anxious subjects showed greater interference on threat-related words. They also demonstrated a specific bias for physical threat words in subjects reporting particular anxiety about physical matters. In addition, through partial correlation analyses, they showed that the main predictor of degree of disruption was state anxiety rather than depression levels, which were also elevated in the anxious patients. In a replication of this study Mogg, Mathews and Weinman (1989) confirmed the selective interference of threat words on colour naming in generally anxious patients compared to controls. They also found clearer evidence for the specificity of the interference effect such that threat words which were congruent with the individual's predominant worries were particularly slowed.

Using a sample of spider phobics Watts, McKenna, Sharrock and Trezise (1986) provided further support for the specificity of disruption, demonstrating that while the phobics' performance was little affected by general emotional words such as "death" and "grief", it was greatly disrupted by spider words, such as "hairy" and "crawl", compared to a control group.

Mogg and Marden (1990) extended this research with the aim of answering 4 questions. The first was to determine whether such processing biases are the sole province of *clinically* anxious subjects (as opposed to anxious but non-clinical subjects). Secondly, they explored the issue of whether the bias operates on all emotional information, or just threat information. Thirdly, is there a bias for



information of personal concern, unrelated to emotionality? Finally, is the bias an artefact of the familiarity of the words for the individuals (termed the frequency effect)? Mogg and Marden tested 4 groups of medical students: high trait anxious, low trait anxiety scorers, students who were active members of a rowing club, and non rowers matched for trait anxiety with the rowers. Six sets of word stimuli were used for the Stroop task: social threat, physical threat, positive emotion, neutral, high frequency neutral (matched for frequency with the social and physical threat words and positive emotion words), and a set of rowing terms. Results indicated that high trait anxiety subjects were relatively slower in naming emotional words than non-emotional words compared to low trait anxious subjects. However, the high trait anxiety subjects did not distinguish between positive emotional and threat words. The results also demonstrated that the processing bias is associated with emotional material and not merely information that is relevant to personal interests, and interference in colour naming is not associated with word frequency. The authors' primary conclusion was that all emotional stimuli are involved in selective processing effects, and not threat stimuli alone.

Martin, Williams and Clark (1991) arrived at a similar conclusion, finding that generalized anxiety disorder patients and controls did not differ in their colour-naming times of positive and negative (threat) emotional words. They also found evidence that the bias is stronger in patients compared to high-anxious controls.

Thus although the results of such Stroop tasks are generally consistent in providing evidence that anxious patients have a processing bias for certain information, they do not agree entirely on the nature or extent of the bias. Unfortunately, the Stroop task is not able to distinguish between a bias in

perceptual/attention processes and a response bias explanation. In other words it may be possible that emotionally disturbed people are "preset" to respond to stimuli of particular relevance to their psychopathology, rather than there being any inherent differences in the way in which they process information compared to control groups.

The other problem in interpreting results from the Stroop experiments lies in the potential confounding of variables such as word frequency, concreteness and imageability. Although Mogg and Marden (1990) assessed the influence of high versus low frequency neutral words on the colour naming task, and found that frequency appeared not to play a mediating role, they did not assess the frequency of the threat/emotional words for the different groups. It is conceivable that the high trait anxiety group were more familiar with the threat/emotional words (and by implication these words were therefore of high frequency for this group) than the low trait anxiety group. However, this explanation of the Stroop results seems implausible in view of the findings of Watts *et al* (1986), who found that emotional Stroop interference was reduced in a group of spider phobics after systematic desensitization treatment involving repeated exposure to the threatening stimuli.

Given the processing bias versus response bias problem inherent in the emotional Stroop paradigm, MacLeod, Mathews and Tata (1986) devised an experiment where subjects make a *neutral* response (pressing a button) to a *neutral* stimulus (dot probe). In this experiment 16 generally anxious subjects and 16 matched controls were presented 2 words simultaneously, one 3cm above the other, on a computer screen. The task was to read out loud the top word of each pair. On some of the trials one of the words was replaced by a small dot (the "dot probe"). The dot probe replaced the upper and lower words on an equal number of trials. When the probe

appeared subjects had to press a button as quickly as possible. On some trials one of the words was a threat stimuli. Results indicated that anxious patients were relatively quicker in detecting the probe when it replaced a threat word in the top position, than when it replaced a neutral word at the top with a threat word at the bottom. On the other hand, if the probe occurred in the bottom position with a threat word at the top, these subjects were relatively slower in detecting/responding to it. Control subjects exhibited the opposite pattern. Thus anxious patients tend to orient towards threat while normal controls tend to orient away from the threat stimuli. In accordance with Mogg and Marden (1990), and Martin, Williams and Clark (1991), but in contrast with Mathews and Macleod (1985) and Mogg, Mathews and Weinman (1989), the results of this experiment also suggested that there is no relationship between the type of threat word and the predominant concerns of the individual. The results of this study are extremely important, in so far as they provide strong evidence that anxious patients *do* differ from controls in the way in which they allocate attention to their environment, and that this cannot be accounted for in terms of a simple response bias.

MacLeod and Mathews (1988) used this visual dot probe task to explore the relationship between state anxiety, trait anxiety and attention bias. They reasoned that if an attention bias were to be found in relation to state anxiety alone, the bias could be seen as a secondary consequence of the mood state. Alternatively, if the bias is found with trait mood alone, it might be viewed as a cognitive mechanism underlying vulnerability to that particular mood state, ie. a tendency to preferentially attend to aversive stimuli in the environment. Fifty-eight medical students, divided into two groups on the basis of STAI trait anxiety scores, underwent the dot probe task on 2

occasions - the first 12 weeks prior to their exams (low state anxiety), then again one week before their exams (high state anxiety). Two sets of threat words were compiled; exam-related and general threat. The results indicated that only high trait anxious subjects shifted their attention to general threat words on both test occasions. However, as the exam drew nearer, while the attention bias towards exam-related words increased in high trait anxiety students, in the low trait anxiety group attention tended to be drawn away from the exam-related words (ie "cognitive avoidance"). This finding can be seen to parallel the pattern of results of the control group in the first visual dot probe experiment. The authors concluded that state and trait anxiety interact in attentional bias to stimuli relating to events currently causing stress. They also suggest that the differences observed reflect rehearsal or avoidance of exam-related issues between the first and second times of testing. Alternatively, the threat value of the words themselves may have increased between the two times and been responsible for the effect, rather than the emotional state *per se*.

In a recent study employing this paradigm, Mogg, Mathews and Eysenck (1992) replicated the findings of MacLeod, Mathews and Tata (1986) in currently anxious patients, and provided evidence that the extent to which these patients selectively attend to social threat is associated with the severity of their social worries.

In a similar paradigm, the "colour perception" task, two words, differing in emotional valence are presented simultaneously, then displaced by 2 bars of colour, also presented simultaneously. The subject has to decide which bar appeared first, the rationale being that if attention is switched to a particular word from the pair, the colour bar replacing that word will be perceived as appearing earlier. The failure of Mogg *et al* (1991) to find evidence for either an attentional bias for threat stimuli

associated with both state (stress) or trait anxiety in medical students, or a bias towards threat in clinically anxious individuals compared with normal controls, runs contrary to predictions and previous findings. Mogg *et al* attempt to account for these null findings primarily in terms of problems inherent in the nature of the task, highlighting the differences between this and the visual dot probe task.

Finally, a lexical decision task has been used in an attempt to identify the exact conditions under which an attentional processing bias exists, in terms of the accessibility of information or assignment of processing priorities. Using this approach, MacLeod and Mathews (1991) provide evidence in support of the contention that anxiety is associated with the assignment of high processing priorities to threat-related information, rather than with facilitated availability of such information from memory. On each trial subjects were presented with either one or two letter strings on a computer screen. The comparison between single and double trials permitted the "competition" for processing resources hypothesis to be tested. Some of the strings were real words, others were non-words. The task involved deciding whether any of the letter strings were non words, and the time taken to do this was measured. Half of the real words were threat-related, the remainder neutral, matched for frequency and length. Equal proportions of each appeared on single and double letter string trials. Two groups of subjects were tested; 16 generally anxious out-patients and 16 non-patient controls. The three way interaction of particular interest (valence: threat, neutral x anxiety: generally anxious patients, controls x string number: one, two) was significant, and its interpretation entirely consistent with the prediction that an attention bias in anxiety is only apparent when there is competition for processing resources between concurrent threat and non-threat

stimuli.

Mogg, Mathews, Eysenck and May (1991) developed this idea, and established, using the same task, that when the neutral words form members of a semantic category (household terms), an anxiety-related bias is only evident when there is competition for processing resources (as before), but also when information is presented outside the focus of the subject's attention. This categorization effect places doubt over the conclusions of the MacLeod and Mathews experiment. Thus the evidence for the "competition" hypothesis is equivocal.

Overall, the available evidence from all paradigms suggests that under certain circumstances anxiety states are associated with pre-attentive biases (eg. Mathews and MacLeod, 1986), post-awareness processing biases (eg. Mathews and MacLeod, 1985; Mogg *et al*, 1989; Mogg and Marden, 1990), visual attention biases (operative outside awareness) (eg. MacLeod, Mathews and Tata, 1986; MacLeod and Mathews, 1988), and the assignment of processing priorities to threat-related information (MacLeod and Mathews, 1991; Mogg *et al*, 1991). Findings conflict regarding the extent to which these processing biases are specific to threat or emotional words in general (Martin, Williams and Clark, 1991; Mogg, Mathews and Weinman, 1989).

### *Memory processes*

The effects of mood on memory can be divided into two sorts: state dependency and mood congruity. The former refers to the finding that information learnt in one state, for example depression, is remembered better when the person is back in that state. Mood congruity effects, on the other hand, occur when material which is congruent with an individual's prevailing mood is recalled better than incongruent

material. Although there is considerable evidence for mood-state dependent memory (Ucross, 1989, provides a meta-analysis of published research from 1975 to 1985), it is mood congruity research which forms the focus of much of this thesis, and will therefore receive attention here.

In contrast to the plethora of evidence supportive of attention processing biases in anxiety, empirical support for selective memory processes in anxiety states is sparse. In an early study, Nunn, Stevenson and Whalan (1984) explored selective memory effects in agoraphobic patients using a prose recall task. Nine agoraphobic patients and 9 controls were presented with 5 prose passages, three of which contained potentially phobic material, the other two containing neutral information. The number of phobia-related propositions recalled was shown to differ significantly between the groups, with patients recalling more of the phobic propositions, but an equal proportion of neutral propositions. However, given the small sample sizes and bizarre statistical manipulations, these results cannot be assumed to be equivocal. In a second experiment the authors presented the same subjects with a word list containing 10 phobia-related and 10 neutral words. Subjects heard the list four times in random order, and attempted to recall them. Again a significant interaction between group and wordtype was found, with the patients remembering comparatively more phobic words and fewer neutral words. However, these results are also open to question, since it was not possible to account for effect of the first part of the experiment on the second, and the experimenter reading out the material was not blind to group membership.

These methodological problems were overcome by Rusted and Dighton (1991), whose results from a prose recall task also showed a recall bias favouring phobia-

related material, this time in spider phobics.

In a carefully controlled study using a free recall paradigm, Mogg, Mathews and Weinman (1987) presented groups of anxious patients and controls with positive, threat-related and negative words, matched for frequency and length, in self versus other-reference conditions. In this type of experiment subjects are required to think about the words either in relation to themselves or another person, in this case a television personality. This experiment failed to provide evidence for a self-referenced recall bias in the anxious subjects, and, contrary to prediction, these patients exhibited relatively poorer recall of threatening material. Interpretation of these results is complicated, however, by the fact that the anxious patients were also significantly more depressed than the controls.

Foa, McNally and Murdock (1989) also failed to find evidence for an anxiety-related mood congruity effect, at either encoding or retrieval stages of processing.

In contrast to these recall studies, Burke and Mathews (1992) found a significant difference in the number of anxious autobiographical memories produced by generalized anxiety disorder patients compared to controls. However, there are major difficulties with the use of autobiographical memory as an indicator of biased processing: firstly, the anxious patients may have experienced a greater number of anxiety-provoking events than the controls, and these patients may label equivalent events as more threatening than controls, either at the time of their occurrence or when they are retrieved as memories.

Thus studies exploring the relationship between anxiety and memory are rare. Selective memory effects appear to occur in prose recall but not free recall tasks, however the number of published studies in this area are too few to draw firm



conclusions.

## **Depression**

### *Attention processes*

Like studies on anxiety and memory, published experimental investigations of depression<sup>and attention</sup> are relatively uncommon. They too have produced mixed results. Gotlib and McCann (1984) used an emotional Stroop task to compare the performance of 15 depressed and 15 non-depressed students (assigned to these groups on the basis of Beck Depression Inventory scores). The words used were depressive, neutral or manic self-descriptive adjectives. The mean colour naming latencies for non-depressed subjects did not differ between wordtypes, however, the depressed subjects showed significant interference on the depressive compared to both manic and neutral words. This effect was not found in subjects in whom depressed mood was induced, suggesting that the bias was more strongly associated with stable patterns of processing than transient mood. Williams and Nulty (1986) arrived at a similar conclusion after investigating Stroop disruption in subjects who were tested one year apart, forming stable depressed, stable non-depressed and 'unstable' depressed groups. These studies suggest that an attention bias is associated with negative mood, however there are several problems associated with this work. Firstly, where non-clinical populations are sampled, it cannot be assumed that the results are generalizable<sup>to</sup> to their equivalent patient populations; caution is particularly required where the role of such biases are implicated in the cause and maintenance of these disorders. More importantly, anxiety levels of the subjects were not assessed in these experiments, and depression is commonly associated with elevated levels of anxiety. Consequently

it is plausible that their findings may have been directly attributable to anxiety, rather than depression.

Williams and Broadbent (1986) employed the Stroop paradigm, with the aim of clarifying these issues. They examined a group of patients who had taken an overdose and compared them with other hospital patients and non-patient controls, using neutral, general negative words (eg. hopeless) and negative words specific to the concerns of the attempted suicide patients (eg. fatal). Results showed that the greatest disruption occurred in the overdose patients on the condition-specific negative words, and that the extent of interruption was most clearly predicted by self-rated depression than by any other mood, including tension-anxiety.

In contrast to these positive results, MacLeod, Mathews and Tata (1986) failed to find evidence for an attention bias related to depression on the visual dot-probe task. However, the stimuli used were anxiety-related, rendering the results inconclusive. Gotlib, McLachlan and Katz (1988) employed the colour perception task (described earlier) and found that depressed subjects attended equally to depressed, manic and neutral-content words, although their depressed group only exhibited a mild level of depression as assessed by the BDI. Mogg *et al* (1991), in their experiment using the colour perception task were also unable to find a relationship between depression and attention bias.

### *Memory processes*

Selective memory effects in negative mood using the free recall paradigm are well established and documented. They are presented in detail in the next chapter when introducing the first experiment of the thesis, and are therefore not covered here. In

addition to this research, the role of negative affect in autobiographical memory and prose recall has been investigated, and will be briefly reviewed.

In an early study Lloyd and Lishman (1975) employed recall of personal memories as their task, and found a significant positive association between severity of depression and time taken to retrieve unpleasant memories. This study is beset by two major flaws. Firstly, the more severely depressed patients may have experienced a greater number of negative events, providing them with a greater choice to retrieve from, and secondly, these subjects may have been more likely to interpret neutral events as negative, again inflating the number available to choose from. Teasdale and Fogarty (1979) used a mood induction paradigm and randomization of allocation of subjects to conditions, and Clark and Teasdale (1982) used clinically depressed patients who exhibited diurnal variations in mood to overcome these problems. The former study showed slowed recall of positive material in depressed mood, while the latter study demonstrated a clear pattern of positive memories being less probable in the depressed phase of the cycle, with the reverse when the same patients were in their less depressed state. Memories had been rated for pleasantness/happiness by independent judges.

Findings of mood-related recall of stories with mixed affective content are also relatively robust. Breslow, Kocsis and Belkin (1981) examined recall of positive negative and neutral aspects of a narrative by depressed patients and matched controls and demonstrated a decrement in the recall of the positive components of the story by the patient group. Bower, Gilligan and Monteiro (1981) also found a selective memory effect in subjects who had undergone a hypnotic mood induction procedure. They found that more sad facts were remembered from the story by the

subjects who had heard the story while in sad mood, compared to those who had heard it while in a happy mood. It appears that the mood at time of encoding the information is the important factor in producing biased recall, since Bower (1981) failed to demonstrate an effect of mood at time of recall, but replicated the encoding congruity result.

To conclude these sections on affect and cognition, empirical evidence suggests that biases in attentive processes are more strongly associated with anxiety states, while selective memory effects are more strongly related to depression, however there is considerable overlap between the two areas, and a complete distinction is not supported.

## **PAIN, AFFECT AND COGNITION**

Given the increasing recognition that pain cannot be conceived solely as a sensory experience, the relationship between chronic pain and depression, and the observation that psychological and cognitive factors play an important role, it is perhaps surprising that so few studies have explored the impact of pain on information-processing. As described earlier, Lefebvre (1981) identified cognitive distortions in the way depressed chronic low back pain patients interpret information, however this approach is able to tell us little about the attention and memory processes more usually examined. Three studies have explored information-processing in relation to chronic pain, using the Stroop, autobiographical memory and free recall paradigms.

Pearce and Morley (1989) provide evidence for a pain-related attention bias in a group of chronic pain patients. Using the Stroop task with negative, sensory and affective words, they demonstrated greater interference on both sensory and affective

stimuli in the chronic pain patients compared to controls. No systematic pattern of correlations was found between interference scores and ratings of fatigue, tension, vigour, despondency, confusion and anger. However, standard measures of anxiety and depression were not obtained, and it remains possible that the observed effect could be attributed to anxiety (or depression) rather than pain per se. A recent study by Pincus *et al* (submitted) supports this possibility: evidence for interference on affective adjectives was provided, but disappeared when differences between the chronic pain and control group's levels of depression (BDI score) were taken into account.

In an investigation of autobiographical memory in female students, Eich, Rachman and Lopatka (1990) compared retrieval of real-life events when the subjects were experiencing menstrual pain, and again when they were pain-free. The events were then rated for pleasantness by the subjects. Results revealed that pain promoted recall of unpleasant events only if the pain was accompanied by increased negative affect, suggesting that the impact of pain on autobiographical memory is mediated by its influence on mood. This investigation did not, however, specifically study recall of pain experiences.

Finally, only one experiment has explored memory for pain-related information in relation to pain status. Pearce *et al* (1990) explored both mood congruity effects and state-dependent learning in chronic pain patients and non-patient controls. These are described in the next chapter, and are therefore not presented here.

## **THEORETICAL PERSPECTIVES**

Research such as that described in the preceding sections can be best conceptualized within an information-processing framework. This framework is characterized by seven basic concepts (Williams, Watts, MacLeod and Mathews, 1988). Firstly, information-processing systems are conceived of as having a limited capacity, which places constraints on the amount of information which can be processed at any one time. This limited capacity can either be due to restriction in resources, or competition for structures or mechanisms within the system. Secondly, as an inevitable consequence of this competition, information is processed selectively. This 'selective attention' may be seen as either the cause, or the consequence of differential processing. Thirdly, information-processing models typically construe an operation as comprising a number of separate, serial, component processes. Each stage of processing requires the output from the previous stage in order to function. The fourth concept is that of parallel processing, where a number of operations can take place simultaneously. The fifth concept contrasts bottom-up with top-down processing. The former operates where basic low-level processes concerned primarily with the physical stimulus, influence higher order representations, including attitudes, expectations, beliefs and prototypical situations. The latter occurs when high-order representations influence low-level processes. Sixth, processing of information is thought to occur through hierarchical systems (cf Leventhal, 1979), the operation of lower level hierarchies being controlled by higher levels. Bottom-up and top-down processing is conceived of as occurring within each hierarchy, with hierarchies working independently. Finally, some processes are thought to be automatic, not requiring attentional resources or conscious effort, either innate or learned, and are

fixed. In contrast, strategic (controlled) processes are flexible, can be modified and are essential in dealing with novel situations. Automatic processes do not suffer from limited capacity constraints since they can occur in parallel, whereas controlled processes are largely serial. Zajonc (1980) has argued that the assessment of the affective quality of a stimulus is an automatic process, proceeding entirely outside awareness.

### *Associative Network Models*

Several variations of the basic associative network model proposed by Quillian (1968) have been devised, including that of Anderson and Bower (1973). Their Human Associative Memory (HAM) Network has been most commonly used to account for the effects of mood on memory. Within the network concepts and events are represented as nodes, with links of varying strength between them - the associative connections. When a word, (or concept) is presented, the corresponding node is activated, with activation spreading along the links to other closely related nodes. The most important aspect of the model as applied to mood and memory research is the assumption of specific nodes for each emotion, associatively linked with clusters of nodes representing descriptions of past events, beliefs, the subjective experience and verbal labels (Bower, 1981). When activation exceeds a threshold level, either through presentation of a stimulus or the result of a prior thought, the contents of the network enter awareness.

Mood congruity effects are thought to occur in free recall tests as a result of increased elaboration (more associative links) between items that are congruent with the subject's prevailing mood, along with biased search strategies. In reviewing the

evidence that cognitions affect depressive mood and mood affects cognitions, Teasdale (1983) concludes that depression may be maintained by a reciprocal relationship between the two. This can readily be accounted for in terms of activation of a depression network. Similarly, Ingram (1984) suggests a feedback loop involving the activation of negative memories which enter consciousness, recycling activation back through the depression node.

Although the model can be successfully applied to interpret much of the mood and memory literature, there are several major problems, discussed by Williams, Watts, McLeod and Mathews (1988). The most important of these is that anxiety and depression do not appear to have comparable effects on attention and memory, with an attention bias more readily demonstrated in anxiety states, but selective memory more strongly evidenced in depression. Associative network theory would not predict this distinction in the effects of the two moods on information-processing.

### *Schemata*

In contrast to the nodes of the associative network model described above, schema are mental representations of all the information relevant to a particular stimulus. Schema interact with new information, influencing the encoding, understanding and retrieval of that information as a result of beliefs, expectations, rules and assumptions, and by guiding attention and memory search (Beck, Emery and Greenberg, 1985). By definition, schema have consistent internal structures, often considered modular such that activation of any part of the schema will result in activation of the whole (Mandler, 1984). In addition schema are commonly considered to comprise prototypes of stimuli, against which new information is



evaluated. Both bottom-up and top-down processing are thought to be involved in schematic processing of a stimulus: identifying the relevant schema and using it to comprehend the stimulus respectively.

These principals of schematic functioning are able to accommodate mood and memory phenomenon. For example, the schemata of depressed individuals may be overactive, and new information which is schema-congruent more likely to be assimilated, leading to biases in interpretation of new events and in recalling information. These schemata are also thought to contain faulty information about the self, world and future (Beck, 1967; Beck *et al*, 1979). In particular, knowledge about the self is thought to exist in "self-schema" (Kuiper, MacDonald and Derry, 1983), resulting in the biased processing of negative information which has been encoded with specific reference to the individual in self/other-reference recall paradigms (eg. Derry and Kuiper, 1981).

However, like associative network models, schema theory is unable to account for the differing effects of anxiety and depression on information-processing.

### *An Integrated Model*

Williams, Watts, MacLeod and Mathews (1988) propose an 'integrated model' which is able to account for the differential effects of anxiety and depression on attentive and memorial processes. The model is based on the concepts of integration (priming) and elaboration (Graf and Mandler, 1984). Priming is the automatic activation of the mental representation of a stimulus, resulting in strengthening of the internal organization of the representation and hence increasing accessibility - the word is more likely to come to mind when only partial cues are presented.

Elaboration occurs when activation spreads from the representation of the stimulus to other associated representations, forming new links and activating old ones. The consequence of elaboration is heightened retrievability of the word. Graf and Mandler (1984) explicitly state in summary that integration makes the word more accessible, whereas elaboration renders the word both more accessible and more retrievable. The implication is that the process of elaboration necessarily involves priming. Williams *et al* (1988) propose that tasks such as the dot-probe assess the level to which words have been primed, and that anxiety acts to bias the extent to which threat information is primed. They also suggest depression specifically affects the elaboration stage of processing, such that negative information is more easily remembered in free recall tasks. In anxiety states priority is given to threat-related information in the allocation of processing resources pre-attentively. In depression the allocation of processing resources is to the elaboration of negative material. Inherent in this model is the assumption that priming and elaboration are entirely distinct, however, this is in direct conflict with the original Graf and Mandler model as described above. If the original model is strictly applied to the data from the mood and information-processing literature, the following predictions would ensue: 1. anxiety causes bias in priming, and hence attention bias; 2. depression leads to bias in priming *and* elaboration, and thus attention bias *and* memory bias. As noted in earlier sections, there is some evidence that negative affect is associated with an attentive bias (Gotlib and McCann, 1984; Williams and Nulty, 1986; Williams and Broadbent, 1986).

## **AIMS OF THE THESIS**

The experiments reported in this thesis are an attempt to explore the impact of chronic pain on a variety of cognitive processes, within an information-processing framework. Particular emphasis is placed on the investigation of schematic and conceptual processing. Schematic processing is explored through an investigation of selective memory for sensory and affective information in depressed and non-depressed chronic pain patients, and acute pain patients. Schematic processes are investigated further by comparing responses of chronic pain patients, health professionals and controls on a word-stem completion task. Conceptual processing is explored by developing a questionnaire examining pain-related beliefs. Changes in both schematic and conceptual processing as a result of surgical and cognitive-behavioural interventions are investigated. Causal relationships between beliefs about pain and psychological variables including depression, health locus of control, cognitive coping strategies and activity levels are examined. The possibility that information-processing biases in chronic pain may be attributable to differences in frequency/usage of pain-related word stimuli between chronic pain patients and non-patient controls is tested. The adequacy of the associative network account of effects of pain on memory is also examined.

## *Chapter 2 Investigation of selective memory for sensory and affective information in chronic pain and depression*

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### INTRODUCTION

#### BACKGROUND

The importance of cognitive factors in emotional and physical disorders has been emphasized in the previous chapter. An analysis of the role of cognitions in depression has been presented by Teasdale (1983), in terms of a reciprocal relationship, such that negative cognitions produce depression, and depression increases the probability that those cognitions will be experienced, causing further depression. Thus a vicious cycle is invoked, perpetuating and intensifying the depression. Based on several standard information-processing concepts, including network theories, depth of processing and cognitive capacity, Ingram (1984) provides a comprehensive analysis of the possible mechanisms of onset and maintenance of depression, accounting for the emotional, cognitive, motivational and physical symptoms of the disorder.

One of the processes thought to play an important role in the relationship between cognitions and depression is memory, and this will now be considered in some depth.

#### EFFECT OF DEPRESSION ON RECALL

Much of the research in this area has centred on an attempt to identify the cognitive processes responsible for the vicious cycle of negative thoughts and

depression. To this end, the role of memory processes, especially mood congruity effects, have<sup>3</sup> been explored with researchers seeking to specify which factors underlie differences between depressed and non-depressed subjects. The approach which allows the greatest control over variables such as the affective tone of the material, is the use of word lists (other approaches have been discussed in chapter 1). Typically, subjects are presented with lists of words, either with or without the knowledge that they are going to be asked to remember them at a later stage. Either each of the individual lists contain words of different emotional valence, or the words are of mixed valence *within* each list.

There are two main types of study in this area; firstly those involving the induction of the desired mood states, usually contrasting depression with elation, in normal subjects. Secondly, subjects are classified as either depressed or non-depressed on the basis of clinical diagnosis or self-report measures. As with all research of this nature, caution should be exercised in generalizing the results of normal subjects to clinical groups.

One of the first relevant studies in this area, undertaken by Isen, Shalke, Clark and Karp (1978) employed a success/failure mood induction procedure. Positive and negative affect was induced prior to, and immediately after, learning a list of 26 words, including positive, negative and neutral personality trait words. Forty-seven psychology students were divided into 4 groups; those who experienced success at a computer game both before and after learning the words, those who failed at both times, a group who succeeded before learning the list but failed at the game afterwards, and finally a group who failed at the first game but succeeded at the second. Results showed no evidence for a state-dependency memory effect - there

was no significant interaction between outcome (success/failure) at the game the first and second times. However, results did indicate a significant interaction between wordtype and mood at time of recall, such that students who had experienced success after learning the list recalled more of the positive trait words. Notably, there was no evidence for an effect of negative mood on the recall of negative information, nor differences in the recall of neutral words. Isen *et al* conclude that selective memory is therefore associated with retrieval rather than encoding processes, and suggest that the results represent the effect of mood on accessibility of cognitions.

McDowell (1984) was able to demonstrate biased recall of negative words in depressed patients only when words were presented in mixed lists of pleasant and unpleasant words, rather than when presented in separate lists. He suggests that the reason for this is that mixed lists produce competition for processing resources between wordtypes, whereas separate lists do not. McDowell also found that, compared to non-depressed controls, depressed patients recalled more unpleasant than pleasant words, but only when not instructed to rate the stimuli in terms of their pleasantness. McDowell concludes that these results indicate the importance of *encoding* processes in selective recall.

