COGNITIVE AND FACIAL STRATEGIES IN THE CONTROL OF EXPERIMENTAL PAIN

by

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ABSTRACT

An investigation was conducted to test the effect of cognitive strategies and changes in facial expression in the control of experimental cold-pressor pain.

Forty-four subjects were divided into four groups matched for sex and age:

1. a cognitive strategy group, instructed to re-interpret pain as cold;

2. a facial strategy group, instructed to 'hide' the facial expression of pain;

3. a combined strategies group which carried out both strategies simultaneously;

4. a no-treatment control group.

A number of factors known to correlate with pain were measured by standardized tests to control for any initial differences in group composition. Experimental measures consisted of a pain threshold measure (immersion time), physiological correlates of pain (heart rate, respiration rate, inspiration-expiration ratio) and Ss' pain ratings on a modified version of the McGill Pain Questionnaire (MPQ).

It was hypothesized that both the cognitive strategy and facial strategy would have a significant effect in controlling pain and that the combined strategies would prove the most effective of the treatments.

Experimental results indicated that only the cognitive strategies had a significant effect on immersion times as compared to

controls. None of the experimental groups differed from controls on MPQ ratings. There were no significant differences between groups on the physiological response measures.

The results were discussed in terms of implications for the hypotheses. The experimental method and the adequacy of the measures used, especially the MPQ, were discussed. The implications of the results for the control of clinical pain were elaborated.

ACKNOWLEDGEMENTS

I would like to express my appreciation to my supervisor, Dr. George Wilson, for his patient and thoroughgoing assistance. I also owe thanks to James Alexander, John Wanless, my subjects and my wife, Joy.

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CHAPTER 1

Introduction

Clinical pain may take many forms. It may be severe or mild, unremitting or occurring only at intervals. In cases of mild to moderate pain lasting a relatively short time but occurring consistently (e.g., some phantom limb pain) conventional medical treatment such as analgesic drugs, nerve blocks and surgical section of nerve tracts may be of limited usefulness (Melzack, 1973; Weisenberg, 1977). Possible side-effects may render long-term analgesic use undesirable or larger doses may be required as the person's body adapts to the drug (Melzack, 1973). Medication taken only when pain occurs may be ineffective because of the delay in the onset of action of the drug. Surgical procedures (e.g. rhizotomy) may not prove effective or may not be considered justified (Melzack, 1973). In such cases psychological methods of pain control assume importance. Indeed, Casey and Melzack (1967) speculate that psychological methods of pain control may in time become far more powerful and be more widely used. It is the aim of this thesis to refine and extend some of these psychological methods by examining the relative effects of two pain control strategies. The first is the relatively well-established technique using cognitive strategies (e.g., Beers & Karoly, 1979; Scott & Barber, 1977 a & b; Spanos, Horton & Chaves, 1975). The second employs the manipulation of facial expression and is an extension of work by Kleck and colleagues (e.g., Lanzetta, Cartwright-Smith & Kleck, 1976; Colby, Lanzetta & Kleck, 1977).

In order to approach this task the various approaches to the definition of pain will be presented and the measurement of pain and theories of its origin and transmission will be discussed. Most stress will be laid on pain theory which makes allowance for psychological influences. There will also be a review of the relevant areas of the literature concerning psychological factors in pain control.

Definitions of Pain

Sternbach (1968), in defining pain, states that it can be seen in these ways concurrently, that is as:

1. a private sensation of hurt,
2. a harmful stimulus signalling current or impending tissue damage,
3. a pattern of responses operating to protect the organism from harm.

Phenomena such as post-herpetic neuralgia where pain occurs in the absence of current or impending tissue damage lie outside the scope of this definition. Nor is there any reference to psychological factors in the pain response.

Hardy, Wolff and Goodell (1952) define pain as a sensation of hurt and classify cognitive and emotional aspects of pain as reactions to the pain sensation (the term 'pain experience' being applied to pain in all its aspects). Pain sensation is seen as a response to noxious stimuli.

Perhaps the most useful current definition of pain is that of Melzack (1973). Pain is conceptualized in terms of a multi-

dimensional space comprising "those subjective experiences which have both somatosensory and negative-affective components and that elicit behavior aimed at stopping the conditions that produce them" (Melzack, 1973, p.46). Melzack holds that all of these conditions are necessary in pain, thus acknowledging the essential place (using different terminology) of a private sensation of hurt. Melzack has not attempted to relate pain to tissue damage in this definition (either in terms of response to it or escape from it). In this definition, unlike the others, explicit reference is made to the role of psychological factors in pain perception and pain behaviour.

For ongoing research, particularly experimental work, operational definitions of pain are necessary to aid in conceptualization and to provide a basis for measurement. Pain in this study is defined and measured in terms of withdrawal from the painful stimulus and a verbal rating of the pain.

Theories of Pain

In the following section the more important current theories of pain will be reviewed and an attempt made to trace recent theoretical developments, particularly as these relate to psychological factors in pain perception.

Specificity Theory of Pain

Von Frey (1895) and later researchers (e.g., Head, 1920; Keele, 1957) postulated four sensory modalities - warmth, cold, touch and pain - and proposed that each modality had specialised receptors in the body. For each modality, specific nerve pathways project to

particular areas of the brain. In this model the pain tract comprises free nerve endings in the skin (as well as around hair roots), Aδ and C fibres in peripheral nerves, the lateral spinothalamic tract and a pain centre in the thalamus (Melzack & Wall, 1965).

The main strength of specificity theory lies in apparent physiological specialisation (i.e., the role of Aδ and C fibres) yet the proposed simple and direct relationship between pain stimulus and pain response has been repeatedly challenged on physiological grounds (e.g., Head, 1920; Mayer, Wolfle, Akil, Carder & Liebeskind, 1971; Melzack, Stotler & Livingston, 1958; Weddell, Palmer & Paillie, 1955). Wall (1978) was still able to write that pain fibres (defined as those always and only carrying pain information) had not been demonstrated. He does concede that large numbers of nociceptive fibres (carrying pain and non-pain information, depending on intensity), appear to exist.

The lack of a simple and direct relationship between pain stimulus and response is also shown by such clinical phenomena as phantom limb pain, causalgia and peripheral neuralgia (Melzack, 1973).

The psychological evidence against specificity theory is also strong. Beecher (1959) showed that a person's motivational state could affect reports of pain even after severe wounds. Many other psychological factors have been shown to be related to pain response, including anxiety, personality traits, social class, and cultural group (Barnes, 1975; Sternbach & Tursky, 1965; Tursky & Sternbach, 1967; Weisenberg, 1977; Woodforde & Merskey, 1972 ; Zborowski, 1952). There is also evidence against specificity

theory from the literature on psychological methods of pain control (e.g., Weisenberg, 1977).

Thus, specificity theory cannot cope with known pain phenomena; some allowance for internal modulation of pain must be made.

Pattern Theory

In general, pattern theories of pain deny receptor specialisation and state that the sensation felt depends on the patterning of the input (i.e., discharges form a code, interpreted centrally). Such theories tend to fall into two categories, stressing either peripheral patterning or central summation of input.

Peripheral patterning. In peripheral patterning theories, pain sensations are considered to vary according to different discharge patterns of nerves, the number of nerves discharging and the location of receptors. Pain is usually said to be felt whenever any kind of stimulus (light, heat, pressure, etc.) is too intense. Sinclair (1955) and Weddell (1955) have proposed theories of this type. Peripheral pattern theory fails because it does not take account of known receptor specialisation, in particular, that nociceptive fibres are usually delta and non-myelinated fibres.

Central summation theories. The above fault is avoided by pattern theories emphasising central summation (usually in the dorsal horn of the spinal cord). One such theory is that of Livingston (1943) dealing with phantom pain. Yet the effects of surgical section of nerve tracts fail to support this theory (Melzack, 1973). As Melzack and Wall (1965) also point out, none

of the pattern theories is fully comprehensive and there is little experimental verification, especially of central summation theories. Nor again is there any allowance for psychological factors except in Livingston's (1943) theory, where that allowance is inadequate.

Gate Control Theory

Scope of theory. Gate control theory (Casey & Melzack, 1967; Melzack, 1973; Melzack & Casey, 1968; Melzack & Wall, 1965; Wall, 1978) represents both an integration and an extension of much previous pain theory. It also takes into account other approaches to pain such as Marshall's (1894) view of pain as an emotion by recognising the negative affective quality of pain. In giving much more weight to the non-sensory aspects of pain the gate control theory greatly extends Beecher's (1959) shift from the strictly sensory view of pain of Hardy, Wolff and Goodell (1952). Beecher has concluded that cognitive and emotional aspects are integral with pain rather than reactions to it. Gate control theory goes further and proposes that the various aspects of pain operate in parallel. It does not give primacy to the sensation of pain.

Account of theory. As first proposed by Melzack and Wall (1965), gate control theory contained three basic postulates:

1. that the substantia gelatinosa (the second and third laminae of the dorsal horn of the spinal cord) exerts a gating effect on pain sensation;

2. that afferent impulses in the dorsal column allow for central influence on the gate;

3. that 'T cells' (first central transmission cells in the dorsal horn) activate the systems concerned with the perception of

and response to pain.

With regard to the first postulate, Melzack and Wall (1965), extended the work of Noordenbos (1959). It was postulated that excitation of the substantia gelatinosa by large sensory fibres led to pre-synaptic inhibition whereas excitation by small fibres reduced pre-synaptic inhibition; therefore large fibre activity can block the slower 'pain' fibre activity. The gate is normally kept relatively open by the small, more slowly adapting, more tonically active fibres (the larger fibres tend to adapt more quickly and may be inactive in the absence of change in stimulation). There is however, usually enough large fibre activity to prevent spontaneous pain. Pain felt depends on the initial level of activity, activity following pain stimulation and the relative balance of large and small fibre activity.

The second postulate concerns central influence on the gating mechanism. It is known that central efferent activity can inhibit somaesthetic afferent conduction (Melzack, Stotler & Livingston, 1958). Melzack and Wall (1965) propose that this takes place via the gate mechanism.

Melzack and Wall (1965) also propose the existence of a central control trigger capable of selective activation of brain processes that inhibit pain. Two known afferent pathways - the dorsal column-medial lemniscus system and the dorsolateral pathway - are capable of this role. The central control trigger constitutes the feed-forward section of the feedback loop to the brain.

The third postulate states that T cells activate systems

responsible for perception and response to pain. Activation occurs when T cells reach a critical firing-level over a period of time. It is proposed that both spatial and temporal summation of impulses occurs in the T cells. This accounts for such phenomena as altered pain threshold following prior stimulation.

Casey and Melzack (1967) and Melzack and Casey (1968) extend the above account of the action system and attempt to provide a neurophysiological basis for several facets of pain experience. Two extra sub-systems other than that of central control are postulated: the sensory/discriminative and motivational/affective sub-systems.

The sensory/discriminative component provides information concerning the spatial and temporal properties of the stimulus as well as its intensity. Casey and Melzack (1967) propose that this processing could take place in the ventrolateral nuclei of the thalamus and in the somatosensory cortex, having been projected there from the T cells via the neospinothalamic projection system.

Processing for the motivational/affective system probably takes place in the reticular core of the brain stem and in the medial thalamus. This area is close to and has many connections with the 'limbic' system around the upper brain stem. The limbic system is known to play a role in aversive drive, emotional and pain-related behaviours (Delgado, Rosvold & Looney, 1956; Foltz & White, 1962; Schreiner & Kling, 1953). Information is projected to this area via the paramedial ascending system comprising spinoreticular, spinomesencephalic and palaeospinothalamic components of the anterolateral somatosensory pathway. Activation of

this system leads to drive and unpleasant affect in turn leading to action (though stimulation below a certain level may lead to positive affect and approach behaviour).

The account of the central control system itself is largely unchanged from that of Melzack and Wall (1965). Central influences are highly selective, for example, excitement may affect both the sensory/discriminative and motivational/affective dimensions while placebo or certain psychological techniques may affect only the latter. Given the complexities of pain response it is likely that there is much cortical involvement. It appears that information may reach the central control system first, there to influence the other pain dimensions directly or to do so indirectly via the gate mechanism. A requirement here is fast input; this is achieved through the central control trigger described above. Diagrams of the gate control system as a whole are shown in Figure 1, while a detailed description of the action system is shown in Figure 2.

The three sub-systems of the action system interact to provide perceptual information, motivational tendency and cognitive information based on past experience, including probable outcome of various responses to noxious stimuli. This interaction determines the person's pain response.

Nathan (1976) has comprehensively detailed six main areas of criticism of the gate control theory.

1. the evidence for hyperpolarization at the first synapse,

2. the inhibition of small fibre activity by large fibre activity,

3. the failure to take account of stimulus specificity in

FIGURE 1

Schematic Diagram of the Gate Control System (Melzack, 1973)

