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Primary emotions in the affective contribution to chronic pain

Fernandez, Ephrem, Ph.D.
The Ohio State University, 1989
PRIMARY EMOTIONS IN THE AFFECTIVE CONTRIBUTION TO
CHRONIC PAIN

DISSERTATION

Presented in Partial Fulfillment of the Requirements for
the Degree of Doctor of Philosophy in the Graduate
School of the Ohio State University

By

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* * * * *

The Ohio State University

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To My Family
I thank my adviser, Dr. Steven Beck who in the spirit of true mentorship allowed me independence to develop my specialized research interests while shaping them with critical feedback. Thanks to Dr. Herbert Mirels for conceptual input and for directing the course of my clinical training over the last four years. I am grateful to Dr. Thomas Nygren for advice on psychometric scaling and statistics, and to Dr. John Corrigan for making available the patient population at the Department of Physical Medicine and overseeing the operation of this study. I also extend my appreciation to Plenum Press and Dr. Carroll Izard of the University of Delaware for permission to utilize and reproduce photographs from Dr. Izard's (1977) book on human emotions. Finally, for their sensitivity and support over the years, I owe much to my parents, brother and sisters.
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CHAPTER I
INTRODUCTION

Pain is an evolving concept. Its history has been traced by Dallenbach (1939) and more recently by Procacci (1980) and Raj (1986); collectively, these accounts describe the transition from religiophilosophical perspectives associating pain with "sin" and "evil", to scientifically-derived notions relating pain to "tissue damage". Perhaps most salient in this evolution has been the ascent from a monolithic view to a multidimensional one in which pain is now conceptualized as a complex experience involving sensations, emotions, cognitions, behaviors and neurochemical events. All but one of these ("emotions"), have become major sub-specialties within the domain of pain research. The few studies relating pain and emotion are themselves scattered in a manner as to be in conducive to clarity on the subject. An attempt is therefore made here to delineate, organize and critically review the amorphous literature on pain and emotions. In this Introductory chapter, empirical studies that distinguish affect from sensation in pain will be evaluated; these are grouped according to methodology, namely, multivariate statistics,
signal detection paradigm, scaling techniques and clinical reports. The affective aspect of pain will then be elaborated with reference to recent psychological and biological findings on emotion. Primary emotions will be identified and linked to pain. Specific hypotheses will be generated about the separate contributions of sensation and affect in pain and the varying degrees to which different emotions make up the affective variable in pain. Furthermore, cognitive and behavioral correlates of each emotional response to chronic pain will be developed. Finally, the effect of multidisciplinary treatment on pain and its emotional, cognitive and behavioral correlates will be addressed. All these issues will be formulated into a research design and investigation that will become the subject matter of succeeding chapters.

**Distinguishing Affect from Sensation in Pain**

Equating pain with tissue damage, is questionable. Firstly, pain can occur in the absence of identifiable tissue injury. Sometimes termed "psychogenic pain", this condition has been observed in patients with conscious or unconscious guilt feelings (Engel, 1959) and among psychiatric patients (Chaturvedi, 1987). It is also prevalent among individuals for whom monetary compensation or other types of "tertiary gain" are contingent upon pain behavior (Bokan, Reis & Katon, 1981). Furthermore, pain
can persist in conditions like causalgia and phantom limb pain where it is apparent that the tissue injury has long healed -- a phenomenon referred to as "spontaneous pain" (Melzack, 1973).

The converse, tissue damage without pain, is also possible. Cases of congenital and other insensitivity to pain have been reported by Jewesbury (1951), Sternbach (1963) and reviewed by Carr (1977).

The lack of constant conjunction between tissue damage and pain is also consistent with observations that in many situations, pain begins long after tissue injury has occurred (Wall, 1979). The injury may culminate in fatality and yet involve disproportionately little pain (Melzack & Wall, 1982).

Pain is a perceptual experience, and one characteristic differentiating it from a mere sensation is its inherent affective quality. It has two defining features: (i) it is a bodily sensation, and (ii) it carries an aversive affect. The first means that pain is characterized with reference to spatial (anatomical) coordinates such as site on body, distribution and depth (cutaneous or deep pain). The second premise asserts that pain is invariably unpleasant. Even in the aberrant instance of masochism, pain is never regarded as pleasant in itself though it may be promoted due to accompanied rewarding consequences (Reik, 1941). In sum, pain is a bodily sensation with an unequivocally
aversive affect (and an association with tissue damage or tissue stress).

In view of the aversiveness of pain, several scholars have propounded sensory and affective components of pain. Early this century, Sherrington (1900) had proposed affective tone as an attribute of all sensation. Hardy, Wolff and Goodell (1952) suggested that pain comprises a sensory component related to stimulation of sensory nerve endings, and a reaction/processing component embodying distress and emotional reactions. Beecher (1957) adopted a similar view using the substitutive terms, "primary" and "secondary" pain components. Melzack (1973) spoke of pain as comprising a sensory-discriminative dimension relating to spatial and temporal properties, a motivational-affective dimension incorporating tension, fear and autonomic events, and a cognitive-evaluative dimension of pain as a whole. Tursky (1976) referred to intensity (quantitative), reactive (e.g., agony, distress) and sensory (qualitative) components of pain.