Teasdale and Russell (1983) investigated the effect of induced mood (elation or depression) at the time of recall on the recall of mixed lists of positive, negative and neutral personality trait words, presented in normal (presumably neutral) mood. They clearly demonstrated a recall bias for positive words in the elation condition, and negative words in the depression condition. However, the experimenters explicitly told the subjects (n=32) that the study concerned the effect of mood on memory, and it is therefore not implausible that the subjects guessed the hypotheses, especially in

view of the induction procedure used - modified Velten (1968) statements.

An important factor in mood-related memory biases appears to be the applicability of the material to the person learning it. Bradley and Mathews (1983), for example, found a bias for negative compared to positive self-referent adjectives in depressed patients compared to non-psychiatric controls, only when asked to think of the words in relation to themselves. When instructed to focus on the applicability of the words to others, the patients showed a positive (normal) recall bias.

In a similar study to that of Teasdale and Russell (1983), Clark and Teasdale (1985) found that women, but not men, recalled more pleasant than unpleasant personality trait words when in induced happy mood and vice versa. The induction procedures were equally effective for men and women, and they did not differ on their ratings of the pleasantness of the words. In a second study they found that women gave higher usage ratings for the trait words, and that within this group usage predicted the extent of preferential recall in the congruent mood state. This is of great importance, since it implies that selective memory may simply be a function of word frequency for the particular population being sampled, rather than any inherent differences in the way in which groups process information. (This issue is addressed in a later chapter, in relation to chronic pain patients.)

The results of these experiments are, clearly, not entirely consistent. However, it is apparent that under certain, well defined circumstances, and given the methodological weaknesses of some of the studies, a memory bias for words of negative emotional valence can be demonstrated, in both normal subjects in whom mood states have been induced, and in subjects with naturally occurring clinical mood states.

## RECOGNITION MEMORY AND MOOD

In addition to exploring the effect of mood on free recall, some investigators have assessed the relationship between mood and recognition memory. In this paradigm the subject is typically presented with a list of words (which he/she may be asked to remember in a free recall task). These words are then presented again (the "old" words), interspersed with an equal number of "new" words. The subject is required to decide whether each word was a member of the original list. The advantage of this approach is that it enables a distinction to be made between a person's "true" memory - their ability to recall information, and response bias. A response bias explanation in its most general sense suggests that selective memory is an artefact of emotional states, and that for example, depressed subjects are "preset" to always respond to negative material in a particular, stereotyped way. In terms of recognition memory, response bias refers more specifically to the relative laxity or strictness of the criterion used by subjects in making each recognition judgement.

Several studies have used the recognition paradigm to investigate the effect of mood on memory, generally with little success (Bower and Cohen, 1982 provide a review). These experiments typically concentrate on hit rates as their dependent variable, ie. the number of words correctly recognised. Several reasons for the lack of significant results have been proposed. Simon (1982) offers an explanation in terms of the "index" and "encyclopedia" in episodic memory; Bower and Cohen (1982) suggest that presenting the "old" word provides such a strong cue that it overrides the relatively weak mood cue, and Williams *et al* (1988) propose a simple ceiling effect.



## SIGNAL DETECTION THEORY

The experiments described above do not, however, use the recognition paradigm to its full potential. Responses on a recognition test fall into four categories - hit (correct recognition of an old word), false alarm (saying a new word was present when it wasn't), correct rejection and false rejection. Following the principles described by Swets *et al* (1961), this data can be submitted to a Signal Detection Theory (SDT) analysis, which separates the effects of response bias ( $\beta$ ) from "true" memory ( $d'$ ). Small values of  $\beta$  are indicative of a relatively lax criterion, high values a more strict criterion. The use of SDT for memory research is not universally accepted, primarily because the assumptions of normality, homogeneity of variance and an optimally located criterion are not always met, and receiver operating characteristics (ROC curves) are rarely calculated to test these assumptions. Despite this, Healy and Kubovy (1978) conclude that in relation to other methods of dealing with recognition data,  $d'$  is the "preferred index of performance".

Zuroff, Colussy and Wielgus (1983) suggest that "most of the existing evidence on selective memory and depression is readily interpreted in terms of response bias...". They therefore tested 3 groups of psychology students, classified as depressed, formerly depressed and non-depressed using the short form Beck Depression Inventory (BDI) and a past tense form of the BDI. Subjects were presented with 10 positive and 10 negative self-relevant adjectives, and tested on free recall, and after 7 days a recognition test comprising the original 20 words and 20 new words matched for valence. The authors claim that the results of the recall tests indicate a recall bias for negative adjectives in the depressed and formerly depressed groups compared to the non-depressed group. The hit rate and false alarm rate data

also apparently tended to support a selective processing account; more (although not significantly) negative adjectives were correctly recognised by the depressed and formerly depressed groups, and depressed subjects produced the most negative false alarms, and non-depressed the least. These results are, however, qualified by significant differences in response bias ( $\beta$ ) between the groups, with depressed subjects employing a less strict criterion than the other two groups. Zuroff *et al* interpret these results as indicative either of differing guessing strategies between the groups, or differences in the "willingness" of the groups to *report* negative adjectives, but not intrinsic differences in the subjects' ability to recall the material.

This study has been heavily criticised by Martin and Clark (1986a,b). They point out the failure to carry out appropriate analyses of variance, by considering positive and negative words separately (a 2-way, groups x wordtype was needed), and that the necessary interaction between them was unlikely to have been significant given the reported means. Depressives appear instead to simply show a tendency to adopt a less strict criterion for all material irrespective of valence. In addition, the Zuroff *et al* study does not permit the distinction between effect of mood on encoding and retrieval processes to be made.

In a much better designed study Dunbar and Lishman (1984) presented 30 clinically depressed and 30 non-depressed controls with positive, negative and neutral words (they were not specifically told to learn them), which were then presented again with an equal number of matched words for the recognition test. The results of interest were a highly significant interaction between group and wordtype for the variable  $d'$ , such that depressed patients had higher  $d'$  values for the negative words than did controls, with the opposite pattern for positive words. They also report a

stricter criterion (higher  $\beta$ ) in depressed patients for positive and neutral words, but, importantly, no difference in  $\beta$  for the negative words.

## A THEORETICAL PERSPECTIVE

Despite the lack of consistency in the results of SDT analysis of recognition memory, the approach remains a useful tool in the attempt to specify the exact nature of selective memory effects. Also, although the results of both recall and recognition memory experiments have not been entirely consistent, where selective memory has been found, it has invariably been accounted for in terms of an associative network model of mood and memory. Hypothesized emotion "nodes" are cognitive representations of clusters of memories associated with a particular emotion, and include the concepts, beliefs, descriptions of past events etc. linked to that emotion (Bower, 1981). Activation of a node and its associated network above a threshold level results in memories entering conscious awareness (see previous chapter for details). It is proposed that there exists a "pain node", which, in addition to the negative affect node, is activated during the experience of pain. This node, and its close associates comprise the sensory attributes of pain, memories of past experiences of pain, and beliefs about the causes of pain etc. It is hypothesized that, over time, when the experience of pain becomes chronic, the pain node and associated network will become permanently activated to such an extent that selective memory effects become apparent. If in addition to the long-term activation of the pain node, the individual becomes depressed, under these circumstances the node representing the affective side of pain would also become chronically activated.

## MEMORY AND PAIN - EMPIRICAL EVIDENCE

Providing general support for this idea, one study used word lists to explore the role of memory in pain. In their first experiment Pearce *et al* (1990) compared 25 chronic pain patients with 25 non-patient controls on a recall test comprising pain-related (sensory), negative and neutral words. Their results provided evidence for a mood congruity effect in the chronic pain group, who recalled significantly more pain-related words than did the controls. In their second experiment non-patient volunteers undergoing experimentally induced pain (a cold pressor task) provided no evidence for selective recall of pain-related information compared to volunteers not subjected to pain. These results suggest that a memory bias in relation to pain may be more related to the status of being a chronic pain patient than the state of being in pain.

However, it could be argued that since the chronic pain sufferers in the Pearce *et al* (1990) study were significantly more depressed than their control group, the selective memory for sensory words demonstrated in their experiment may have been an artefact of the negative affect in the chronic pain group. The differences in depression levels could, incidentally, account for the lack of memory bias in the induced pain group. Also, in terms of a pain node, induced pain would not necessarily be assumed to activate the same network as that for chronic pain. Although sensory attributes of the experience would activate similar "sensory" concepts, the meaning of the pain, its implications etc. are likely to be very different. The whole package of activation, over a long time, may be required for selective processing.

## STUDY AIMS

The aims of this study are twofold. Firstly, to test the prediction that, in accordance with network theory, patterns of selective recall in chronic pain and depression are related to both pain and depression status. A word list recall paradigm will be used, with sensory and affective pain related adjectives, and neutral adjectives, in four groups of subjects: chronic pain patients who are also depressed, chronic pain patients who are not depressed, depressed patients with no pain, and non-patient controls. Secondly, the relative contributions of true memory and response bias in selective memory in these groups will be investigated using a recognition paradigm and Signal Detection Theory analysis.

## METHOD

### DESIGN

A mixed design was employed with two between groups variables and one within groups variable - chronic pain status, depression status and wordtype respectively. The four subject groups comprised patients with either chronic pain, depression, both pain and depression or neither. The wordtype categories consisted of two classes of pain-related adjectives, sensory and affective, and neutral adjectives unrelated to pain. The sensory and affective adjectives were chosen from the McGill Pain Questionnaire (MPQ) (Melzack, 1975), and were matched for frequency and number of syllables with the neutral adjectives (Carroll, Davies and Richman, 1971). The affective category included some words from the "evaluative" scale of the MPQ. This scale has been shown not to be separable from the MPQ affective scale using factor

analysis (Brennan *et al*, 1987). The words used are presented in Table 2.1.

Table 2.1 Sensory, affective and neutral words used in the recall tests.

| Sensory   | Affective     | Neutral   |
|-----------|---------------|-----------|
| scalding  | horrible      | flexible  |
| stabbing  | unbearable    | windswept |
| pressing  | mild          | imprecise |
| boring    | discomforting | amazing   |
| pounding  | fearful       | educated  |
| tender    | cruel         | polished  |
| tingling  | miserable     | legal     |
| flashing  | gruelling     | selective |
| throbbing | distressing   | leaking   |
| crushing  | troublesome   | promising |
| tugging   | vicious       | nimble    |
| hurting   | terrifying    | angular   |

The experiment was divided into two sections, recall and recognition. The design of each is outlined below.

*Recall Tests.* Three recall tests each comprised 4 sensory, 4 affective and 4 neutral words in pseudo-random order, along with 3 neutral "fillers" at the beginning and end of each list. The fillers were included to minimize primacy and recency effects, and were excluded from statistical analyses, except to obtain a measure of the proportion recalled. The main dependent variable was the number of words correctly recalled from each wordtype.

*Recognition Test.* The 54 words of the recall lists were matched for wordtype,

frequency and number of syllables with an equal number of new adjectives, constituting the 108 words of the recognition test. The additional words are shown in Table 2.2. Again the order of presentation was pseudo-random. Here the dependent variables were the number of words correctly recognized ("hits") and the number of words "recognized" which were new words ("false alarms"), for each wordtype category.

Table 2.2 Additional sensory, affective and neutral words for the recognition test.

| Sensory   | Affective    | Neutral    |
|-----------|--------------|------------|
| beating   | killing      | spreading  |
| shooting  | tiring       | grand      |
| pricking  | suffocating  | reputable  |
| drilling  | wretched     | swaying    |
| cutting   | blinding     | prime      |
| pinching  | sickening    | protruding |
| gnawing   | frightful    | youthful   |
| wrenching | excruciating | transient  |
| searing   | punishing    | resounding |
| itchy     | exhausting   | knotty     |
| aching    | annoying     | stony      |
| splitting | intense      | informal   |

## SUBJECTS

The subjects recruited for the study were chronic pain patients from Pain Relief Clinics at the Whittington and Wanstead Hospitals, clinically depressed patients attending the Middlesex Hospital and normal, non-patient controls. The control group comprised mainly adults attending Extra-Mural Studies evening classes, and a small number who replied to advertisements requesting volunteers for a psychological

experiment. Patients were not included if, in the opinion of the physician responsible for their care, they were suffering global memory, attention or concentration dysfunction as a result of organic brain disease, psychosis, brain damage, Alzheimer's Disease, alcohol intoxication, Korsakoff Syndrome or other amnesic syndrome. The Beck Depression Inventory (BDI), (Beck et al, 1961) was used to classify the pain patients into two groups: those who were not depressed (score 0 - 8), and those who exhibited significant depressive symptomatology (score 15 or above) at the time of testing. Similarly, the criterion for inclusion of psychiatric depressed patients was a minimum score of 15, and for controls a maximum score of 8 on the BDI.

All the chronic pain patients had a history of at least six months pain, and were in pain at the time of testing. Ideally the psychiatric depressed patients would have had no pain, however it proved impossible to fulfil this condition since most of these patients reported some pain. Patients were therefore excluded from this group if they reported a current pain intensity rating of 35 or more on a 0 - 100mm Visual Analogue Scale [VAS]. This group had a mean current pain intensity score of 8.50, which contrasts with that of the pain patients, whose mean pain intensity rating was 54.9. A total of 121 subjects was recruited; of these 49 were excluded. Reasons for exclusion were as follows: twenty one chronic pain patients scored in the range 6 to 14 on the BDI; 10 of the depressed patients either failed to score the minimum required on the BDI or had pain intensity ratings of greater than 35; and 16 controls were excluded for analogous reasons. The remaining 72 comprise the final groups.

The characteristics of the subjects in these groups are shown in Table 2.3. The diagnoses classified as "other" in this table include neck/shoulder, limb, intestinal and



Table 2.3 Characteristics of subjects.

|   | Pain, Not<br>Depressed<br>n=19 | Pain,<br>Depressed<br>n=16 | Depressed,<br>Not Pain<br>n=18 | Control<br>n=19 |
|---|--------------------------------|----------------------------|--------------------------------|-----------------|
| <b>Biographical</b>                             |                                |                            |                                |                 |
| Mean age (sd)                                   | 45.79 (14.8)                   | 52.38 (8.54)               | 45.39 (14.6)                   | 39.42 (9.9)     |
| Sex ratio (M:F)                                 | 1:0.9                          | 1:2.2                      | 1:1.25                         | 1:1.71          |
| <b>Pain Intensity</b>                           |                                |                            |                                |                 |
| Mean pain intensity <sup>a</sup> (sd)           | 54.37 (24.4)                   | 55.71 (26.4)               | 8.50 (12.4)                    | 1.79 (4.0)      |
| <b>Depression Status</b>                        |                                |                            |                                |                 |
| Mean BDI <sup>b</sup> score (sd)                | 4.47 (2.1)                     | 23.13 (8.2)                | 29.06 (10.5)                   | 3.26 (2.4)      |
| <b>Chronicity of Condition</b>                  |                                |                            |                                |                 |
| <i>Pain patients</i><br>Mean (mths) (sd)        | 66.74 (44.1)                   | 65.07 (70.0)               | -                              | -               |
| <i>Psychiatric patients</i><br>Mean (mths) (sd) | -                              | -                          | 43.31 (48.1)                   | -               |
| <b>Diagnostic Status</b>                        |                                |                            |                                |                 |
| <i>Pain patients</i><br>Low back pain (%)       | 31.58                          | 20.00                      | -                              | -               |
| Arthritis (%)                                   | 10.52                          | 20.00                      | -                              | -               |
| Neuralgia (%)                                   | 5.25                           | 13.33                      | -                              | -               |
| Other (%)                                       | 52.65                          | 46.67                      | -                              | -               |
| <b>Medication Status</b>                        |                                |                            |                                |                 |
| Prescribed analgesics (%)                       | 26.32                          | 42.86                      | 00.00                          | -               |
| "Over-the-counter" analgesics (%)               | 21.05                          | 14.29                      | 5.56                           | -               |
| Anti-depressants (%)                            | 00.00                          | 7.14                       | 55.56                          | -               |
| Anxiolytics (%)                                 | 10.53                          | 21.43                      | 22.22                          | -               |

<sup>a</sup> 0 -100mm Visual Analogue Scale

<sup>b</sup> Beck Depression Inventory

myofascial pain. With regard to the medication classification, it should be noted that some patients were taking more than one type of drug at the time of testing. There was no significant difference in the ratio of males to females in the four groups ( $\chi^2 = 1.971$ ,  $df=3$ ,  $p>0.50$ ).

## PROCEDURE

For the recall task subjects were asked to listen to the word lists presented using a Sony "Walkman"-type recorder. Subjects were instructed to attempt to learn the words, and were told that they would be asked to repeat out loud all those they could remember. The inter-stimulus interval (ISI; onset to onset) was 2 seconds. After each list the subject was allowed 2 minutes for free recall.

Instructions were then given for the recognition task. The words were again presented using the Sony Walkman. After hearing each word the subject was required to decide whether the word had previously been in any one of the three recall lists, responding "yes" if they thought it was, and "no" if they believed it was not. If they were uncertain, they were instructed to guess. Here the ISI was 3 seconds, to allow sufficient time for the subject's verbal response. All responses were recorded by the experimenter. No attempt was made to obtain ratings of how confident subjects were in the responses they made on each item, which would have allowed ROC curves to be determined. This was because in a pilot study Dunbar and Lishman (1984) found this procedure too demanding for their patient population.

The 21 item, full length BDI was then completed, and the VAS rating of current pain intensity was obtained using a 0 -100mm VAS anchored at 'no pain' and 'the most intense pain I have ever experienced'. The BDI and pain intensity rating were

administered after the memory tests to avoid possible priming effects, which would confound the results of the memory tests.

The duration of the procedure was approximately 30 minutes. Subjects were unaware of the hypotheses under investigation; they were told merely that the experiment concerned memory ability.

## RESULTS

### *Recall*

The recall score was expressed as a proportion of the total correct recall accounted for by each wordtype was calculated for each subject. This data was subjected to a 3-way, split-plot Analysis of Variance using the program BMDP 2V with pain and depression as the between groups variables and wordtype as the repeated measure variable. None of the main effects or two-way interactions were significant. A significant pain by depression by wordtype interaction emerged using the Greenhouse-Geisser probability adjustment for multiple levels of the repeated measure,  $F(2,136)=3.68$ ,  $p=0.0285$ . To control for the possibility that age may influence accuracy of recall, since the groups were initially different in their mean age, the analysis was repeated with age as a covariate. No change in the patterns of results occurred and the pain by depression by wordtype interaction was unaltered.

Table 2.4 presents the proportion recall means for each group and wordtype, along with their standard deviations. Figure 2.1 depicts graphically the nature of the interaction. It can be seen that the pain patients who were not depressed, and the

Table 2.4 Mean proportion recalled (SD) by group and wordtype

|           | Pain, Not<br>Depressed<br>n=19 | Pain,<br>Depressed<br>n=16 | Depressed,<br>Not Pain<br>n=18 | Control<br>n=19 |
|-----------|--------------------------------|----------------------------|--------------------------------|-----------------|
| Sensory   | 0.221 (0.13)                   | 0.194 (0.13)               | 0.206 (0.10)                   | 0.170 (0.05)    |
| Affective | 0.139 (0.12)                   | 0.205 (0.19)               | 0.122 (0.10)                   | 0.193 (0.07)    |
| Neutral   | 0.154 (0.09)                   | 0.138 (0.08)               | 0.147 (0.08)                   | 0.181 (0.05)    |

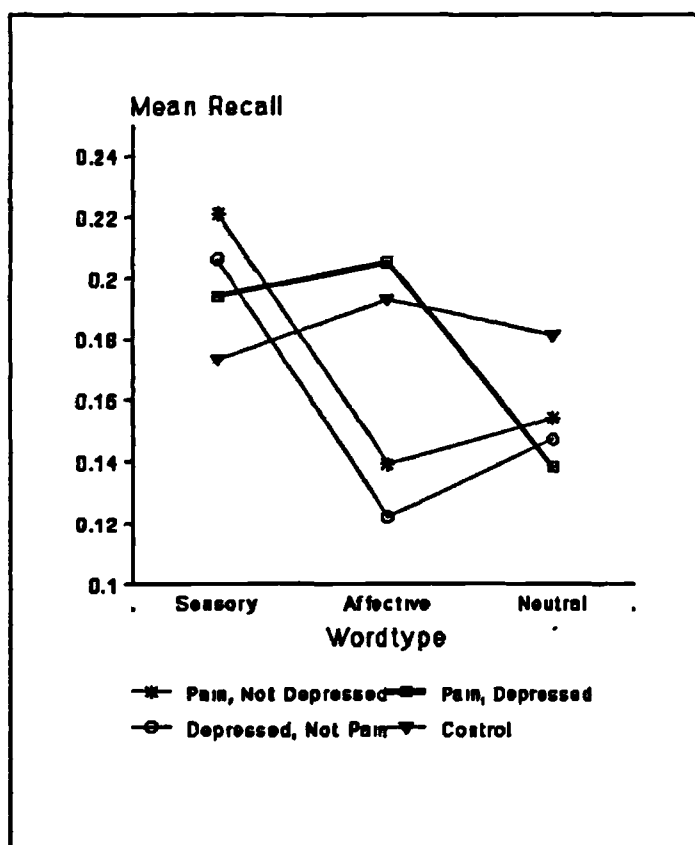


Fig. 2.1 Mean proportion recalled by group and wordtype.

psychiatric depressed patients, recalled more sensory than affective words, while the depressed pain patients recalled approximately equal proportions of sensory and

affective words, and fewer neutral words. The control group recalled similar numbers of words from the three wordtype categories. A simple effects analysis of wordtype for each group was performed to identify the location of the significant effect. The simple effect of wordtype for the four groups, each with 1.96,133.39 df using the Greenhouse Geisser adjustment, were as follows: pain, not depressed,  $F=3.07$ ,  $p=0.0584$ ; pain, depressed,  $F=1.69$ ,  $p=0.1919$ ; depressed, not pain,  $F=2.74$ ,  $p=0.0726$  and control,  $F=<1$ ,  $p=0.8018$ . In accordance with predicted patterns of recall, a priori contrasts were carried out on the simple effects for the first three groups. In the pain, not depressed group, significantly more sensory than affective or neutral words were recalled,  $F(1,18)=4.75$ ,  $p=0.0428$ . In the depressed pain group the comparison between the neutral and the sensory and affective words did not reach significance,  $F(1,15)=2.27$ ,  $p=0.1528$ . Finally, in the depressed, not pain group the contrast comparing affective versus sensory and neutral was also significant,  $F(1,17)=5.56$ ,  $p=0.0360$ . Since such a procedure is unable to distinguish between a V-shape and an inverted V-shape pattern, it is clear from the graph of the results that the significance lies in the opposite direction to that predicted, ie. fewer affective words were recalled than either sensory or neutral words.

### *Recognition*

The variables  $d'$  and  $\beta$  (derived from signal detection analysis procedures), were computed from the "hit rate" and "false alarm rate" variables using tables from Hochhaus, (1972), for each subject and wordtype. The  $\beta$  distributions for each wordtype were significantly skewed, and were therefore subjected to log transformation which returned each distribution to an adequate degree of normality.

The means and standard deviations of these results are presented in Table 2.5. These variables were subjected to separate 3-way split-plot ANOVAs, with pain and depression as between groups variables and wordtype as the repeated measure variable. The significant results of these analyses are displayed in Table 2.6.

The main effect of pain in the  $d'$  data can be accounted for by higher  $d'$  values in non-pain patients compared to chronic pain patients. Similarly, the main effect of depression is accounted for by lower  $d'$  values in depressed compared to non-depressed patients. The interaction between pain and wordtype approached significance ( $p=0.08$ ). The pattern of this result, which was also apparent but not significant in the depressed patients, suggests that higher  $d'$  values occur for sensory words compared to affective or neutral. Finally, in general, affective words produced lower  $d'$  values than the other two wordtype categories (main effect of wordtype)

The only significant effect found in the  $\beta$  data under this analysis was a main effect of wordtype. A post-hoc contrast analysis using Scheffe's adjustment revealed that neutral words resulted in significantly higher  $\beta$  values than did either sensory or affective words ( $F(1,71)=40.25, p<0.001$ ).

## DISCUSSION

The results of the recall section of this study are generally consistent with the initial predictions. Chronic pain patients who were not depressed showed a pattern of selective recall directly related to pain and depression status. In other words, those pain patients who showed minimal or no depressive symptomatology had a recall

Table 2.5 Mean (SD)  $d'$  and  $\text{Log } \beta$  by group and wordtype.

|                                       | Pain, Not<br>Depressed<br>n=19 | Pain,<br>Depressed<br>n=16 | Depressed,<br>Not Pain<br>n=18 | Control<br>n=19 |
|---------------------------------------|--------------------------------|----------------------------|--------------------------------|-----------------|
| <b><math>d'</math></b>                |                                |                            |                                |                 |
| Sensory                               | 1.507 (0.70)                   | 1.442 (0.78)               | 1.450 (0.76)                   | 1.469 (0.67)    |
| Affective                             | 1.007 (0.67)                   | 0.950 (0.66)               | 0.996 (0.75)                   | 1.560 (0.81)    |
| Neutral                               | 1.221 (0.73)                   | 1.186 (0.47)               | 1.264 (0.73)                   | 2.133 (1.10)    |
| <b><math>\text{Log } \beta</math></b> |                                |                            |                                |                 |
| Sensory                               | 0.049 (0.49)                   | -0.128 (0.45)              | 0.096 (0.51)                   | 0.142 (0.45)    |
| Affective                             | 0.144 (0.47)                   | -0.241 (0.50)              | -0.054 (0.38)                  | 0.075 (0.57)    |
| Neutral                               | 0.363 (0.50)                   | 0.183 (0.50)               | 0.426 (0.41)                   | 0.357 (0.49)    |

Table 2.6 Significant effects of separate ANOVAs for  $d'$  and  $\beta$ .

|                     | Main Effect | F Value         | p     |
|---------------------|-------------|-----------------|-------|
| $d'$                | Pain        | (1,68) = 4.40   | 0.040 |
|                     | Depression  | (1,68) = 4.69   | 0.034 |
|                     | Wordtype    | (2,136) = 6.04  | 0.003 |
| $\text{Log } \beta$ | Wordtype    | (2,136) = 21.87 | 0.000 |

bias for sensory adjectives alone. Although the expected bias for the depressed chronic pain patients did not quite reach statistical significance, the pattern found was as predicted - high recall for both sensory and affective material compared to neutral material. These effects may have been more significant had the subjects been instructed to encode the material self-referentially. The non-patient controls showed no bias for any wordtype. All these findings are in line with predictions that pain and

depression are associated with specific selective information-processing biases.

In theoretical terms, the results of this experiment are supportive of the notion that there are separate nodes for pain and depression, with distinct associated networks. However, the findings do to some extent conflict with those of Eich *et al* (1990), who suggest that pain impedes the retrieval of pleasant material and aids the retrieval of unpleasant material *only* when the pain is accompanied by negative affect. In other words pain per se has no impact on memory. If this were invariably the case it would be predicted that no recall bias be found for either sensory or affective adjectives, *unless* the chronic pain patients were also depressed, ie. pain alone would not result in any selective processing of pain-related material. This clearly was not the case in this study, nor that of Pearce *et al* (1990). This apparent discrepancy may have been the direct result of differences between autobiographical memory as in Eich *et al*, and memory for recently presented words as in these experiments. Alternatively, pain may aid only the retrieval of pain-related events, rather than any unpleasant event, as was assessed by Eich *et al* (1990).

One recall result, however, runs contrary to predictions. This is that patients who were depressed but not suffering chronic pain showed no recall bias for the affective pain words, these adjectives being proportionally less well remembered than either sensory or neutral words. This is surprising and runs contrary to the prediction derived from previous findings that clinically depressed patients selectively recall negative material. Two possible explanations can be found for these results. Firstly, the affective adjectives in this study may not have been sufficiently salient to the depressed patients to increase the activation of the depressive associative network, in the absence of a forced encoding strategy. This may have been because these



words, although negative, were more strongly linked to pain than depression. Mood congruity effects, when found, appear to be largely dependent on the subject being required to think of the material with particular reference to themselves (eg. Bradley and Mathews, 1983). In previous studies subjects have been forced to process material in greater depth than would result from just hearing the words: for example subjects have been asked to rate themselves on each adjective (eg. Roth and Rehm, 1980). Although this explanation is a plausible account of the failure to demonstrate a bias for the affective words it does not account for the significant V-shaped pattern of results, nor indeed for the fact that an alternative contrast comparing sensory with both affective and neutral words was also significant, ie. that the affective and neutral words are significantly less well remembered than the sensory. There are two possibilities. Firstly that the depressed non-pain patients have a memory bias for sensory pain-related words (the alternative contrast). Secondly, and more plausibly, that these results represent an alternative information-processing mechanism, "cognitive avoidance". This concept is discussed in relation to anxiety disorders by Foa and Kozack, (1986), who point out that in this group of patients "concentrating on the non-fearful elements of a situation is a common pattern". Thus cognitive avoidance is believed to occur when emotional stimuli, after identification as such, are less well processed than neutral stimuli. This would result in such stimuli being less well remembered. The phenomenon has been demonstrated by Watts et al (1986), who found that spider phobics are increasingly less able to identify spiders in a recognition task, as the size of the spider is increased. In the present study cognitive avoidance appears to have operated such that the most 'distressing' words, the "affective" ones, were actively avoided by the clinically depressed patients.