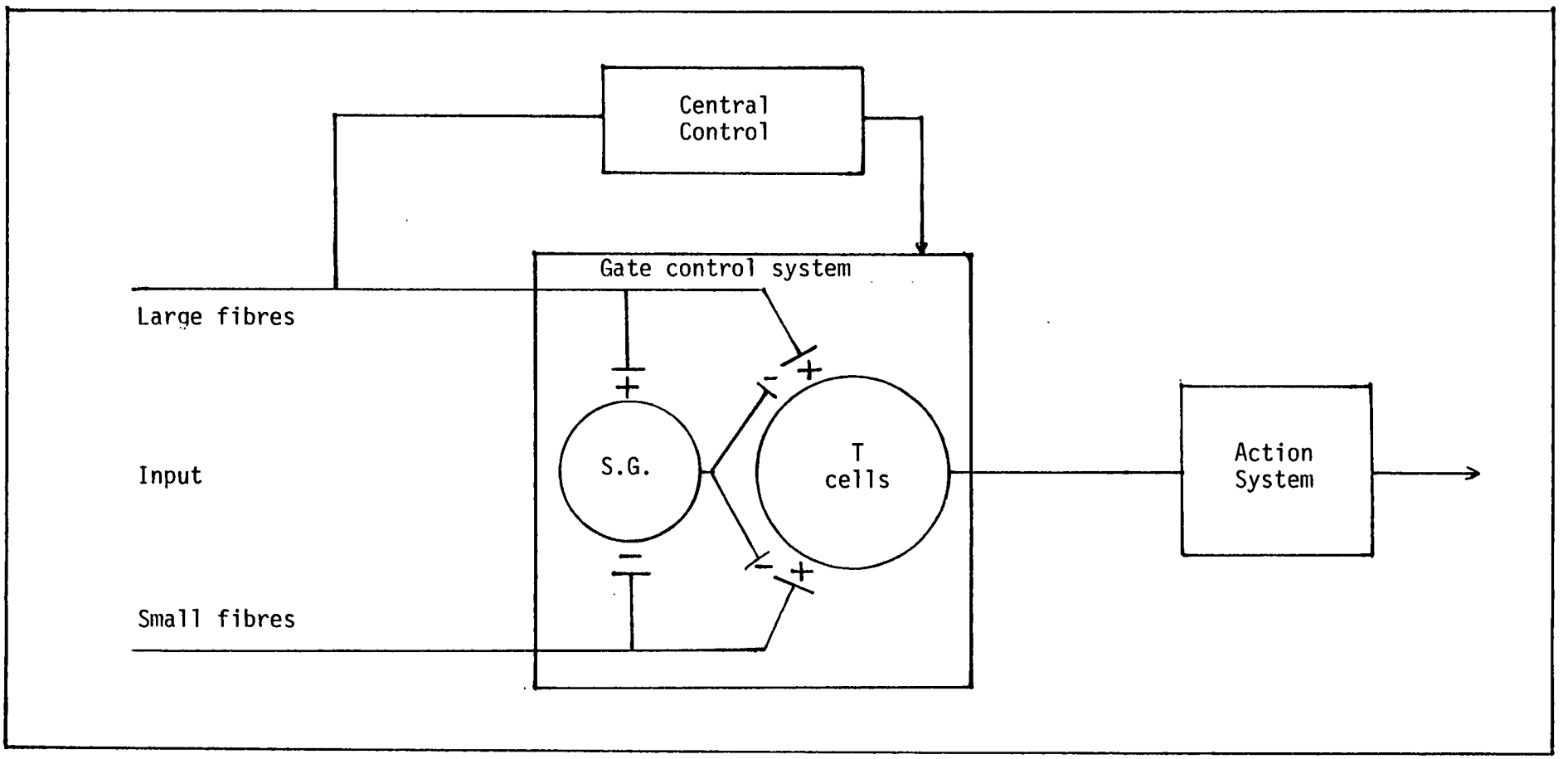
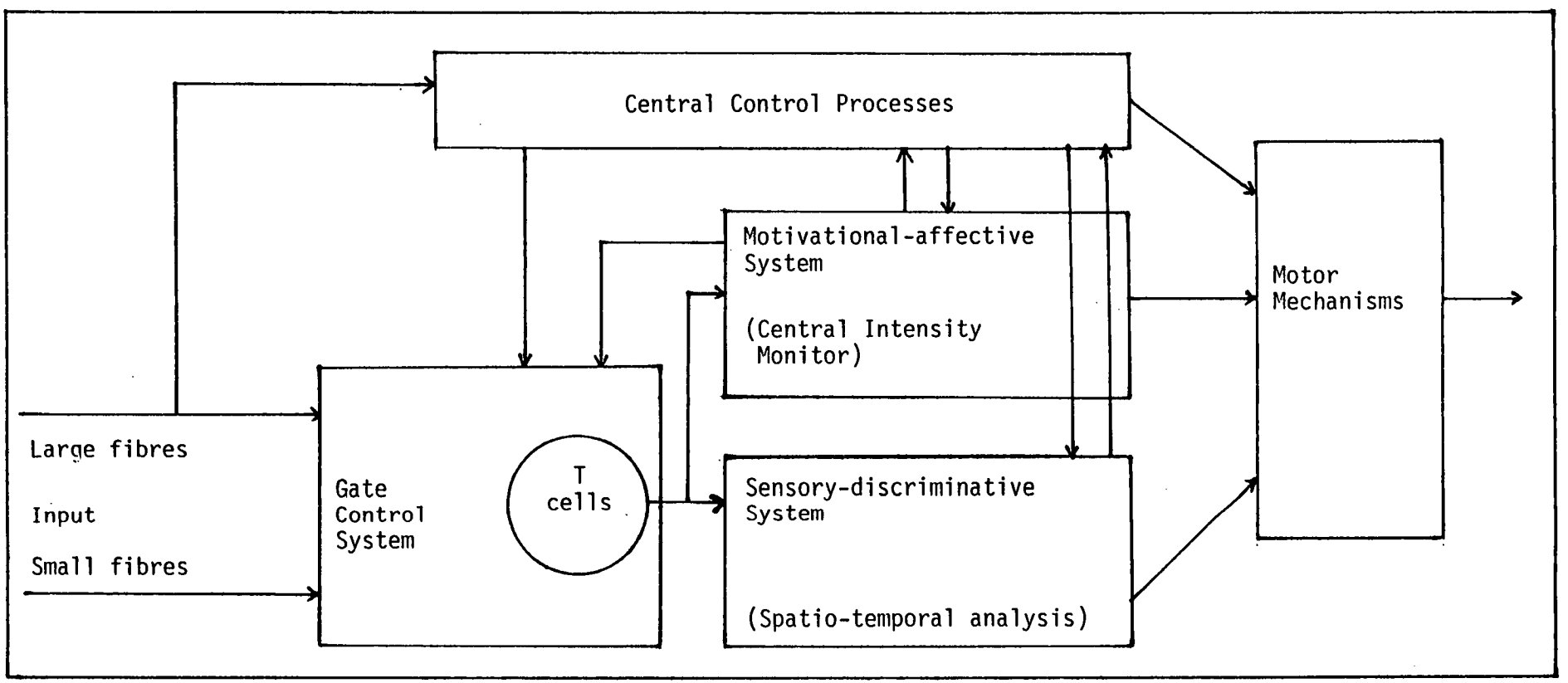


FIGURE 2

Schematic Diagram of the Cognitive, Motivational-affective and Sensory Determinants of Pain (Melzack, 1973)



peripheral fibres,

4. the lack of allowance for different types of pain,

5. pain occurrence in the peripheral neuropathies not as predicted by gate control theory,

6. the applicability of the theory to human beings (since much basic experimental work used decerebrate cats).

Nathan (1976) should be consulted if specific arguments and evidence are required for further elaboration.

Overview of gate control theory. Gate control theory derives its strength partly from the fact that it is largely stated in testable form (and has in fact generated much research). It is also robust in that it draws together into a comprehensive neurophysiological theory a great many normal and abnormal pain phenomena and many explanatory mechanisms. It makes use of the known characteristics of large and small peripheral fibres but at the same time allows for variation in the intensity of stimulation and for summation of information. Most importantly, it incorporates the idea of inhibition of pain fibre activity. Later additions to the theory provide a possible mechanism for the different facets of the pain experience - sensation, negative affect, avoidance. It is a dynamic theory stressing the plasticity of the pain experience. Lipton (1979) suggests that gate control theory could form part of an even broader theory of pain. Such a theory would have to include the important recent biochemical work on the endogenous opioid neuropeptides such as enkephalin, the endorphins and substance P (Duggan, 1979; Henry, Sessle, Lucier & Hu, 1980; von Knorring, Almay, Johansson & Terenius, 1978; Olson, Olson, Kastin & Coy, 1980).

Most research generated by gate control theory has focussed on

the peripheral and spinal mechanisms yet it is the account of the action system (Casey & Melzack, 1967; Melzack & Casey, 1968) which is of crucial importance to the present thesis. In specifically setting out a mechanism for central influence, great scope is left for the investigation of psychological influences on pain and of psychological methods of pain control.

CHAPTER 2

Measurement of Pain

In any scientific investigation of pain, the question of the measurement of pain arises. This is far from straightforward, due ultimately, as noted earlier, to the fact that pain is a subjective experience and can only be measured indirectly. In order to discuss the many measures that have been devised it is useful to distinguish between clinical pain and pain that is induced experimentally.

The differences between clinical and experimental pain arise from their origins. Clinical pain is likely to be sufficiently severe to have led to the person seeking treatment for it, its physiological origin may not be known, nor may its implications for the person's future well-being. It may not be known how long it will continue or whether treatment for pain will itself be painful. Clinical pain is frequently associated with a high level of anxiety, partly for the above reasons. Experimental pain, on the other hand, is usually of known type, intensity and duration. In normal circumstances the subject knows no permanent harm will come to him and the pain carries no implications concerning his future health. Experimental pain is likely to be less severe and of shorter duration than clinical pain and is associated with lower levels of state anxiety.

Though experimental pain can be more closely controlled, described and measured, its relevance to the study of clinical pain has been questioned. For example, Beecher (1959) considered that

clinical and experimental pain were different in that only clinical pain appeared to respond to morphine. He attributed this to the 'reaction component' (partly anxiety) evident in clinical pain. However it is likely that the experimental pain Beecher used was less severe than the clinical pain to which it was being compared. It has subsequently been shown that severe experimental pain does respond to narcotic analgesics (Wolff, Kantor, Jarvik & Laska, 1966). Beecher (1966) attributes this still to greater levels of anxiety though Sternbach (1968) argues that in the absence of independent measures of anxiety and a demonstration of the relationship of anxiety to other variables this is not an adequate explanation. In any case, as Wolff (1978) points out, milder experimental pain (not associated with great anxiety) has been shown to be relieved by the less potent analgesics, e.g. aspirin.

Wolff (1971) compared surgery patients on a variety of experimental and clinical pain measures and factor analysed the results. He concluded that there was evidence of a 'pain endurance' factor. The main experimental measure contributing to this factor was the pain sensitivity rating (described later). An independent study by Timmermans and Sternbach (1974) provides support for this hypothesis. The study by Crocket, Prkachin and Craig (1977) found pain dimensions common to both experimental and clinical pain.

Thus it seems that clinical and experimental pain have much in common. The value of studying experimental pain may depend partly on the appropriateness of the pain induction method used. Experimental pain has the further significant advantage that it permits accurate measurement both of pain stimulus and pain response. These considerations strongly support the utility of experimental pain

research.

The current measures of experimental pain fall into a number of groups - psychophysical, verbal, physiological. Because of the complex variations found in pain response it is desirable to obtain several independent measures both to cross-validate the measures themselves and to provide more information on the pain response.

Psychophysical Measures

The three potentially most useful psychophysical measures for this study are pain threshold, pain tolerance and signal detection theory (SDT). The latter initially appears to be capable of providing more information than the other two measures. SDT attempts to separate sensory and response bias aspects of response (including motivational, emotional and learning factors) by requiring Ss to detect the presence of a signal against a background of noise. Thus there would seem to be a possibility of determining whether particular experimental manipulations change sensitivity or response bias or both (Lloyd & Appel, 1976). Many SDT studies have been carried out, for example, Dougher (1979), Clark and Goodman (1974), Clark and Mehl (1971), Chapman, Murphy and Butler (1973).

McBurney (1975), however, made the point that whereas the basic SDT model attempts to measure absolute sensitivity (by separating response bias and sensitivity, in the application to pain only differential sensitivity could be measured. This is because a pain stimulus has to be at least at threshold point (by definition). Problems arise because absolute and differential sensitivity may

vary and thus the applicability of the SDT model to pain may be questioned.

Rollman (1977) made a similar point in distinguishing between detection and discrimination. He concluded that it could not be held that the resulting measures are of sensitivity and response bias. This criticism has not been satisfactorily answered though Clark, Yang and Hall (1975) argue that McBurney's (1975) criticism does not apply to their experiment. Rollman (1977) also asserts that SDT pain researchers have claimed that SDT can separate sensitivity from emotional factors as such. Chapman (1977) convincingly denies that this is so by referring to the pain model used by his research group (a model derived from Casey & Melzack, 1967). Rollman's other criticisms: slow data collection, training of Ss, difficult statistical analysis and E's theoretical background, are problems common to other areas of psychophysical research and can be coped with in practice.

The validity of the application of SDT to pain research has been questioned and the argument continues. Gracely (1979), for example, holds that d' is not just a measure of sensitivity but includes a cognitive component. β similarly, as well as being a measure of response bias and expectations may also reflect changes in the affective quality of the stimulus apart from sensory effects. Other recent articles on the application of SDT to pain research are by Jones (1979) and Rollman (1979).

A long established psychophysical measure in pain research (e.g. Hardy, Wolff & Goodell, 1952) is the pain threshold, defined by Wolff (1978) as 'that point at which S just begins to feel pain

in an ascending trial or at which pain just disappears in a descending trial' (p.150). Pain threshold is also defined as 'that point where pain is felt on 50% of trials' (Wolff, 1978, p.150). Pain is described by reference to stimulus parameters (Wolff, 1978, 1980).

The validity of pain threshold as a measure of pain has been questioned on the basis of unreliable results in cross-model studies (Wolff, 1978) and because pain threshold is often difficult to establish (Merskey & Spear, 1964). Yet threshold measures have been shown to be sensitive to non-narcotic analgesics (Wolff, 1980). The reliability of pain threshold as a measure may depend to some extent on the pain-induction method used (Wolff, 1978) and on being taken over a number of trials.

Procacci (1979) states that pain thresholds can be reliably measured subject to four conditions:

1. adequate training of S,
2. use of verbal measures of pain and adequate experimental controls,
3. non-damaging pain induction,
4. control of other factors, for example, circadian rhythms.

It has been hypothesised (Beecher, 1959; Gelfand, 1964) that pain threshold reflects largely sensory response while pain tolerance (to be described) has a greater psychological component. Blitz and Dinnerstein (1968) showed that this may be due partly to experimental instructions. In their experiment they were able to change both pain threshold and tolerance with appropriate instructions. There is evidence that pain threshold varies according to a

number of factors, e.g. several cognitive strategies (Beers & Karoly, 1979) and subject control of the administration of experimental pain (Bowers, 1968).

The third major psychophysical measure of pain is pain tolerance which may be defined as that point at which S will no longer tolerate the pain induced and withdraws from or makes a signal for the termination of the stimulus. For ethical reasons, stimulation is usually not increased or continued past this point (simply because there is an implicit agreement by E not to do so). Indeed, there are ethical considerations even in inducing this much pain. Nevertheless the pain induced to reach tolerance would usually be much less than in severe clinical pain. Reliable pain tolerance measures can be obtained with several pain induction methods such as those using radiant heat and electric shock (Wolff, 1978). As with threshold, validity on the basis of cross-modal matching is equivocal (Wolff, 1978) but tolerance may compare better with clinical pain and has proved a useful measure in analgesic assays (Wolff, 1977).

In view of the controversy regarding the use of SDT in pain research, the choice of psychophysical measure lies between threshold and tolerance measures. Because threshold measures the lower limit of pain experience and tolerance measures the upper threshold endured, tolerance is considered to bear more relation to clinical pain. A threshold measure may be more relevant where mild and moderate pain is being studied. This is especially so in view of its usefulness in research on milder analgesics (Wolff, 1980).

A further argument for the use of a threshold measure rests on the finding of Barber and Cooper (1972), Barber and Hahn (1962) and Blitz and Dinnerstein (1971) that the effect of cognitive strategies appears most powerful early in the exposure to the noxious stimulus. It is likely that cognitive strategies would be better indexed by a threshold rather than tolerance measure of pain.

The last reason for the use of a threshold measure is an ethical one: that it is difficult to justify the induction of severe pain in the present exploratory study.

The reliability of a threshold measure should be satisfactory provided the requirements set out by Procacci (1979) and Wolff (1978) are fulfilled. That is, measures should be taken over a number of trials, extraneous factors controlled and Ss given prior exposure to the stimulus.

Physiological Measures

Experimental pain is associated with changes in physiological variables and like the emotions of anger and fear, may give rise to the pattern of autonomic changes preparing the body for fight or flight (Cannon, 1929). Chronic clinical pain may be associated with the hormonal stress reaction of Selye (1946, 1956).

Detailed information on the effect of particular painful stimuli is also available, for example, that of Wolf and Hardy (1943) on reaction to exposure to ice water (cold pressor pain). They found increased systolic and diastolic blood pressure,

increased pulse rate, decreased finger pulse amplitude. They concluded that the response pattern appears primarily to be to pain rather than to cold since administration of analgesics reduced changes in the physiological indicators. Schachter (1957) found a noradrenaline-like response pattern in cold pressor pain. This response is consistent with Wolf and Hardy's (1943) findings as a simple cold stimulus would be expected to produce an adrenaline-like pattern, part of which is superficial vaso-constriction and conservation of body heat.

Engel (1959) confirmed Wolf and Hardy's (1943) results on heart rate, blood pressure and peripheral vasoconstriction. He found no significant differences in skin temperature at three sites, in skin conductance, or in respiration (though measures tended upwards in the latter two). There were some differences in the pattern of changes between experimental sessions.