Processing was therefore diverted to the neutral, and in particular sensory words. Sensory words may have been more salient than the neutral words, probably because this patient group is likely to have had a significantly higher incidence of pain problems in the past than normal populations, (who did not show this pattern of recall). The processes involved in cognitive avoidance are poorly understood, and as Williams et al (1988) point out, the boundary conditions under which it occurs are as yet unknown. However, it appears that depressed mood, as well as anxiety, may play an important role in the process. This account of the results in terms of cognitive avoidance clearly requires further investigation, but is consistent with the debriefing reports of patients who remarked that many of the words were very relevant to them, but they couldn't recall them because they were too 'painful' or 'difficult'. The first experiment reported in the next chapter aims to address this issue in a group of clinically depressed patients.

Two conclusions may be drawn from the results of the recognition data. The  $d'$  results suggest that both people who suffer chronic pain, and people who are depressed have poorer overall true memory than normal controls, but there is some evidence that in the patient groups true memory is better for sensory information. This follows logically from the recall results, and provides insight into the possible nature of this information-processing bias. However, once again the results of the psychiatric depressed group are anomalous. The recall data and its interpretation provides a clue as to why this might be so. A cognitive avoidance mechanism operating in this group could account for these results. Since processing is diverted away from affective words and onto sensory and neutral ones, the  $d'$  scores (true memory) for affective adjectives were lower than for sensory and, (to a lesser extent)

neutral adjectives. The precise nature of these differences in true memory can, as yet, only be intimated. Whether they reflect encoding, storage or retrieval processing dissimilarities needs to be empirically explored. No conclusions can be drawn at this point since the subjects were in the same "mood" at both encoding and retrieval.

The second conclusion concerns the contribution of response bias to the selective memory effect. The  $\beta$  results indicated a propensity for subjects in all groups to adopt a stricter criterion for deciding that they have heard a word before if it has a neutral valence. In this respect the results are analogous to those of Dunbar and Lishman (1984). The reliability of this finding needs further investigation, but the results do suggest that memory biases in chronic pain and depression can to some extent be accounted for by differences in true memory ability, and are not solely the consequence of response biases.

From a clinical perspective, the findings of this study have implications for the maintenance of chronic pain problems. Selective memory processes are likely to play a role in instigating a vicious cycle similar to that proposed by Teasdale (1983) in depressed patients, although the exact mechanisms, and relationships between sensory and affective components of pain remain undetermined. A further issue which requires clarification is the role of duration of pain in selective memory, and therefore the second experiment to be reported in the next chapter examines the recall of sensory, affective and neutral adjectives in a group of patients experiencing acute, clinical pain.

## *Chapter 3 Tests of cognitive avoidance in depression and selective memory in acute clinical pain*

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### INTRODUCTION

One of the interesting and puzzling findings of the previous chapter was that a group of clinically depressed patients failed to exhibit the predicted memory bias for affective adjectives, compared to sensory and neutral adjectives. Indeed, a contrast analysis of this group's data demonstrated that significantly *fewer* affective words were recalled compared to the other wordtypes. This unexpected result was accounted for in terms of cognitive avoidance. An empirical example of this phenomenon is provided by Watts, Trezise and Sharrock (1986). In the first of two experiments they showed that spider phobics have poorer recognition memory for spiders than do controls, but only for large spiders (this was a post hoc comparison of small versus large spiders). They interpreted this as indicative of larger spiders provoking greater emotional arousal. Their second experiment failed to demonstrate the predicted remedial effect of elaborative encoding. It was hypothesized that forcing phobic subjects to look carefully at, and describe 2 distinguishing features of each spider would result in the amelioration of the poorer recognition memory for big spiders, ie. under this condition phobics would have similar memory to controls. This was not statistically supported under the *a priori* analysis.

In a recent study Watts and Dalgleish (1991) provided further evidence for poor memory for spider-related information in spider phobics. Using a wordlist recall

memory paradigm, they found that phobics show significantly poorer recall of spider words (compared to a group of words also from a category - baby-related words) in relation to non-phobic controls. The results were consistent with a shift in processing resources towards baby words and away from spider words in the phobics. Watts and Dalgleish suggest that cognitive avoidance provides a plausible account of these results.

Although cognitive avoidance effects have, to date, been associated solely with anxiety disorders, it is not unreasonable to suggest that a similar process operates, under certain conditions, in depression.

The first experiment reported here aims to test the hypothesis that clinically depressed patients cognitively avoid certain types of negative information, and that this avoidance results in poorer memory for this information. Within a simple recall paradigm, groups of clinically depressed patients and non-depressed controls are presented with the same words as previously (sensory, affective and neutral), with an additional category, adjectives associated with depressive feelings. They were included in addition to the MPQ affective/evaluative words in order to check that these words were not under-recalled in the previous experiment because they were not sufficiently connected with depression to activate the depressive associative network. The alternative possibility was that this group had a bias *for* sensory information. This will be clarified in this experiment.

The second issue to be explored in this chapter concerns the specificity of selective memory effects to *chronic* pain. At this point it seems appropriate to draw an analogy between pain and anxiety. Anxiety is often divided into state (the current, largely situation-dependent level of anxiety) and trait (seen as an enduring personality

characteristic) components. In terms of information-processing, chronic pain may be analogous to trait anxiety, and acute pain more akin to state anxiety.

There is evidence to suggest that attentional bias in anxiety is most reliably related to trait rather than state anxiety. Broadbent and Broadbent (1988), in a series of experiments using the visual dot-probe paradigm demonstrated that the bias "characterizes the individual and [is] not solely a change that appears in anybody who enters a temporary state of anxiety". The authors also point out that this finding is of theoretical interest, since it lends credence to the assertion that biased processing is a causal factor in the development of the clinical disorder.

If this analogy holds, it would be anticipated that in acute pain conditions no selective processing of pain-related information be apparent. In support of this, Pearce *et al* (1990) found no evidence for biased recall of sensory adjectives in a group of subjects in whom pain had been experimentally induced. However, it would be invalid to assume that induced pain is equivalent to acute clinical pain. Therefore, in the second experiment reported here a comparison will be made between a group of patients suffering acute pain (of less than one months duration), and controls. The same measures and procedures as those employed in the original experiment will be used.

## **EXPERIMENT 1. Test of cognitive avoidance in depression**

### **METHOD**

#### **DESIGN**

A mixed design was employed, with one between groups variable, depression

status (clinically depressed or non-depressed), and one repeated measure variable, wordtype. The wordtype categories comprised sensory, affective, neutral and depressive adjectives. Four lists of words, with 3 words from each category, were presented in fixed pseudo-random order, with 3 fillers at the beginning and end of each list. Thus the independent variables were depression status and wordtype, and the dependent variable was number of words correctly recalled.

## MATERIALS

The same sensory, affective and neutral words as those used in the previous experiment were employed. A pool of 12 adjectives reflecting feelings associated with depression were generated. These were as follows: pessimistic, lethargic, lonely, hopeless, discouraged, worried, pathetic, gloomy, inadequate, bleak, worthless, despairing. All words were matched as closely as possible for frequency and length. The depressive adjectives did not come from a recognised source (unlike the words typically chosen for memory research in depression, they were not members of the list of personality trait words rated for likeableness by Anderson, 1968, eg. hostile, impolite, dishonest). Personality trait words were not chosen because they do not describe the feelings associated with depression, and are therefore not comparable with the pain-related adjectives and the experience of pain. An attempt was made to ensure that these words were appropriate. A group of 15 psychology undergraduates rated how negative each of the depressive and neutral words were on a 5-point scale from "not at all negative" to "extremely negative". They were also asked to rate how depressed they were currently feeling on a scale from 0 (not at all depressed) to 10 (extremely depressed). A matched t-test confirmed that the two categories of words

were completely distinguishable on this basis,  $t(14)=16.75$ ,  $p<0.0001$ . There was no significant correlation between levels of depression and ratings of how negative the adjectives were.

## SUBJECTS

Twenty-seven clinically depressed subjects were recruited from out-patient clinics and the psychiatric day hospital at Watford General Hospital. Patients were required to score a minimum of 15 on the BDI for inclusion in this group. Patients were not included if they had any history of chronic pain, or were experiencing any current pain. Of the 27 patients interviewed, 8 failed to meet these criteria and were therefore excluded from statistical analyses.

The control group comprised 25 people randomly chosen from a pool of volunteer members of the general public. Inclusion in this group required a maximum score of 9 on the BDI, and no history of chronic pain. Four of the 25 subjects were excluded on this basis. The characteristics of these groups are shown in table 3.1.

Table 3.1 Characteristics of subjects.

|  | Depressed Patients<br>n=19 | Controls<br>n=21 |
|--|----------------------------|------------------|
| Mean age (sd)                              | 41.74 (14.40)              | 37.00 (11.68)    |
| Sex ratio (M:F)                            | 8:11                       | 5:16             |
| Mean BDI <sup>a</sup> score (sd)           | 25.68 (9.12)               | 6.14 (2.13)      |
| Duration of depression<br>mean (mths) (sd) | 32.38 (58.28)              | -                |

<sup>a</sup> Beck Depression Inventory



## PROCEDURE

Subjects were tested individually. They were informed that they would hear four lists of words, and that after each list they would be asked to say out loud as many of the words as possible. Lists were presented aurally using a Sony "Walkman" recorder at a rate of one word every two seconds (stimulus onset to onset). Subjects were allowed two minutes for free recall (this length of time was, in fact, never completely filled). The 21-item, full length BDI was then administered, and it was ascertained whether the individual had ever experienced a chronic pain condition, or was currently in any pain. Subjects were given no indication of the true nature of the experiment prior to participating, but were fully debriefed afterwards.

## RESULTS

Mean recall scores, expressed as proportions of the total recall including fillers, were calculated. This data was subjected to a 2-way, split plot ANOVA using the program BMDP 2V, with depression status (depressed versus control) as the between groups variable, and wordtype (sensory, affective, neutral or depressive) as the repeated measure variable. The analysis revealed a significant main effect of wordtype,  $F(3,114)=4.49$ ,  $p=0.0051$ , accounted for by better overall recall of neutral adjectives. There was no evidence for either a main effect of depression status,  $F(1,38)<1$ ,  $p=0.7674$ , nor a wordtype by group interaction,  $F(3,114)=1.30$ ,  $p=0.2783$ . However, an *a priori* contrast analysis in accordance with the cognitive avoidance hypothesis in the depressed patients (ie. comparing recall for sensory and neutral adjectives with

Table 3.2 Mean proportion recalled (SD) by group and wordtype.

|            | Depressed Patients<br>n=19 | Controls<br>n=21 |
|------------|----------------------------|------------------|
| Sensory    | 0.132 (0.09)               | 0.094 (0.06)     |
| Affective  | 0.093 (0.06)               | 0.097 (0.04)     |
| Neutral    | 0.143 (0.06)               | 0.154 (0.06)     |
| Depressive | 0.112 (0.07)               | 0.124 (0.07)     |

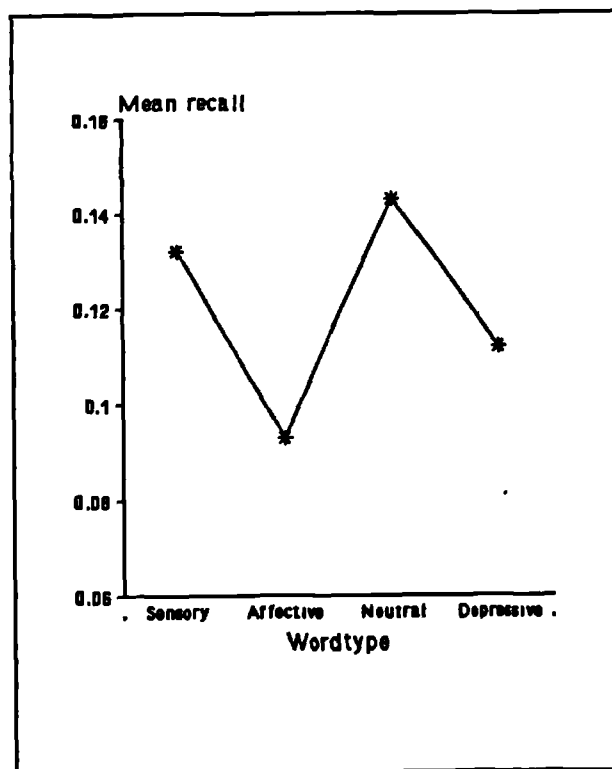


Figure 3.1 Mean proportion recalled by depressed patients in each wordtype category.

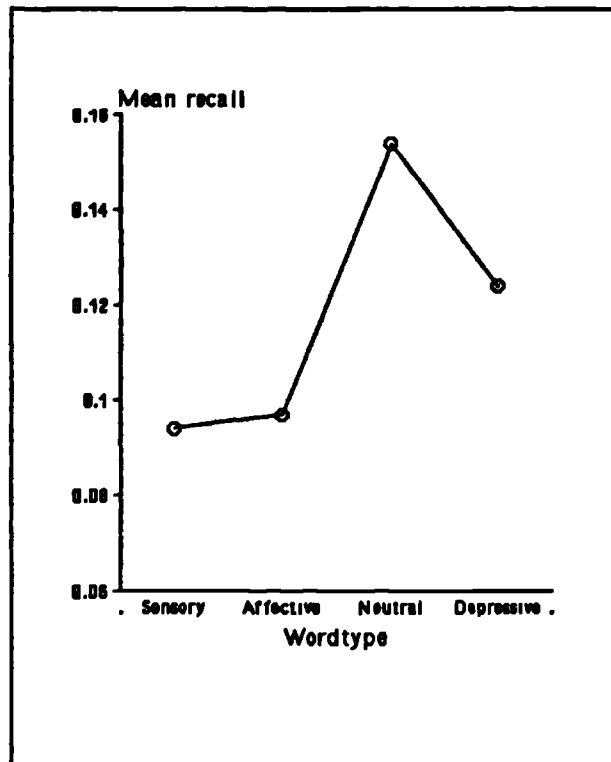


Figure 3.2 Mean proportion recalled by control subjects in each wordtype category.

that for the affective and depressive adjectives) was significant,  $F(1,18)=5.74$ ,  $p=0.0276$ . Also, a *post hoc* contrast on the control group's results, comparing recall of neutral words with the other 3 categories was significant,  $F(1,20)=10.45$ ,  $p=0.0042$ . The mean proportions recalled, along with standard deviations are presented in Table 3.2, and graphically for each group separately (since there was no interaction) in Figures 3.1 and 3.2.

## **EXPERIMENT 2. Test of selective memory in acute clinical pain.**

### **METHOD**

#### **DESIGN, MATERIALS and PROCEDURE**

In this experiment acute pain patients and controls were compared on recall and recognition memory tests. Again a mixed design was used, with one between groups variable, pain status (acute pain versus control) and one repeated measure variable, wordtype (sensory, affective and neutral). The wordlists were identical to those described in Chapter 1 and the procedures for both recall and recognition tests were replicated.

#### **SUBJECTS**

Sixteen patients from the gynaecology ward at Whipps Cross Hospital were recruited for this experiment. Patients were invited to participate in the research if they had recently (within the last 4 days) undergone planned surgery for previously non-painful conditions, or had surgery for ectopic pregnancy. This condition typically causes intense pain which rapidly necessitates treatment. Patients whose condition had caused pain for longer than 4 weeks were excluded.

Patients were tested between one and four days after surgery, mean=2.75 days, (sd=0.75). At pain assessment, after the memory test, it became apparent that 3 patients were no longer in pain, and were therefore excluded from the statistical analysis. The mean age of the acute pain group was 30.31 years (sd=5.63). Their mean current pain intensity rating on a 0-100mm VAS was 40.54 (sd=25.11), and

their mean BDI score was 7.00 (sd=5.54). 53.85% of the group were taking analgesics.

The control group comprised the female members of the non-patient control group of the experiment reported in Chapter 1. Their mean age was 39.50 years (sd=10.30), current VAS pain intensity rating was 1.83 (sd=4.32), and mean BDI score was 3.17 (sd=2.52).

## RESULTS

### *Recall*

The proportion recall data was subjected to a split-plot ANOVA with pain status (acute pain versus control) as the between groups variable, and wordtype (sensory, affective and neutral) as the repeated measure variable. There was no evidence for main effects of pain status ( $F(1,23)=1.23$ ,  $p=0.2783$ ) or wordtype ( $F(2,46)=2.28$ ,  $p=0.1162$ ), nor an interaction between the two variables ( $F(2,46)=1.66$ ,  $p=0.2015$ ). Although the two groups differed significantly in their levels of depressive symptomatology (as assessed by the BDI,  $t(23)=2.195$ ,  $p<0.025$ ), the use of BDI scores as a covariate in the above analysis did not alter the results. The mean proportion recalled for each group and wordtype, with corresponding standard deviations, can be found in Table 3.3. The results are presented graphically in Figure 3.3. Although further statistical analysis is inappropriate given the lack of significant effects, it can be seen that in the acute pain group a trend is emerging for sensory words to be remembered best, and neutral words least well.

Table 3.3. Mean proportion recalled (SD) for each group and wordtype.

|           | Acute Pain Patients<br>n=13 | Controls<br>n=12 |
|-----------|-----------------------------|------------------|
| Sensory   | 0.244 (0.09)                | 0.176 (0.05)     |
| Affective | 0.198 (0.12)                | 0.187 (0.07)     |
| Neutral   | 0.153 (0.08)                | 0.168 (0.06)     |

### *Recognition*

The variables  $d'$  (true memory) and  $\beta$  (response bias) were derived from the hit rate and false alarm rate results, using tables from Hochhaus (1972). Mean  $d'$  and  $\beta$  scores and their standard deviations are presented in Table 3.4.  $D'$  and  $\beta$  scores were subjected to separate two-way ANOVAs with pain status as the between groups variable and wordtype as the repeated measure variable. Under the first analysis ( $d'$ ), a significant main effect of wordtype was found using Greenhouse-Geisser adjusted probabilities,  $F(2,46)=6.55$ ,  $p=0.0033$ . A significant interaction between pain status and wordtype also emerged,  $F(2,46)=3.60$ ,  $p=0.0360$  (again with Greenhouse-Geisser probabilities). The main effect can be accounted for by superior true memory for neutral adjectives. A simple effects analysis of wordtype for each group was performed to identify the location of the significant effect(s) within the interaction. The simple effect of wordtype for the acute pain patients and controls, with Greenhouse-Geisser adjusted probabilities were  $F(1.97,45.25)=5.08$ ,  $p=0.0106$  and  $F(1.97,45.25)=5.07$ ,  $p=0.0106$  respectively. It can be seen from the graph of these results (Figure 3.4) that acute pain patients have significantly poorer true memory for affective compared to sensory and neutral adjectives, whereas control subjects have

significantly higher  $d'$  scores for neutral adjectives alone.

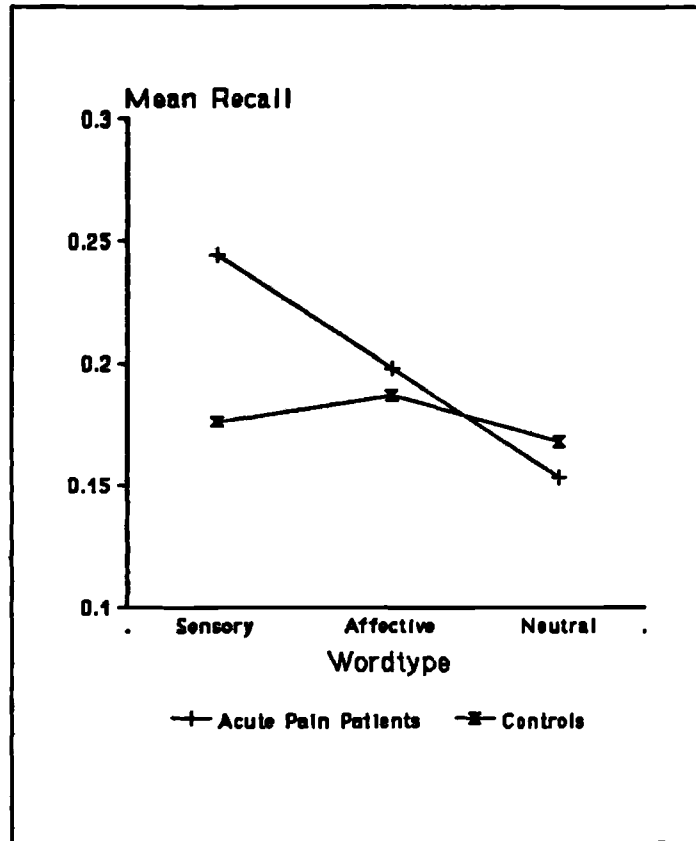


Figure 3.3. Mean proportion recalled by group and wordtype.

Under an equivalent analysis of  $\log \beta$ , significant main effects of pain status and wordtype were found,  $F(1,23)=9.61$ ,  $p=0.0050$  and  $F(2,46)=3.76$ ,  $p=0.0308$  respectively, but no significant interaction emerged. The main effect of pain status reflects a stricter criterion (higher values of  $\log \beta$ ) in the acute pain patients across all wordtypes. The main effect of wordtype can be accounted for by higher  $\beta$  values for neutral adjectives. For clarity, the pattern of these results is presented graphically in Figure 3.5.

Table 3.4 Mean (SD) scores for the variables  $d'$  and  $\log \beta$ .

|                                | Acute Pain Patients<br>n=13 | Controls<br>n=12 |
|--------------------------------|-----------------------------|------------------|
| <b><math>d'</math></b>         |                             |                  |
| Sensory                        | 2.605 (0.75)                | 2.423 (0.53)     |
| Affective                      | 1.843 (0.83)                | 2.598 (0.93)     |
| Neutral                        | 2.577 (0.82)                | 3.274 (1.17)     |
| <b><math>\log \beta</math></b> |                             |                  |
| Sensory                        | -0.184 (0.47)               | 0.252 (0.34)     |
| Affective                      | -0.179 (0.29)               | 0.249 (0.55)     |
| Neutral                        | 0.139 (0.22)                | 0.403 (0.22)     |

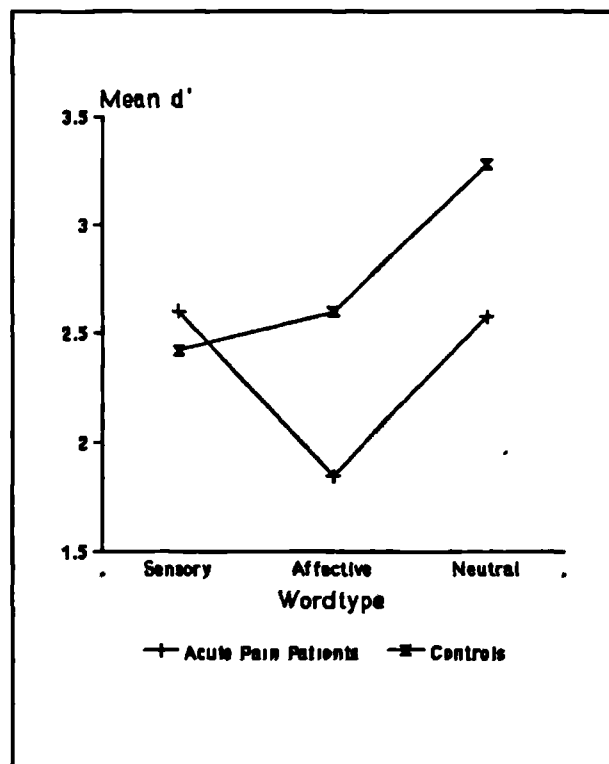


Figure 3.4 Mean  $d'$  scores by group and wordtype.



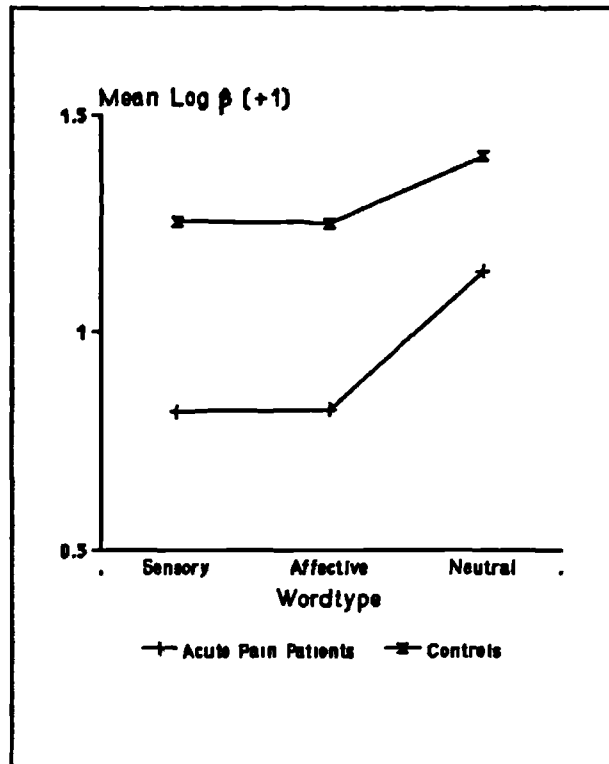


Figure 3.5 Mean Log  $\beta$  by group and wordtype.

## DISCUSSION

### COGNITIVE AVOIDANCE IN DEPRESSION

The results of the clinically depressed patients in the first experiment can be seen to provide evidence for cognitive avoidance of information which is related to the emotional components of depression. These results support this interpretation of the findings in the clinically depressed group in the previous chapter, and render the alternative possibility (a bias for sensory words) less likely. Indeed, in the present experiment sensory adjectives were marginally less well remembered than neutral adjectives. However, before considering the possible mechanisms and implications of the cognitive avoidance interpretation, it is worthwhile to appraise, and eliminate

alternative explanations.

Firstly, rather than placing emphasis on the relative under-recall of the affective and depressive adjectives, it may be that sensory and neutral were relatively over-recalled, ie. that this group of patients have a memory bias *for* sensory and neutral adjectives. This is unlikely and makes little sense. Care was taken to ensure that none of the patients had any previous or current pain complaints, and even if the group were in some way unusual with regard to pain, this would not account for the equally high recall of neutral adjectives.

Secondly, the concreteness and imageability of the words may have differed significantly between categories, and therefore played an important role in recall. It is well established that words which are more concrete and hence more imageable are more easily remembered (Paivio, 1969). It could be argued that the neutral words were slightly more concrete than the other three categories, resulting in superior recall of this category. However, this explanation could not account for the relatively better recall of the sensory adjectives.

Thirdly, following the equally well established finding that presenting words belonging to a common semantic category increases recall (eg. Deese, 1959; Jenkins and Russell, 1952; Bousfield, 1953 and Cohen, 1966), it would be predicted that affective, depressive and (not just) sensory words would have been recalled more than the neutral words. This was clearly not the case. In support of the improbability of this as an explanation, Watts and Dalglish (1991) showed less free recall of spider words in spider phobics in comparison to another set of words which also belonged to a category - baby related words.

Fourthly, and lastly, it may be that the words chosen, despite the additional

category, still did not adequately represent the emotional component of depression. Again this seems unlikely, since if these words were simply undifferentiated from the sensory and neutral words, no difference in the number of words recalled from each category would be predicted.

Thus, the most plausible explanation for the results of the clinically depressed patients in this experiment is that information associated with feeling depressed is cognitively avoided.

There are several possible mechanisms potentially involved in a cognitive avoidance process. Depressed patients may attend less to depression-relevant material, leading to poor encoding and subsequently poor recall. If this were the case, on a theoretical level in terms of the associative network model, the depressive network would not become activated, and hence the words would not readily be recalled (and they certainly would not be selectively remembered). Alternatively, the depressed patients may attend equally to all information, but only elaborate non-personally relevant material, perhaps as a safety mechanism. This is not consistent, of course, with the clinical observation that depressed individuals tend to ruminate, and fail to prevent negative thoughts from leaving conscious awareness. Hypotheses concerning the roles of attention and elaboration in cognitive avoidance clearly need empirical investigation.

The last issue to be considered in relation to these results, is why was cognitive avoidance found to be operating in this group, when research in this area typically points to a recall bias *towards* remembering negative information? There are two main differences between this study and those in which selective memory for negative information was demonstrated. In the present study subjects were not

required to perform a self-referential encoding procedure. In addition, the words chosen were very different to those used in previous experiments. The words used were chosen very specifically to reflect the *emotions* associated with depression, rather than personality trait words which are more closely related to self-image. The emotion words may have caused greater arousal in the depressed subjects resulting in cognitive avoidance and poorer processing. This explanation is in accord with that provided by Watts, Trezise and Sharrock (1986), who suggested that the large spiders in their study caused greater arousal in the phobics than the small spiders, leading to poorer recognition of the large spiders.

Finally, with regard to this experiment, the controls subjects' results require some comment. There was evidence to suggest that in this group neutral words were recalled better than any of the other three categories. It is possible that this is simply an expression of the tendency of non-patient subjects to remember words which have either a positive valence, or non-emotional content.

#### SELECTIVE MEMORY IN ACUTE CLINICAL PAIN

The results of the second experiment provide some evidence to support the hypothesis that patients suffering acute clinical pain will not show a memory bias. Although the interaction between pain status and wordtype did not approach significance, the data do suggest that a higher proportion of sensory adjectives are recalled compared to neutral adjectives. The proportion of affective adjectives recalled took an intermediate position. This is not surprising since some of the patients scored in the moderately depressed range on the BDI, but there were too few subjects in the group to justify a comparison between depressed and non-depressed

patients. Given the slightly ambiguous nature of the results, it would be premature to draw firm conclusions on this issue. Larger groups of subjects may have revealed a significant interaction, and this clearly requires further investigation.

If the statistical findings are considered conclusive, and the trend ignored, the analogy between acute and chronic pain, and state and trait anxiety suggested in the introduction, seems applicable. Like the role of selective attention in anxiety, which has been shown to be more closely associated with state anxiety, or at least an interaction between state and trait anxiety (Broadbent and Broadbent, 1988; MacLeod and Mathews, 1988), selective memory seems to be associated more closely with chronic than acute pain.

The results of the signal detection analysis of recognition data do little to illuminate the situation. Neither the true memory nor response bias measures are able to account for the free recall results, and hence some doubt must be cast over their utility in investigating information-processing in pain.

Although the results of the recall test, when taken at face value, do point to the importance<sup>of</sup> chronicity in the development of information-processing biases in pain, other factors such as beliefs, and attitudes, exerting a "top-down" processing influence are also likely to be of great significance. In the terms of Leventhal (1979), conceptual processes influence lower level schematic processes. The conceptual processing of an individual experiencing an acute pain episode, with the expectation of full recovery, is likely to differ markedly from that of patients for whom pain is a permanent problem. One of these factors, beliefs, forms the focus of the next chapter, in which differences in beliefs about pain are explored in chronic pain patients and non-patient controls.

## *Chapter 4 The development and validation of a questionnaire assessing beliefs about pain*

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### INTRODUCTION

In recent years research has increasingly focused on beliefs about pain in chronic pain patients as important factors in pain report, psychological functioning and treatment compliance (eg. Williams and Thorn, 1989). In a comprehensive review of the literature Jensen, Turner, Romano and Karoly (1991) identified 62 articles which examined the relationship between beliefs, coping efforts and adjustment to chronic pain. At a general level, an internal locus of control has been associated with positive adaptation to chronic pain - greater use of active coping strategies and less depression (Crisson and Keefe, 1988 ; Skevington, 1983). More specifically, there is evidence for a relationship between beliefs about the ability to control pain and coping/adjustment (eg Strong *et al*, 1990; Jensen *et al*, 1987; Crisson and Keefe, 1988; Keefe and Williams, 1990; Jensen and Karoly,1991). Beliefs concerning helplessness in relation to chronic pain have been associated with passive coping style, and greater levels of psychological and physical disability, pain intensity and interference with activity (Nicassio *et al*, 1985; Smith *et al*, 1988; Flor and Turk, 1988). Although there is a fair degree of consistency in findings between studies, the majority employed correlational designs and therefore the *causal* role of the beliefs investigated cannot be assumed.

The concept of attributional style, which is closely linked to that of locus of

control, has received some attention in relation to chronic pain. Three primary attributional styles have been identified as playing a role in chronic pain and psychological functioning: internal attributions - the belief that outcomes are the result of something about the individual; stable attributions - beliefs that outcomes are a result of non-transient factors and are therefore long-lasting, and finally global attributions - beliefs that many situations will have the same outcome (Abramson, 1978). The Attributional Style Questionnaire, developed by Peterson *et al* (1982) as a measure of internal, stable and global attributions has been applied to chronic pain patients with mixed results. In the first study Love (1988) found that depressed chronic pain patients, in comparison to non-depressed chronic pain patients, are more likely to exhibit all three attributional styles for negative, but not positive outcomes. In a second study, a composite score of the three styles for negative outcomes was found to be related to depression in a group of chronic pain patients (Cheatle *et al*, 1990). However, no significant association between depression and attributional style was found by Ingram *et al* (1990).

Taken as a whole, despite a few inconsistencies, these results clearly demonstrate the importance of beliefs and attributions in the adjustment to chronic pain.

Thus, to date, great emphasis has rested on the impact of beliefs about the ability to *control* pain on measures of psychological and physical functioning. In contrast, there is a relative paucity of research investigating beliefs and attributions about the *causes* of pain, its consequences, and factors affecting the experience of pain. It is thought that if symptoms are attributed to a neutral "external" cause, they will be less disabling and cause less distress than if attributed to personal "internal" causes. (Storms and McCaul, 1976). For example, Storms and Nisbett (1970) investigated

this phenomenon in a group of insomniac patients, who typically experience heightened arousal symptoms on retiring to bed. Two groups of subjects were given a placebo pill, the first with the information that it would cause increased heart rate, alertness and body temperature, the other group being informed that the pill was a sedative. The second group took longer to get to sleep, apparently because they were unable to attribute their symptoms to an external cause - the pill. Similar results were found by Liebhart, (1974), who provided cardiac neurosis patients (who worry excessively about relatively minor cardiac problems) with a pill to which they could attribute their symptoms. Compared with waiting list and irrelevant placebo control groups, these patients showed significantly less somatic, affective and behavioural deterioration. Although not without criticism (Watts, 1983, pg 139), these studies provide evidence that if patients are given the opportunity to reattribute their symptoms externally they are less troubled by their symptoms.

Psychological and organic beliefs about the causes of symptoms may be seen to parallel internal and external attributions, and have been identified as having impact on psychological functioning. Watts (1983) suggests that patients who "attribute somatically based symptoms to internal, psychological causes may be unnecessarily disturbed by them". For example, in obesity it is found that altered circadian rhythms induced by hormones may be misattributed by patients to emotional arousal (Rodin, 1978). More commonly, however, predominantly psychological symptoms may be believed to be due to an organic cause. Imboden *et al* (1961) found that some patients who had recovered from influenza and who continued to complain of symptoms three weeks later believed that they still had an organic disease. Since these patients could be predicted from the depression scale of the MMPI administered



prior to the onset of the infection, it is likely that their symptoms were indicative of depression and misattributed to the influenza. It follows that the way in which a patient explains their illness to themselves will to some extent determine the way in which they cope with that illness and respond to interventions. The model proposed by Leventhal *et al* (1980) describes how a patient's representation of their illness can play a role in both preventing and dealing with illness, by acting as part of a regulatory system guiding coping efforts and setting goals by which coping efforts are evaluated.

Clearly attributions concerning the causation of pain and recovery from it will influence the way in which pain is experienced and communicated to others. As pain persists, pain behaviours and emotions associated with the experience of pain may reinforce the notion of illness and the sick role, thus inhibiting coping and reducing the probability of effective treatment. An understanding of the chronic pain patient's attributional belief framework is therefore likely to aid accurate assessment and the development of appropriate management strategies (Watts, 1983).

In an attempt to provide a method of assessing abnormal behaviour Pilowsky and Spence (1983) developed an instrument, the Illness Behaviour Questionnaire, which comprises 7 scales - general hypochondriasis, disease conviction, psychological versus somatic perception of illness, affective inhibition, affective disturbance, denial and irritability. Of particular interest is the third scale which assesses the tendency to blame oneself and be accepting of the need for psychiatric help at one end of the scale, and rejection of the possibility that psychological factors are important and greater focus on somatic problems at the other end. Although at face value this measure appears to satisfy the need for an assessment tool for psychological versus

somatic beliefs, many of the items in the questionnaire as a whole have been found not to meet adequate psychometric standards (Main and Waddell, 1987). In another large scale study the third scale disappeared altogether on factor analysis (Zonderman, Heft and Costa, 1985).

Three studies have described the development of measures designed to assess beliefs about pain. The Pain Beliefs and Perceptions Inventory (Williams and Thorn, 1989) has three dimensions - self-blame, perception of pain as mysterious and beliefs about the duration of pain. They found a positive association between pain intensity and the belief that pain is enduring. In addition, their results indicated that such beliefs are also related to decreased compliance with health psychology and physical therapy interventions. A strong belief in the mysterious nature of pain had similar impact on compliance with physical therapy, along with lack of improvement post treatment with psychological distress and somatization. The belief that pain is enduring and mysterious was shown to be linked with decreased likelihood of using cognitive coping strategies, greater probability of catastrophization and less likelihood that coping strategies would be rated as effective.

The second, the Beliefs about Pain Control Questionnaire (Skevington, 1990) is derived from the Multidimensional Health Locus of Control Questionnaire (MHLC, Wallston and Wallston, 1978). It assesses the extent to which people believe that they have personal control over their pain (Internal scale), or fate, or doctors and other influential people control their pain (Chance and Powerful Others scales). As yet there is little data on the relationship between these scales and indices of pain experience.

(Jensen et al)

Finally, the Survey of Pain Attitudes (1987) provides a measure of attitudes in 5

subscales; medical cure, pain control, solicitude, disability and medication. Although it is relatively comprehensive and has adequate psychometric properties, this questionnaire cannot readily assess the constructs of current interest. Also, it does not permit a variety of degrees of agreement with, or belief in a concept, given its true/false response options. A recent study (Jensen and Karoly, 1991) has also examined the relationship between patients' beliefs about the degree to which they could control pain and psychological functioning, medical services use and activity level. The authors demonstrated the importance of control beliefs in well-being and activity levels in chronic pain patients.

Although these measures and findings provide insight into certain aspects of belief systems they do not encompass specific beliefs about the causes and consequences of pain in terms of organic and psychological components, the importance of which have been outlined.

Thus there is evidence to support the contention that attributional beliefs about pain aetiology and consequences play a significant role, however there is as yet no standardized instrument with which they can be assessed. The aim of this study was to develop an instrument to assess such beliefs concerning the experience of pain. The new questionnaire was then used to compare the beliefs of a heterogeneous group of chronic pain patients and non-patient controls, and to investigate the relationship between beliefs, the MHLC and other pain-related measures.

## **Phase 1: Scale Development**

### **METHOD**

#### **DESIGN**

Twenty items were chosen for the questionnaire, reflecting common beliefs about the experience of pain - its causes, consequences and factors influencing its severity. Each item contained a statement concerning pain, and at the appropriate place in the sentence a choice of six qualifying adverbs. The instructions read as follows:

'For each item please indicate your opinion by underlining one of the following words in each sentence: always, almost always, often, sometimes, rarely, never. There are no right or wrong answers: it is important that you respond according to your actual beliefs, not according to how you feel you should believe, or how you think we want you to believe.'

A sample of 294 people, comprising 100 chronic pain patients and 194 non-pain subjects completed the questionnaire.

#### **SUBJECTS**

(a) **Chronic Pain Patients.** Patients suffering pain of mixed aetiologies, for a minimum of six months were recruited for the study. Thirty-eight (out of 45 who were invited to participate) completed the questionnaire, while waiting for routine

Pain Clinic appointments at Whipps Cross Hospital. In addition patients attending the Pain Clinic at the Hammersmith Hospital were mailed the questionnaire: 152 were sent out and 62 of these were returned usable. Reasons for the non-return of questionnaires could not be assessed. It is assumed that those patients who did respond provided a representative sample of the chronic pain population. The mean age of these subjects was 53.8 years (SD=14.05); 62% were female.

(b) Non-Patient Controls. The criteria for inclusion in this group was an absence of pain complaint, and maximum scores of 2 out of 10 for current pain intensity and 3 out of 10 for average pain intensity over the past week respectively. Subjects were recruited from two sources. Firstly, 140 students attending Extra Mural Studies evening classes at the University of London were mailed the questionnaire. Forty-five of the 65 returned were usable. Secondly, 116 undergraduate students of University College London completed the questionnaire - 102 were acceptable. The mean age of these controls was 26.34 years (SD=10.76); 66.2% were female.

(c) Others. This group comprised 47 members of the general public approached outside Euston Station who agreed to fill in the questionnaire. This London station was selected at random as a place where a large number of people congregate and have time to fill in a questionnaire. It was also thought that such a location would provide a representative sample of the adult population. Their mean age was 34.3 years (SD=16.16); 57% were female.

## PROCEDURE

All subjects who were mailed the questionnaire were invited in a covering letter to participate in a study concerning the experience of pain. They were assured of the

confidentiality and anonymity of their responses, and were provided with a stamped, addressed envelope for the return of the questionnaire. The remaining chronic pain patients were approached by a member of the pain clinic nursing staff and invited to participate in the project, under the same conditions. The undergraduate students completed the questionnaire after attending psychology lectures. The subjects approached outside Euston Station were given brief details of the nature of the study, and invited to participate in a similar manner to all other subjects. Despite the apparently complex format of the questionnaire, no subject experienced difficulty in its completion.

## **RESULTS**

Responses of all 294 subjects on the Pain Beliefs Questionnaire were subjected to a Factor Analysis with oblique rotation using the Varimax procedure. Two factors were obtained accounting for 68.15% of the variance. Items were included in a factor if they loaded greater than 0.4 on either but not both factors. Table 4.1 presents the items with their factor loadings. Where these are less than 0.4 they are replaced by zero. The rotated factors did not correlate significantly (0.078). The squared multiple correlations of the factors with their items were 0.80 and 0.73 for factors 1 and 2 respectively. This indicates good internal consistency of the factors.

Factor 1, accounting for 43.89% of the variance consists of 10 items, primarily concerning the organic aspects and implications of pain. The four items of factor 2 accounted for 24.26% of the variance, and were psychological in nature. Five of the items did not meet the loading criteria to be included in either factor (items 2,4,6,12

and 14), and one item loaded on both factors, and was therefore dropped from the solution (item 13). The two items loading least heavily on the first factor were also then removed from the solution, since they were very similar in content to two other items loading on this factor. This led to greater equality of number of items on each of the resultant questionnaire scales. Thus the final solution comprised two factors, the first with eight items, the second with four.

All items loaded positively onto their respective factors. A second factor analysis, using identical procedures but on these 12 items confirmed the factor structure. Each item loaded at least 0.4 on the same factor as in the original analysis and no item loaded on both factors or on neither factor. This solution accounted for 82.37% of the total variance.

### Scale Reliability

Total factor scores were calculated for each individual, scoring the items from 1, 'never', through to 6, 'always'. Using Cronbach's coefficient alpha to assess the internal reliability of the test showed good internal consistency for each scale; 0.73 and 0.70 for the organic and psychological scales respectively. Considering the chronic pain patients alone, the coefficients were 0.71 and 0.73.

Scores on the scales (adjusted for the difference in number of items on each) were subjected to a two-way split-plot analysis of variance with group (pain vs non-pain) as the between groups variable and scale as the repeated measures variable. Subjects from Euston Station were excluded from this analysis due to concerns about the reliability of the information concerning their pain status. Results indicated main effects of both group ( $F(1,236)=4.86, p<0.05$ ) and scale ( $F(1,236)=54.36, p<0.0001$ )

Table 4.1 Rotated factor loadings for the Pain Beliefs Questionnaire administered to 294 subjects

| Item  | Factor Loadings |         |
|---|-----------------|---------|
|   | Factor 1        | Factor2 |
| 1. Pain is the result of damage to the tissues of the body.                 | 0.428           | 0.000   |
| 2. Doctors/GPs are the people best able to relieve pain.*                   | 0.000           | 0.000   |
| 3. Physical exercise makes pain worse.                                      | 0.506           | 0.000   |
| 4. Taking medication is the best way to relieve pain.*                      | 0.000           | 0.000   |
| 5. It is impossible to do much for oneself to relieve pain.                 | 0.550           | 0.000   |
| 6. When in pain it is advisable to rest.*                                   | 0.000           | 0.000   |
| 7. Being anxious makes pain worse.  | 0.000           | 0.580   |
| 8. Experiencing pain is a sign that something is wrong with the body.       | 0.563           | 0.000   |
| 9. When relaxed pain is easier to cope with.                                | 0.000           | 0.449   |
| 10. Being in pain prevents you from enjoying hobbies and social activities. | 0.513           | 0.000   |
| 11. The amount of pain is related to the amount of damage.                  | 0.530           | 0.000   |
| 12. A cause for pain can be found by doctors.*                              | 0.000           | 0.000   |
| 13. Pain can be reduced by concentrating on other things.*                  | -0.418          | 0.401   |
| 14. Women can tolerate more pain than men.*                                 | 0.000           | 0.000   |
| 15. Thinking about pain makes it worse.                                     | 0.000           | 0.667   |
| 16. Pain can be dealt with by ignoring it.*                                 | -0.401          | 0.000   |
| 17. When injured one feels pain.*   | 0.425           | 0.000   |
| 18. It is impossible to control pain on your own.                           | 0.484           | 0.000   |
| 19. Pain is a sign of illness.  | 0.426           | 0.000   |
| 20. Feeling depressed makes pain seem worse.                                | 0.000           | 0.616   |

Factor loadings of less than 0.40 have been replaced by zeros.

\* These items were excluded from the final solution.

and a significant interaction ( $F(1,236)=54.03, p<0.0001$ ). Since there was a large difference in age between the two groups this variable was entered into the above analysis as a covariate. Its effect was to render the main effect of group insignificant,



but did not alter the main effect of scale or the interaction term, both of which remained significant at the  $p < 0.0001$  level. The adjusted means did not differ from the original means by more than 0.08 in any instance. The means and standard deviations are shown in Table 4.2, and unadjusted means are presented graphically in Figure 4.1.

It can be seen that chronic pain patients are more likely than controls to endorse items on the 'organic beliefs' scale, but less likely to show agreement with items on the 'psychological beliefs' scale. This provides supportive evidence for the validity of the scales.

Although chronic pain patients show mean scores of around the midpoint on both scales, the greatest proportion of these individuals selecting the same response for any one item was only 47%, not sufficient to cause concern over the discriminability of the scales.

Table 4.2 Unadjusted and adjusted (using age as covariate) means (SD) for the organic and psychological scales of the PBQ, in chronic pain patients and controls.

|                            | Chronic Pain Patients (n=93) | Controls (n=145) |
|----------------------------|------------------------------|------------------|
| <b>Organic scale</b>       |                              |                  |
| Mean <sup>a</sup> (sd)     | 4.012 (0.71)                 | 3.420 (0.42)     |
| Mean <sup>b</sup>          | 3.947                        | 3.438            |
| <b>Psychological scale</b> |                              |                  |
| Mean <sup>a</sup> (sd)     | 4.013 (1.00)                 | 4.284 (0.69)     |
| Mean <sup>b</sup>          | 3.968                        | 4.310            |

<sup>a</sup> unadjusted    <sup>b</sup> adjusted

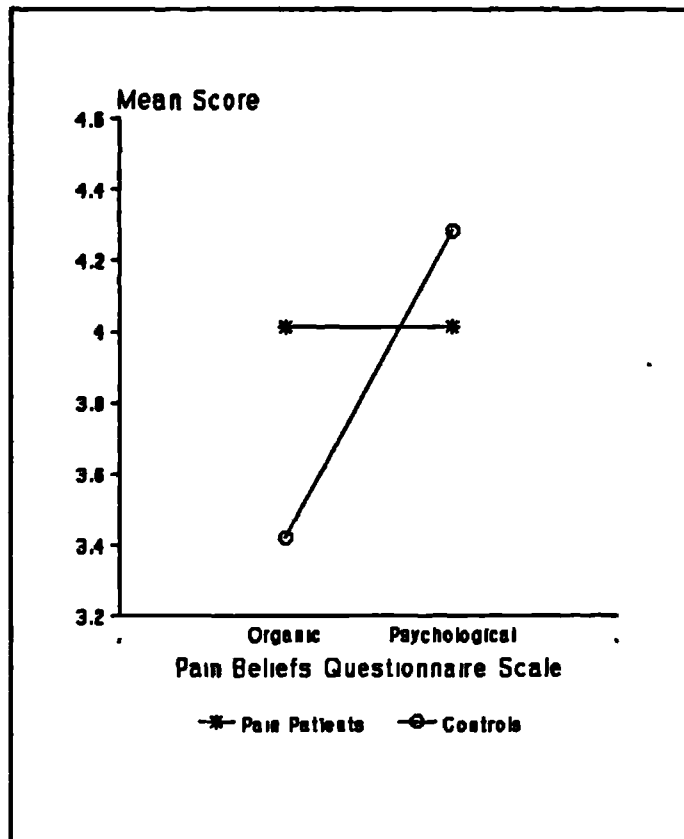


Figure 4.1 Mean scores (adjusted for number of items per scale) for the organic and psychological scales of the PBQ.

## Phase 2: Criterion Related Validity

### METHOD

#### DESIGN

The 12-item Pain Beliefs Questionnaire (PBQ) was administered to a separate group of 40 pain patients, along with the MHLC and a measure of current pain intensity.

## MEASURES

Patients completed the PBQ, as described in the development section of this paper, the MHLIC, and indicated the duration of their pain and their current pain intensity on a 0 - 100mm Visual Analogue Scale (VAS).

## SUBJECTS

Forty patients attending Rheumatology Out-Patient Clinics at the Whittington Hospital were recruited for this part of the study. Since pain chronicity was a variable of interest, no restriction was placed on the length of time pain had been experienced. The mean age of the subjects was 51.0 years (sd=14.19); 75% were female, 57.9% married, 18.4% single and 23.7% divorced, separated or widowed. The mean duration of pain was 99.6 months (SD=105.08, range 1 -444 months). The VAS yielded a mean current pain intensity of 48.38 (SD=31.0, range 0 - 100).

## PROCEDURE

Patients waiting for routine out-patient appointments in the Department of Rheumatology at the Whittington Hospital were invited to take part in an investigation into the experience of pain. After giving consent patients completed the questionnaires in their own time, although an experimenter was always available to answer any questions that arose. Completion of both questionnaires, and collection of the other information took around 10 minutes.

## RESULTS

Pearson product-moment correlations were computed between the PBQ and all other measures, and are presented in Table 4.3. Results provide support for the construct validity of the PBQ. As anticipated, scores on both the Chance and Powerful Others scales of the MHLC correlate significantly with scores on the PBQ organic beliefs scale. Also, scores on the internal MHLC scale and the psychological beliefs PBQ scale correlate significantly. Correlations between the two PBQ scales and each of the other measures did not reach significance.

Table 4.3 Correlations between the Pain Beliefs Questionnaire scales and all other measures.

|                         | PBQ Organic Scale | PBQ Psychological Scale |
|-------------------------|-------------------|-------------------------|
| MHLC-Internal           | 0.0013            | 0.3869*                 |
| MHLC-Chance             | 0.4316**          | 0.1310                  |
| MHLC-Powerful Others    | 0.4037**          | 0.1420                  |
| Duration of pain (mths) | 0.2338            | 0.1635                  |
| Current pain intensity  | -0.3032           | -0.0510                 |

MHLC: Multidimensional Health Locus of Control

\* p<0.05

\*\* p<0.01

## DISCUSSION

Analysis of the results suggest that the PBQ is a reliable and valid measure of beliefs about the cause and consequence of pain. It identifies two clear, discrete classes of beliefs about pain. The first encompasses beliefs about the importance of organic factors in the experience of pain, and the logical sequelae of this position. The other concerns the personal, psychological factors that influence the experience of pain. The choice of labels for the two scales requires some clarification, since the intention is not to promote the concept of pain as readily divisible into organic and psychogenic. However, after much deliberation these labels were adopted because they appeared to reflect most accurately the way in which the lay population in our sample, including chronic pain sufferers, viewed pain. The emphasis needs to be on the beliefs held by these individuals, whether they are correct, or adaptive or not.

The PBQ appears to have adequate validity and reliability. However, it is possible that, along with all self-report measures of this type, there are some biases in the results. For example, chronic pain patients may place psychological investment in reporting an organic cause for their condition, while de-emphasizing psychological factors. However this is thought unlikely in this case, and that the responses reflect the patients' true beliefs, since varying degrees of endorsement of the beliefs were possible, and the statements did not overtly suggest an organic/psychological dichotomy of aetiologies. Indeed, the psychological scale in particular assesses the extent to which people believe psychological factors can influence pain, rather than cause it *per se*.

In the group of chronic pain patients tested, beliefs concerning the organic

component of pain were significantly associated with the belief that other people with power (usually doctors), and chance or fate control health status, inferring a sense of dissociation between the experience of pain and the individual themselves. In contrast, belief that psychological factors may play a role was significantly associated with the belief that the individual has control over their own health and well-being. Since perceived personal control over pain (ie. internal locus of control) has been associated with positive adaptation to chronic pain, it follows that those patients who recognise that psychological factors can influence pain experience may show greater use of coping strategies. It might also be anticipated that the PBQ scales are related to activity levels, measures of mood and pain intensity. In this study pain beliefs were not associated with the intensity of pain, although the correlation approached significance, suggesting that such beliefs may have an enduring quality, more strongly related to personality than current physical state. Thus it is likely that pain beliefs are more closely associated with adjustment to pain than intensity per se, and further studies are needed to address this issue.

Of particular interest is the finding that chronic pain patients and non-patient controls differ in their beliefs about pain. Chronic pain patients place greater emphasis than controls on the organic aspects of pain, whereas non pain-patients are more likely to believe that psychological factors play a role in pain experience. The explanation for this difference requires investigation. It is possible that changes in beliefs occur as a consequence of the long-term experience of pain. Alternatively it may be the case that holding certain beliefs about pain predisposes an individual to becoming a long-term pain patient. In this study no conclusion regarding this issue can be drawn since although no association was found between the duration of pain

and the nature of the patients beliefs, relatively few patients in the sample had experienced pain for less than six months. Hence the hypothesis that changes in beliefs occur over time is not adequately tested here.

The results of this study have wide implications. On a clinical level, despite the frequent misconceptions held by patients about pain, it would be unwise to attempt to alter beliefs in organic aetiology to beliefs in a purely psychological one. This position is obviously as incorrect as the premise that all pain is organic in origin. As Watts (1983) points out, patients often have a "crude dichotomy of aetiologies, believing that they must either have genuine symptoms with an organic aetiology, or that they have psychological symptoms that are simply 'all in the mind'". The results of this study highlight the fact that the beliefs held by chronic pain patients differ from those of non-patients. Furthermore, this difference is in a direction which may be maladaptive, insofar as denial that psychological factors can influence pain may prevent optimal adjustment and effective use of coping strategies. This points to the need for the education of chronic pain patients, to explain, for example how stress can interact with biological processes to produce heightened pain sensation.

The beliefs held by a patient suffering pain are also likely to influence the way in which they present their problem to health professionals, along with the way in which they respond to both conventional physical, psychological and multidisciplinary approaches to the management of pain. Leventhal, Meyer and Nerenz (1980) present a model for the "common sense representation of illness danger" in which they emphasize the role of beliefs in forming an organized system or "theory" which helps the patient interpret and explain their illness. Three sources of information are proposed to shape the belief system: bodily experience,

information from the external social environment (health professionals, family, media etc.) and information based on past experience of illness. They also suggest that where the beliefs held by the patient conflict with those of the medical practitioners, compliance is diminished.

In the next chapter the impact of surgical and cognitive behavioural interventions for chronic pain conditions on organic and psychological pain beliefs will be investigated.



## *Chapter 5 Investigation of the effects of surgical and cognitive-behavioural interventions for chronic pain on cognitive processes.*

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### INTRODUCTION

Beliefs about pain - what it is, what causes it, what factors affect its severity, and what it means for the individual, all combine to form a patient's conceptualization of pain. In developing an understanding of pain and illness, patients may retain beliefs, or adopt new ones, which in the eyes of health professionals appear incorrect or maladaptive. Becker *et al* (1977) suggested that if beliefs about illness are discordant with the treatment offered, compliance is greatly diminished. Following this work, Williams and Thorn (1988) examined the relationship between beliefs about pain and subjective pain intensity, treatment compliance, psychological distress and attributions concerning health care. The authors assessed beliefs using their questionnaire, the Pain Beliefs and Perceptions Inventory, which has three scales; "pain stability" - beliefs about the duration and continuous/ intermittent nature of pain, "pain as a mystery", and "self blame". The dependent measures used were subjective pain intensity ratings, therapists ratings (physiotherapists, occupational therapists and psychologists) of patient compliance mid-treatment, the Rosenberg Self-Esteem Scale, the MMPI and the Multidimensional Health Locus of Control. Results indicated that beliefs in long endurance of pain, and the belief that pain is mysterious are both associated with lower compliance with physiotherapy. They also

found a tendency for patients who held the belief that pain is enduring to show poorer compliance with health psychology interventions. Beliefs in pain as mysterious were associated with little post-treatment improvement on measures of psychological distress and somatization, and beliefs in pain endurance were associated with low internal locus of control.

Self-efficacy beliefs or expectancies form another subset of an individual's belief system. These beliefs have been postulated by Bandura (1977, 1982) to be associated with both the prediction and maintenance of behaviour. Dolce, Crocker and Doleys (1986) suggest that self-efficacy ratings may provide useful predictors of the maintenance of therapeutic gains in chronic pain patients. Following a four-week multidisciplinary pain management programme patients were found to have significantly higher ratings for the perceived efficacy of exercise, medication-free coping and work in relation to their pain. High post-treatment efficacy beliefs were related to higher follow-up exercise levels, less use of medication and better work status.