However, it has also been known for some time (Lewis, 1929) that if immersion continues then reflexive vasodilatation may occur. When a limb is immersed for an extended period, phasic dilatation (termed cold-induced vasodilatation) occurs on a background of strong vasoconstriction. Both phenomena are associated with reports of pain. It has more recently been shown (Teichner, 1965, 1966) that there are individual differences in the occurrence of cold-induced vasodilatation and that the phenomenon can be influenced by a number of factors such as ambient temperature (Teichner, 1965, 1966) and threat of shock (Teichner, 1965).

In studies using cold pressor pain at least some of the above cardiovascular measures should be useful. Respiration also, since

it is under voluntary control, needs to be monitored since it is known that respiration itself interacts with all cardiovascular responses (Greenfield & Sternbach, 1972; Sternbach, 1968).

Verbal Measures of Pain

In research on clinical pain, verbal measures are widely accepted as simple and direct (Beecher, 1959; Sternbach, 1968; Melzack, 1973; Wolff, 1978, 1980). Other measures are used too, for example, ratings of pain based on a number of factors - complaints of pain, apparent comfort of patient, physical signs. Verbal report has been less frequently used in the assessment of experimental pain. The main emphasis here has been on the precise description of the pain stimulus, on psychophysical measures and on the physiological correlates of pain. Yet verbal report has its advocates with experimental pain; for example, Hilgard and Hilgard (1975) regard verbal reports as the most lawful and reliable measure of cold pressor and ischaemic pain.

Verbal measurement varies according to its sophistication. Ss may be asked simply to report the presence or absence of pain or to estimate the degree of pain. The latter may be achieved by asking S to estimate its magnitude relative to previous pain. This is a direct scaling technique. A development of it is cross-modality matching where another measurable response (e.g. handgrip force) is matched to the pain felt (Gracely, 1979). Or S may place pain on a scale with a number of fixed points. These may be given numbers or particular descriptors (e.g. mild, moderate, severe) and are known in pain research as categorical and verbal rating scales, respectively. A more recent development in pain research is the visual

analogue scale in which S estimates pain by marking any point on a scale with only the end-points marked. The end-points are usually 'no pain' and 'pain as bad as it could be' or an equivalent term (Huskisson, 1974).

All three of these scales have produced acceptable results but it is not clear which is best, or whether one or the other is better for particular situations. Gracely (1979) reviews the research comparing verbal rating scales and visual analogue scales and concludes that visual analogue scales are probably not markedly more reliable than verbal rating scales though several authors (Ohnhaus & Adler, 1975; Scott & Huskisson, 1976) consider them superior. In other areas of psychological research well-designed rating scales have been shown to be highly reliable (Anastasi, 1968).

Verbal measures of pain also vary according to their theoretical base. The McGill Pain Questionnaire (MPQ; Melzack, 1975; Melzack & Torgerson, 1971) reflects this and was devised for the assessment of clinical pain. There are three sections where S locates his pain on a diagram of the body, states how it changes across time and rates its intensity (the last two sections being verbal rating scales). In a fourth section S selects words from 20 groups to provide a description of the pain in terms of four categories: intensity (the words in each group are ranked according to intensity), sensory, affective and evaluative dimensions of pain. The latter three categories parallel the dimensions of pain proposed by Melzack and Wall (1965) and Casey and Melzack (1967) in the gate control theory of pain. The MPQ, being multidimensional, provides a broad account of pain felt.

Much research has been undertaken on the reliability and validity of the MPQ, the bulk of which is in the clinical area. For the most part these studies confirm the ability of the MPQ to measure pain and the utility of the sensory, affective and evaluative dimensions (e.g. Nehemkis, Charter, Stampp & Gerber, 1981; Graham, Bond, Gerkovich & Cook, 1980; Prieto, Hopson, Bradley, Byrne, Geisinger, Midax & Marchiselo, 1980; Dubuisson & Melzack, 1976; Melzack, 1975). Reading,Everitt and Sledmere (1981) in a replication of the construction of the MPQ using different methods (card sort, cluster analysis and independent ratings) found considerable overlap between their word-groups and those of the MPQ.

Several factor analytic studies have produced factors differing from the dimensions of the MPQ. Reading (1979) found most support for the sensory dimension with less for each of the others. He felt though that the type of pain studied (in this case dysmenorrhoea) may have affected the factors extracted.

Crocket, Prkachin and Craig (1977) and Leavitt, Garron, Whisler and Sheinkop (1978) found support for the affective and sensory dimensions but not for the evaluative one. These studies have methodological problems however. The Crocket study uses heterogeneous groups of Ss, instructions and pain aetiologies so that spurious factors are possible. The Leavitt study used too low a ratio of Ss to items in their factor analysis and a factor analysis method that often produces spurious results (Prieto et al, 1980).

Martinez-Urrutia (1975) confirmed the usefulness of the sensory

dimension and its relation to state anxiety (i.e. a positive correlation post-surgically) but found no significant changes or interactions in the evaluative dimension or on intensity. However, in an apparent attempt to improve the MPQ psychometrically, the word-groups in the fourth section had been much altered (16 of the 20 word-groups were either reduced to four words or were eliminated if they had contained less than four words). In the process all the word-groups for the affective category were eliminated so that this dimension could not be measured at all. Van Buren and Kleinknecht (1979) found significant changes before and after oral surgery in all measures except the affective dimension where there was a non-significant change. They also found a correlation between anxiety and the MPQ measures. They suggested that more psychometric work was necessary to provide a better separation of the dimensions. The clinical studies provide good support for the sensory and rather less support for the affective and evaluative dimensions of pain. Where reported, the intensity measure (from a separate verbal rating scale) is also well supported. The studies also show that the MPQ needs further refinement.

Crocket et al (1977) included experimental pain in their study but, as noted, pooled the data from both clinical and experimental Ss before analysis. The only other study of the use of the MPQ in experimental pain is by Klepac, Dowling and Hauge (1981). Two types of pain - cold pressor and electrical tooth pulp stimulation - are used. The study provides strong confirmation of all the MPQ measures except the Number of Words Chosen in the final section. This was intended to be an extra measure of intensity. The study also shows clear differences between the MPQ results for cold pressor and tooth pulp pain, with the former being found more severe.

Generally, the measurement of pain is likely to be more valid and reliable where several different types of measures - psychophysical, verbal and physiological - are taken and their interrelationships studied.

Pain-Induction Methods

In general the requirements of experimental pain are that it be reliable, valid, convenient, repeatable, clear cut and having one pain quality only. The pain stimulus should be non-damaging, closely measurable and able to be finely controlled over a large range (Hardy, Wolff & Goodell, 1952; Beecher, 1959). Beecher (1959) also lists several requirements pertaining to drug studies only. There are a number of experimental pain-induction methods each with different characteristics, advantages and disadvantages.

Electrical pain. Electrical stimulation (e.g. Barber & Hahn, 1962; Blitz & Dinnerstein, 1968; Lanzetta, Cartwright-Smith & Kleck, 1976), providing that correct voltage, etc., is used, is quite safe. Fine control of degree and duration of stimulation is possible. Its reliability is high if due regard is paid to current characteristics (wave form, constant current), type of electrode used and part of body stimulated (which may be cutaneous, sub-cutaneous or visceral). However electrical stimulation does not lend itself to a slow build-up of pain. Ss also frequently classify it as discomfort rather than pain in experimental situations (Wolff, 1978).

Heat pain. Induction of pain by the application of radiant heat is a long-established method with good reliability and validity

(Hardy et al, 1952; Wolff, 1977, 1978), though there are some disadvantages. The most important is the possibility of tissue damage in the single trial method where over a constant time the amount of heat is varied until threshold or tolerance is reached. Where the method of limits is employed, only ascending trials can be used and use of this method is also very slow. In Wertheimer's (1952) variation of the method, holding heat constant and varying exposure time, threshold may be reached very quickly - in one to three seconds (Wolff, 1977).

Cold pressor pain. A second thermal pain-induction method utilises the cold pressor response (Hines & Brown, 1932) and usually requires that S immerses a hand or foot in near-freezing water. Like heat pain, stimulation is cutaneous yet the pain itself is not simply cutaneous but is of a deep, aching nature. (A burning skin pain often occurs too with cold-induced vasodilatation). Unlike radiant heat pain though, there is little possibility of damage to S. The major disadvantages of cold pressor pain are that it is slow to administer (since the limb must return to normal before further pain stimulation) and that it is less reliable than some other pain induction methods, e.g. of electrical stimulation and heat (Wolff, 1977, 1978).

The advantages of cold pressor pain lie partly with the familiarity of the stimulus (so that it should not arouse much anxiety) and partly in its excellent validity. Wolff (1978) submits that this outweighs considerations of reliability. Hilgard and Hilgard (1975) state that cold pressor pain is more like clinical pain than heat, electrical or pressure pain. They also identify two further disadvantages of cold pressor pain in

experimental use - that cardiovascular responses also occur reflexively and that S must distinguish between cold and painful sensations. The first of these is worthy of study in itself and also constitutes a useful correlate of pain. The second may at times be turned to good account, e.g. cold pressor pain lends itself to re-interpretation as cold.

Further evidence of the validity of cold pressor pain is provided by Klepac et al (1981) who showed that cold pressor pain was rated as more painful by Ss than electrical tooth pulp stimulation. Certainly much other current experimental pain research utilises cold pressor pain (e.g. Girodo & Wood, 1979; Leventhal, Brown, Shacham & Engquist, 1979; Rosenbaum, 1980; and Knox, Gekoski, Shum & McLaughlin, 1981).

Ischaemic pain. A more recent experimental pain-induction method is that of ischaemic pain where blood supply, usually to the arm, is occluded and pain allowed to develop. It may be hastened by having S exercise the limb in a standard manner. Beecher (1966) has praised this method as producing pain comparable to clinical pain and Hilgard and Hilgard (1975) support this view. As with cold pressor pain, pain develops slowly and is not simply cutaneous. However, also like cold pain, it is slow to administer and its reliability has been questioned (Wolff, 1978). There are other problems in that the discomfort of the sphygmomanometer cuff used to occlude blood flow may be important to S as may fatigue (Wolff, 1978). The lack of blood supply, though not dangerous, may increase anxiety in S (Wolff, 1977).

Pressure pain. A final pain-induction device is the pressure algometer where a known pressure is applied via a spring or weight usually to a bony surface of the body, for example, the shin, or knuckles (Merskey & Spear, 1964). Like several other methods this is slow to administer, and can only be used in ascending series. There is not much information on reliability and validity. Wolff (1977) states that reliability is less than several other methods. Merskey and Spear (1964) place reliability between that of electrical stock and heat pain (therefore quite high). However, one of the two pain measures used (the Pain Reaction Point - when pressure 'hurts a lot') may be criticised as being non-standard and not tied to a clear criterion (such as withdrawal from the stimulus).

From the above brief survey of experimental pain-induction methods it is clear that no one method is entirely satisfactory. A method should be chosen that is most suitable and practicable for a particular study, for example, relatively slow pain control strategies cannot be tested where intolerable pain is induced within seconds.

CHAPTER 3

Psychological Methods of Pain Control

Cognitive Strategies and Pain

A good deal of psychological research has been carried out on modifying experimental pain in man. A number of techniques have been applied to pain control, e.g. hypnosis (Barber & Hahn, 1962; Hilgard, 1973; Hilgard & Hilgard, 1975; Orne, 1980; Spanos, Radtke-Bodorik, Ferguson & Jones, 1979), relaxation (Bobey & Davidson, 1970; Lehrer, 1972; Stevens & Heide, 1977), biofeedback (Budzynski, Stoyva & Mullaney, 1973; Sargent, Green & Walters, 1973; Gannon & Sternbach, 1971), advance warning of pain (Sime, 1976; Cohen & Lazarus, 1973; Langer, Janis & Wolfer, 1975; Staub & Kellett, 1972), modelling (Chaves & Barber, 1974; Melamed & Siegel, 1975; Craig, 1978) and stress inoculation training (Meichenbaum & Turk, 1976; Girodo & Wood, 1979).

The use of cognitive strategies in the control of pain has also been investigated. Research on this technique has been based on several paradigms (e.g. the behavioural self-control paradigm of Skinner (1953) or the cognitive one of Scott & Barber (1977a)). This section will deal with the effectiveness of cognitive strategies as a pain control technique and the parameters of this effectiveness.

An early study by Barber and Hahn (1962) using cold pressor pain induced by a stimulus exposure of fixed duration compared four groups. The first was hypnotized and given suggestions that the

experimental arm would be numb. The second, cognitive strategies group was given, in a waking state, instructions to interpret the ice water stimulus as pleasantly cool. The third group was a cold water control and the fourth a warm water control. At the end of the three-minute exposure, Ss rated pain for each of the one-minute periods. Physiological measures were also taken during exposure. Results showed that the hypnosis and cognitive strategies groups did not differ significantly but that both reported significantly less pain than the cold water controls. Cold water controls reported significantly more pain than the warm water controls. On physiological measures (and this study is one of the few in the area to use them), there were likewise no significant differences between the hypnosis and cognitive strategies groups. The two experimental groups and the cold water controls showed significantly higher heart rate and lower skin resistance than warm water controls. The two experimental groups showed significantly reduced muscle tension and respiratory irregularities compared to cold water controls. Both experimental groups also recorded muscle tension not significantly different from warm water controls though this result was not achieved with respiratory irregularities. There was also a quasi-tolerance measure (some Ss were unable to tolerate the three-minute exposure). No significant differences were found on this measure. This study then, speaks strongly to the effectiveness of cognitive strategies in that cognitive strategies were as powerful as hypnosis and suggestion despite the extra elements in the latter. It also demonstrates the usefulness of physiological measures in providing additional detailed information.

A study by Spanos, Radtke-Bodorik, Ferguson and Jones (1979) tests hypnotic susceptibility, hypnotic induction and the tendency

to catastrophize (Meichenbaum, 1977) in their relation to pain control. This study supports the conclusion of Barber and Hahn (1962) that hypnotic induction per se has little effect in reducing reported pain. Spanos et al (1979) cite several other studies with similar results (e.g. Hilgard & Hilgard, 1975; Spanos, Barber & Lang, 1974) and criticise on methodological grounds two studies confirming the effectiveness of hypnotic induction in pain reduction (Hilgard, Macdonald, Morgan & Johnson, 1978; Stacher, Schuster, Bauer, Lahoda & Schulze, 1975).

In an experiment similar to that of Barber and Hahn (1962), Barber and Cooper (1972) tested three cognitive strategies - listening to a story, adding aloud by sevens and counting aloud (in reality repeating '1, 2, 3, 4'). In difference scores computed from pre- and post-test pain ratings, there was a significant reduction in pain for the first two experimental groups. The effect was weak however and the authors attribute this to the spontaneous use of cognitive strategies by the control group.

A much stronger study is that by Scott and Barber (1977a). In this study cold pressor and pressure pain were used. There were four experimental conditions with each of the two types of pain. These were:

1. cognitive strategies and long instructions,
2. cognitive strategies with short instructions,
3. a single cognitive strategy (thinking of pleasant events),
4. control - Ss were asked to tolerate pain as long as possible.

Strategies comprised trying not 'to be bothered' by the pain, concentrating on other things, dissociating oneself from the pain, reinterpreting sensations as not painful and imagining the stimulated area

to be numb. Dependent variables were pain tolerance and ratings of pain and distress.