In a similar study O'Leary, Schoor, Lorig and Holman (1988) tested the hypothesis that enhancement of perceived self-efficacy to manage rheumatoid arthritis would mediate the magnitude of cognitive-behavioural treatment effects. Thirty female rheumatoid arthritis patients completed the Arthritis Self-Efficacy Scale, which assesses perceived ability to control symptoms of pain, depression and fatigue, along with "Self-Efficacy to Manage Pain" and "Self-Efficacy to Function" scales. These scales, in addition to measures of depression, stress and loneliness were obtained pre and post-treatment. The results suggested that high post-treatment self-efficacy was associated with low pain intensity. Post-treatment perceived efficacy for

physical functioning was related to low post-treatment disability, and similarly, higher post-treatment arthritis efficacy was associated with less depression and stress post-treatment. In this paper changes in perceived efficacy were correlated with changes in outcome measures - a procedure thought to be inadvisable, for reasons outlined in the results section of the current chapter. The only significant finding of this type was that changes (increases) in efficacy beliefs for physical functioning were correlated with changes (decreases) in disability.

Recently Kores *et al* (1990) used a modified self-efficacy scale with 5 categories - walking distance, lifting ability, pain coping, working ability and social and recreational engagement - to examine the relationship of perceived self-efficacy to treatment outcome in a chronic pain population. They found that higher post-treatment self-efficacy scores were related to increased sitting and standing tolerance at follow-up, and that patients with higher self-efficacy scores after treatment had lower pain behaviour levels at follow-up.

Thus it can be seen that beliefs play a role in both predicting outcome of interventions for chronic pain, and also to some extent in understanding the processes involved. However, it appears that self-efficacy beliefs have limited capacity in this respect, given that significant findings are on the whole evident only for measures of physical functioning.

Two groups of patients form the focus of the studies reported in this chapter: patients who have been offered either a surgical or a cognitive-behavioural intervention for their chronic pain condition. Since the choice of treatment is made on a clinical basis (rather than as the result of a double-blind randomized trial basis), no attempt will be made to draw direct, statistical comparisons between the groups;

the studies should be considered 'naturalistic', not experimental. It is anticipated that these two treatment approaches will have contrasting outcomes - the surgery is expected to result in complete pain relief, whereas the cognitive-behavioural approach aims primarily to improve coping, not specifically to reduce pain. The two groups therefore provide an excellent opportunity to examine the impact of different interventions on cognitive processes.

The first aim of the studies reported here is to explore the relationship between organic and psychological beliefs and recovery from surgical and cognitive-behavioural interventions. Outcome measures employed include measures of pain, anxiety, depression, cognitive coping strategies and psychological and physical functioning.

The second aim of the studies reported here is to investigate the influence of interventions for chronic pain on memory processes in chronic pain patients. Research on selective memory in depression has provided mixed evidence regarding memory biases in patients who have recovered from a clinical depression. Zuroff, Colussy and Wielgus (1983) found that recovered clinically depressed patients responded in a manner akin to that of currently depressed patients on a recall memory test ie. they recalled relatively more negative words than normal controls. However, it should be noted that the authors conducted separate analyses on the numbers of positive and negative words recalled, rather than providing the necessary evidence of an interaction between group and wordtype. Indeed, inspection of the means shows that more positive than negative words were recalled by members of all three groups. Therefore caution should be exercised in attaching importance to these results. In a more recent study Bradley and Mathews (1988) investigated

memory bias for negative versus positive adjectives in 11 recovered unipolar depressives, 12 non-psychiatric controls and 9 current depressives. Words were presented in either self or other person referent conditions. Results showed that depressives have a recall bias for negative self-referent adjectives while the recovered depressives and normal controls recalled more positive information in the self-referent condition. Unexpectedly, the recovered depressed group recalled more negative adjectives in the other-person referent condition. The authors interpreted these results as suggesting that self-referent recall bias is a function of both current mood state *and* more enduring cognitive structures, with consequent implications for vulnerability to depressive disorders. Lending support to these results, although with different types of cognitive processes, Dohr, Rush and Bernstein (1989) found that remitted depressives and normal controls did not differ in their attributional biases, endorsement of dysfunctional attitudes, or interpretation of schema-relevant ambiguous events, although both of these groups differed significantly from currently depressed patients.

In the first of the two studies reported here, a group of patients undergo surgery for a chronic pain condition, an operation which results in a high probability of the patient being pain-free within a few weeks post-surgery. This group may therefore form a "recovered chronic pain group", analogous to the remitted/recovered depressives. It is predicted that these patients will recall more pain-related than non pain-related words prior to surgery (ie. a pain-related memory bias), but will exhibit the opposite pattern of recall when pain-free, post surgery.

The other study in this chapter explores the influence of a multidisciplinary cognitive-behavioural pain management programme on pain-related memory biases.

Evidence provided by Watts, Trezise and Sharrock (1986) suggests that a behavioural treatment for spider phobia (desensitization) leads to changes in recognition memory for large spiders. Recognition memory was found to improve after desensitization, but also improved in the no-treatment control group, although to a lesser extent. Thus practise effects are likely to have played a significant role in these results, since the same spiders were used on both occasions of testing. As a result of the treatment, patients were also found to be less vigilant towards, and less preoccupied with spiders, and showed less use of avoidance coping strategies. This suggests that cognitive processes which are condition-related (ie. to depression or anxiety) may be altered by psychological interventions.

However, it would be unwise to draw comparisons between this result and possible changes in processing in chronic pain patients. Chronic pain patients appear to show biases in processing which bear greater resemblance to those in depression than in anxiety - memory rather than attention biases. Also, recall, as opposed to recognition memory biases are the focus of interest, and findings clearly differ in these domains. In the present study, the cognitive-behavioural programme aims primarily to increase coping, rather than reduce pain intensity per se. Thus in contrast to the spider phobics, whose treatment resulted in reduction in symptoms of their phobia, the chronic pain patients are unlikely to experience substantial relief from their physical pain symptoms. Therefore, if the memory bias is strictly a result of pain sensations, no change in patterns of recall would be anticipated. If, on the other hand, the bias is the result of being a "pain patient", with the related cognitions, emotions, and sensations, it would be predicted that after treatment, the bias would remain.

## **STUDY 1 Effect of surgical intervention on cognitive processes in chronic pain.**

### **METHOD**

#### **DESIGN**

A sample of chronic pain patients undergoing surgical intervention were interviewed on three occasions - the day before their operation, and around 2 months and 6 months post-operatively. At these times the patients completed a recall memory task, the Pain Beliefs Questionnaire (PBQ; Appendix A) and other mood and pain-related measures.

#### **MEASURES**

The recall memory test on each occasion comprised 4 lists of words, each containing 2 sensory and 2 affective adjectives from the McGill Pain Questionnaire (Melzack, 1975), 2 neutral words and 2 gardening words. The gardening words were included since they belong to a common semantic category, to provide a control for the pain-related adjectives, which also belong to a semantic category. (As noted in Chapter 3, words which are related in this way are remembered better than those between which there is no connection). Items were matched as closely as possible for frequency and length. They were presented aurally at a rate of one word per 2 seconds, (ie. ISI onset to onset = 2 secs), in fixed random order, with three fillers at the beginning and end of each list. Different lists were used at pre-treatment, 2 months, and 6 months post-treatment (thus there were a total of 12 matched lists). The order of the blocks of 4 lists was systematically varied across subjects. The sensory, affective, neutral and gardening words are presented in Table 5.1. In

addition to the PBQ, the following questionnaires were administered: the Beck Depression Inventory (BDI), the Spielberger State and Trait Anxiety Inventory (STAI), and the 28-item General Health Questionnaire (GHQ-28; Goldberg, 1978), which has 4 scales - somatic symptoms, anxiety and insomnia, social dysfunction and severe depression. Current and average weekly pain intensity ratings were obtained using 0 - 100mm visual analogue scales (VAS).

Table 5.1 Sensory, affective, neutral and gardening words used in the recall task.

| Sensory        | Affective     | Neutral    | Gardening   |
|----------------|---------------|------------|-------------|
| <b>Block 1</b> |               |            |             |
| pounding       | intense       | educated   | growing     |
| tingling       | fearful       | selective  | shovelling  |
| tugging        | cruel         | promising  | seedling    |
| itchy          | vicious       | nimble     | leafy       |
| pinching       | terrifying    | informal   | budding     |
| splitting      | sickening     | protruding | grassy      |
| throbbing      | punishing     | resounding | weeding     |
| pricking       | suffocating   | reputable  | bedding     |
| <b>Block 2</b> |               |            |             |
| stabbing       | tiring        | stony      | watering    |
| boring         | horrible      | flexible   | sowing      |
| searing        | annoying      | leaking    | pruning     |
| crushing       | distressing   | angular    | planting    |
| shooting       | troublesome   | prime      | wilting     |
| wrenching      | discomforting | imprecise  | fertilizing |
| cutting        | blinding      | spreading  | fencing     |
| aching         | excruciating  | transient  | spraying    |
| <b>Block 3</b> |               |            |             |
| scalding       | mild          | legal      | annual      |
| pressing       | unbearable    | polished   | potting     |
| tender         | gruelling     | amazing    | clipping    |
| flashing       | miserable     | windswept  | digging     |
| beating        | killing       | grand      | reaping     |
| gnawing        | frightful     | youthful   | flowering   |
| hurting        | wretched      | knotty     | mowing      |
| drilling       | exhausting    | swaying    | blooming    |



## SUBJECTS

Women undergoing hysterectomy and oophorectomy for pelvic venous congestion at the Samaritan Hospital, London were invited to participate in a study on the effects of surgery on psychological functioning. Twenty-four were recruited for the study: of these, 20 were interviewed at two months, and 12 at six months post surgery. The primary reason for missing data was non-attendance at out-patient follow-up appointments, largely because those patients who were pain-free had no reason to attend. The measures were not mailed to patients since the memory test was not amenable to self-administration.

The mean age of the patients was 35.958 (sd=6.16), their mean duration of pain prior to surgery was 106.7 months (sd=78.30), and their mean current and average (weekly) pain intensity ratings pre-treatment were 55.88 (sd=30.54) and 72.29 (sd=16.28) respectively.

## PROCEDURE

Patients were first interviewed on the ward, on the day prior to surgery (in a room allowing privacy). After obtaining informed consent to participation in the investigation, subjects first completed the recall memory task, followed by all the questionnaire and VAS measures. At 2 and 6 months post-operatively patients were interviewed at routine out-patient appointments, before their consultation with the doctor. Again, the recall test was administered, followed by all other measures, in the same order as previously.

## RESULTS

### A. Memory

The mean number of words recalled in each category at the three times of testing were calculated. This data was subjected to 2-way, repeated measures ANOVAs, with "wordtype" and "time" as the within groups variables. Greenhouse-Geisser adjusted probabilities are reported wherever appropriate. First, a comparison between the number of neutral and gardening words recalled on the three occasions revealed no significant main effects, nor a significant interaction. Therefore these two categories were combined to produce a "non pain-related" word category. The number of sensory and affective words recalled were also combined, forming a "pain-related" category. It will be remembered that the results of an earlier experiment suggested that a memory bias for affective adjectives may only be present if the chronic pain patient is also depressed. Since it was not possible to anticipate the depression levels in this group, both sensory and affective words were included in the lists. Insufficient numbers of patients were tested to legitimately compare depressed with non-depressed groups, and therefore the most sensible option appeared to be to combine these two wordtype categories.

A comparison of recall of pain-related versus non pain-related words over the three occasions of testing showed no significant main effects or interaction (means and standard deviations are shown in Table 5.2). However, a trend towards a decrease in the number of pain-related words recalled coupled with an increase in the number of non pain-related words recalled appears to be emerging (Figure 5.1). An ANOVA performed on the data from pre-treatment and 6 months post-treatment

alone revealed that although there were still no main effects, the interaction approached significance,  $F(1,11)=3.61$ ,  $p=0.0840$ .

Table 5.2 Mean (SD) number of pain-related and non pain-related words recalled before surgery, 2 months post-surgery and 6 months post-surgery (n=11).

|                  | Pre-surgery  | 2 mths post-surgery | 6 mths post-surgery |
|------------------|--------------|---------------------|---------------------|
| Pain-related     | 4.364 (1.91) | 3.727 (1.95)        | 3.545 (2.30)        |
| Non pain-related | 3.636 (2.34) | 3.818 (3.28)        | 4.000 (2.68)        |

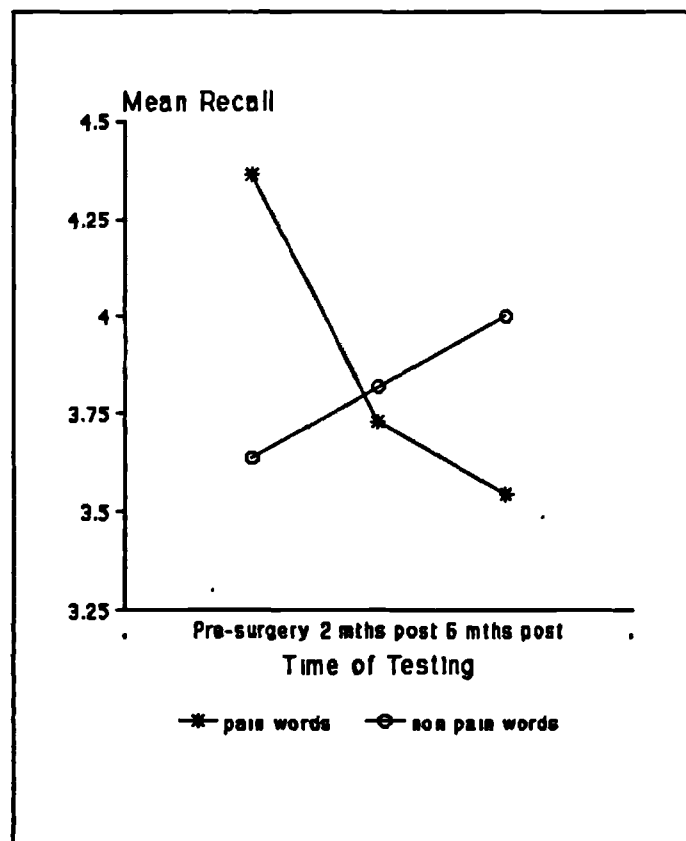


Figure 5.1 Mean recall of pain-related and non pain related words before surgery, 2 months post-surgery and 6 months post-surgery. (n=11)

## B. Pain Beliefs Questionnaire

Mean scores on the organic and psychological scales of the PBQ were obtained and subjected to 2-way repeated measures ANOVAs with time of testing (pre-surgery, 2 months and 6 months post-surgery) and scale as the within groups variables. This revealed non-significant main effects (time of testing,  $F(2,22)=0.66$ ,  $p=0.5178$ ; scale,  $F(1,11)=0.21$ ,  $p=0.6533$ ), but a significant interaction,  $F(2,22)=9.18$ ,  $p=0.0017$ . The nature of this interaction is clearly demonstrated graphically in Figure 5.2 (the means and standard deviations are presented in Table 5.3). Prior to treatment patients show greater agreement with organic than psychological beliefs, whereas at 2 months post-treatment the reverse pattern is evident. This change is maintained at 6 months post-treatment. The graph suggests that the change in beliefs occurred between pre-treatment testing and 2 months post-treatment, and this was confirmed by a highly significant interaction between time of testing (pre-treatment vs 2 months post-treatment) and scale,  $F(1,19)=25.30$ ,  $p=0.0001$ , but not between time of testing (2 months versus 6 months post-treatment) and scale,  $F(1,11)=0.05$ ,  $p=0.8235$ .

Table 5.3 Mean scores (SD) on the organic and psychological scales of the Pain Beliefs Questionnaire, at the three times of testing (n=12).

|                     | Pre-surgery  | 2 mths post-surgery | 6 mths post-surgery |
|---------------------|--------------|---------------------|---------------------|
| Organic Scale       | 3.708 (0.54) | 3.323 (0.34)        | 3.479 (0.48)        |
| Psychological Scale | 3.271 (0.73) | 3.729 (0.99)        | 3.833 (0.67)        |

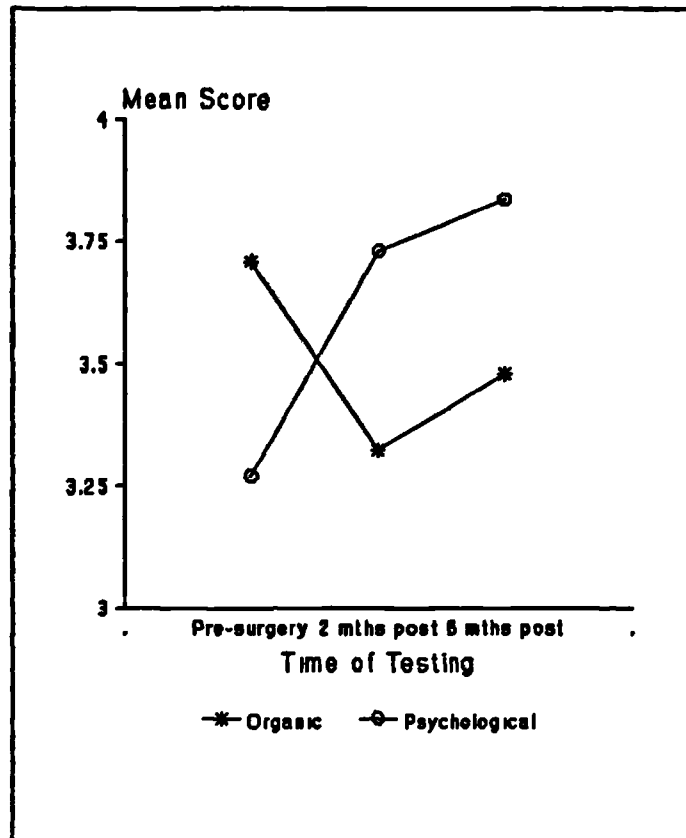


Figure 5.2 Mean scores on the Pain Beliefs Questionnaire pre-surgery, 2 months post-surgery and 6 months post surgery. (n=12)

### C. Relationship between beliefs and outcome

Table 5.4 shows the mean scores and standard deviations on the BDI, Spielberger STAI, GHQ scales and VAS pain intensity ratings at pre-treatment and first follow-up, along with the associated 1-tailed probabilities (matched t-test). None of these measures changed significantly between first and second follow-up, and 6 month post-treatment data is therefore not presented. Patients showed significant reduction in scores on the BDI, Spielberger State and Trait anxiety scales, the somatic symptoms and anxiety & insomnia scales of the GHQ and in both current and

average weekly pain intensity ratings.

Pearson product-moment correlations were calculated between scores on the organic and psychological scales of the PBQ prior to surgery and all other measures at 2 and 6 months post-surgery. These correlations are shown in Table 5.5.

It can be seen that strong organic beliefs prior to treatment correlate with high current pain intensity at 6 months post-treatment. High endorsement of questions on the psychological beliefs scale pre-treatment correlates with high trait anxiety, high levels of somatic symptoms and high levels of social dysfunction at 2 months post-treatment. These beliefs also correlate significantly with greater depression (BDI), state and trait anxiety, somatic symptoms and severe depression (GHQ) at 6 months post-treatment.

Table 5.4 Means (SDs) on the BDI, Spielberger, GHQ and pain intensity ratings.

|                      | Pre-surgery   | 2 mths post-surgery | p (1-tail) |
|----------------------|---------------|---------------------|------------|
| BDI                  | 14.60 (3.39)  | 6.20 (6.49)         | 0.0003     |
| Spielberger          |               |                     |            |
| State anxiety        | 49.60 (11.71) | 36.10 (11.52)       | 0.0000     |
| Trait anxiety        | 44.56 (7.05)  | 37.92 (7.95)        | 0.0001     |
| GHQ-28               |               |                     |            |
| Somatic symptoms     | 9.55 (4.45)   | 4.25 (4.22)         | 0.0014     |
| Anxiety & insomnia   | 9.75 (4.48)   | 4.40 (4.54)         | 0.0005     |
| Social dysfunction   | 10.15 (3.62)  | 7.85 (4.21)         | 0.0535     |
| Severe depression    | 3.65 (4.79)   | 1.65 (4.49)         | 0.0000     |
| Pain intensity (VAS) |               |                     |            |
| Current              | 56.65 (32.25) | 7.65 (18.94)        | 0.0000     |
| Average              | 73.25 (15.66) | 17.40 (25.62)       | 0.0000     |

**Table 5.5 Pearson product-moment correlations between pre-surgery scores on the Pain Beliefs Questionnaire scales and other measures at 2 months and 6 months post surgery. NB. Degrees of freedom vary.**

|                      | Organic             | Beliefs             | Psychological       | Beliefs             |
|----------------------|---------------------|---------------------|---------------------|---------------------|
|                      | 2 mths post-surgery | 6 mths post-surgery | 2 mths post-surgery | 6 mths post-surgery |
| BDI                  | 0.2211              | -0.1990             | 0.3086              | 0.5615*             |
| Spielberger          |                     |                     |                     |                     |
| State                | 0.3323              | -0.1577             | 0.3065              | 0.6395*             |
| Trait                | -0.0477             | 0.0224              | 0.4806*             | 0.5840*             |
| GHQ-28               |                     |                     |                     |                     |
| Somatic symptoms     | 0.2653              | -0.2292             | 0.4169*             | 0.5786*             |
| Anxiety & insomnia   | 0.2484              | -0.1066             | 0.2409              | 0.4688              |
| Social dysfunction   | -0.1292             | -0.1252             | 0.6798***           | 0.2555              |
| Severe Depression    | 0.2254              | -0.1290             | 0.0677              | 0.7623**            |
| Pain Intensity (VAS) |                     |                     |                     |                     |
| Current              | 0.1784              | 0.7363**            | -0.1418             | -0.1677             |
| Average              | -0.0878             | 0.3436              | 0.0073              | -0.0506             |

\*  $p < 0.05$     \*\*  $p < 0.01$     \*\*\*  $p < 0.005$

#### **D. Causal relationship between beliefs and other measures**

It is clear from section B above that the beliefs about pain which are assessed by the Pain Beliefs Questionnaire change between pre-surgery and 2 months post-surgery assessments. The most obvious way of examining the relationship between alterations in beliefs and improvements in the outcome measures would be to simply correlate change in beliefs with change in other measures, ie. correlate difference scores with difference scores. However, Plewis (1985) states that use of this method is inadvisable for three primary reasons. Firstly, it may be unreasonable to assume that a difference in scores on the same test on two or more occasions is valid, given the possibility of repeated measurement bias. Secondly, for many variables,

particularly questionnaire measures, the scale used is arbitrary and differs across tests, making difference scores uninterpretable. Thirdly, social science variables are generally subject to a significant degree of measure error, resulting in difference scores which are inherently unreliable.

An alternative approach, recommended by Plewis, allows the direction of causality between two variables to be determined if a population is studied on two or more occasions; if measures are obtained on a single occasion, only simple correlations can be computed, with no possibility of determining direction of causality. It is this method which will be adopted in both the present study and the following one. The procedure is to compute stepwise hierarchical regression analyses. For example, for two variables, A and B, measured on two occasions,  $A_1$ ,  $A_2$  and  $B_1$ ,  $B_2$ , A causes B if  $A_1$  predicts  $B_2$  after  $B_1$  is taken into account, and B causes A if  $B_1$  predicts  $A_2$  after  $A_1$  is taken into account. As Mann and McManus (1991) note, it is quite feasible for both directions of causality to co-exist, reflecting a "vicious circle" effect. In the present studies investigating the causal relationship between beliefs and for example depression, scores on an "outcome" measure eg. BDI pre-treatment are entered into the analysis as the first step, with pre-treatment scores on a beliefs scale as the second step (independent variables). The dependent variable is the outcome measure post-treatment. To conclude that beliefs, as measured by a particular scale, are causally related to, for example depression, requires a significant improvement in "fit" between the first and second steps of the analysis. In other words, the addition of the second independent variable must account for a significantly greater proportion of the variance than the first independent variable alone. If evidence for a causal relationship is found, it can be inferred that if a change in one variable occurs, this



will cause a change in the other variable.

Using this approach, with mean substitutions for missing data, it was found that psychological beliefs are causally related to social dysfunction (GHQ);  $F(2,17)=8.49$ ,  $p<0.01$ , state anxiety;  $F(2,17)=4.91$ ,  $p<0.025$ , and trait anxiety;  $F(2,17)=7.20$ ,  $p<0.01$ . Causal relationships were also found in the opposite direction for these variables, eg. social dysfunction, and state and trait anxiety are causally related to psychological beliefs,  $F(2,17)=10.45$ ,  $p<0.01$ ;  $F(2,17)=9.23$ ,  $p<0.01$  and  $F(2,17)=9.16$ ,  $p<0.01$  respectively.

## **STUDY 2 Effect of a cognitive-behavioural intervention on cognitive processes in chronic pain.**

### **METHOD**

#### **DESIGN**

A sample of chronic pain patients undergoing a cognitive-behavioural management programme were interviewed on three occasions - prior to the intervention, 8 weeks later at the end of the intervention, and 4 months post-intervention (ie. 6 months after the baseline measures). At each of these times patients completed a recall memory test, the Pain Beliefs Questionnaire and other mood and pain-related measures.

## **MEASURES**

The recall lists and their presentation were identical to those employed in the previous study. In addition to the Pain Beliefs Questionnaire, the BDI, Spielberger and GHQ-28, which were again administered, the Sickness Impact profile, The Pain Cognitions Questionnaire (PCQ), the Multidimensional Pain Inventory (MPI) and Multidimensional Health Locus of Control (MHLC) were also employed. These additional measures were included since the current study was carried out as part of a larger study on the cognitive-behavioural management of chronic pain. The MPI comprises 13 scales: pain severity, interference, life control, affective distress, support, punishing responses, solicitous responses, distracting responses, household chores, outdoor work, activities away from home, social activities and general activity level (the sum of the previous 4 scales). The SIP has 7 scales providing measures of the extent to which pain affects the following functioning: physical, psychosocial, sleep and rest, recreational pastimes, eating, work and household management. An overall disability measure can also be obtained from the sum of these scales. Given the large number of measures used, and the length of time required to obtain them, the SIP was dropped from the battery for the end of treatment testing, and is therefore used for validation of the Pain Beliefs Questionnaire only.

## **SUBJECTS**

Subjects were chronic pain patients attending an 8-week, out-patient cognitive-behavioural pain management programme at the Whittington Hospital, London. Twenty-five patients were recruited for the study; of these, 24 were assessed at the

end of the programme, and 17 at 4 months post-treatment.

The mean age of the subjects was 48.56 years (sd=11.97), their mean duration of pain prior to the intervention was 121.83 months (sd=103.92), and 72% were female. Their mean current and average VAS pain intensity ratings pre-treatment were 53.96 (sd=20.06) and 64.17 (sd=20.96) respectively. The primary location of pain in this sample was the back (56%).

## PROCEDURE

Patients were first assessed one week prior to the start of the programme, as part of a "preparation" day, during which they were provided with information concerning the structure and content of the course, and individualized goals were obtained for each patient. Subjects completed the memory task first, followed by the questionnaire and VAS measures (a few of the questionnaires had been completed some weeks previously as part of routine assessment for the programme - these were not repeated at this time). On the last day of the programme, and again at a four-month follow-up session, the recall test and other measures were administered.

## RESULTS

### A. Memory

Mean number of correctly recalled words per category were subjected to 2-way repeated measures ANOVAs, with "wordtype" and "time" as the within groups

variables. Greenhouse-Geisser adjusted probabilities are presented where appropriate throughout. As before, the number of neutral and gardening words recalled was compared first - no significant main effects of time or wordtype were revealed, nor a significant interaction, and therefore these two categories were again combined to form a "non pain-related" category". Similarly, the recall of sensory and affective adjectives did not differ, and these too were again combined formed the "pain-related" category. A comparison of recall of pain-related and non-pain related words over the three occasions of testing showed no main effect of time,  $F(2,32)<1$ , or wordtype,  $F(1,16)<1$ , nor an interaction between the two,  $F(2,32)<1$ . Means and standard deviations are shown in Table 5.6, and are presented graphically in Figure 5.3.

Table 5.6 Mean (SD) number of pain-related and non pain-related words recalled prior to a cognitive-behavioural management programme, at the end of the programme, and at 4 months post-treatment. (n=17)

|                  | Pre-treatment | End of treatment | 4 months post-treatment |
|------------------|---------------|------------------|-------------------------|
| Pain-related     | 2.882 (1.45)  | 2.941 (2.14)     | 2.824 (2.24)            |
| Non pain-related | 2.529 (1.59)  | 2.529 (1.46)     | 2.412 (1.77)            |

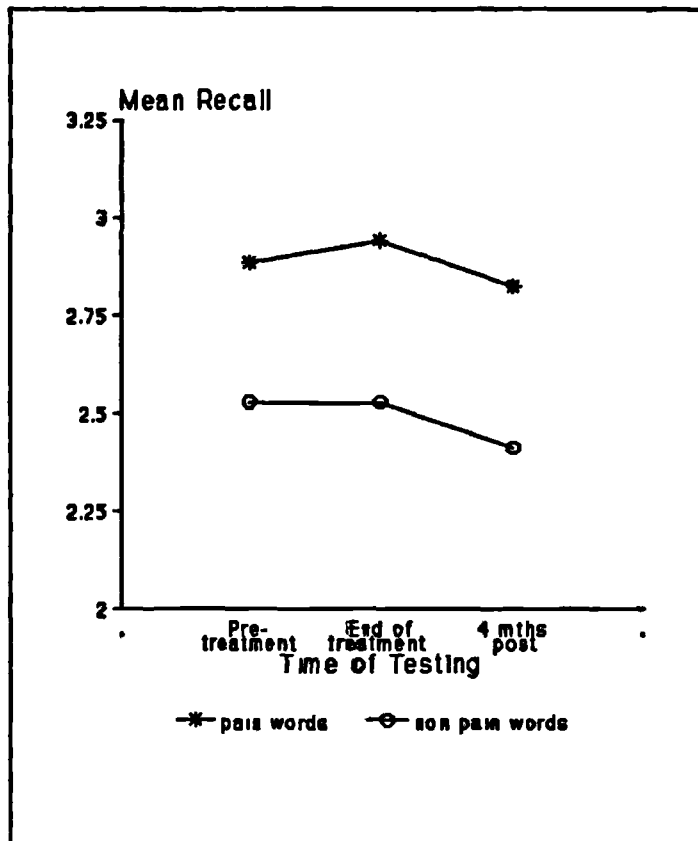


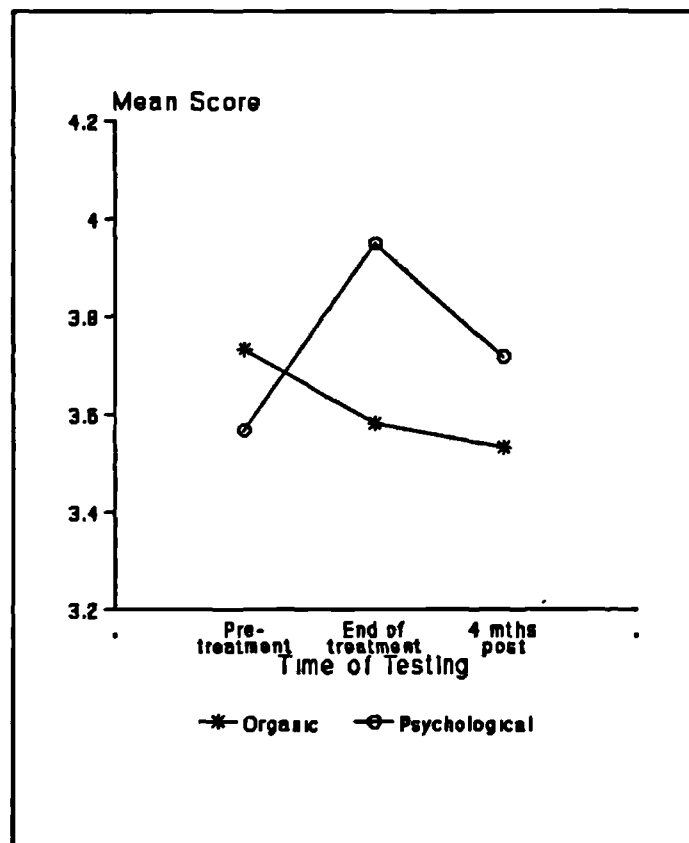
Figure 5.3 Mean recall of pain-related words and non pain-related words pre-treatment, at the end of treatment and 4 months post-treatment (n=17) (cognitive-behavioural pain management)

## B. Pain Beliefs Questionnaire

Repeated measures ANOVAs were performed on the PBQ, with time of testing (pre-treatment, end of treatment and 4 months post-treatment) and scales (organic versus psychological) as the within groups variables. Again, neither main effect was significant: time of testing,  $F(2,28)=2.10$ ,  $p=0.1413$ ; scale,  $F(1,14)<1$ . However, the analysis revealed an interaction which is just significant,  $F(2,28)=3.34$ ,  $p=0.0568$ . The nature of this interaction is depicted graphically in Figure 5.4, with the corresponding

**Table 5.7** Mean scores (SD) on the organic and psychological scales of the Pain Beliefs Questionnaire, at the three times of testing (n=15).

|                     | Pre-treatment | End of treatment | 4 months post-treatment |
|---------------------|---------------|------------------|-------------------------|
| Organic Scale       | 3.733 (0.62)  | 3.583 (0.64)     | 3.533 (0.71)            |
| Psychological Scale | 3.467 (0.89)  | 3.950 (0.85)     | 3.717 (0.91)            |



**Figure 5.4** Mean scores on the Pain Beliefs Questionnaire pre-treatment, at the end of treatment and 4 months post treatment (cognitive-behavioural management (n=15)).

means and standard deviations presented in Table 5.7. It can be seen from the graph that the interaction occurs between the pre-treatment and end of treatment times of testing, and this is confirmed statistically,  $F(1,22)=6.44$ ,  $p=0.0188$ .

### **C. Relationship between beliefs and outcome**

Table 5.8 shows the mean scores and standard deviations on all measures (except memory and pain beliefs), pre-treatment and at the end of treatment, along with their associated 1-tailed probabilities (matched t-test). Significant reductions were observed in depression, trait anxiety, social dysfunction, interference of pain, affective distress, and the extent of perceived support received from the spouse. Patients were found to be engaging in greater levels of outdoor work, activities away from the home and showed an increased general activity level. Significant increases in life control, active positive coping cognitions and support and trust cognitions were found, with significant decreases in hopelessness cognitions. Decreases in pain severity, state anxiety, severe depression (GHQ) and average pain intensity were noted which approached significance.

Pearson product-moment correlations were calculated between scores on the organic and psychological scales of the PBQ prior to the intervention and all other measures at the end of treatment and 4 months post-treatment. These correlations are shown in Table 5.9.

Strong organic beliefs prior to treatment can be seen to correlate significantly with high 'powerful others' locus of control at the end of treatment, along with high 'chance' locus of control, punishing responses from the spouse, greater current pain intensity, and lower severe depression (GHQ) four months after the end of treatment.

**Table 5.8 Means (SDs) on the BDI, Spielberger, GHQ-28, PCQ, MHLC, MPI and pain intensity ratings, pre-treatment and at end of treatment.**

|                             | Pre-treatment        | End of treatment     | p (1-tail)    |
|-----------------------------|----------------------|----------------------|---------------|
| <b>BDI</b>                  | <b>14.440 (6.89)</b> | <b>11.120 (7.06)</b> | <b>0.0386</b> |
| <b>Spielberger</b>          |                      |                      |               |
| State                       | 39.750 (8.84)        | 36.833 (9.85)        | 0.0858        |
| Trait                       | 44.417 (9.32)        | 41.292 (8.94)        | 0.0341        |
| <b>GHQ-28</b>               |                      |                      |               |
| Somatic symptoms            | 7.083 (3.68)         | 6.125 (2.64)         | NS            |
| Anxiety & insomnia          | 6.583 (3.48)         | 6.167 (3.36)         | NS            |
| Social dysfunction          | 8.625 (1.93)         | 6.083 (2.21)         | 0.0000        |
| Severe depression           | 3.500 (4.06)         | 2.250 (3.18)         | 0.0547        |
| <b>Pain Intensity (VAS)</b> |                      |                      |               |
| Current                     | 52.417 (21.65)       | 46.583 (20.09)       | NS            |
| Average                     | 64.667 (20.70)       | 57.583 (21.45)       | 0.0605        |
| <b>PCQ</b>                  |                      |                      |               |
| Active positive coping      | 2.950 (0.60)         | 3.163 (0.58)         | 0.0342        |
| Hopelessness                | 2.015 (0.50)         | 1.727 (0.46)         | 0.0017        |
| Helplessness                | 1.788 (0.56)         | 1.583 (0.42)         | NS            |
| Support & trust             | 2.489 (0.73)         | 2.815 (0.44)         | 0.0304        |
| <b>MHLC</b>                 |                      |                      |               |
| Internal                    | 3.518 (0.91)         | 3.770 (0.90)         | NS            |
| Chance                      | 3.107 (0.66)         | 2.935 (0.77)         | NS            |
| Powerful Others             | 2.936 (1.01)         | 2.630 (0.76)         | NS            |
| <b>MPI</b>                  |                      |                      |               |
| Pain severity               | 4.379 (0.77)         | 3.972 (1.16)         | 0.0539        |
| Interference                | 4.337 (1.15)         | 3.801 (1.22)         | 0.0114        |
| Life control                | 3.473 (1.13)         | 4.073 (1.06)         | 0.0141        |
| Affective distress          | 3.662 (1.16)         | 2.750 (1.01)         | 0.0128        |
| Support                     | 4.517 (1.26)         | 4.042 (1.35)         | 0.0282        |
| Punishing responses         | 1.626 (1.34)         | 1.218 (1.20)         | NS            |
| Solicitous responses        | 3.720 (1.39)         | 3.449 (1.40)         | NS            |
| Distracting responses       | 2.443 (1.31)         | 2.337 (1.45)         | NS            |
| Household chores            | 3.629 (1.86)         | 3.844 (1.90)         | NS            |
| Outdoor work                | 1.450 (1.51)         | 2.180 (1.81)         | 0.0496        |
| Activities away from home   | 2.333 (1.06)         | 2.875 (0.96)         | 0.0010        |
| Social activities           | 2.659 (1.17)         | 2.740 (1.16)         | NS            |
| General activity level      | 2.513 (0.94)         | 2.909 (0.93)         | 0.0047        |



Table 5.9 Pearson product-moment correlations between pre-treatment scores on the Pain Beliefs Questionnaire scales, and other measures at the end of treatment and 4 months post-treatment. NB. Degrees of freedom vary.

|                           | Organic Beliefs  |                       | Psychological Beliefs |                       |
|---------------------------|------------------|-----------------------|-----------------------|-----------------------|
|                           | End of treatment | 4 mths post-treatment | End of treatment      | 4 mths post-treatment |
| BDI                       | -0.3058          | 0.1270                | -0.2983               | 0.0288                |
| Spielberger               |                  |                       |                       |                       |
| State                     | -0.1389          | -0.0870               | -0.1362               | 0.0269                |
| Trait                     | -0.2180          | -0.1589               | -0.1652               | 0.2192                |
| GHQ-28                    |                  |                       |                       |                       |
| Somatic symptoms          | -0.0627          | 0.3761                | -0.2297               | -0.0469               |
| Anxiety & insomnia        | -0.1355          | 0.1431                | -0.0500               | 0.0547                |
| Social dysfunction        | 0.0695           | 0.3892                | -0.0849               | -0.1165               |
| Severe depression         | -0.1322          | 0.2632                | -0.2450               | -0.0675               |
| Pain intensity (VAS)      |                  |                       |                       |                       |
| Current                   | 0.0672           | 0.4902*               | -0.1208               | -0.2941               |
| Average                   | -0.1040          | 0.0909                | -0.1503               | 0.0544                |
| PCQ                       |                  |                       |                       |                       |
| Active positive coping    | -0.0461          | -0.3212               | 0.7322****            | 0.6846***             |
| Hopelessness              | 0.1889           | 0.0913                | -0.1666               | -0.0905               |
| Helplessness              | -0.2098          | -0.0525               | 0.0002                | 0.0640                |
| Support & trust           | -0.0881          | -0.5016**             | 0.4744**              | 0.5086**              |
| MHLC                      |                  |                       |                       |                       |
| Internal                  | -0.1920          | -0.2574               | 0.4146*               | 0.4239*               |
| Chance                    | 0.2630           | 0.7272****            | -0.1146               | -0.1639               |
| Powerful others           | 0.5249****       | 0.3804                | -0.0984               | 0.2594                |
| MPI                       |                  |                       |                       |                       |
| Pain severity             | -0.0480          | 0.3932                | -0.1951               | -0.0364               |
| Interference              | -0.0071          | 0.3039                | -0.1537               | -0.603 ***            |
| Life control              | -0.2146          | -0.6020***            | 0.4805**              | 0.3578                |
| Affective distress        | 0.0004           | 0.3687                | -0.3304               | -0.3747               |
| Support                   | 0.0079           | 0.0846                | -0.3704*              | -0.2579               |
| Punishing responses       | 0.2839           | 0.4347*               | 0.2627                | 0.4502*               |
| Solicitous responses      | -0.0503          | 0.0859                | -0.1319               | -0.0669               |
| Distracting responses     | -0.3221          | -0.0985               | 0.1642                | 0.2188                |
| Household chores          | -0.0123          | -0.1421               | 0.6006***             | 0.6621***             |
| Outdoor work              | -0.1608          | -0.1739               | 0.0949                | 0.3368                |
| Activities away from home | -0.2562          | -0.3696               | 0.5896***             | 0.5711***             |
| Social activities         | -0.1845          | -0.3975               | 0.2679                | 0.6267***             |
| General activity level    | -0.2046          | -0.3221               | 0.5896***             | 0.7110***             |

\* p<0.05 \*\* p<0.025 \*\*\* p<0.01 \*\*\*\* p<0.001

High endorsement of items on the psychological beliefs scale pre-treatment were found to correlate significantly with high active positive coping and support and trust cognitions, strong internal locus of control, high life control, low support from the spouse, and high levels of engagement in household chores, activities away from the home and general activity at the end of treatment. These beliefs before the intervention were also found to be related to the following 4 months after the end of treatment: high levels of active positive coping and support and trust cognitions, internal locus of control, high degree of 'punishing responses' from the spouse, and high household chores, activities away from the home, social activities and general activity levels.

#### **D. Causal relationships between beliefs and other measures**

The same procedure as that employed in the preceding study was used to examine causal relationships between organic and psychological beliefs and other measures. Mean substitutions were again made for all analyses. Where significant relationships are evident in both directions of causality, the results of the "beliefs" → "other measure" direction is presented first. Each analysis has 2 and 21 degrees of freedom. Significant, bi-directional, causal relationships were identified between organic beliefs and internal locus of control  $F=7.76$ ,  $p<0.01$  and  $F=4.30$ ,  $p<0.05$ ; chance locus of control  $F=6.22$ ,  $p<0.01$  and  $F=5.00$ ,  $p<0.05$ ; powerful others locus of control  $F=10.11$ ,  $p<0.001$  and  $F=5.33$ ,  $p<0.05$ ; and hopelessness cognitions  $F=9.27$ ,  $p<0.01$  and  $F=6.18$ ,  $p<0.01$ . Bi-directional causal relationships were found between psychological beliefs and the following: depression  $F=6.71$ ,  $p<0.01$  and  $F=8.07$ ,  $p<0.01$ ; active positive coping cognitions  $F=20.11$ ,  $p<0.0001$  and  $F=29.57$ ,

$p < 0.0001$ ; support and trust cognitions  $F = 6.32$ ,  $p < 0.01$  and  $F = 14.22$ ,  $p < 0.001$ ; life control  $F = 6.04$ ,  $p < 0.01$  and  $F = 15.13$ ,  $p < 0.001$ ; household chores  $F = 93.01$ ,  $p = 0.000$  and  $F = 14.14$ ,  $p < 0.001$ ; activities away from home  $F = 16.42$ ,  $p < 0.001$  and  $F = 26.49$ ,  $p < 0.0001$ ; and general activity level  $F = 18.54$ ,  $p < 0.0001$  and  $F = 17.50$ ,  $p < 0.0001$ . These relationships are summarized in Figure 5.5.

## DISCUSSION

There are four main areas to be discussed as a result of these two studies:

- 1) the impact of interventions for chronic pain on patients' beliefs,
- 2) the impact of interventions on memory processes,
- 3) the utility of beliefs in predicting outcome of interventions,
- 4) the role of beliefs in understanding the processes of change which occurred as a result of intervention.

Comment will also be made on the additional evidence for the validity of the Pain Beliefs Questionnaire.

The results of the studies suggest that both physical (ie. surgical), and psychological <sup>treatments</sup> (ie. cognitive-behavioural management) can have a <sup>profound</sup> effect on the beliefs about pain held by patients. As a consequence of both forms of treatment, endorsement of the organic type beliefs decreased, while agreement with psychological beliefs increased. This interaction was less pronounced in the cognitive-behavioural group. The reason for this may have been in part due to the extensive preparation for the programme received by the patients, which included

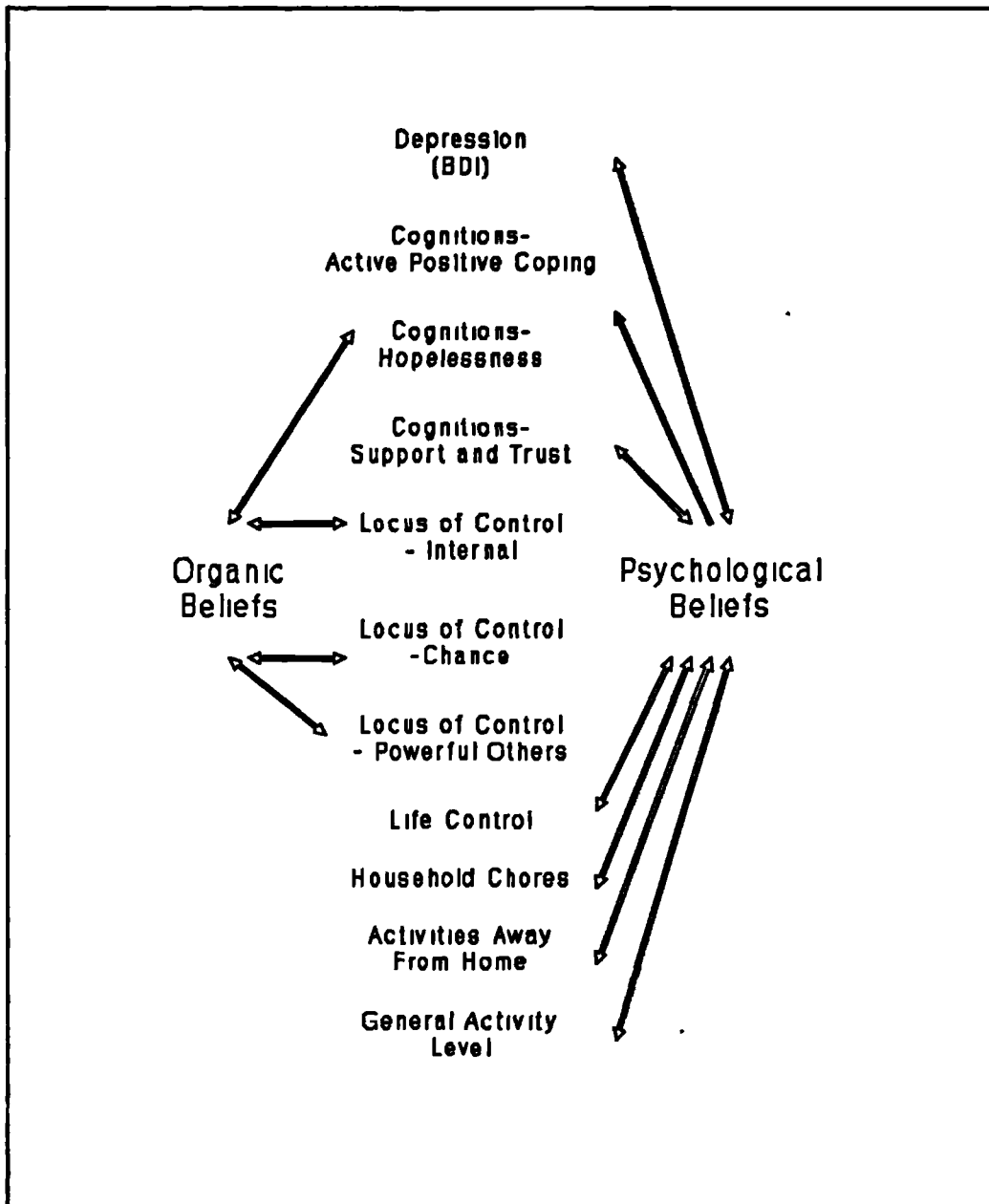


Figure 5.5 Causal relationships between organic and psychological beliefs and other measures. Unfilled arrows represent bi-directional relationships; filled arrow represents causality in one direction only - that indicated.

information about the content and emphasis (ie. psychological) of the course, which may have influenced beliefs prior to the baseline assessment of beliefs. Also, patients were selected for the programme partly on the basis of their ability to accept that psychological factors play a role in pain. Thus the 'room for movement' in terms of altering beliefs may have been substantially reduced. Having said this, significant changes in beliefs were observed, probably as a direct result of the intervention. However, it was not possible to determine whether beliefs may change spontaneously, as a control group (no intervention) was not employed, but this is thought unlikely. Major components of the programme were education (including the Gate Control Theory), skills training (cognitive coping strategies such as attention diversion), and graded physical exercises, all of which are likely to influence beliefs about pain.

The change in beliefs exhibited by the surgical intervention group is perhaps more puzzling. This group received no education about pain, nor any form of treatment other than the surgery itself, yet a dramatic change in both organic and psychological beliefs was observed. The most plausible explanation for this finding is that these patients have an investment in strongly endorsing organic-type beliefs prior to surgery. These beliefs would be entirely compatible with the intervention being offered and undertaken - beliefs at the opposite end of the spectrum would be expected to result in cognitive dissonance, described by Festinger (1957) as the awareness of implicit contradiction anywhere within ones beliefs, preferences or thoughts about behaviour. The theory also proposes that cognitive dissonance of this sort will prompt the individual to change cognitions in order to restore a kind of balance. This type of process may be occurring in these patients at this time. After

surgery, however, when in the majority of cases pain is largely or entirely eliminated, the psychological 'need' for these women to strongly hold organic type beliefs is reduced, and the influence of psychological factors in pain can be acknowledged. Furthermore, those women who did continue to feel pain weeks after the operation when normal healing has occurred, may then feel that organic beliefs must be inaccurate, and therefore place greater emphasis on psychological type beliefs.

The second issue to be discussed concerns the influence of different interventions on memory processes in chronic pain. In the surgical intervention group, on the recall memory task, it was found that prior to surgery subjects recalled more pain-related than non pain-related words, but at six months post-surgery, relatively more non pain-related words were remembered. That the interaction did not quite reach significance, may have been due to the small sample for whom data was available for both these times of testing, however the pattern of results emerging is very clear. The result may also be an indication that changes in schematic processing occur very slowly over time, and that the memory bias in chronic pain is a function of pain per se, rather than a result of some kind of 'pain personality' or vulnerability factor. These results may also be seen to parallel those of Bradley and Mathews (1988) who found evidence that recovered depressives behave in a manner similar to that of normal controls on memory tests comparing recall of positive and negative self-referent adjectives. Thus selective memory in chronic pain may be more related to state than trait factors.

In contrast, there was no change in the number of pain-related and non pain-related words recalled as a consequence of the cognitive-behavioural pain management programme. At each time of testing subjects recalled more pain-related

than non pain-related words, although the difference (main effect of wordtype) was not significant. This implies that there was no memory bias in these patients. This may have been largely due to the fact that there were only 2 sensory and 2 affective pain-related words in each list (through necessity - there are a finite number of suitable words on the McGill Pain Questionnaire), fewer than in previous experiments demonstrating selective memory effects. Alternatively, it is possible that the assessment procedures and preparation for the programme each patient received prior to the intervention may have had a "top-down" influence on schematic processes, altering these processes before the first memory test.

This study provides additional evidence to suggest that selective memory processes in chronic pain are a function of pain itself rather than being a 'pain patient'. However, unlike Watts, Trezise and Sharrock (1986), study 2 failed to provide evidence that psychological intervention influences biased processing. Further experiments are required to clarify this issue, to provide information regarding what type of memory (ie. recall/recognition), in which patient groups, after what type of intervention, such changes in processing are demonstrable.

The results of the present two studies do, however, provide evidence to suggest that selective memory in chronic pain is not found simply because words belonging to a common semantic category ie. pain-related words, are easier to remember than unrelated words - the neutral category in most experiments. In both of the studies reported here there was no difference in the recall of neutral and gardening words.

The organic and psychological scales of the PBQ were found to be predictive of various measures of outcome of both the surgical and cognitive-behavioural treatments for chronic pain. Considerable consistency was found in the measures

related to beliefs at the two and six month times of testing, particularly in the cognitive-behavioural treatment group. However, some of the associations were not in the direction anticipated. Notably, in the surgical intervention group the finding that strong psychological beliefs were associated with high levels of depression, anxiety, somatic symptoms or social dysfunction was not expected. (Interestingly these patterns were not found in the cognitive-behavioural treatment group). In women who have experienced chronic pelvic pain it may be the case that simply *knowing* that psychological factors can influence pain is insufficient to prevent disruption of emotional functioning, given that they may be unable to make use of this information in dealing with their pain. Not all of the patients were pain-free at the follow-up assessments, and therefore the relationships between psychological beliefs and outcome may be a reflection of this subgroup of women. If this is the case, it is less surprising that these beliefs are associated with poor functioning, since the women are not provided with alternative approaches to managing their pain, in the way that patients attending the cognitive-behavioural pain management programme are. Clearly, this requires further investigation, but if supporting evidence is obtained there are important implications for the long-term post-surgical management of pelvic pain patients.

The results of the subjects in the pain management programme suggest that in general, patients who hold strong organic beliefs prior to commencing the programme benefit less from the treatment than those who hold strong psychological beliefs. Strong organic beliefs were related to chance and powerful others locus of control. These patients also perceive that they have little control over their life and their pain, and have little ability to deal with problems or stressful situations. On the other hand,



recognition that psychological factors can play a role in pain is related to internal locus of control, the belief that the individual has personal control over their health, which in turn is typically associated with positive outcome (eg. Rock *et al*, 1987; Toomey *et al*, 1991). These beliefs were also associated with greater use of active positive cognitive coping strategies such as reassuring yourself about your ability to cope with the pain and thinking of ways to distract yourself from the pain, and support and trust cognitions, for example believing the doctor can help, after the intervention. Finally, these beliefs were related to greater participation in a variety of activities.

The role that changes in beliefs play in causing the improvements in measures of emotional and physical functioning found as a result of both types of intervention, was assessed using a statistical technique called 'cross-lagged panel correlations'. In the surgical intervention group reciprocal causal relationships were found between psychological beliefs and depression, state anxiety and trait anxiety. This result carries the inference that increases in endorsement of psychological beliefs found in this group was responsible, at least in part, in causing increases in depression and anxiety. As discussed earlier, this group was not homogeneous in terms of pain intensity levels at the follow-up assessments, with important implications for psychological functioning.

Similarly, the results of the group of patients who attended the pain management programme suggest that as beliefs become less organic, patients perceive that they have greater personal control over their health, while at the same time believing less that fate and significant other people such as health professionals control their health. Decreases in organic beliefs were also implicated as playing a causal role in

decreasing the extent to which patients report having negative, 'hopelessness' thoughts when experiencing pain. Increases in psychological beliefs were found to influence depression, as well as increasing the use of cognitive positive coping strategies and support and trust cognitions. In addition evidence was found to suggest that these beliefs were causally involved in increasing activity levels.

## *Chapter 6 Further investigation of schematic processing*

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### INTRODUCTION

In the attempt to investigate schematic processing in chronic pain, emphasis has been placed on recall and recognition memory task performance. This has revealed processing biases for pain-related information in patients suffering a chronic pain condition. The aim of the current chapter is to explore further the nature of schematic processing using an alternative paradigm to the recall and recognition tests previously employed.

For this purpose a word-stem completion task is used, where subjects are asked to complete a number of three-letter word stems such as 'sen....' with the first English word which comes to mind. All stems can potentially be completed with pain-related endings. On this task subjects are not required to *process* information which has previously been presented to them, as in a memory test. Instead, they are simply producing responses to ambiguous stimuli.

In addition to the chronic pain patients and controls, in this experiment a third group of subjects, health professionals will be tested to assess the significance of contextual effects in schematic processing. Previous studies where information-processing biases have been demonstrated in relation to mood (typically depression and anxiety states), and more recently pain, have not examined processing styles in people who have had considerable contact with these disorders through their

occupation but not through personal experience. Such studies have typically emphasized the implications of their results for the development and maintenance of the disorder under investigation (eg. Teasdale, 1983; MacLeod *et al*, 1986). However, if biased processing is found to the same extent in these health professionals as in the patients, such inferences are clearly unwarranted.

## METHOD

### DESIGN

The experiment was presented to subjects as an investigation into language. This was to avoid the unquantifiable confounding (as a result of activation of the pain schema) of results which would occur if subjects were aware that it concerned pain.