The authors report that pain tolerance was raised by about 100% over base level in the groups using multiple cognitive strategies and given either long or short instructions. Tolerance times of the single strategy group fell between these on the one hand and the times of the control group on the other. They were significantly different from neither. None of the experimental conditions had a significant effect on self-ratings. There were no significant differences in tolerance times, pain ratings or distress ratings between the pressure and cold pressor pain groups.

In an earlier experiment (Chaves & Barber, 1974), subjects were exposed to experimenter modelling and also used cognitive strategies to reduce pain. In this study the length of time the pain was to be tolerated was both fixed and known in advance by all Ss. The only measure used in this experiment was S's rating of pain, on an eleven-point scale ranging from 'no pain' to 'very severe pain'. Cognitive strategies were the most effective element in reducing ratings. Modelling was effective only with Ss who had high pain ratings on pre-test and expectation of pain reduction was also identified as an operative factor. Scott and Barber (1977a) conclude that either measure (tolerance or ratings) may be affected but probably not both at once.

Scott and Barber (1977a) also discuss the results of their experiment in terms of the demands made on Ss, that is to tolerate more pain and experience it less. This conclusion is supported by a further study (Scott, 1980). Experimental demand has also been

shown to be a significant variable in psychologically-based pain research generally (Bowers, 1966; Orne, 1962).

In addition, Scott and Barber (1977a) note that greater pain control seems to derive from giving Ss a range of cognitive strategies to use. There is some support for the view that simply having a number of cognitive strategies available lends power to the technique, e.g. Scott and Barber (1977b) in a follow-up report, found that experimental Ss added their own cognitive strategies or stopped using the given strategy part way through exposure to the stimulus. Girodo and Wood (1979) studied stress inoculation training (Meichenbaum & Turk, 1976), a technique incorporating cognitive strategies. They compared their own relatively weak results with those of Meichenbaum and Turk (1976) and Horan, Hackett, Buchanan, Stone and Demchik-Stone (1977). They noted that these studies provided a greater range of cognitive strategies, taking Ss more time to use and allowing them less time (apparently) to disconfirm them. The study earlier referred to, by Spanos et al (1979) also provides evidence on the effect of a range of strategies. These authors found that for non-catastrophizers the extent of pain reduction on the post-treatment trial was a function of the number of strategies used (though no explicit strategies were suggested). Catastrophizers did not report reduced pain regardless of strategies. This effect did not vary according to hypnotic induction.

A second relevant aspect of the success of multiple strategies may have been the choice given to Ss in the use of strategies though this particular choice situation does not seem to fit into any of the three categories given in the extensive review by Averill (1973)

of the literature pertaining to S's control over aversive stimuli.

Averill's first category is behavioural control, that is, control over the timing of the stimulus or who administers it (e.g. Staub, Tursky & Schwartz, 1971, Kanfer & Goldfoot, 1966) or modification or avoidance of the stimulus itself (e.g. Bowers, 1968). The latter is referred to as perceived control and may interact with other factors such as the locus of control of the individual (Rotter, 1975). The second category is cognitive control ('control' may be by re-interpretation of the stimulus itself, e.g. of ice water as pleasantly cool). The third category is decisional control where S can choose between different sets of responses.

In fact though, this area appears confused, with the terms 'control' and 'self-control' being freely interchanged and being given various meanings. Kanfer and Goldfoot (1966) for instance, seem to attach at least four meanings to the term 'self-control'. These only partly overlap Averill's (1973) categories. The categories listed by Kanfer and Goldfoot (1966) are Skinner's (1953) self-control paradigm, S's controlling his own reactions to a stimulus, S controlling the stimulus itself and S controlling the strategies used to control reaction to pain. An experiment by Kanfer and Seidner (1973) falls into this last category. This study tested the effect on pain tolerance of S's or E's control of distraction (via slides) from the pain stimulus. S's control over aversive stimuli has nevertheless been shown to be an important variable in experimental pain research.

Spanos, Horton and Chaves (1975) tested a further parameter of cognitive strategies - that of the relevance of the strategies to

the pain stimulus situation. Cold pressor pain was used. The prime measure was pain threshold and a secondary measure was of involvement in using cognitive strategies. The first experimental group was instructed to concentrate only on the coolness of the water and to interpret it as pleasant and refreshing. The second experimental group was asked to imagine being in a particular lecture theatre with a particular lecturer. The control group was given no instructions. All Ss were pre-tested and divided into high and low threshold groups. After the post-treatment test, experimental Ss completed a rating scale of self-involvement in cognitive strategy use. Analysis of thresholds showed that group means for low threshold Ss did not differ significantly. For high threshold Ss, the relevant-strategy group thresholds were significantly higher than those of the irrelevant strategy group which in turn were higher than those for the control group. Further analysis showed greater elevation in pain threshold for Ss highly involved in strategies. The relative effectiveness of relevant and irrelevant strategies did not vary according to involvement.

A study by Beers and Karoly (1979) is similar; it tested the relative effectiveness of four cognitive strategies. The first was rational thinking, involving positive self-statements and minimising the unpleasant nature of the stimulus (again ice water). The second was task-irrelevant cognition (counting backwards by threes). The third was compatible imagery (a pleasant winter scene) with the fourth being incompatible imagery (a pleasant but warm scene). There were two control groups. One was given a positive expectation of pain reduction but no strategies while the other was a no-treatment control group. Analysis of co-variance was performed for each measure, using pre-test scores as the co-variate. Pain tolerance times for

the rational thinking, compatible imagery and incompatible imagery groups were significantly greater than for the no-treatment control group. Pain tolerance differences between the rational-thinking, compatible imagery and incompatible imagery groups were not significant. For threshold measures, rational-thinking, compatible and incompatible imagery groups differed significantly from both the wait-only (no treatment control) group and the wait-expectancy group. There were no significant differences on self-rated discomfort. Differences between Ss in imaginal ability did not correlate with any of the differences found between groups. The study provides moderate support for the Spanos, Horton and Chaves (1975) study regarding relevance of strategies.

Another interesting finding of the Beers and Karoly (1979) study however, was that the effect of cognitive strategies could not simply be attributed to expectation since on neither the threshold nor tolerance measures did the wait-only and wait-expectancy groups differ significantly. This finding is supported by Chaves and Barber (1974) who showed that pain ratings in an expectancy group were significantly lower than those of controls but significantly higher than for Ss using cognitive strategies (imagining a pleasant event). Scott and Leonard (1978) however, found expectancy to be as effective in raising pain threshold above the level for the control group as a re-interpretative strategy. In this experiment though, covert reinforcement was significantly more effective than either of the other treatments. The covert reinforcement group reinterpreted the stimulus as non-painful and then imagined a scene or object pleasurable to them.

The degree of distraction from pain afforded by cognitive

strategies has also been hypothesised to account for their effectiveness. Barber and Cooper (1972) viewed the cognitive strategies in their experiment as distractors. Although significant differences were found between strategies, the differences between the successful strategies and the controls, though significant, were not as great as expected.

Spanos, Horton and Chaves (1975), by using Ss' self-ratings of involvement, showed that the more successful strategies in their study were no more distracting than the less successful strategies. This conclusion depends on acceptance of the measure of involvement as a measure of distraction. The more direct measure of distraction used by Barber and Cooper (1972) did not vary significantly between groups.

Adoption of Blitz and Dinnerstein's (1971) distinction between distraction (diverting attention completely away from the noxious stimulus) and dissociation or re-interpretation (focusing on a particular aspect of the noxious stimulus) would lead to the conclusion that results in the above studies may have varied mainly because of the different types of strategies used rather than because of the degree of distraction. In any case, the role of distraction in pain control still needs to be clarified.

Thus several factors - distraction, relevance, control, expectation and number of strategies available have been identified as influencing the effectiveness of cognitive strategies. It would seem likely that for cold pressor pain a re-interpretative cognitive strategy focusing on cold should be successful in reducing pain and would provide a standard against which to compare other pain control

techniques

Facial Expression and Pain

Much of the research that has been carried out on human facial expression is concerned with it as an indication of emotion (Ekman, Friesen & Tomkins, 1971; Ekman & Oster, 1979; Engen, Levy & Schlosberg, 1958).

A number of instruments have been developed, some of which attempt to measure facial expression as such and others which attempt to measure displayed emotion. In the former category are Grant's (1969) facial expression checklist, providing a framework for standardized description and coding of facial expression. The Facial Action Coding System (Ekman & Oster, 1979) has a similar aim.

In the latter category are the electromyographic studies of Schwartz and colleagues (e.g. Schwartz, Fair, Salt, Mandel & Klerman, 1976). These represent perhaps a renewal of Duchenne's (Tomkins, 1961) attempt to link particular facial muscle activity to particular affects. The Facial Affect Scoring Technique (Ekman, Friesen & Tomkins, 1971) is the latest in a series of classifications aimed at matching facial expression and emotions and of providing a research tool for the study of emotions. Similar classifications of facial expressions have been developed previously e.g. the Frois-Wittman Scale (Frois-Wittman, 1930) and the Lightfoot Scale (Engen, Levy & Schlosberg, 1957). A good deal of work has been done with such scales, e.g. Woodworth's (1938) attempt to place six basic emotional expressions on a continuum and the work of

Schlosberg and colleagues on a three-dimensional rating of emotion from facial expression (Engen, Levy & Schlosberg, 1958).

Much of the above research assumes simply that emotion affects facial expression. Yet there is also a body of research dealing with the possible influence of facial expression on emotion. A basic concept in psychoanalytic theory is of the build-up and discharge (via speech, action, facial expression) of emotions (Fenichel, 1946) and there is some experimental evidence for this view (Jones, 1950). More recently, Notarius and Levenson (1979) tested the effects of natural facial expressiveness in response to threat of pain and obtained results consistent with discharge theory. The authors were careful to limit their conclusions and stated that they did not contradict those of Kleck and colleagues which are discussed below (e.g. Kleck, Vaughan, Cartwright-Smith, Vaughan & Lanzetta, 1976). This is because expressions were uncontrolled in the former study and controlled in the latter.

A greater amount of research in experimental psychology has been carried out on the possibility of facial expressions enhancing emotional activity. Four hypotheses are current. The first is that facial and postural expression may lead to visceral changes and that the perception of both skeletal and visceral feedback leads to the experience of emotion (James, 1884). Tomkins (1961) and Izard (1971) hold similar views. Izard holds that emotion is the result of the interaction of neural activity, voluntary muscle activity and subjective experience. Gellhorn (1964) states a similar theory in neurophysiological terms.

The second explanation is that of attribution theory. Laird

(1974) holds that emotion is an attribution based on the degree of felt autonomic arousal for intensity information and on an inference from the context for quality information. Schachter (1964) and Bem (1972) hold somewhat similar views.

The third explanation comes from Lazarus's work on cognitive reappraisal. Lazarus and Alfert (1964) and Lazarus and Opton (1966) propose that the response to threat is based partly on S's cognitive evaluation of it. If the evaluation can be changed (perhaps by use of the cognitive reappraisal technique) then the response may be altered.

The fourth hypothesis (Kleck et al, 1976) is based on classical conditioning. It is held that facial expressions precede autonomic arousal and by contiguity come to serve as conditioned stimuli for arousal. Recent work by Orr and Lanzetta (1980) and Lanzetta and Orr (1980) has demonstrated some effect of facial expression on autonomic arousal. Much of the experimental work on the last hypothesis concerned the relationship of facial expression and pain.

Lanzetta, Cartwright-Smith and Kleck (1976) examined the effect of non-verbal dissimulation of pain on emotional experience and autonomic arousal. Ss were twelve male and six female undergraduates who were individually given a series of shocks and led to believe that they were not being visually observed (i.e., pain tolerance baseline was being established). Ss first rated shock intensity on a four-point scale then received 20 shocks (five at each of four levels) in random order. After a further interval there was another block of trials preceded by instructions to attempt to hide

responses until the time the shock was to be given. Ss were told that a videotape would be made and observers would try to guess if shock were given and to rate the strength of the shock.

The investigators found that the 'hide' condition led to reduced skin conductance changes in both shock and non-shock trails. Ss rated lower shock levels as less aversive but did not alter their rating of the highest shock level.

The second experiment in the series tested an alternative explanation viz that the results may have been due to distraction or because requested responses were incompatible with fear responses. This was done by introducing a second condition, that Ss should freely express anticipatory fears. It was found again that the 'hide' condition produced autonomic and self-reported reduction in pain response whether or not Ss knew that they were being filmed.

In the third study of the series both male and female Ss were used and the 'hide' and 'reveal' (express) conditions applied as well to the actual reception of shock, not just its anticipation. It was found that posing the anticipation and reception of intense shock produced more intense facial displays and led to greater signs of emotional arousal than posing no-shock. This effect was significant both for physiological indices and self-report. The result is inconsistent with an explanation based on simple distraction since in this case posing intense shock could have been expected to lead to a reduction rather than an increase in arousal.

A later study by Colby, Lanzetta and Kleck (1977) found that skin conductance charges were monotonically and positively related

to level of expression but only in the presence of shock. Pain tolerance levels were not related to the level of expression and were higher for rapidly than for slowly ascending shock.

The study by Kleck, Vaughan, Cartwright-Smith, Vaughan, Colby and Lanzetta (1976) found reduced facial expressiveness and reduced change in physiological indices when Ss knew they were being observed. Gender of the observer made no significant difference to scores.

From these studies there is evidence for consistent physiological and self-rating changes in response to facial expression change. The evidence relating facial expression to psychophysical measures is weaker, though only pain tolerance has been measured. These results raise the possibility of the use of facial expression as a technique aimed at modifying experimental and perhaps clinical pain.

One way of testing the effectiveness of the modification of facial expression as a pain-reduction technique would be to compare it on that basis with cognitive strategies. To this end, Ss would need to know (unlike the Ss in the experiments reviewed here) that facial expression will be manipulated with a view to pain reduction since this is how cognitive strategies are usually presented to Ss. Cold pressor pain would be more appropriate than electric shock pain: because of the slower onset, cold pressor pain is more like clinical pain and would provide a good basis for comparison of the facial expression and cognitive strategies techniques. A last reason for comparing facial expression and cognitive strategies is to explore interaction between them. For example, it is possible that if S is concentrating on a cognitive strategy he may be likely to assume a facial expression not indicative of pain.

Aims and Hypotheses

The current study has three aims:

1. comparison of the relative effectiveness of cognitive strategies and facial expression in pain reduction;

2. clarification of the relationship between cognitive strategies and facial expression in the control of pain threshold;

3. clarification of the relationship between strategies and physiological responses and between pain and physiological responses.

The experimental predictions to be investigated in this study are:

1. That cognitive strategies will reduce felt pain as measured by threshold time and pain self-ratings.

2. That facial expression changes (to 'hide' expression of pain) will reduce felt pain as measured by threshold pain and pain self-ratings.

3. That cognitive strategies will modify physiological responses to pain stimuli.

4. That facial expression changes will modify physiological responses to pain stimuli.

5. That the combination of the two strategies will produce the

greatest reduction in pain and the greatest modification in physiological responses to pain stimuli.

CHAPTER 4

Method

Subjects

Forty-four Ss (12 male, 32 female) were tested. All Ss were caucasian; 20 were university students, 20 were matriculation college students and four were recent university graduates. Ages of Ss ranged from 16 - 33 years with a mean of 20.4 years. Ss volunteered after being informed that the study was to investigate physiological responses to various stimuli. They were also told that a cold thermal stimulus would be used which might produce discomfort or mild pain. All Ss completed the experimental session even though advised that they could withdraw from the experiment at any time. An additional eight Ss were tested but were excluded from the experiment because they could not be matched according to sex and age.

Design

The 44 Ss were allocated to one of four groups of 11 Ss each - a cognitive strategy group, a facial strategy group, a combined cognitive and facial strategies group and a no-treatment control group. The matching procedure used to allocate Ss to groups is described later under the heading "Control Measures". The experiment was a single factor design with repeated measures on Ss for immersion times as an index of pain threshold and physiological responses. The aim was to test the effect of the strategies on

pain threshold when a hand is immersed in cold water. A baseline trial was given to establish basal immersion time and physiological responsiveness. Then experimental instructions were given and three identical experimental trials followed. Each S was tested under only one experimental condition to avoid the confounding of experimental variables. The no-treatment control group was included to check that treatment effects were not due solely to extraneous variables (e.g. the demand characteristics of the experimental instructions and possible habituation or sensitization effects of the thermal stimulus). In addition a combined treatment group was used to allow assessment of any additive or interference effects resulting from the combination of facial and cognitive treatments.

Appropriate water temperature and experimental procedure were determined in an informal pilot study. A pain threshold measure was chosen as being most relevant to the aims of the study (the development of techniques to control intermittent pain). Water temperature of 5° C. was chosen: a colder stimulus rapidly induces severe pain in many people. The range of pain threshold immersion times with a colder stimulus may have been too restricted to have shown differences between the strategies used in this experiment. S placed his hand in water of neutral temperature (29° C.) before and after exposure to the cold stimulus on each trial (including baseline) to assist temperature recovery of the hand to minimise habituation or sensitisation effects.

In addition to the objective immersion time measure of pain threshold a modified version of the McGill Pain Questionnaire (Melzack, 1975) was administered. This formed the second main

experimental measure. To attempt to assess the physiological correlates of pain, three physiological variables - respiration, finger pulse amplitude and finger blood volume - were also measured. Physiological baselines were taken while S was at rest; immersion time baselines were taken during S's first exposure to the cold stimulus. The physiological baselines were taken after, rather than before the immersion time baseline so that pulse baselines could be taken from the same hand as experimental pulse measures. The change from one hand to the other was to ensure that S's hand was in as normal a condition as possible for the first experimental trial.

Before testing, Ss were matched for sex and age by grouping into blocks of four, on the basis of same sex and five-year age range (16 - 20, 21 - 25, 26 - 30, 31 - 35 years). Ss in each block were then randomly assigned to one of the four experimental groups. All Ss were naive to the pain control strategies. No attempt was made formally to control for social class. Nor was there any control on cultural background though later enquiry showed that only one S was not of Australian or British birth. Room temperature was not controlled, though room temperature was monitored and variation was minimal (15.5 - 19.0° C.).

Several important control variables related to pain response were measured with standardised tests. These variables were used as post-hoc controls to ensure that any differences in the experimental variables could not be attributed to differences in group composition. The variables so controlled were neuroticism, extraversion (Levine, Tursky & Nichols, 1966; Lynne & Eysenck, 1961; McLaughlin & Harrison, 1973), state anxiety and trait anxiety

(Bowers, 1968; Bobey & Davidson, 1970; Weisenberg, 1977; Woodforde & Merskey, 1972). S's ability to imagine events vividly and to control imagery bear a possible relationship to the cognitive strategy treatment (Beers & Karoly, 1979). These were also measured with standardised tests.

A videotape recording was made of all Ss during experimental trials to monitor facial expression and in particular as a check that Ss in the facial group were able to 'hide' any expression of pain or discomfort.

Apparatus

Thermal stimuli. Apparatus consisted of two large bowls to contain water and a Digitron 275 digital thermometer. Water temperature was adjusted immediately before each trial to ± 0.1° C. of the standard temperatures of 5° C. for the cold stimulus and 29° C. for the neutral stimulus.

Immersion time measurement. A switch was positioned beneath the bowl containing cold water. When S immersed his hand in the cold water he was required to press lightly on the bottom of the bowl, closing the switch which operated an event marker on a Beckman R511A multi-channel physiological recorder. The event marks defined immersion times. A buzzer was used to signal the beginning and end of each trial.

Physiological measurement. All physiological recording was on three channels of a Beckman R511A Dynograph using a paper speed of 2.5 mm/sec.

Respiration. A D.C. recording of respiration was taken using a Parks mercury-in-rubber strain gauge placed around the upper part of S's chest. The strain gauge was connected to a Parks 270 Plethysmograph and the latter was connected to a Beckman 9853A general-purpose coupler on the recorder. Sensitivity setting was 10 mv/mm. Maximum pen deflection for a normal breath was 35mm; for most Ss, pen deflection was 10 mm for a normal breath.

Finger blood volume. Finger blood volume (F.B.V.) was measured using a Beckman photoplethysmograph pick-up, a bridge circuit and a 9853A general-purpose coupler on DC mode at a sensitivity setting of 5 mv/mm. The photoplethysmograph pick-up was placed on the first phalanx of the forefinger of S's non-dominant hand. This produced an F.B.V. record with finger pulse amplitude responses of less than 2 mm.

Finger pulse amplitude (F.P.A.). The measure of finger pulse amplitude was recorded by taking the output of the finger blood volume channel back into another channel with a Beckman 9806A AC/DC coupler. The sensitivity setting was .02 v/mm and a time constant of 0.3 sec. was used. The F.P.A. height was 1 to 6 mm.

The physiological recording apparatus was in a room adjacent to the S's room. Electrical connections were made via a plug-board between the rooms.

McGill Pain Questionnaire. Parts 2 - 4 of the McGill Pain Questionnaire (Melzack, 1975) were extracted and modified for use with experimental pain. A copy of the modified questionnaire is presented in Appendix A. Questions A and C were unchanged except

that Ss were asked only to consider the experimental pain with no reference to ongoing clinical pain. In Question B Ss were asked to select word-groups only. In the standard questionnaire they may select individual words as well. Two further questions concerning pain onset and cultural background were asked by E after the completion of the questionnaire.

Control measures. As noted previously, standardised tests were used as control measures. These were the Eysenck Personality Inventory, Form B (Eysenck & Eysenck, 1964), the State-Trait Anxiety Inventory (Spielberger, Gorsuch & Lushene, 1970), the Betts Q.M.I. Vividness of Imagery Scale (Sheehan, 1967) and the Gordon Test of Visual Imagery Control (Gordon, 1949).

Video recording. Recordings were made using a Sony AVC 3200 CE camera and Sony AV3620 CE recorder with standard video recording tape. Samples of recordings were later re-recorded using video editing equipment.

Procedure

All Ss were tested individually in a single 100 - minute session. The following procedure was used.

Baseline. For all Ss the procedure was outlined. The dominant hand (defined as the writing hand) was then determined to control lateral dominance as a factor in pain response (Weisenberg, 1977). The non-dominant hand was used for the baseline trial. S was asked to place his hand in neutral water (in order to control initial skin temperature). After five minutes, on the first buzzer, S placed his

hand in cold water. S pressed a switch as his hand entered the water, pressing it again when pain was felt. After a further five seconds the second buzzer signalled S to withdraw his hand from the cold water and place the hand in the neutral water bath for two minutes. Maximum immersion times were fixed at 90 seconds for both baseline and experimental trials. Seventeen Ss exceeded 90 seconds on one or more trials. The neutral water bath in the inter-trial interval was used to reduce pain and stabilise skin temperature since this hand would later be used for physiological recording. The procedure is shown in Figure 3.

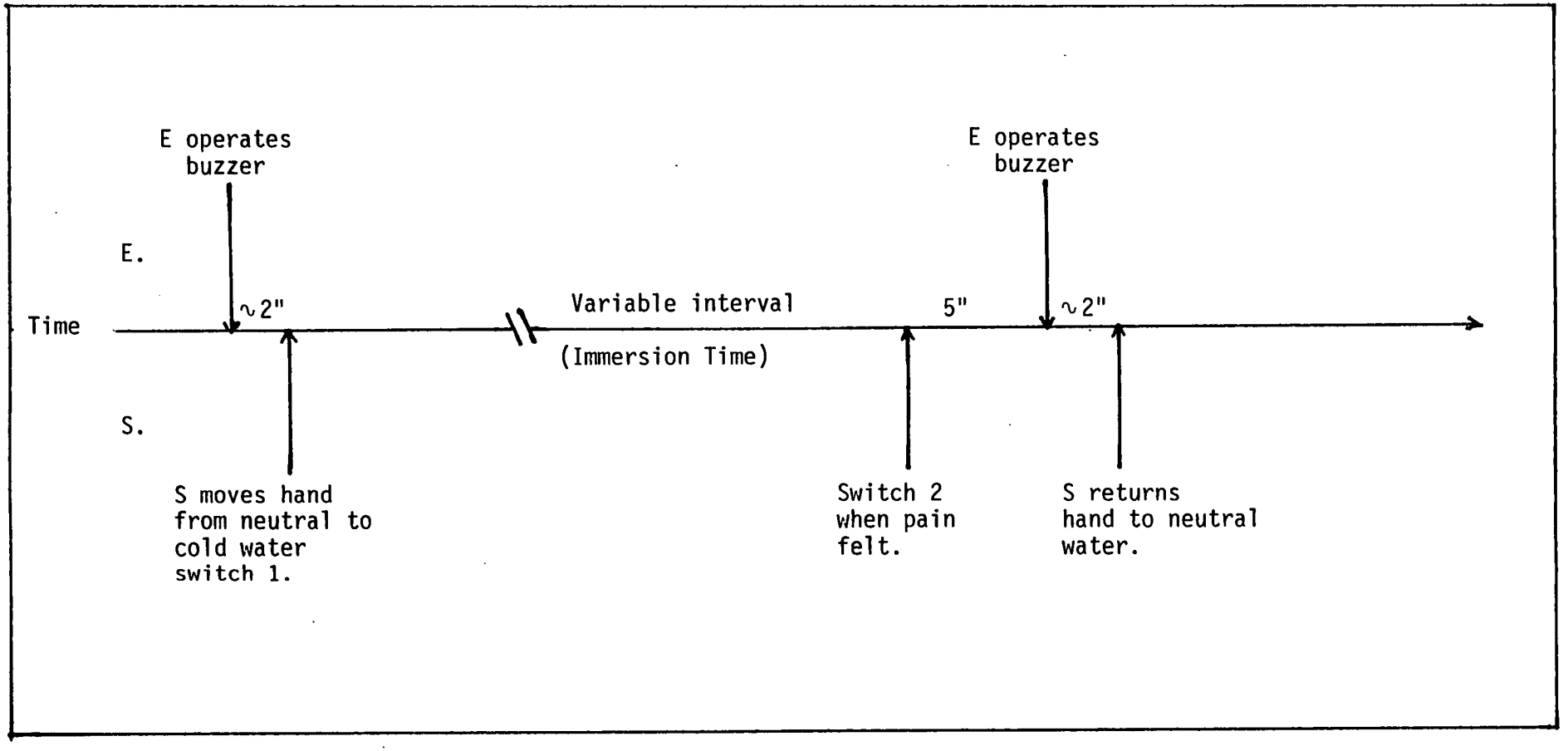
Questionnaires. In the interval following the baseline trial S commenced the questionnaires. These were always given in the same order: Betts Q.M.I., Gordon Test, S.T.A.I. and E.P.I.

General procedure. Prior to the first experimental trial a mercury-in-rubber strain gauge was placed around S's chest just below the armpits. The photoplethysmograph pick-up was then attached to the forefinger of the non-dominant hand. The dominant hand was exposed to cold in all experimental trials. This arrangement allowed S to complete questionnaires in the inter-trial intervals. The video recording was started, after which S placed his hand in neutral water. Prior to the trial S was given the relevant instructions for his group.

Control group (Z). These Ss were given the baseline instructions again. They were also asked to pay particular attention to the stimulus so that they could describe the sensations experienced. The purpose of the second instruction was to increase the face-validity of the procedure for the control group; this was furthered

FIGURE 3

Flow-chart of Trial Procedure



by the setting-up of the physiological and video recording apparatus.

Cognitive strategy group (C). Ss in this group were given a short explanation of cognitive strategies together with an account of their use in modifying pain. Ss were asked to employ a single strategy in which they repeated sub-vocally "It's only cold!". This sentence was to be repeated during exposure to the cold stimulus. No mention was made of facial expression.

Facial strategy group (F). Ss were asked to maintain a neutral facial expression, 'hiding' pain during the exposure to cold. The type of facial expression required was precisely described (though not modelled) so that Ss did not 'hide' pain in other ways, for example by smiling.

Combined strategies group (B). This group was given details of both strategies and asked to perform both together while exposed to cold. In the instructions, the facial strategy was mentioned first, though both strategies were given equal emphasis.

Second and third experimental trials. The three experimental trials were carried out using the same procedure employed in the baseline determination. Shortened instructions were repeated before trials 2 and 3. After each trial S placed his hand in neutral water for two minutes.

Trials were at 20 minute intervals during which time questionnaires were to be completed. After the last trial physiological and video recording apparatus was removed and S was asked to complete the modified McGill Pain Questionnaire.

After the experimental requirements had been finalised, S was given a full explanation of the nature of the experiment and was requested not to discuss the experiment with anyone else.

Video ratings. In the rating of facial expression, samples were edited from the videotaped record and were presented in temporal order. Three five-second segments of each trial were recorded. The periods were after the first switch-press at the beginning of the trial and the second switch-press to signal pain as well as a segment from mid-trial. For those Ss who did not signal pain, the 90-95 second segment was used. The full range of conditions which Ss experienced was thus sampled.

Two independent raters were trained using close specification of the rating task, discussion and extended video recordings of Ss who could not be included in the data analysis. Raters were able to reach adequate agreement using a three-point scale, (1. No pain or slight pain - 2. Moderate pain - 3. Severe pain). The coefficient of reliability calculated from 9 pairs of ratings for each S was $r(395) = .80$, $P < .01$.

Quantification of Data

Immersion time. To calculate hand immersion time to pain threshold, the distance between event recorder marks on the polygraph record of each trial was first measured. This was then divided by the paper speed (2.5 mm/sec.). The result was expressed in seconds.

Physiological measures. Experimental physiological measures were taken from the section of polygraph record between event

recorder marks for each trial. The pre-trial baseline was taken over 30 seconds from the record preceding the first experimental trial for each S. To aid assessment of the effect of pain within experimental trials, separate scores for first and second halves of trials were derived for all the physiological measures (these are referred to as half-trial measures).

Respiration rate. In calculating respiration rate the number of respiratory cycles was counted and divided by the elapsed time. Only complete cycles were counted.

Inspiration-expiration ratio. This ratio was calculated for completed respirations by dividing the total time taken for inspirations by the total time taken for expirations.

Heart rate. Heart rate was calculated by counting the number of completed pulse beats in the finger pulse amplitude record and dividing by the elapsed time.

Half-trial measures. For all the above measures (except baselines), half-trial measures were calculated by dividing trials into halves on the basis of the time per trial. The halves were then scored separately in the manner stated above.

For all measures, half-trial scores were averaged over the three trials to give average first-half and average second-half measures. Because heart rate was the one physiological measure to show systematic variation across trials, first and second half-trial measures were retained for individual trials on this measure.

McGill Pain Questionnaire. The same measures were derived from the modified as from the standard McGill Pain Questionnaire (Melzack, 1975). From question A was derived the Present Pain Intensity measure (PPI) which is, for groups, the mean pain rating on the given five-point scale. From question C two measures were obtained. These were the total number of words chosen (NWC) and the Pain Rating Index-Ranked (PRI-R). The latter, for each S, is the total ranked value of words selected. A related measure, the PRI-Scaled, was not used as scale values were not available for words related to cold from Melzack and Torgerson (1971). Probably little sensitivity is lost as Melzack (1975) has demonstrated very high correlations between PRI-Ranked and PRI-Scaled.