A total of 102 subjects in three groups (chronic pain patients, health professionals, and controls) completed two tasks which comprised 12 word endings (for example ...ell, ...ed), and 12 three-letter word stems (for example sha., fea...). Subjects were asked to write down the first two words of any length which came to mind, for each of the 12 endings and stems. The first task was included solely to lend credence to the assertion that the experiment concerned language. The word endings were chosen as being the most common in the English language. The responses on this task were not included in any statistical analysis. In the second task all the stems had at least one possible pain-related completion, and a minimum of three (and usually many more) possible non-pain-related completions of equivalent or greater frequency (Carroll, Davies and Richman 1971). Given these strict criteria for choosing words

to include in the experiment, relatively few of those available were suitable, resulting in the list of only 12 words. Four words were chosen from each of the sensory and affective categories of the McGill Pain questionnaire and the remaining four stems could be completed with words associated with the experience of illness (eg. disease). These words are presented in Table 6.1. The design was therefore a 3 (group:chronic pain, health professionals, controls) x 3 (wordtype: sensory, affective, illness-related). The first factor was between subjects, the second within subjects.

Table 6.1 Pain-related words presented as three letter word stems.

| Sensory words  | Affective words  | Illness-related words |
|----------------|------------------|-----------------------|
| <i>tender</i>  | <i>horrible</i>  | <i>ambulance</i>      |
| <i>hurting</i> | <i>miserable</i> | <i>disease</i>        |
| <i>burning</i> | <i>fearful</i>   | <i>healthy</i>        |
| <i>sharp</i>   | <i>cruel</i>     | <i>accident</i>       |

## SUBJECTS

The chronic pain group consisted of 38 patients attending routine out-patient appointments in the Rheumatology department and the Whittington Hospital. Their mean age was 46.8 years (SD=17.2) and 71% were female. Their mean duration of pain was 114.1 months (SD=112) and mean current pain intensity on a 0 - 100 mm visual analogue scale was 41.6 (SD=27.3). The health professionals group comprised 28 nurses and physiotherapists (approximately 1:1 ratio) working at the Whittington

Hospital London. Their mean age was 31.3 years (SD=7.7) and all were female. The mean duration since qualification as a nurse/physiotherapist was 132.4 months (SD=84.1) and none were currently in pain or suffered a long-term painful condition. Finally, the control group were volunteer members of the general public, whose mean age was 29.6 years (SD=14.1) and 50% of whom were female. None were in pain at the time of testing or suffered a long-term painful condition.

## PROCEDURE

Subjects were invited to take part in an experiment on language. Having obtained verbal consent, they first completed the two language tasks, and were then asked to provide demographic details, a measure of current pain intensity, and to indicate how long they had suffered pain/been qualified as a nurse or physiotherapist, as appropriate. All subjects were then fully debriefed.

## RESULTS

The total number of pain-related words produced on the word completion task was calculated for each subject. Variants of the original words on the McGill Pain Questionnaire were included, eg. discomfort/discomforting. If subjects produced two variants of a word both were counted, but only if the variant did not involve the addition of 'ing', eg. horrible, horrendous. In all cases data was normally distributed and plots of means against standard deviations indicated that no transformations were necessary. These results were subjected to a one-way analysis of variance with group

as the between groups factor. Since the ratio of men to women clearly differed significantly between the groups, sex was entered into the analysis as a covariate. A highly significant difference in the number of pain-related words produced by the three groups was identified,  $F(2,99) = 6.84, p < 0.002$ . (The covariate had no effect on the difference between the groups, and was therefore not used in further analyses.)

In the main analysis the number of word completions in each of the three wordtype categories was subjected to a two-way split plot ANOVA with group as between groups factor and wordtype as the repeated measure factor. Here, the main effect of wordtype was significant;  $F(2,198) = 5.77, p < 0.01$ . This is accounted for by a smaller number of illness-related completions compared to sensory or affective completions. The interaction between group and wordtype approached significance,  $F(4,198) = 2.24, p < 0.07$ . Figures 6.1 and 6.2 depict these results graphically, and in particular clarify the nature of the interaction. The means and standard deviations of pain-related completions for each wordtype and group can be found in table 6.2. A simple effects analysis of group for each wordtype highlighted the significant difference between chronic pain patients compared to health professionals and controls in the number of sensory words produced,  $F(2,99) = 3.89, p < 0.025$ . No difference between groups was found for affective completions,  $F(2,99) = 0.26, p > 0.75$ , but a difference approaching significance emerged between controls compared to pain patients and health professionals,  $F(2,99) = 2.87, p < 0.06$ , for illness-related completions.

Table 6.2 Mean (SD) number of pain-related word completions by each group for each wordtype.

|                 | Pain Patients | Health Professionals | Controls     |
|-----------------|---------------|----------------------|--------------|
| Sensory         | 2.139 (1.10)  | 1.464 (0.96)         | 1.605 (1.05) |
| Affective       | 1.889 (1.10)  | 1.714 (0.85)         | 1.816 (0.93) |
| Illness-related | 1.500 (1.25)  | 1.607 (0.99)         | 1.026 (0.94) |

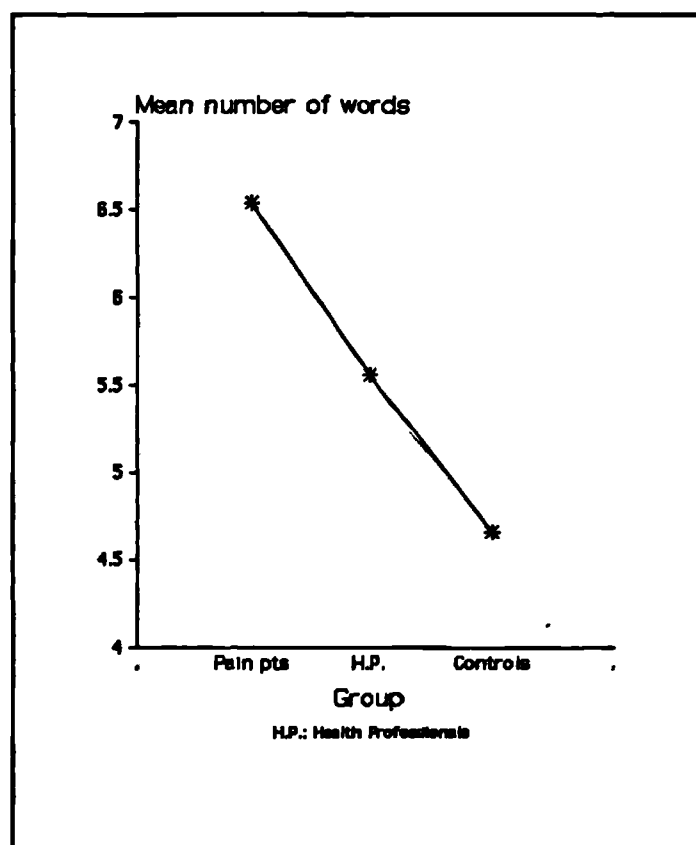


Figure 6.1 Mean number of pain-related word stem completions by chronic pain patients, health professionals and controls.



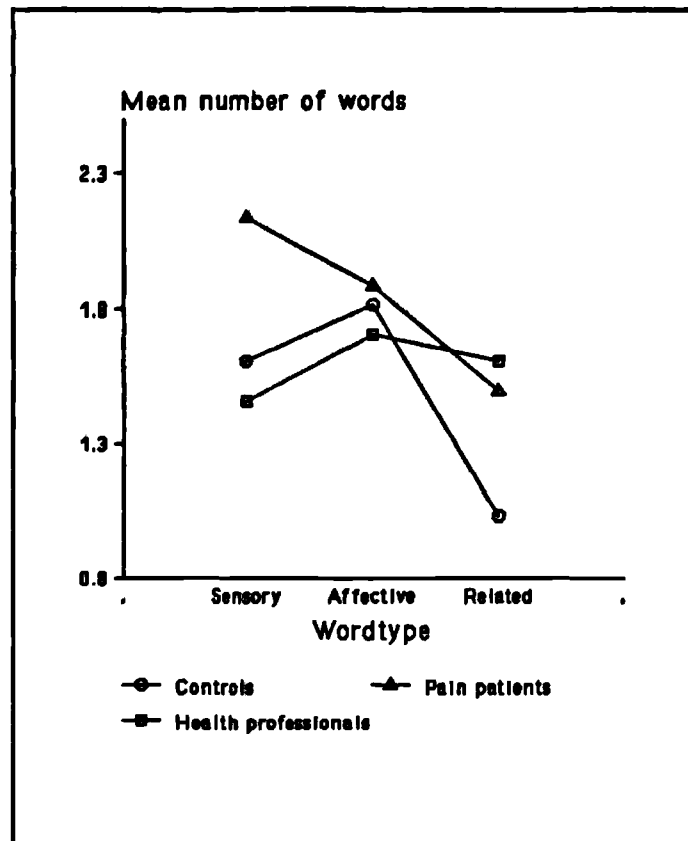


Figure 6.2 Mean number of sensory, affective and illness-related word stem completions by chronic pain patients, health professionals and controls.

## DISCUSSION

On a word completion task chronic pain sufferers produced significantly more pain-related word completions than did non-patient controls. A group of health professionals, exposed to pain through their occupational responsibilities, produced a mean number of pain-related words intermediate to the chronic pain and control groups, despite a mean number of years since qualifying which was greater than the

pain patients' pain duration. These results can best be accommodated within a "schema" model of mental representations, using the definition of schema presented in Chapter 1. The findings suggest that the pain schema of chronic pain patients differ from those of non-patients controls; their schematic representation of pain may be more highly organised. Although the health professionals also produced more pain-related stem completions relative to controls, it was not statistically significant. The intermediate position of this group in the one-way analysis of variance is thought to imply that personal experience of pain is the crucial factor in developing altered patterns of information-processing in chronic pain, rather than vicarious experience of pain and exposure to distress experienced via patient contact or education/training. In other words, these results could be accounted for by the existence of a 'self-schema', which in chronic pain patients incorporates highly elaborated pain representations. Pincus (submitted) provides evidence for a such a self-schema in chronic pain patients, with the finding that these patients show a self-referent (but not other person-referent) recall bias for sensory compared to neutral words.

This study also provides evidence to suggest that the way in which schema are elaborated is specific to the nature of exposure to pain information. The simple effect analysis of group on sensory word completions indicated that the chronic pain patients produced significantly more sensory completions than either the health professionals or controls. In contrast, both the health professionals and chronic pain patients produced more illness-related completions than the controls, although this difference just failed to meet the 5% significance criterion. This is consistent with much of the research in information processing in emotional disorders, which suggests that biased processing is typically, (but not exclusively) associated with

categories of words which hold personal relevance for the subjects, eg. Zeitlin and McNally, 1991; Mathews and MacLeod, 1985. In the present study the groups did not differ in the number of affective word completions produced. This is explicable, taking into account the finding reported in Chapter 2, that biased processing of affectively valenced pain-related information occurs only in depressed chronic pain patients. Neither the pain patients in this study, nor the health professionals and controls, could be assumed to be a homogenous group in terms of levels of depression. This requires clarification in future research.

Whilst these results have been discussed in terms of their implications for the organization of pain schema, it is also possible to explain them in terms of differences in baseline levels of activation of the schema. In chronic pain patients the baseline level of activation may exceed that of non-pain patients, resulting in their 'biased' performance on this task. Baseline schema activation levels have been investigated in other emotional disorders using implicit memory tasks which are an extension of the paradigm described here. Implicit tasks contrast with explicit tasks such as recall and recognition, which require the individual to consciously attempt to retrieve information which has previously been presented. Implicit memory does not require subjects to retrieve information from specific episodes (for example a previously presented word list) and is evidenced in priming tests such as word identification (where words are presented in degraded form), word fragment completion (eg. em\_ \_ \_ ke\_), and word stem completion (eg. spe.....). In a typical paradigm subjects are presented with a list of words, followed by a distraction procedure such as counting backwards in 7s, then the implicit task, for which they are asked to complete the items with the first word which comes to mind. A

dissociation between implicit and explicit memory function has been shown to occur in patients who have brain damage resulting in amnesia for new information. In these patients, performance on explicit memory tests shows very poor retention, whereas implicit test performance is unimpaired (Warrington and Weiskrantz, 1970). Comparable dissociations have been demonstrated in normal subjects, eg. Jacoby, 1983, 1988. Such results have been used to support the assumption that implicit memory test performance reflects unconscious or unaware retention of information. Also, it appears that these findings cannot be attributed to the subjects realizing that it was possible for the task to be completed by explicitly retrieving information from an earlier part of the experiment (Bowers and Schacter, 1990).

The distinction between implicit and explicit memory has inspired research which has resulted in the demonstration of implicit memory biases in anxiety states, where evidence for explicit selective memory effects for threat material has generally proved elusive. Three studies have provided evidence consistent with the idea that anxious subjects show an implicit memory bias. Mathews, Mogg, May and Eysenck (1989) found an implicit memory bias in generalised anxiety disorder patients using a word stem completion task. Subjects were presented with words and instructed to imagine a situation involving themselves and the word (elaborative encoding). Following 6 minute, unrelated filler tasks, subjects completed cued recall (explicit memory task) and word stem completion tasks in random order. Half of the stems could be completed with threat words previously presented (primed completions), while the remainder could be completed with other threat words *not* previously presented (unprimed completions). Results indicated that for primed (but not unprimed) completions, control subjects showed a pattern of processing favouring

non-threatening information, compared to the anxious patients who produced equal numbers of threat and non-threat completions.

In a similar study Zeitlin and McNally (1991) explored implicit memory in post traumatic stress disorder (PTSD) patients, for combat, social threat, positive and neutral words. In this study more substantial evidence was provided for an implicit bias - the PTSD patients produced *more* combat primed and unprimed completions than any other category, while controls produced equivalent levels of completions from all categories. A response bias explanation for this finding was ruled out since significantly more primed than unprimed combat completions were found in the PTSD group alone.

The only other study which has examined this issue used a word-fragment completion task, and compared self-referenced encoding with a 'read-only' condition. In high trait anxious subjects, an implicit memory bias was demonstrated for threat-related stimuli under the self-reference encoding condition only. This finding may, of course, have been due to the depth of processing involved, rather than the self-referencing *per se*, (Richards and French, 1991).

There are at least three theoretical approaches to account for implicit memory phenomena (discussed by Schacter, 1987), however, the most applicable here is the activation approach. The basic model used by Graf and Mandler (1984) to account for dissociations between implicit and explicit memory performance is presented in Chapter 1, along with the Williams *et al* (1988) extension of the model to account for the dissociation of the effects of anxiety and depression on memory and attention. In summary, according to Graf and Mandler (1984) activation of a schema makes its contents more accessible, whereas elaboration renders the contents both more

accessible and more retrievable. However, unlike Graf and Mandler (1984), Williams *et al* (1988) suggest that integration and elaboration are separate, distinct processes, and therefore items can be more easily retrieved without necessarily being more accessible. They propose that anxiety acts on integration resulting in attention biases, whereas depression influences elaboration, and hence retrieval processes.

To date, no studies have examined implicit memory in relation to depressive disorders. From the above theoretical perspectives, differing predictions would be made from the initial premise that depression acts on elaborative processes. The Graf and Mandler version of the model would lead to the prediction that since integration must precede elaboration, an implicit memory bias will occur in depression, whereas the Williams *et al* (1988) model does not. In support of the first position there is some evidence to suggest that depressed mood is related to Stroop interference (ie, an attention bias), which would be expected if negative words are primed; Gotlib and McCann, 1984; Williams and Nulty, 1986; Williams and Broadbent, 1986. However, another study has failed to find evidence for attentive biases in depression (MacLeod, Mathews and Tata, 1986).

Taking into account these conflicting models and empirical findings, the question of whether an implicit memory bias occurs in chronic pain is equivocal, and it remains an issue which requires full investigation. Never-the-less, within the implicit memory framework it is possible to say that the chronic pain patients in the present experiment produced more unprimed pain-related word completions than controls, and the most plausible explanation is that the internal representations of pain in these patients is in a constant state of activation as a result of the personal experience of long-term pain. However, there is another possible explanation for the results

obtained which might imply that they are artifacts of the experimental design, which needs to be excluded before we can be confident of this interpretation. The heightened number of pain word completions in the chronic pain group could potentially be due to a frequency effect, ie. chronic pain patients are exposed to pain words more often, and hence they are more common in their language, and the results merely reflect this effect. This is thought improbable since if completion simply reflects frequency, it would be predicted that nurses and physiotherapists, who are constantly exposed to pain vocabulary, would show the same bias as the chronic pain patients. This was not the case. However, it is an important issue, and forms the focus of the next chapter, where a lexical decision task is used to examine frequency of pain words in chronic pain patients and controls.

## *Chapter 7 Tests of frequency effect and associative network explanations for information-processing biases in chronic pain.*

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### INTRODUCTION

In previous chapters considerable emphasis has been placed on the effects of mood on memory. Numerous studies have demonstrated selective processing of mood congruent information, primarily threat-related information in anxiety states and negative material in depression. Similarly, a recall bias has been found for pain-related words in chronic pain sufferers, along with differences in pain-related word stem completion. From both clinical and theoretical perspectives a great deal of importance has been attached to this type of finding. However, all of these findings can potentially be accounted for by a simple frequency effect: although studies invariably match words for frequency across word categories, it may be erroneous to assume that the frequency of particular words is the same for more than one population. For example, it is conceivable that a word such as "throbbing" may be high frequency ie. common, for the chronic pain patient population, but of lower frequency for the general, non-pain population. If this is the case the classical finding that high frequency words are easier to remember (in a free recall paradigm) than low frequency words would account for the superior recall of pain-related words in chronic pain sufferers.

It appears that only one study in the literature has attempted to tackle this



problem. Clark and Teasdale (1985) aimed to investigate the possibility that women use personality trait words and concepts more frequently than men, and that this resulted in their finding of differential recall of pleasant and unpleasant words in induced happy and unhappy moods in women but not men. Subjects were asked (post experimentally) to rate a list of trait words, including those from the recall test, for how much they would notice and think about them when talking about peoples' behaviour, on a scale from never (1) through moderately frequently (4) to extremely frequently (7). In women, a significant positive correlation was found between word usage ratings and preferential recall. Clark and Teasdale fail to discuss the implications of this finding: clearly, however, selective memory effects may be more strongly associated with word frequency/usage than biases in information processing.

Other studies have implications for this issue, although they did not directly set out to investigate the problem. For example, Watts, McKenna, Sharrock and Trezise (1986) found that an exposure treatment for spider phobia (desensitization) reduced interference on the Stroop task, despite increased exposure to the threat stimuli. Other work on perceptual processing biases which has also suggested that abnormalities disappear after treatment includes that of Foa and McNally, (1986) and Gotlib and Cane (1987).

There is no empirical evidence on this issue in chronic pain. However, before it is possible to draw safe conclusions from previous experiments, it seems necessary to investigate the possibility of a frequency effect. Obtaining usage ratings from subjects provides one method of assessing frequency, but gives only a subjective measure and is therefore not ideal. Lexical decision research provides an alternative approach.

In a lexical decision task subjects are typically asked to decide as quickly and accurately as possible whether on each trial a single letter string forms an English word, for example "yellow" or a non-word, for example "yillow". Non-words are usually formed by replacing one or more of the vowels in a real word, as in the previous example, making the non-words pronounceable. Basic research using this paradigm has revealed that word frequency is a consistent predictor of response time, with high frequency words being responded to more quickly (eg. Landauer and Freedman, 1968; Rubenstein *et al*, 1970). Also, when the same word is presented twice, as in 'repetition priming', subjects respond significantly faster to the second presentation, even when there is a considerable lag of 15 other words between the first and second presentations. Interestingly, and of importance to the current problem, is the robust finding that frequency and repetition interact, such that the size of the repetition effect (ie. the decrease in response time) is larger for low frequency (uncommon) words than for high frequency words, (eg. Norris 1984; Scarborough, Cortese and Scarborough, 1977). This observation allows the following prediction to be made with regard to chronic pain: if pain-related words are relatively *high* frequency for pain patients but relatively *low* frequency for controls, a greater repetition effect for these words would be expected for control subjects. If the words are of equivalent frequency for both groups, the extent of repetition priming would be equal across groups. These possibilities are represented graphically in Figures 7.1 and 7.2.

Thus the first and primary aim of this study is to address the issue of potential frequency effects in information processing biases in chronic pain. It is predicted that there will be no difference in the extent of repetition priming in a lexical decision

task on pain-related words, between chronic pain patients and controls.

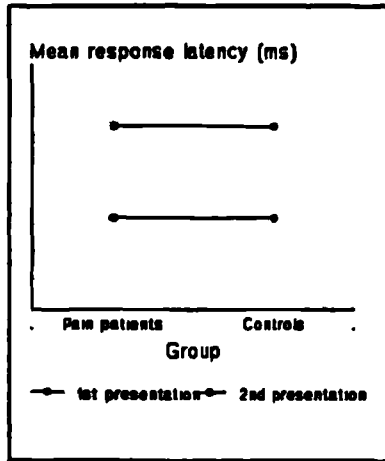


Figure 7.1 Predicted pattern of results if pain-related words are high frequency for pain patients and low frequency for controls.

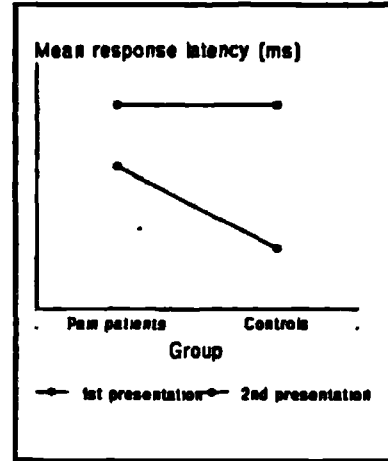
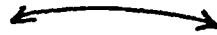


Figure 7.2 Predicted pattern of results if pain-related words are of equal frequency for pain patients and controls.

One of the most replicated findings in lexical decision research in the 1970s was that the lexical decision time for a word is more rapid if it follows a word with which it is associated (eg. Meyer and Schvaneveldt, 1971; Meyer *et al*, 1975). The classic example is that the response latency for "doctor" is faster if it is preceded by the word "nurse" than if it is preceded by the word "butter". This type of finding has generally been interpreted within an associative network/spreading activation model, such that when a representation of a concept is presented, for example the word

"doctor", its corresponding node is activated, along with links in the network with other related nodes, thereby increasing their activation levels. If the representation of a related concept is then presented, lexical decision time is reduced since this node is already activated to some extent.

Explanations for information-processing biases in mood states (and of course chronic pain) in terms of this model have proposed emotion nodes, along with the nodes for descriptors of the emotional/physical states. Using a variant of the lexical decision paradigm, Clark, Teasdale, Broadbent and Martin (1983) tested the prediction that emotional states (elation and depression in this instance) will prime or activate nodes related to that emotion, such that relative to neutral words, lexical decision times for positive words would be faster when subjects were in an induced happy mood than induced depressed mood. The converse would be true for negative words. In the paradigm used, subjects were presented with a priming word to which they did not have to respond, followed by the target word. Contrary to expectations, there was no interaction between mood and word valence. The authors suggest three possible explanations. Firstly, the strength of association between the mood state and words may have been insufficient to produce facilitation in the lexical decision task. However, differential recall of these words was found in induced moods by Teasdale and Russell, (1983). Secondly, they suggest that semantic information and information from personal experience of an emotion may be stored separately, but this would not be predicted from the original model. Finally, mood congruity effects may occur only when the subject has to generate the emotionally-valenced response as in recall (and word completion). In similar studies, Martin and Clark (1985) and MacLeod *et al* (1987) failed to find differences in lexical decision times in relation

to depressed mood.

Although associative network models have more recently been claimed inadequate to account for the different effects of anxiety and depression on information-processing (Williams *et al*, 1988), it would be unreasonable to assume that associative networks for emotions do not exist: it may be that they simply do not differ in patient groups and controls. The alternative is that the variant of the lexical decision paradigm used in previous research was not appropriate, and it is this possibility which will be explored here in chronic pain patients. The primary questions are therefore 1) is there an associative network for pain?, and 2) if so, can chronic pain patients and non-patient controls be distinguished on the basis of the degree of activation of nodes and their links? The second part of the experiment reported in this chapter uses association priming in the following manner: subjects (chronic pain patients and non-patient controls) are presented with pain-related words consecutively, and the response latency for *both* is recorded. These pairs are interspersed with neutral words and non-words. It is predicted that the extent of priming of the first pain-related word will be greater for the chronic pain patients than the controls, in other words the difference in response latency between first and second of the pairs of pain words will be greater for chronic pain patients than controls (see Figure 7.3.)

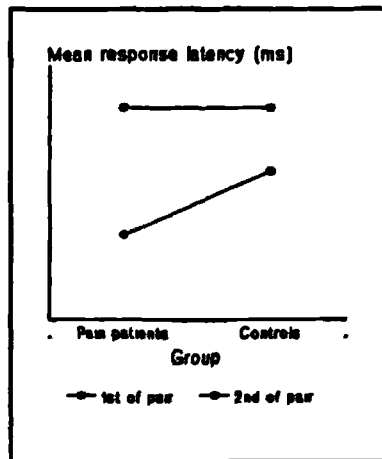


Figure 7.3 Predicted pattern of results for association priming of pairs of pain-related words.

## METHOD

### DESIGN AND STIMULI

The experiment is divided into two sections, part A: repetition priming and part B: association priming. Groups of chronic pain patients and controls completed both sections. The design of each will be considered separately, however certain aspects are common to both. Although only pain-related words are strictly required for the hypotheses to be tested, neutral fillers are included in an attempt to prevent subjects from guessing the purpose of the experiment. However, fewer neutral than pain-related words are used to keep the duration of the experiment to a minimum. All pain-related adjectives were from the McGill Pain Questionnaire (Melzack, 1975), and include those used in previous studies of this thesis. Neutral adjectives were approximately matched for length and frequency with the pain-related words so that they did not stick out like a sore thumb. The non-words were compiled by changing

one or two vowels (depending on the length of the word) in a further set of neutral words. Fifty per cent of the non-words appeared in both sections of the experiment. Neutral and non-words were not statistically analysed.

Half of the subjects completed part A first, the remainder started with part B. Words were presented in the centre of the screen, on a Toshiba T3100SX, gas plasma display portable computer. Each trial consisted of a "\*" fixation point presented for 1 second, followed by a blank screen for 1 second, then the word.

#### *Part A. Repetition priming*

Thirty pain-related adjectives (18 sensory and 12 affective), 15 neutral words each presented twice, and 45 nonsense words were presented to subjects in fixed random order. The following constraints were placed on the randomization: no more than 4 words (pain-related, neutral or a mixture) presented consecutively, and the lag between first and second presentation of words varied between 2 and 4 words. This was to ensure that subjects could not learn (consciously or unconsciously) what type of word to expect next.

#### *Part B. Association priming*

Fifteen pairs of pain-related adjectives (9 sensory and 6 affective), 15 neutral words and 45 non-words were presented to subjects in fixed random order under the following constraints (for the same reasons as previously): a minimum of 1 neutral or non-word and maximum of 7 between each pain-related pair, and no more than 4 words or non-words consecutively. The order of presentation of words within the pain-related pairs was reversed for half of the subjects.

The BDI was completed by all subjects to obtain a measure of level of depression.

## **SUBJECTS**

The chronic pain patient group comprised 20 patients attending routine out-patient appointments at the Whittington Hospital Rheumatology clinic. All patients had experienced pain for a minimum of 6 months. Their mean age was 47.20 years (sd=9.24) and 90% were female. Their mean duration of pain was 91.350 months (sd=93.28), mean current and average VAS pain intensity ratings were 41.200 (sd=23.66) and 54.474 (sd=22.61) respectively, and their mean BDI score was 12.00 (sd=7.56). In this sample, 12 patients had rheumatoid arthritis, 4 had systemic lupus erythmotosis, 1 ankylosing spondylitis, 1 Sjogans disease, 1 tendonitis and 1 unclassified arthritis.

Twenty volunteer members of the general public formed the control group, recruited primarily through advertisements. Their mean age was 44.95 (sd=13.71) and again 90% were female. Their mean score on the BDI was 7.25 (sd=6.03), and none of these subjects had experienced a long-term painful condition.

The two groups differed significantly in their scores on the BDI ( $t(38)=2.637, p<0.05$ ).

## **PROCEDURE**

Subjects were invited to participate in a study investigating how quickly people can decide whether a word is "real" (in English) or not. Subjects were instructed to press the right hand button of a hand-held set if they thought the word was real, and the left hand button if it was not, using right and left hands respectively. Subjects



were warned that some words would be repeated once. (Instructions using the terms "letter-string" and "non-word" were avoided to prevent confusion). Subjects were asked to respond as quickly but as accurately as possible. They first completed a block of 10 practice trials, 5 of which were words, and 5 non-words, compiled from high frequency nouns. This was followed by the 2 experimental blocks, as described in the design. Subjects were allowed to rest for as long as they wished between these two blocks. The BDI was then administered. The procedure lasted approximately 30 minutes.