Four sub-measures were derived from PRI-R and constitute total rank values of words selected in four categories of descriptors. These were sensory (word-groups 1-9 and 17-19), evaluative (group 16), affective (groups 11-14) and miscellaneous (groups 10, 15 and 20). On all of the above measures higher scores indicate greater pain.

A last numerical measure from this questionnaire used ordinal rather than interval data. Derived from question B, it classified the pattern of pain felt into three categories - transient, periodic and continuous.

Ss were finally classified according to their experience of pain onset as sudden or gradual.

Control measures. All questionnaires were scored by the standard methods except the Betts QMI Scale. In addition to the total score, a separate subscale of Tactile Imagery was derived because

it was considered to be of particular relevance to this study (especially item 15 which refers to a thermal stimulus). This sub-scale comprises the summed scores of items 11-15 of the Betts Scale.

CHAPTER 5

Results

Control Measures

One-way analyses of variance were carried out on all control measures, that is, on age of Ss, E, N and L scores of the Eysenck Personality Inventory, the State-Trait Anxiety Inventory (State Anxiety and Trait Anxiety Scores), the Betts Scale (Total score and Tactile Imagery Subscale score) and the Gordon Test. A list of F ratios for the control measures is given in Table 1; analysis of variance summary tables are given in Appendix B. These analyses showed that there were no significant initial differences between the experimental groups on any of the control measures.

Video ratings. The rated facial expression from the edited videotape showed non-significant differences between groups in a 4 x 2 analysis of variance with repeated measures on one factor ($F(3, 40) = 1.74$, $P < .25$). In the same analysis significant increases in pain were found across each trial when ratings at the beginning and middle of each trial were compared with the rating at the end of each trial. $F(1, 40) = 7.45$, $P < .01$ which showed significantly higher rated pain at the end of trials. Interaction effects were not significant ($F(3, 40) = 1.82$, $P < .25$). A summary of this analysis is given in Appendix C.

TABLE 1

F Ratios from Analyses of Variance Testing Group Differences on Control Measures

Measure	d.f.	F	P
Ages of Ss	3, 40	0.03	> .25
EPI - Neuroticism	3, 40	0.82	> .25
EPI - Extraversion	3, 40	0.31	> .25
EPI - Lie Scale	3, 40	1.15	> .25
STAI - State Anxiety	3, 40	0.90	> .25
STAI - Trait Anxiety	3, 40	0.35	> .25
Betts - Total Score	3, 40	0.35	> .25
Betts - Tactile Imagery	3, 40	0.50	> .25
Gordon Test	3, 40	0.77	> .25

Immersion Times

A 4 x 3 (Groups x Trials) analysis of variance with repeated measures was carried out initially on experimental immersion-time data; the analysis of variance summary table is presented in Table 2. Because there were no significant trials or trials x group interaction effects subsequent analysis of immersion times used data averaged over trials. Baseline and averaged experimental immersion times appear in Table 3. One-way analysis of co-variance was used so that group differences on baseline immersion times could be used to adjust experimental immersion times. In this analysis, for the baseline data, $Fx\ (3, 40) = 0.31$, $P > .25$, while for the experimental data $Fy\ (3, 40) = 1.70$, $P > .1$. For the adjusted experimental data $Fy'(3, 39) = 3.19$, $P < .05$. Baseline and averaged experimental immersion times appear in Table 3. Table 4 shows the analysis of co-variance summary table. Subsequent Tukey tests on the adjusted means showed the cognitive group mean immersion times to be significantly higher (indicating longer time to pain threshold) than those of the control, facial and combined strategies groups. There were no other significant differences between adjusted group means. Unadjusted immersion-time scores of the cognitive and combined strategies groups tended to rise as compared with baseline; control and facial group scores showed a non-significant downward trend. The graph showing baseline and adjusted experimental means for the four groups is presented in Figure 4.

Physiological Measures

Trials effects. For the physiological measures, 4 x 3 (Groups x Trials) analyses of variance with repeated measures were performed

TABLE 2

Summary of Analysis of Variance of Experimental Immersion Times

Source of Variation	S.S.	d.f.	M.S.	F
Between Ss	72233.1	131	2698.16	1.68
A (groups)	8094.5	3	1603.47	
Ss within groups	64138.6	40		
Within Ss	6926.9	88		
B (trials)	99.3	2	49.65	0.61
AB	271.4	6	45.23	0.55
B x Ss within groups	6556.2	80	81.95	

TABLE 3

Baseline Immersion Times and Averaged Experimental Immersion Times (secs.)

	GROUPS							
	Control		Cognitive		Facial		Combined	
	Base	Expl	Base	Expl	Base	Expl	Base	Expl
	28.7	23.8	90.0	90.0	50.5	40.3	71.2	65.2
	25.7	12.7	20.2	26.1	34.5	34.8	27.7	42.2
	17.1	22.1	44.7	40.9	48.0	27.4	24.8	36.7
	24.8	19.7	31.0	73.4	26.4	33.3	90.0	62.0
	39.7	59.1	90.0	58.0	25.3	22.2	29.9	90.0
	35.8	41.8	26.9	50.1	90.0	90.0	51.3	52.3
	90.0	70.0	90.0	90.0	90.0	90.0	70.9	51.4
	63.6	53.8	90.0	90.0	90.0	52.6	46.9	64.9
	62.0	34.6	90.0	90.0	26.0	30.4	33.7	39.6
	90.0	90.0	28.7	50.9	90.0	50.1	90.0	75.5
	66.4	34.5	21.3	35.6	90.0	90.0	47.5	49.2
Means	49.4	42.0	56.6	63.2	60.1	51.0	53.1	57.2

TABLE 4

Summary of Analysis of Co-variance of Experimental Immersion Times, Adjusted for Baseline Immersion Times

Anova for X variable (Baseline)

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	712.0	3	237.33	0.31
Within groups	30463.2	40	761.58	
Total	31175.2	43		

Anova for Y variable (Experimental times)

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	2721.3	3	907.10	1.70
Within groups	21377.6	40	534.44	
Total	24098.9	43		

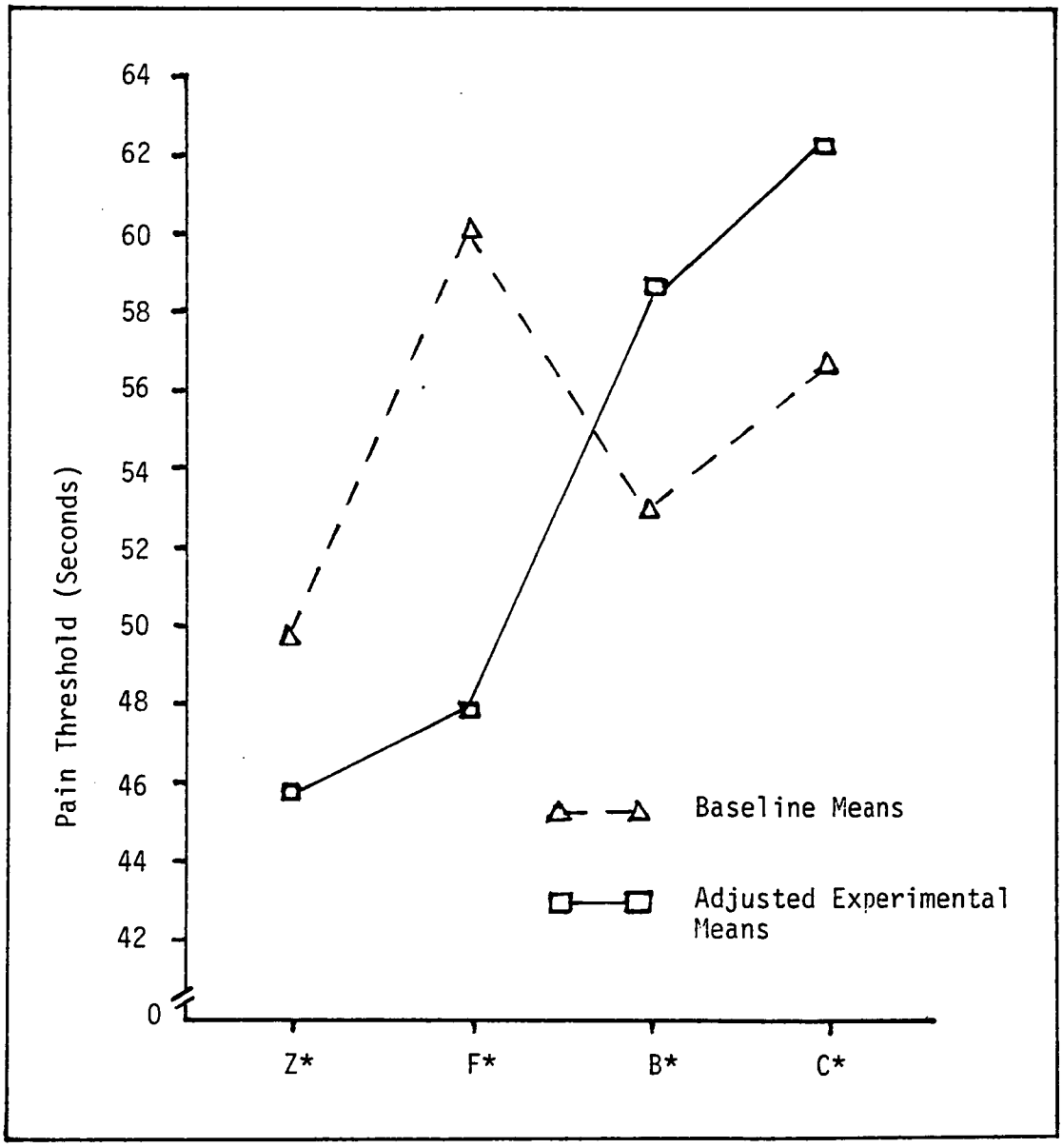
Co-variance analysis (Y')

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	2140.0	3	713.33	3.19*
Within groups	8709.0	39	223.31	
Total	10849.0	42		

* $P < .05$

FIGURE 4

Graph showing Mean Immersion Times (Arranged in Rank Order of Experimental Means)



* Z = Control group B = Combined group

F = Facial group C = Cognitive group

to test variation across trials. Inspiration-expiration ratio and respiration rate showed non-significant trials and trials x group interaction effects. For heart rate the trials effect was highly significant. Here $F(2, 80) = 23.61$, $P < .001$. On Tukey test all means differed from each other at the 1% level of significance showing a consistent reduction in heart rate from first to last trial. Group and interaction effects were non-significant. A summary of the three analyses is given in Table 5.

First and Second Half-trial Analyses. Further analysis of physiological measures was by 4 x 2 analysis of co-variance comparing groups and averaged first and second halves of trials, scores being adjusted for baseline scores. These analyses will now be considered in detail.

Heart rate. Summary data for this analysis are shown in Table 6; a summary of the analysis of co-variance is shown in Table 7. Heart rate decreased significantly across the two trial halves ($F(1, 39) = 30.79$, $P < .001$). Group differences on baseline were non-significant. With experimental data group effects were non-significant both before and after the co-variance adjustment. The means of cognitive and combined strategies groups tended to be below those of the facial and control groups. Interaction effects were non-significant.

Inspiration-expiration ratio. Baseline and experimental data are shown in Table 8; the analysis of co-variance summary is presented in Table 9. Again there was significant variation between halves of trials, though it was not so highly significant as for heart rate ($F(1, 39) = 8.00$, $P < .01$). A relatively greater time

TABLE 5 (a)

Summary of Analysis of Variance of Experimental Heart Rate

Source of Variation	S.S.	d.f.	M.S.	F
Between Ss	21037.5	131		
A (groups)	2563.6	3	854.53	1.85
Ss within groups	18473.9	40	461.85	
Within Ss	1906.9	88		
B (trials)	641.6	2	320.80	23.61**
AB	177.6	6	29.60	2.18
B x Ss within groups	1087.7	80	13.59	

** $P < .001$

TABLE 5 (b)

Summary of Analysis of Variance of Experimental Inspiration-Expiration Ratios

Source of Variation	S.S.	d.f.	M.S.	F
Between Ss	1.55	131		
A (groups)	0.13	3	0.043	1.19
Ss within groups	1.42	40	0.036	
Within Ss	1.84	88		
B (trials)	0.0023	2	0.0012	0.06
AB	0.06	6	0.01	0.5
B x Ss within groups	1.78	80	0.02	

TABLE 5 (c)

Summary of Analysis of Variance of Experimental Respiration Rate

Source of Variation	S.S.	d.f.	M.S.	F
Between Ss	988.6	131		
A (groups)	148.0	3	49.33	2.34
Ss within groups	840.6	40	21.02	
Within Ss	416.5	88		
B (trials)	20.7	2	10.35	2.15
AB	11.3	6	1.88	0.39
B x Ss within groups	384.5	80	4.81	

TABLE 6

Baseline and Experimental Heart Rates (Beats/Min.)*

	GROUPS											
	Control			Cognitive			Facial			Combined		
	Base	HR1	HR2	Base	HR1	HR2	Base	HR1	HR2	Base	HR1	HR2
	82.9	102.2	96.6	79.5	81.6	75.4	110.6	115.6	115.4	72.9	74.6	67.0
	74.2	74.2	71.4	62.3	74.6	59.8	86.2	82.8	80.6	69.1	79.4	79.4
	73.2	76.8	70.8	73.9	74.0	71.0	97.5	99.0	94.0	74.6	72.0	66.6
	79.1	83.7	78.0	73.6	82.8	78.4	86.2	90.8	86.0	73.8	75.0	69.6
	60.0	68.8	66.6	72.7	78.4	77.4	72.1	76.7	67.5	72.2	76.0	72.2
	83.3	93.4	89.1	70.9	72.8	71.9	75.0	77.9	73.2	83.0	86.1	78.7
	80.2	76.3	74.1	100.4	88.5	85.1	61.2	90.9	84.0	85.2	101.3	98.4
	74.0	78.0	74.5	55.2	55.0	53.2	66.1	71.0	66.0	67.1	62.3	64.7
	93.2	100.1	89.6	86.2	95.3	97.7	75.3	84.7	86.4	86.5	80.9	78.8
	86.0	87.8	87.7	92.3	86.0	88.9	85.2	87.6	83.4	51.7	72.1	56.1
	111.5	107.2	111.1	59.3	56.9	58.0	74.0	78.7	75.5	83.3	87.8	88.1
Means	81.6	86.2	82.7	75.1	76.9	74.3	80.9	86.9	82.9	74.5	78.6	74.5

* HR1 and HR2 = Average Rates for First and Second Halves of Trials, Respectively.

TABLE 7

Summary of Analysis of Co-variance of Experimental Heart Rate Adjusted for Baseline Heart Rate.