## RESULTS

### *A. Repetition priming*

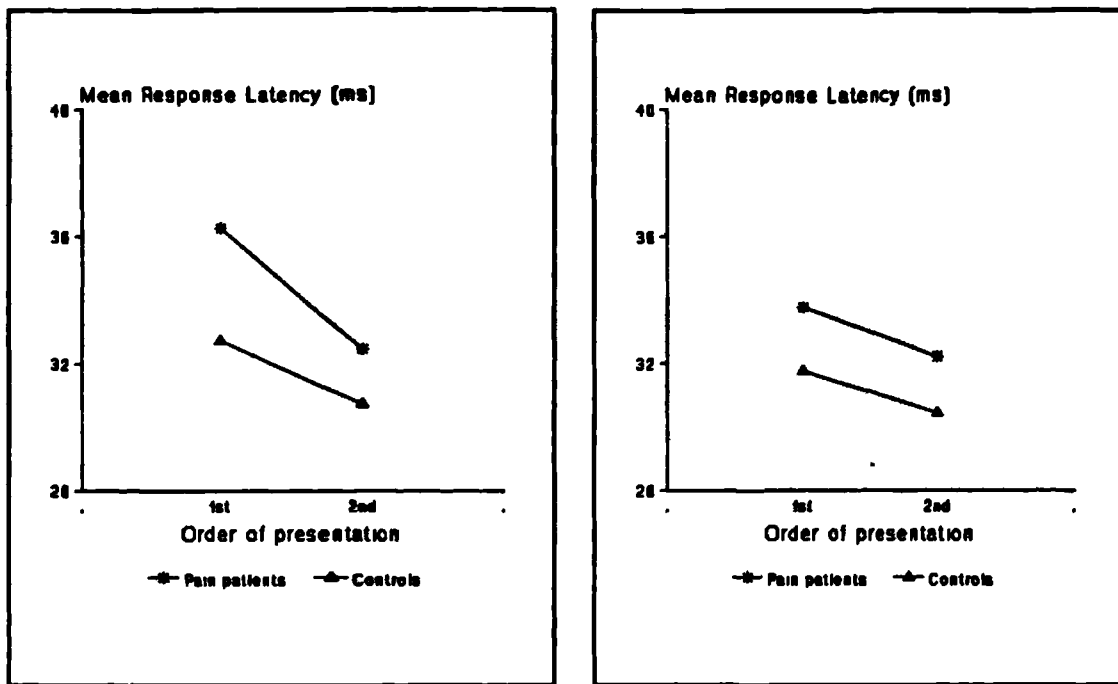
Mean response latencies in milliseconds for the pain-related words (Table 7.1) were subjected to a 3-way split plot ANCOVA (after undergoing a square root transformation), with group (chronic pain patients, controls) and order (repetition priming first, association priming first) as the between groups variables, presentation (first versus second) as the repeated measure variable, and BDI score as the covariate. Covariates do not affect the repeated measure variable or interactions with this variable; their only influence in this and all other analyses in this study was to render previously significant differences between the groups insignificant (chronic pain patients consistently respond more slowly than controls in all conditions). This analysis therefore revealed no significant main effects of group or order, but a highly significant main effect of repetition, such that the response latency for the second

presentation of a pain word was faster than the first presentation of that word,  $F(1,36)=55.50$ ,  $p=0.000$ . The interaction between repetition and order was also significant, indicating that there was a greater repetition effect in subjects who completed this section of the experiment first than in those who completed the association priming section first. These results, using adjusted means, are shown graphically in Figure 7.4 (a, b).

**Table 7.1** Unadjusted and adjusted (with BDI as covariate) mean response latencies in milliseconds (after square root transformation) for repeated pain-related words.

| Order                   | Chronic Pain Patients (n=20) |                | Controls (n=20) |               |
|-------------------------|------------------------------|----------------|-----------------|---------------|
|                         | 1 <sup>c</sup>               | 2 <sup>d</sup> | 1               | 2             |
| <b>1st presentation</b> |                              |                |                 |               |
| Mean <sup>a</sup> (sd)  | 37.368 (6.65)                | 33.635 (5.78)  | 32.001 (3.373)  | 31.511 (5.22) |
| Mean <sup>b</sup>       | 36.265                       | 33.777         | 32.734          | 31.768        |
| <b>2nd presentation</b> |                              |                |                 |               |
| Mean <sup>a</sup> (sd)  | 33.571 (4.79)                | 32.085 (5.98)  | 30.014 (2.70)   | 30.195 (5.15) |
| Mean <sup>b</sup>       | 32.467                       | 32.288         | 30.747          | 30.453        |

<sup>a</sup> Unadjusted mean    <sup>b</sup> Adjusted mean    <sup>c</sup> Repetition priming first.    <sup>d</sup> Association priming first



(a) Repetition priming first

(b) Association priming first

Figure 7.4 Mean response latencies (square rooted, in milliseconds) for first and second presentation of pain-related words in chronic pain patients and controls.

### B. Association priming

Mean response latencies in milliseconds (after square root transformation; Table 7.2), for pain-related words were subjected to a 4-way ANCOVA, with group (chronic pain patients, controls), order (repetition priming first, association priming first), and order of pain-related words within the pair as the between groups variables, position in pair (first or second) as the repeated measure variable, and BDI score as the covariate. There were no significant main effects or interactions involving either 'order' variable; these were therefore dropped from further analysis. The results showed neither main effects of group or position of words in the pair, nor

an interaction between the two (Figure 7.5). Since the sensory words may have been more strongly associated with pain than the affective, these were considered separately. For the sensory adjectives there was no main effect of group, and the interaction between group and position in pair failed to reach significance. However, the analysis revealed a significant main effect of position, with faster response latencies for the second word in the pair compared to the first, across both groups;  $F(1,38)=8.84$ ,  $p=0.005$ . Considering the affective adjectives, there was again no evidence for any main effects or interactions.

Table 7.2 Unadjusted and adjusted (with BDI as covariate) mean response latencies in milliseconds (after square root transformation) for association priming of pain-related words.

|                           | Chronic Pain Patients<br>(n=20) | Controls<br>(n=20) |
|---------------------------|---------------------------------|--------------------|
| 1st of pair of pain words |                                 |                    |
| Mean <sup>a</sup> (sd)    | 34.643 (5.45)                   | 32.185 (4.42)      |
| Mean <sup>b</sup>         | 34.274                          | 32.554             |
| 2nd of pair of pain words |                                 |                    |
| Mean <sup>a</sup> (sd)    | 33.934 (5.37)                   | 32.875 (5.11)      |
| Mean <sup>b</sup>         | 33.565                          | 32.244             |

<sup>a</sup> Unadjusted mean    <sup>b</sup> Adjusted mean

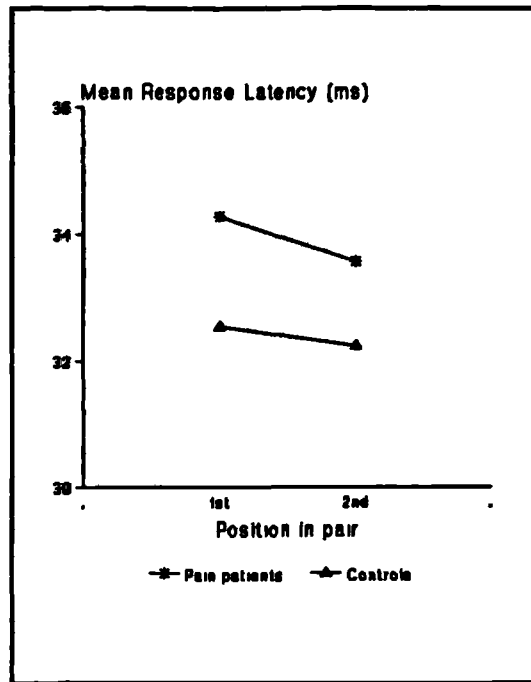


Figure 7.5 Mean (square rooted) response latencies in milliseconds, for the first and second of pairs of pain-related adjectives, in chronic pain patients and controls.

## DISCUSSION

The results of the first part of this experiment provide evidence which suggests that differences in information processing between chronic pain patients and non-patient controls cannot be explained solely in terms of a word frequency effect. The interaction between repetition (first or second presentation) of pain-related adjectives and group did not reach significance - control subjects did not respond faster to the second presentation *relative* to the first presentation of the pain-related words, compared to the chronic pain patients. However, in accord with research on general

repetition priming effects, all subjects showed significantly shorter response latencies to the second presentation of words. Together, these findings carry the implication that pain-related words are not of higher frequency for chronic pain patients than controls; they are no more common, or used more frequently in either population. This finding is apparently in contrast to that of Clark and Teasdale (1985), who found that women use personality trait words more frequently than men, and that this could account for the superior recall of these words in congruent mood states in women alone. However, in this study a subjective measure of word frequency was obtained, and the population sampled was 'normal' students, in whom depression and elation was induced. Therefore direct comparisons between the current study and that of Clark and Teasdale (1985) cannot be drawn, and future research is needed to replicate this result, also using alternative paradigms. If the result can be taken at face value, previous findings that chronic pain patients selectively recall pain-related information, and produce more pain-related stem completions than controls, are indicative of biased information processing in these patients.

One surprising aspect of this part of the experiment was that the repetition effect was found to interact with the order in which subjects completed the repetition and association priming sections of the experiment. The reason why the repetition effect was greater when this part of the experiment was completed first is unclear; however there were no other significant effects involving this variable, and no mention of such an occurrence in the relevant literature, suggesting it is of little importance.

The results of the association priming part of the experiment are more difficult to explain. The predicted interaction between group and position of the word within the pain-related pair failed to emerge. In previous studies on mood congruity in

lexical decision tasks, emphasis was placed on decision times for mood congruent words relative to neutral words, in induced happy and unhappy moods. Here the explanation that nodes representing the mood itself, and nodes representing the words associated with that mood may not be strongly connected, is plausible (if not readily predicted from Bowers (1981) network model). However, in the present experiment response times were compared for *two pain-related* words and therefore this possibility is not viable. All subjects would be expected to possess an associative network for pain, as all must have experienced pain at some time in their lives. The associative network of chronic pain patients should, however, be more structured, with a greater number of links, and higher levels of activation. Yet there was no evidence for association priming for pain-related information in either group, under the analysis of all the available pain-related words.

There are two possible approaches in accounting for this finding. Firstly, the methodology used may have been inappropriate. Lupker (1984) points out that the automatic activation of a concept and spread of activation decays rapidly, perhaps within 40 milliseconds (Fischler and Goodman, 1978), unless the subject continues to attend to the stimulus. Clearly, it would be impossible to obtain inter-stimuli intervals (onset to onset) of this order in chronic pain patients and matched controls, who typically show response latencies alone of greater than 1000 ms, without considering the presentation of the fixation point or interval between the fixation point and presentation of the stimulus. In addition, association priming has consistently been demonstrated for other classes of words, where inter-stimulus intervals exceed 40ms, and the subjects have not been forced to continue to attend to stimuli (eg. Meyer and Schaveneveldt, 1971; Meyer *et al*, 1975).

Further methodological issues include the possibility that the pain-related words chosen were not closely associated enough with each other to promote priming. In an effort to obtain as many stimuli as possible, all the words on the McGill Pain Questionnaire were used. Some of these words, such as 'hot' and 'jumping' may not have been linked to pain by the subjects. This requires investigation using association norms, such as those provided by Postman and Keppel (1970). Other types of pain-related words such as 'disabled' and 'hospital' may be more powerful primers of pain concepts, particularly compared to the affective adjectives used in this experiment, since the pain patients did not generally exhibit a particularly high level of depression. This study provides some evidence to this effect - association priming by the sensory, but not the affective adjectives was found in both groups.

The alternative explanation for this finding is that the theoretical assumptions being tested are at fault, resulting in the failure to corroborate the network model, at least for a pain network encompassing both physical and emotional pain descriptors. This is perhaps a less compelling argument, given that some evidence was found for association priming, with the sensory pain descriptors. The problem remains, though, that there was no evidence for a superior network, even for the sensory pain-related words in the chronic pain patients. Thus an account of previous findings of processing biases in relation to pain in terms of associative network theory may be premature.



## *Chapter 8 Discussion and Conclusions*

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In this thesis eight studies have been described which investigate cognitive processes in pain and depression. The aim of this chapter is to draw together the findings from all these studies and to consider their theoretical and clinical implications.

The model which originally guided these experiments was that of Leventhal and Everhart (1979), who proposed 3 levels of processing in pain; expressive-motor, schematic and conceptual, along with the parallel processing of sensory and distress/emotional components of pain. The model proposes that the emotional component of pain is processed largely pre-consciously, and is produced rapidly and "virtually simultaneously with the pain experience", with pain entering "focal awareness" (or consciousness) as a unified sensory and emotional experience. Thus unless specific anaesthesia instructions are given to the patient while under hypnosis, or other attention diversion strategies are employed, whenever pain is experienced, the emotional component is also necessarily experienced. The extent to which the sensory and distress components can be considered independent pre-consciously is unclear.

The results of the studies reported in this thesis can be divided into those with relevance to the schematic level of processing, and those which can be considered conceptual. Findings will therefore be discussed under these two broad headings, although a complete segregation is not possible given the interactive nature of the processes.

### ***Schematic Processing***

The first study of this thesis explored one aspect of the schematic level of processing: memory function, with the primary aim of assessing the impact of both chronic pain and depression on information-processing. The results of this study, like those of Pearce *et al* (1990), provided evidence supporting the hypothesis that chronic pain patients selectively remember pain-related information in preference to neutral, non pain-related material. In addition, in this study chronic pain patients who showed minimal depressive symptomatology selectively recalled the sensory, but not the affective adjectives of the McGill Pain Questionnaire. One possible conclusion is that these affective pain descriptors do not tap the distress/emotional component of pain as conceived by Leventhal and Everhart. Their model would lead to the prediction that all patients, whether depressed or not, will show a memory bias for both sensory and affective adjectives. Thus the affective adjectives may provide an index of the affective state of the individual, and be more strongly associated with general mood state, rather than reflect the emotional component of pain itself, or pain intensity on an affective dimension. Indeed, Reading *et al* (1982) argue as invalid the assumption that these affective adjectives can be ordered along a single intensity dimension.

The results of this experiment can be conceptualized within two main theoretical frameworks for the mechanisms of mood and memory - associative network and schema theory, described in Chapter 1. Although Leventhal and Everhart (1979) use the terms schemata and schematic processing they do not suggest how exactly they might operate. However, in a general theory of emotions, the "perceptual-motor theory of emotion", Leventhal (1984) clearly states that he "does not believe that it

[the schematic memory structure] can be represented as a set of nodes in an associative network". Instead he suggests that they can be thought of as a set of components which fall in a common cortical field or column - a vertical section through the layers of the cortex that allows a combination of sensory and motor events to be represented in each layer. Thus in this conception of schemata, they are discrete, automatically activated units, with excitation flowing downward through the column from the topmost layer. He argues against a network of nodes with links which are of the same type for the sensory, expressive and other components, as well as for the verbal labels for pain experience. Support for this position stems from the fact that it is possible to talk about past experiences of pain, thus activating verbal labels, without re-experiencing the subjective feeling of pain. However, it remains unclear how this can occur under the unified schema model, since activation of an entire pain schema through reading or talking about pain should result in all other aspects of pain also becoming activated. Presumably this activation is assumed to fail to reach sufficient levels for the subjective experience of pain to enter conscious awareness.

Leventhal therefore points to the distinction between associative network and schema models, and rejects the idea of associative networks for emotions. However, such network explanations are most common for the effects of mood on memory. Ellis and Ashbrook (1991) clarify the distinction, while also attesting to the fact that they are also conceptually very similar. The models differ primarily in that associative network models generally assume *spreading* activation whereas schema models typically do not. However the two models are not necessarily contradictory, and Ellis and Ashbrook (1991) suggest a "fully-developed model of how mood states

influence cognition will see an integration of these two approaches". Both models are able to account for the effects of chronic pain and depression on memory, but *only* if the affective words in the current experiment are assumed to tap a general emotional state and not the affective/distress component of pain, ie. separate nodes/networks or schemas for physical pain and negative affect. If, on the other hand, the affective adjectives describe an integral part of the pain experience, then neither model is acceptable. Also, there is a danger of all these "explanations" becoming tautological. It is however, possible to answer the original question, and conclude that pain has an effect on information-processing distinct from that of negative mood, and that the memory bias demonstrated by Pearce *et al* (1990) cannot be attributed solely to the chronic pain patients' elevated levels of depression.

The use of Signal Detection Theory in this first experiment was an attempt to separate differences in true memory from response bias between the groups. Unfortunately it proved largely unfruitful, with no clear results evident. This is perhaps an indication that the method is inappropriate for this purpose in these groups - receiver operating characteristic (ROC) curves were not obtained (for practical reasons outlined in Chapter 2), which would have permitted validation of the method.

The results of the recall tests in the acute pain sufferers were not conclusive, given the relatively small numbers of subjects in the groups. If future research confirms that there is no memory bias associated with either acute clinical or induced pain, it would suggest that selective memory is a consequence of the long-term experience of pain. Theoretically, this may be the result of the gradual strengthening

of schema, or build up and activation of links in an associative network. On the other hand the meaning of the pain may have prevented the development of recall biases. If patients believed that their pain would be transitory, and manageable, resulting in minimal permanent impact on their lives, a top-down influence from conceptual processes would be exerted on schematic processes, a notion proposed by Leventhal and Everhart's (1979) model of pain processing. An interaction between these factors (duration and meaning of the situation) is perhaps most probable.

Alternatively, if the trend towards selective recall of sensory words which was becoming apparent in the acute pain patients, becomes statistically significant with larger sample sizes, it would indicate that the meaning of the situation for the individual *is* of greater importance than the duration of pain. Although acute, the pain may be considered of serious immediate and long-term threat to the individual. For example, if patients believe that the pain they are currently experiencing heralds the start of a long-term illness, with implications for employment, family and social activities etc., this is likely to influence schematic processing. Indeed, the patients who participated in this study may have formed an unusual group in this respect, since the surgical operation they received carried major implications for future fertility, and therefore their conceptual processing may not have been representative of all patients experiencing an acute pain condition. Although all experience of acute clinical pain is likely to carry some degree of threat, certain conditions, such as appendicitis, in which the threat is minimized may provide a more useful group of patients to investigate. Clearly all of these issues require further investigation and clarification.

One of the more surprising findings in this thesis was evidence for cognitive avoidance of negative information in clinically depressed patients. In a free recall paradigm, a group of clinically depressed patients remembered fewer affective pain descriptors and negative adjectives describing the feelings associated with depression compared to neutral and sensory words. No evidence for cognitive avoidance in disorders other than anxiety states has been found, and not generally as the result of the use of recall paradigms. Conclusions and theoretical interpretation must therefore be considered tentative. As associative network and schema theories stand, neither are able to account for this finding, since both assume heightened activation of mood-congruent nodes/schema, which would only lead to the over-recall of affective information. However, the Williams *et al* (1988) integrated model, which relies on the concept of resource allocation, provides a plausible alternative. Under this model is it possible for processing resources to be allocated either towards, *or away from* material. Allocation away from negative material might have occurred in this group as a result of the combination of encoding procedure used (non self-referential) and the nature of the stimuli. Words were specifically chosen to reflect the feelings associated with depression, rather than personality-trait words used in previous research.

The suggestion that selective memory effects are a consequence of the long-term personal experience of pain received further support from the results of the experiment comparing pain-related word-stem completion amongst chronic pain patients, health professionals and non-patient controls. Chronic pain patients produced the most, and controls the least, pain-related word stem completions. In addition, the

type of words produced generally coincided with the nature/extent of pain experience. Differences in activation levels of schemas or associative networks could be considered responsible for the findings. However, the difference between the responses of the chronic pain patients and health professionals provides strong evidence that simply talking, hearing or reading about pain is not sufficient to cause biased processing, suggesting that a modification of the models is necessarily - the idea of a self-schema incorporating information obtained from experiencing pain personally, may be sufficient. The associative network model is less readily adapted in this manner.

The results of the two studies reported in Chapter 5, which explored the impact of surgical and cognitive-behavioural interventions for chronic pain, should be considered preliminary given the small sample sizes at follow-up. Patients who were largely pain-free after surgery showed an increase in the number of non pain-related words remembered, coupled with a decrease in the recall of pain-related adjectives. In contrast, cognitive-behavioural management appeared to have no effect on the type of information remembered. Together these studies suggest that memory biases in chronic pain are "pain-driven". In other words, only when pain is removed does selective recall of pain-related information disappear: cognitive-behavioural intervention apparently has no impact on biased processing, since the experience of pain sensations remains. However the process of remediation appears to be gradual, perhaps suggesting that schema or associative networks are slow to "de-activate", or that the links in a network do not break down instantaneously. Studies exploring cognitive biases in anxiety and depression have frequently alluded to the possibility

that the mechanisms underlying such biases may represent enduring "vulnerability" factors for those conditions. While evidence has been mixed in relation to anxiety disorders (Eysenck *et al*, 1991; Mathews *et al*, 1990), studies comparing clinically depressed, recovered depressives and normal controls have typically provided evidence that the depressed patients in remission respond in a manner which is closer to that of controls than currently depressed patients, for example Bradley and Mathews, 1988; Dohr and Rush, 1989. This suggests that selective memory is state-related, and therefore cannot be considered an index of vulnerability.

It is therefore possible to tentatively conclude that pain-related memory biases in pain are more strongly related to state than trait factors, although, taking into consideration the results of the acute pain group and induced pain experiments (Pearce *et al*, 1990), long-term experience of pain appears necessary. Also, the results perhaps suggest that schematic processing exerts a more powerful influence than conceptual processing, or over-rides changes in conceptual processing; the changes in functioning which occurred as a result of the pain management programme (for example reductions in depression, anxiety, hopelessness thoughts and social dysfunction, and increases in activities and positive pain-related thoughts, all of which involve some degree of conceptual processing) apparently failed to influence memory function. The chronic pain patients consistently remembered more pain-related adjectives, however, the difference was not significant at any stage. It is important to note, however, that the return to a "normal" pattern of recall would involve the superior recall of neutral information: there was no evidence for such a process occurring. This could be interpreted as indicative of the greater influence of conceptual processing, as a result of the psychological assessment and preparation



prior to the start of the intervention.

Thus the relative dominance of schematic and conceptual processing in pain remains an issue for conjecture. Although Leventhal (1979, 1984) states that conceptual processing can alter schematic processing, and that conceptual processing may not accurately reflect schematic "knowledge", he does not suggest which level of processing may exert greater influence at any particular time. Future research could attempt to solve this issue.

Further support for the inadequacy of associative network models in accounting for selective memory effects was provided by the second part of the experiment reported in Chapter 7. Some evidence compatible with an associative network conceptualization of sensory pain descriptors was found; all subjects responded faster to the second of a pair of sensory adjectives. However, significantly, no differences in the levels of activation or organization of the network between chronic pain patients and controls could be inferred, since the amount of priming provided by the first of the pair was no greater for chronic pain patients than controls.

Two final conclusions can be drawn from the investigation of schematic processing in pain patients in this thesis. Firstly, no evidence was found to support the hypothesis that selective memory effects in chronic pain are found as a result of the use of pain-related stimuli which form members of a semantic category: chronic pain patients did not recall more gardening words than neutral words. Secondly, the results of the first part of the experiment in Chapter 7 suggest that pain-related

memory biases cannot be attributed to differences in the frequency with which chronic pain patients and non-patient controls encounter sensory or affective pain descriptors. This finding supports the indirect evidence from studies which have demonstrated <sup>uctions in bias</sup> reduced ~~reduced~~ despite repeated exposure to the mood-congruent stimuli.

In considering all of these findings it becomes apparent that the models previously adapted to account for the effects of mood on memory, ie. associative networks and schemas require some modification before they can account for the effects of pain on unconscious processes. Williams *et al* (1988) provide a model which is able to accomplish this to a large extent, by distinguishing between the processes of priming and elaboration and proposing that biases can occur in one but not the other.

The experimental findings of this thesis, in conjunction with research on attentive processes in chronic pain, suggest that this model has potential for understanding cognitive processes in chronic pain. It appears that the effects of pain on memory processes are analogous to those of depression, with chronic pain influencing the allocation of processing resources to the elaboration of pain-related information.

Components of each of these models, in particular activation and resource allocation, along with the concepts of feedback and cognitive loops (Ingram 1984), may provide a theoretical account of greater utility in guiding future research in this area. Such research could aim to define more clearly the exact conditions under which biased processing in pain is evidenced, establishing whether findings are generalizable from word stimuli, and developing specific remedial strategies based on this knowledge.

Several authors have proposed that information-processing biases play a role in the development and maintenance of mood disorders (eg. Teasdale, 1983; MacLeod *et al*, 1986), with mechanisms including cumulative activation and feedback thought to be responsible (Ingram, 1984). In anxiety states it has been suggested that the clinical manifestation of elevated levels of activation of cognitive structures results in intrusive thoughts and re-experiencing of symptoms such as flashbacks, which are characteristic of these emotional disorders. The analogous implication for chronic pain is that such activation plays a role in the continued experience of pain after healing has occurred or where no organic pathology is found, typical of chronic pain syndromes. Clearly, such a causal relationship requires empirical verification.

### ***Conceptual Processing***

With the aim of providing a measure of some aspects of conceptual processing, a questionnaire was devised which, on factor analysis, was shown to comprise two scales, labelled "organic" and "psychological" (Chapter 4). Groups of chronic pain patients and non-patient controls were found to differ significantly in the extent to which they endorsed these two classes of beliefs. The chronic pain patients placed greater emphasis on organic beliefs, and less on psychological beliefs compared to controls. The extent to which these differences are a function of chronicity of pain is unclear, and it is possible that rather than changing over time, the contrast in beliefs may be apparent *before* the patients become chronic pain sufferers. In other words, beliefs may be one factor which distinguishes those individuals at risk for developing a chronic pain condition. Blumer and Heilbronn (1982) proposed a model

of chronic pain in which pain is a somatic expression of repressed emotion conflict, and assumed that psychosocial disturbance was a significant factor in the onset of pain. Clearly this is an extreme point of view, and has since been strongly refuted. For example Gamsa (1990) found that emotional disturbance was ~~more~~<sup>less</sup> likely to be a precipitator than a consequence of chronic pain. More recently, Gamsa and Vikis-Freibergers (1991) have argued that psychological factors are both risk factors in, and consequences of chronic pain. They demonstrated an association between chronic pain and less emotional repression and excessive work habits ("ergomania"). These results are in direct conflict with Blumer and Heilbronn's concept of a "pain prone" personality. However, these were retrospective, correlational studies, and therefore it is not possible to conclude causality in either direction.

The studies presented in Chapter 5, through employing a prospective research design, were able to demonstrate causal links between beliefs and a number of pain-related measures. In a group of patients with chronic pain of a variety of aetiologies (who may therefore be considered generally representative of chronic pain sufferers) causal relationships were found between psychological beliefs and depression, positive pain-related cognitions and several indices of activity. In each case increases in endorsement in psychological beliefs were associated with improvements in psychological and physical functioning. Organic beliefs were found to be causally related to hopelessness cognitions and health locus of control beliefs. Decreases in emphasis on this class of beliefs was associated with reduction in the frequency of hopelessness cognitions, heightened internal locus of control and lowered reliance on chance factors and powerful others in controlling health. The majority of these

relationships were in both directions, suggesting that a vicious circle may be operating, with a large number of disparate factors playing important roles in both precipitating and perpetuating chronic pain. Thus it is likely that conceptual processes are able provide an index of vulnerability, where schematic processes (as measured by memory function) were not.

Clinically, the importance of these findings lies in the need to be able to predict which individuals are at risk of developing chronic pain, after, for example a back injury or illness, on the basis of non-medical variables such as beliefs, attitudes, coping styles and emotional state. Appropriate interventions could then be targeted.

In conclusion, the distinction between schematic and conceptual processing has provided a useful starting point for the examination of cognitive processes in chronic pain. Where it suffers is in its failure to propose the exact mechanisms underlying these levels of processing, and in the nature and extent of their interaction. Future research might be directed at exploring these interactions.

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## Appendix A

# PAIN BELIEFS QUESTIONNAIRE

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*For each item please indicate your opinion by underlining one of the following words in each sentence:*

*always / almost always / often / sometimes / rarely / never*

*There are no right or wrong answers: it is important that you respond according to your actual beliefs, not according to how you feel you should believe or how you think we want you to believe.*

*Please make sure that you answer ALL the questions.*

---

- 1) Pain is (always/almost always/often/sometimes/rarely/never) the result of damage to the tissues of the body.
- 2) Physical exercise (always/almost always/often/sometimes/rarely/never) makes pain worse.
- 3) It is (always/almost always/often/sometimes/rarely/never) impossible to do much for oneself to relieve pain.
- 4) Being anxious (always/almost always/often/sometimes/rarely/never) makes pain seem worse.
- 5) Experiencing pain is (always/almost always/often/sometimes/rarely/never) a sign that something is wrong with the body.
- 6) Being in pain (always/almost always/often/sometimes/rarely/never) prevents you from enjoying hobbies and social activities.
- 7) When relaxed pain is (always/almost always/often/sometimes/rarely/never) easier to cope with.
- 8) The amount of pain is (always/almost always/often/sometimes/rarely/ never) related to the amount of damage.
- 9) Thinking about pain (always/almost always/often/sometimes/rarely/never) makes it worse.
- 10) It is (always/almost always/often/sometimes/rarely/never) impossible to control pain on your own.
- 11) Pain is (always/almost always/often/sometimes/rarely/never) a sign of illness.
- 12) Feeling depressed (always/almost always/often/sometimes/rarely/never) makes pain seem worse.