Anova Summary - Baseline

Source of Variation	S.S.	d.f.	M.S.	F
A (groups)	918.0	3	306.00	0.92
Ss within A	13352.7	40	333.82	
Total	14270.7	43		

Co-variance Summary - Experimental Data

Source of Variation	S.S.	d.f.	M.S.	F
A(groups)	1644.0	3	548.00	1.78
Ss within A	12291.3	40	307.28	
B (1st half/2nd half)	279.0	1	279.00	30.79**
AB	7.0	3	2.33	0.26
Residual	353.5	39	9.06	
A (adjusted)	276.6	3	92.20	1.05
Ss with A (adjusted)	3526.6	39	88.16	

** $P < .001$.

TABLE 8

Baseline and Experimental Inspiration-Expiration Ratios*

GROUP											
Control			Cognitive			Facial			Combined		
Base	IE1	IE2	Base	IE1	IE2	Base	IE1	IE2	Base	IE1	IE2
0.81	0.68	0.70	0.63	0.72	0.70	0.32	0.76	0.74	0.59	0.71	0.83
0.50	0.49	0.66	0.69	0.61	0.86	0.53	0.59	0.63	0.66	0.74	0.80
0.56	0.43	0.50	0.66	0.56	0.64	0.65	0.65	0.57	0.83	0.45	0.64
0.47	0.64	0.73	0.51	0.59	0.83	0.73	0.56	0.59	0.55	0.49	0.31
0.49	0.64	0.66	0.87	0.60	0.67	0.48	0.53	0.67	0.71	0.58	0.56
0.59	0.85	0.83	0.58	0.68	0.58	0.74	0.76	0.79	0.58	0.65	0.56
0.69	0.52	0.58	0.87	0.78	0.79	0.50	0.50	0.46	0.57	0.59	0.52
0.62	0.54	0.71	0.58	0.54	0.54	0.58	0.73	0.67	0.60	0.62	0.61
0.75	0.54	0.70	0.52	0.58	0.66	0.53	0.50	0.71	0.41	0.49	0.67
0.47	0.45	0.42	0.58	0.57	0.82	0.42	0.46	0.53	0.52	0.55	0.52
0.49	0.83	0.74	1.97	0.48	1.20	0.55	0.48	0.48	0.64	0.83	0.70
0.59	0.60	0.66	0.77	0.61	0.75	0.55	0.59	0.62	0.61	0.61	0.61

* IE1 and IE2 = Average Inspiration-Expiration Ratios for First and Second Halves of Trials, Respectively.

TABLE 9

Summary of Analysis of Co-variance of Experimental Inspiration-Expiration Ratios adjusted for Baseline Inspiration-Expiration Ratio

Anova summary - Baseline

Source of Variation	S.S.	d.f.	M.S.	F
A (groups)	0.63	3	0.21	1.91
Ss within A	4.3	40	0.11	
Total	4.93	43		

Co-variance summary - Experimental Data

Source of Variation	S.S.	d.f.	M.S.	F
A (groups)	0.08	3	0.027	1.17
Ss within A	0.93	40	0.023	
B (1st half/2nd half)	0.08	1	0.080	8.00**
AB	0.06	3	0.020	2.00
Residual	0.4	39	0.010	
A (adjusted)	0.04	3	0.013	0.62
Ss within A (adjusted)	0.85	39	0.021	

** $P < .001$

was taken in inspiration in the second halves of trials. There were no significant differences between groups on baseline data or on adjusted or unadjusted experimental data. However the cognitive group displayed relatively greater inspiration than expiration times.

Respiration rate. Table 10 contains baseline and experimental data; a summary of the analysis of co-variance is presented in Table 11. No significant variation was shown across halves of trials on this measure. Nor were there significant unadjusted or adjusted experimental group effects or interaction effects. Groups were not significantly different on baseline data. Mean scores were evenly spread; those of the combined strategies group were the lowest.

McGill Pain Questionnaire

One-way analyses of variance were performed for all the McGill interval measures. Experimental data for these measures are given in Tables 12 and 13; analysis of variance summaries are shown in Table 14. All of the measures showed the same tendency to lower pain ratings by the cognitive and facial groups.

Of the main measures, for NWC, $F(3, 40) = 4.67$, $P < .01$. A Tukey test showed cognitive and facial group means to be lower than the combined strategies group mean at the 5% level of significance (though neither was significantly different to the control group mean).

TABLE 10

Baseline and Experimental Respiration Rates (Resp./Min.)*

GROUP											
Control			Cognitive			Facial			Combined		
Base	Resp1	Resp2	Base	Resp1	Resp2	Base	Resp1	Resp2	Base	Resp1	Resp2
20.3	21.8	21.1	13.6	14.3	14.3	16.8	18.3	18.1	16.9	14.9	13.1
16.6	15.4	20.8	18.5	14.4	15.7	14.0	17.9	16.5	10.6	12.2	12.1
15.8	15.9	15.9	15.7	14.2	15.0	22.0	18.6	17.0	11.5	19.3	15.9
16.8	16.8	16.1	15.2	16.7	16.2	17.9	16.4	17.3	14.1	11.3	7.3
17.3	15.9	17.1	14.9	16.9	17.2	17.0	20.3	18.7	17.3	17.5	18.0
19.4	19.6	18.4	19.7	13.0	12.6	17.0	15.7	15.8	13.4	12.5	10.9
17.2	14.2	14.3	25.0	19.2	17.8	18.8	14.6	13.2	16.6	13.4	14.2
18.1	14.9	18.7	20.8	19.0	18.6	14.0	14.8	14.8	15.5	14.4	14.0
13.9	14.9	17.4	22.2	22.0	22.7	18.9	15.4	17.4	18.9	16.7	18.7
21.3	19.1	18.0	13.9	19.1	23.2	14.2	15.1	15.9	17.5	14.1	14.0
20.3	15.4	16.1	16.3	16.4	27.6	15.0	12.8	13.0	21.0	19.8	21.4
Means 17.9	16.7	17.6	17.8	16.8	18.3	16.9	16.4	16.2	15.8	15.1	14.5

* Resp1 and Resp2 = Average Rates for First and Second Halves of Trials, Respectively.

TABLE 11

Summary of Analysis of Co-variance of Experimental Respiration Rate adjusted for Baseline Respiration Rate

Anova summary - Baseline

Source of Variation	S.S.	d.f.	M.S.	F
A (groups)	66.1	3	22.03	1.26
Ss within A	697.5	40	17.44	
Total	763.6	43		

Co-variance summary - Experimental Data

Source of Variation	S.S.	d.f.	M.S.	F
A (groups)	98.5	3	32.83	2.25
Ss within A	584.6	40	14.62	
B (1st half/2nd half)	4.0	1	4.00	1.40
AB	14.7	3	4.90	1.71
Residual	111.4	39	2.86	
A (adjusted)	47.1	3	15.70	1.25
Ss within A (adjusted)	500.7	39	12.52	

TABLE 12

Main Measures* from Modified McGill Pain Questionnaire

	GROUP											
	Control			Cognitive			Facial			Combined		
	PPI	NWC	PRI-R	PPI	NWC	PRI-R	PPI	NWC	PRI-R	PPI	NWC	PRI-R
	2	12	26	1	4	6	2	7	20	3	15	40
	4	13	36	3	11	26	2	9	16	2	12	22
	2	7	25	2	10	20	3	9	23	2	11	30
	2	6	16	2	9	17	2	8	23	3	14	37
	2	13	30	2	5	14	2	5	12	3	11	32
	2	11	24	2	5	18	2	8	18	2	8	13
	2	10	25	1	7	13	1	7	12	1	5	11
	2	9	17	1	6	13	1	6	14	2	5	17
	2	8	22	2	9	22	2	8	20	2	15	47
	2	5	9	2	6	17	2	9	23	2	13	34
	2	13	33	3	8	29	1	5	6	2	5	12
Means	2.2	9.7	23.9	1.9	7.3	17.7	1.8	7.4	17.0	2.2	10.4	26.8

* PPI = Present Pain Intensity; PRI-R = Pain Rating Index - Ranked; NWC = Number of Words Chosen.

TABLE 13

PRI-R Component Measures* (McGill Pain Questionnaire)

	GROUP															
	Control				Cognitive				Facial				Combined			
	Sens	Aff	Eval	Misc	Sens	Aff	Eval	Misc	Sens	Aff	Eval	Misc	Sens	Aff	Eval	Misc
	18	1	2	5	5	0	0	1	19	0	1	0	31	2	4	3
	26	3	4	3	21	0	4	1	14	0	1	1	18	0	1	3
	25	0	0	0	17	1	1	1	21	0	1	1	28	0	1	1
	14	0	2	0	13	0	2	2	18	0	4	1	24	7	4	2
	26	1	1	2	14	0	0	0	12	0	0	0	28	0	4	0
	17	2	2	3	14	0	4	0	16	0	1	1	11	0	1	1
	24	0	0	1	11	0	0	2	11	0	1	0	7	0	4	0
	14	0	2	1	13	0	0	0	13	0	1	0	17	0	0	0
	18	0	4	0	21	0	1	0	18	0	2	0	36	3	3	5
	6	2	1	0	15	0	0	2	22	0	1	0	28	0	1	5
	26	1	4	2	17	2	5	5	6	0	0	0	12	0	0	0
Means	19.5	0.9	2.0	1.5	14.6	0.3	1.5	1.3	15.5	0	1.2	0.4	21.8	1.1	2.1	1.8

* PRI - Sensory; PRI - Affective; PRI - Evaluative; PRI - Miscellaneous.

TABLE 14

Summaries of Analyses of Variance on McGill Pain Questionnaire Measures

PPI

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	1.2	3	0.40	1.00
Within groups	15.8	40	0.40	
Total	17.0	43		

NWC

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	84.1	3	28.03	4.70**
Within groups	238.4	40	5.96	
Total	322.5	43		

** P < .01.

PRI-R

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	753.5	3	251.17	3.48*
Within groups	2886.7	40	72.17	
Total	3640.2	43		

*P < .05.

TABLE 14 (continued)

PRI-Sensory

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	378.4	3	126.13	2.90*
Within groups	1739.5	40	43.49	
Total	2117.9	43		

* P < .05.

PRI - Affective

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	8.8	3	2.93	1.83
Within groups	64.0	40	1.60	
Total	72.8	43		

PRI - Evaluative

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	5.9	3	1.97	0.79
Within groups	99.3	40	2.48	
Total	105.2	43		

TABLE 14 (continued)

PRI - Miscellaneous

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	13.2	3	4.40	1.97
Within groups	89.1	40	2.23	
Total	102.3	43		

For PRI-R, $F(3, 40) = 3.48$, $P < .05$ but there were no significant differences on Tukey test. One of the sub-scales - PRI-Sensory - yielded a significant F ratio though the Tukey test was again non-significant.

PPI, the third main measure, varied non-significantly.

Experimental data for the pattern of pain and pain onset measures are shown in Table 15. The cognitive and combined strategies groups tended to report more continuous pain though there was no significant difference between groups in a Kruskal-Wallis one-way analysis of variance ($H(3)= 6.73$, $P < .10$). On the additional question concerning gradual or sudden pain onset, 29 of the 44 Ss reported gradual onset. There were no significant differences between groups on this variable ($\chi^2(3) = 1.92$, $P < .70$).

Correlations

Product-moment correlations were calculated for all measures using the SPSS II computer package. Most correlations are low and usually non-significant. They add little to the preceding analysis. But it is interesting to note that the correlations between MPQ interval measures are frequently as high or higher than those reported by Melzack (1975). Table D1 in Appendix D contains inter-correlations from the present study. Table D2 presents the inter-correlations reported by Melzack (1975) for purposes of comparison.

Summary of Results

In general, adjusted immersion time results show a significant

TABLE 15

Secondary Measures from McGill Pain Questionnaire - Pain Pattern* and Pain Onset+

GROUP							
Control		Cognitive		Facial		Combined	
Pattern	Onset	Pattern	Onset	Pattern	Onset	Pattern	Onset
3	G	1	G	3	G	2	S
2	S	3	S	3	S	3	G
2	S	3	G	2	G	3	S
1	G	3	S	3	G	3	G
2	G	3	G	2	G	2	G
2	G	3	G	3	G	3	G
3	G	1	S	3	G	2	G
3	G	3	G	3	G	3	G
1	S	3	G	3	S	2	S
1	G	3	S	3	G	3	S
3	S	3	S	3	G	3	G

* 1 = Transient pain; 2 = Periodic Pain; 3 = Continuous Pain.

+ G = Gradual Onset of Pain; S = Sudden Onset of Pain.

increase in pain threshold for the cognitive strategy group only with the facial and combined cognitive and facial groups not differing from the control group. On the McGill Pain Questionnaire there were significantly lower ratings of pain on the Number of Words Chosen measure by the cognitive and facial strategies group compared to the combined strategies group only. Physiological measures failed to differentiate between groups though the inspiration-expiration ratio varied significantly within trials and heart rate varied significantly both within and across trials. There were no significant differences between groups on the control measures. Video ratings showed a significant increase in expressed pain within trials but not between groups.

CHAPTER 6

Discussion

The results of this experiment will be discussed in relation to the aims and hypotheses of the experiment and the findings of other work in the field. Issues of measurement and methodology will also be discussed as will problems with the study and suggestions for further research.

The implications of the above results for the hypotheses of this study are not altogether clear-cut owing to discrepancies between immersion time and McGill Pain Questionnaire (MPQ) measures and a lack of significant variation between groups on physiological indices.

The first hypothesis concerning the effectiveness of cognitive strategies in pain reduction is confirmed. The immersion-time measure was significantly increased for the cognitive strategies group. However, changes in MPQ ratings and physiological indices were non-significant. This result is also consistent with the studies reviewed earlier. An attempt was made to select a single cognitive strategy likely to be successful, in order to provide a basis of comparison for the facial expression strategy and this appears to have been achieved.

The evidence does not support the second hypothesis. The lack of effect on threshold time is consistent with the failure of facial expression change to lead to increased tolerance in the study by

Colby, Lanzetta and Kleck (1977). Effects on threshold had been expected, however, for two reasons. The first was that a threshold rather than a tolerance measure was used which (as discussed in the measurement section) should have been more sensitive to changes which were small or of short duration. The second reason for expecting an immersion-time change was that such a change would be consistent with autonomic and self-report changes found by Kleck and colleagues in each of their studies (Kleck et al, 1976; Lanzetta et al, 1976; Colby et al, 1977).

The MPQ measures also failed to confirm the effectiveness of the facial expression strategy in that none of the experimental group means differed significantly from those of controls. (Though both the cognitive and facial groups made significantly lower pain ratings than the combined strategies group). Weak support was provided however in that both the cognitive and facial groups produced lower pain ratings of about the same extent on the Number of Words Chosen and PRI-R measures. The lack of significant effects here may be part of a general problem in obtaining consistency in measures (e.g., Beers & Karoly, 1979; Girodo & Wood, 1979; Scott & Barber, 1977a), or may be due to problems in the application of the MPQ in this study. This will be discussed more fully in the section dealing with the MPQ.

An additional difficulty was the failure of the physiological measures in this study to support the effectiveness of facial expression as a pain control strategy. The one physiological correlate used in the studies by Kleck and colleagues - skin conductance - was not used in this study. It is possible that skin conductance may be a more sensitive measure of autonomic arousal in

experiments of this type. Barber and Hahn (1962) found significant correlations of several physiological indices, including skin resistance, with subjective ratings of pain. However, Engel (1959) found that skin conductance did not vary significantly according to length of exposure to ice water (up to three minutes). It is not clear whether a skin conductance or skin resistance measure would be more sensitive to the effects of the strategies used in this study. In any case, even the physiological indices usually associated with pain, for example, heart rate, inspiration-expiration ratio and respiration rate (Sternbach, 1968) did not vary significantly between groups.

There was no support for the third hypothesis. Instead of additive effects of the two strategies having occurred there appears to have been interference effects. Use of combined strategies may have been viewed by Ss as too difficult or lacking credibility. This may be reflected in the MPQ result where combined strategies pain ratings were higher than those of controls on the Number of Words Chosen and PRI-R measures. On the more objective immersion time measure, the interference appears to have resulted in the lowering of threshold times to a little below those of the cognitive group.

The implications of the results for the adequacy of the measures themselves also needs examination. While the immersion time measure appeared to change lawfully (and significantly) the study was not primarily testing the reliability and validity of measures as such so only limited conclusions can be drawn. The most serious difficulty with the threshold measure used was that nine of the 44 Ss did not reach pain threshold before maximum trial

time on any of the experimental trials. This would have reduced the effect of experimental variables as these Ss were not feeling pain.

The physiological measures failed to differentiate between groups. Some interesting findings were nevertheless made. The most important was the highly significant reduction for all groups of heart rate within and across trials although heart rate would be expected to increase within trials as a function of increased arousal as pain was anticipated and experienced. This may indicate that the pain stimulus was not sufficiently intense. The reduction across trials is consistent with the expectation that arousal decreased because pain reduction strategies were becoming more effective; however, the lack of differential effects between groups appears to indicate that the effect was due to simple stimulus habituation over repeated exposures.

The second finding of a significant increase in inspiration-expiration ratio from the first to the second half of each trial may support this interpretation. There is evidence that pain leads to a reduction in the inspiration-expiration ratio in guinea pigs (Schiavi, Stein & Sethi, 1961) and that unpleasant emotional states, simulated or real, can also lead to a reduced inspiration-expiration ratio (Feleky, 1914; Stevenson & Ripley, 1952). Woodworth and Schlosberg (1954) also cite an early study (Drozyński, 1911) which found lower inspiration-expiration ratios to be associated with a feeling of tension.

It is difficult to interpret the findings regarding inspiration-expiration ratio in this experiment, however. Because a threshold

measure of pain was used, any pain would have been experienced only during the last small portion of a trial. Physiological responses to the pain would be apparent only for a relatively short time at the very end of the trial. Because of the scoring procedures (whole and half-trial measures), any response to pain would most likely be masked (or swamped) by the physiological activity occupying the major portion of the scoring period.

With regard to the MPQ, the most important point requiring explanation is the discrepancy between the MPQ and immersion time measures in between-group analyses. In immersion times, the cognitive strategy group showed significantly less pain than controls, with the combined strategies being the next most effective treatment. On MPQ ratings both the cognitive and facial strategy groups experienced significantly less pain than the combined strategies group, that is MPQ pain ratings were highest for the combined strategies group (and next to lowest on immersion times).

One explanation of the discrepancy might be that the MPQ measures in this study were more subject to experimental demand than were immersion time measures. This is because instructions referred to physiological reactions to cold, mentioned pain ratings, did not mention immersion times and stated explicitly that it was not the purpose of the experiment to determine how much pain S could bear. This may have led to the apparent effectiveness of facial-expression change on the MPQ. However, because the cognitive strategy group also showed increased immersion times (increased pain threshold), it is likely that part of the effect of cognitive strategies on pain ratings was treatment effect (and not just the effect of

demand). Yet this fails to explain why the combined strategies led to such high pain ratings. One explanation is that the demand effects may have operated most strongly on those treatments that were credible to Ss (Orne, 1962) and that the combined strategies may have been less believable than each of the strategies separately. This accounts only for the demand component, however. One might have expected the combined strategies group results to have fallen between the controls on the one hand and the other two experimental groups on the other. In fact, the combined strategies group rated pain most highly of all the groups. There is no obvious interpretation of this anomaly.

Another reason for the discrepancy between immersion time and MPQ may be that the MPQ was administered to each S after pain inductions had concluded. Instead of being a measure of ongoing pain it was a post-trial measure. Furthermore, Ss were asked to use the MPQ as an overall measure of the pain experienced in the experimental trials which had taken place over the previous 60 minutes. But it is likely that Ss made ratings on the basis of the final experimental trial, that being most easily remembered. By comparison, immersion times were an objective measure of felt pain taken during each pain induction.

An additional consideration relevant to the above discrepancy is the possibility of fatigue in that the MPQ came after approximately 90 minutes of baseline and experimental trials and several sets of other post-trial measures (EPI, Betts QMI and Gordon test). The concentration of Ss may have lapsed. This may also have affected the validity of the measure.

Perhaps it is difficult in this field, as Scott and Barber (1977a) claim, to achieve consistency in measures. This argument is supported by the results of Beers and Karoly (1979), Girodo and Wood (1979), Scott and Barber (1977a). The difficulty in obtaining consistency in measures may be spurious. Sternbach (1968) and Melzack (1973) point out that, pain being a multi-dimensional experience, the various indices of it may vary quite widely.

If the difficulty is not spurious, it would appear that the discrepancy between immersion time and MPQ results may be due to problems in the application of the MPQ. The MPQ also yielded little information on the several pain dimensions described by Casey and Melzack (1967), Melzack (1973) and Melzack and Casey (1968). If there was any systematic effect on pain perception, then it was manifest over all treatments (and controls) and produced no differences on MPQ dimensions relative to each other.

Despite this, there were two interesting findings regarding the MPQ. The first is that the inter-correlations between MPQ sub-measures are similar to those reported by Melzack (1975) in the major early study on the MPQ. This result indicates reasonably high consistency in pain measurement in the two studies and argues for the reliability of the MPQ as a measure. The second finding is in relation to the only (published) investigation of the MPQ in measuring experimental pain, that is, the study by Klepac, Dowling and Hauge (1981) referred to in the section on pain measurement. In the two comparable groups of the Klepac study and the current study (i.e., the cold pressor pain threshold group and the control group, respectively) the word-groups chosen from question C of the modified MPQ are similar. Details of word-groups chosen by Ss are

presented in Appendix E. The most important feature to be noted is that in both cases only a small proportion of Ss chose words from the affective category (word-groups 11-15 in the Klepac study and 11-14 in the current study). It was previously argued that a major difference between experimental and clinical pain was the much stronger affective/anxiety component in clinical pain. Both the Klepac study and the present one have found a low affective component in experimental pain and this constitutes good prima facie support for the construct validity of the MPQ.

Methodological issues. Perhaps the main problem with studies dealing with cognitive strategies is the lack of observability of such strategies. This has two implications, the first being that one cannot accurately determine if and how the cognitive strategy was used. The second aspect is that spontaneous idiosyncratic strategies may be used both in control and experimental groups. As a consequence one is limited to drawing conclusions concerning the effect of instructions about cognitive strategies.

Problems with the study. In the course of the study a number of problems in its design has become clear. The main one was that too many Ss (nine of 44, comprising one S from the control and combined strategies groups, three from the facial and four from the cognitive strategy group) reached the maximum stimulus exposure time. Informal pilot testing to establish stimulus parameters was obviously unrepresentative of the whole experimental subject population. The effect of this 'ceiling effect' was to reduce variation between groups following treatment. This problem may have been avoided if colder water had been used. Another solution may have been to divide Ss into high- and low-threshold groups on the basis of baseline as was done by Chaves and Barber (1974) and Spanos, Horton and

Chaves (1975). This would also have allowed analysis of a threshold variable.

Another variable that could have been analysed was gender differences in the response to pain control strategies. The number of Ss in the present study was too small to provide useful information on this point. Of the studies reviewed, only that of Blitz and Dinnerstein (1971) analyses gender differences (finding a greater elevation of pain threshold for males than for females using cognitive strategies; there was no differential effect on pain tolerance).

A finer analysis of changes in physiological measures would have been possible if the scoring system had been altered, that is, if the last 3-5 seconds of each trial had been scored and compared to 3-5 second periods from the beginning and middle of each trial. This would be a more sensitive comparison to isolate short-term (phasic) effects in physiological systems. The scoring system used was appropriate for relatively long term (tonic) changes in response.

The other main problem in the study concerns the MPQ. It may have been preferable to have relied less on immersion times and to have administered the MPQ after the baseline trial, then again after each experimental trial. This would have provided information on any trial-by-trial changes in felt pain. But the validity of such a procedure is not clear. For example, Ss' memory of words chosen initially may have affected their choice on subsequent trials. As noted previously, the Number of Words Chosen score may show reduced variation over several trials (Melzack & Perry, 1975).

Conclusion. The general conclusion of this study is that the effectiveness of the cognitive strategy as a pain control device has been confirmed. Also confirmed is the finding by Colby, Lanzetta and Kleck (1977) of the lack of effect on psychophysical measures of changes in facial expression. While Ss considered that the facial expression strategy was helpful, there is not enough indication of the effectiveness of facial expression change to warrant further investigation of its use as a clinical technique. Nor did facial expression add to the effect of the cognitive strategy in the combined strategies group so this combination of strategies does not appear to be viable.

With regard to experimental measures, the utility of taking several measures has been shown, perhaps most clearly with the physiological indices which indicated problems regarding stimulus habituation and sub-optimal stimulus intensity. It would be difficult to identify these problems without the evidence provided by the physiological measures.

The threshold immersion time measure has shown lawful variation according to the strategies used and enough information has been derived from and about the MPQ to warrant further investigation of its use in experimental pain studies. For example, further study is required concerning its ability to discriminate the various types of experimental pain to facilitate comparisons between studies using various pain induction methods.

Like much research on experimental pain, this study has shown that experimental pain can be reliably induced and measured and to some extent controlled.

The necessary refinement and development of psychological pain control techniques can be most efficiently and effectively achieved by using experimentally-induced pain in normal subject populations. The knowledge gained from these studies can then be 'trialed' on patients with clinical/chronic pain and eventually may lead to an extension of viable pain control techniques which can be used to reduce human suffering.

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APPENDIX A

Modified McGill Pain Questionnaire

If you felt pain during the last three exposures to cold water;

A. Please rate it on the following scale (by circling appropriate word)

1	2	3	4	5
Mild	Discomforting	Distressing	Horrible	Excruciating

B. Please choose one of the following word groups describing the pattern of pain.

	continuous, steady, constant
	rhythmic, periodic, intermittent
	brief, momentary, transient

C. Please describe the pain itself by circling the words that best describe it. In this section use only a single word. In each appropriate category the one that applies best. Leave out any category that is not suitable.

D. Any comments of your own?

1	2	3	4
1 Flickering	1 Jumping	1 Pricking	1 Sharp
2 Quivering	2 Flashing	2 Boring	2 Cutting
3 Pulsing	3 Shooting	3 Drilling	3 Lacerating
4 Throbbing		4 Stabbing	
5 Beating		5 Lancinating	
6 Pounding			

5	6	7	8
1 Pinching	1 Tugging	1 Hot	1 Tingling
2 Pressing	2 Pulling	2 Burning	2 Itchy
3 Gnawing	3 Wrenching	3 Scalding	3 Smarting
4 Cramping		4 Searing	4 Stinging
5 Crushing			

9	10	11	12
1 Dull	1 Tender	1 Tiring	1 Sickening
2 Sore	2 Taut	2 Exhausting	2 Suffocating
3 Hurting	3 Rasping		
4 Aching	4 Splitting		
5 Heavy			

13	14	15	16
1 Fearful	1 Punishing	1 Wretched	1 Annoying
2 Frightful	2 Gruelling	2 Blinding	2 Troublesome
3 Terrifying	3 Cruel		3 Miserable
	4 Vicious		4 Intense
	5 Killing		5 Unbearable

17	18	19	20
1 Spreading	1 Tight	1 Cool	1 Nagging
2 Radiating	2 Numb	2 Cold	2 Nauseating
3 Penetrating	3 Drawing	3 Freezing	3 Agonizing
4 Piercing	4 Squeezing		4 Dreadful
	5 Tearing		5 Torturing

APPENDIX B

Summaries of Analyses of Variance of Control Measures.

Ages of Ss

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	2.4	3	0.80	0.03
Within groups	1151.8	40	28.80	
Total	1154.2	43		

EPI - Neuroticism

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	42.6	3	14.20	0.82
Within groups	690.3	40	17.26	
Total	732.9	43		

EPI - Extraversion

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	10.5	3	3.50	0.31
Within groups	452.2	40	11.31	
Total	462.7			

APPENDIX B (continued)

EPI - Lie Scale

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	5.2	3	1.73	1.15
Within groups	60.0	40	1.50	
Total	65.2	43		

STAI - State Anxiety

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	204.2	3	68.1	0.90
Within groups	3036.5	40	75.9	
Total	3240.7	43		

STAI - Trait Anxiety

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	58.4	3	19.47	0.35
Within groups	2222.4	40	55.56	
Total	2280.8	43		

APPENDIX B (continued)

Betts QMI - Total Score

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	713.4	3	237.80	0.35
Within groups	26967.8	40	674.20	
Total	27681.2	43		

Betts - Tactile Imagery

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	29.2	3	9.7	0.50
Within groups	781.3	40	19.5	
Total	810.5	43		

Gordon Test

Source of Variation	S.S.	d.f.	M.S.	F
Between groups	21.3	3	7.10	0.77
Within groups	371.3	40	9.28	
Total	392.6	43		

APPENDIX C

Summary of Analysis of Variance of Videotape Ratings.

Source of Variation	S.S.	d.f.	M.S.	F
Between Ss	63.3	7		
A (groups)	7.3	3	2.43	1.74
Ss within groups	56.0	40	1.40	
Within Ss	74.7	80		
B (parts of trial)	10.5	1	10.50	7.45**
AB	7.7	3	2.57	1.82
B x Ss within groups	56.5	40	1.41	

** P < .01

Summary of Point Biserial Correlations between Videotape Ratings and Parts of Trial.

Group	r_{phi}	t	d.f.	P
Z	0.29	1.54	20	< .20
C	0.41	2.39	20	< .05 *
F	0.21	1.06	20	> .20
B	0.26	1.35	20	< .20
All Ss.	0.27	2.93	86	< .01**

APPENDIX D

Table D1. Inter-correlations (r) of PRI (R) and PPI scores on standard MPQ reported by Melzack (1975).

	SENS	AFF	EVAL	MISC	TOTAL (PRI-R)
SENS					
AFF	.41				
EVAL	.27	.42			
MISC	.35	.45	.22		
TOTAL (PRI-R)	.87	.70	.49	.69	
PPI	.29	.42	.49	.18	.42

APPENDIX D (continued)

Inter-correlations (r) of Modified and Standard McGill Pain Questionnaires.

Table D2. Inter-correlations (r) PRI (R) and PPI scores on modified MPQ as used in the present study (N = 44).

	SENS	AFF	EVAL	MISC	TOTAL (PRI-R)
SENS					
AFF	.35* (P<.02)				
EVAL	.35 (P<.02)	.44 (P<.003)			
MISC	.45 (P<.02)	.45 (P<.002)	.31 (P<.04)		
TOTAL (PRI-R)	.94 (P<.001)	.56* (P<.001)	.55 (P<.001)	.62* (P<.001)	
PPI	.58 (P<.001)	.47 (P<.001)	.53 (P<.001)	.33 (P<.03)	.65 (P<.001)

* Inter-correlations lower than Melzack (1975).

APPENDIX E

Percentage of Ss Choosing Each Word-group on the MPQ

Word-group	Control group of the current study (N = 11)	Cold pressor pain threshold group of the Klepac et al (1981) study (N = 20)
Sensory		
1	82	65
2	46	25
3	64	60
4	55	40
5	73	60
6	27	5
7	9	25
8	64	80
9	73	70
10	46	40
Affective		
11	9	10
12	0	5
13	0	10
14	46	20
15	0	5
Evaluative		
16	82	70
Miscellaneous		
17	91	90
18	82	80
19	82	100
20	46	35