Efforts at separating sensory from affective components of pain, may be grouped under: (a) theoretical accounts, (b) clinical impressions, (c) multivariate statistics, (d) signal detection theory applications, and (e) scaling studies. Each of these groups will be reviewed, with regard to the question of whether a sensory-affective distinction in pain is tenable.
Theoretical Accounts

There has been astonishingly limited theoretical discourse on the nature of pain, outside the discipline of philosophy. The few conceptual articles bearing titles about pain and emotion, merely acknowledge the inherence of emotion in pain without elaborating on dynamic aspects of this relationship (e.g., Copp, 1974; Swanson, 1984; Walters, 1952). Three noteworthy exceptions are the work of Szasz (1957), Merskey and Spear (1967) and Leventhal and Everhart (1979), each of which will be discussed in turn.

Szasz (1957) writing at a time when it was customary to view pain solely as an organic disturbance, asserted that pain is neither exclusively sensation nor exclusively affect. Adopting a psychoanalytic orientation, he construed pain in the same light as Freud’s concept of anxiety i.e., pain, like anxiety, is a principal threat to the ego. (The ego as in object relations theory, is regarded as relating not only to other persons like the mother, but also to the body of the self as an object). Sensory features of pain serve to signal damage or danger to body parts and consequent threats to the structural and functional integrity of the ego; affective features play a communicative role in securing help for the endangered body. Szasz then went on to identify symbolic meanings within affective responses to pain, namely, the plea for help, the feeling of rejection when that plea is
frustrated, and the emergence of aggression against the frustrating agent. The psychoanalytic terms have since been replaced with cognitive-behavioral concepts (Szasz, 1975), but one contribution of this classic remains -- a challenge of the old idea that pain is predominantly a bodily sensation.

The succession of arguments favoring a distinction between sensory and affective components of pain, has not gone undisputed however. Merskey and Spear (1967) contended that as common as it has become to distinguish between sensory and affective pain, the distinction could be dismissed as a semantic error leading to misconceptions in theory and practice. They argue that it is impossible to introspectively separate pain sensation and perception and that it may be more appropriate to speak of contributory causes instead of separable components of pain. In the authors' words: "the idea of sensation and reaction components as distinguishable parts of experience is less well established, has less justification anyhow, and should probably be dropped" (p. 62). It is instructive to note that the authors do not dispute the existence of sensory and affective variables underlying pain, but simply maintain that pain is experienced in a holistic way unamenable to decomposition into constituent parts. This is essentially an empirical issue, and some of the research to be reviewed later will bear directly on it.
Leventhal and Everhart (1977) have gone to much technical detail in presenting a "parallel processing model" linking emotion and pain. They postulate that unlike earlier views (such as those of Beecher) wherein the reactive component accompanied its sensory counterpart, sensory information and affective events are processed almost simultaneously. The processing occurs preconsciously with only some of the processed material passing from perception into focal awareness via attentional filters. Separation of the sensory from the affective appears at the onset of stimulus encoding because of the kind of receptor specialization described by Melzack (1973). The separate pathways thus generated, interact at the site of the "gate" in Melzack and Wall's theory of pain. Thereafter, the pathways receive input from perceptual-motor systems (that are automatic), schemas (constructed from memory of past pain experiences) and conceptual processing (based on memory of past pain episodes). These hypothesized stages are regarded as compatible with neurological accounts of pain transmission. The authors also buttress their model with some findings on modification of pain by hypnosis, expectancy and informational variables, attentional and cognitive strategies.

Some of the above abstractions of Leventhal and Everhart are not easily testable, but the model as a whole
is putatively consistent with neuropsychological opinion. To the extent that it recognizes sensory and affective components in the pain experience, it is in keeping with Szasz's ideas. However, it departs from the position of Merskey and Spear, by stipulating two discrete (albeit parallel) systems for processing sensory information and affect, each of which can be individually altered while the other remains invariant. A review of empirical findings may help shed light on these divergent perspectives.

Clinical Impressions

Historically, some of the first impressions regarding the interplay of sensation and affect in pain, arose in the context of clinical treatment. The two principal modes of medical treatment to be considered here are psychosurgery and psychotropic medication. These rarely adhered to proper research design and control, and results were frequently published as anecdotal reports -- thus the word "impressions" in the above heading. These impressions were nevertheless instrumental in the aggregation of support for a sensory-affective conceptualization of pain. The survey of this body of literature will of necessity be brief, and only key publications will be cited.

Psychosurgery. Psychosurgery refers to the removal or destruction of a region of the brain in order to remedy
severe psychiatric disorders (Valenstein, 1980). The area operated on is itself usually free of discernible disease but presumed to be responsible for the aberrant psychological condition. Traditionally, such surgery was performed on the mentally infirm, but it has also been used as a last resort for extreme, intractable pain.

Nineteen different types of psychosurgery have been identified for the treatment of intractable pain (Bouckoms, 1984). Perhaps the best known of these is the leukotomy procedure developed by Moniz (1936) and popularized by Freeman and Watts (1942). This entailed the insertion of a knife (leukotome) into the frontal lobes through trephine holes drilled on both sides of the skull. The knife was then pivoted up and down to make a cut in the desired plane; usually, the white matter in both frontal lobes was sectioned in the plane of the coronal suture directly down to the sphenoidal ridge, thus effecting a lobotomy. The operation was ordinarily performed under local anaesthesia except in cases of severely disturbed psychiatric patients. As a result of such surgery, pain patients frequently reported no change in the sensation of pain, but a substantial decrement in the emotional distress ensuing from pain.

Otenasek (1948) has provided some case reports of lobotomized patients. One of these concerns a 58-year old man admitted to a medical services unit for pain in the
chest and back of two months' duration. The patient soon
developed paralysis in the legs and was diagnosed as having
prostrate carcinoma and widespread metastases of the
backbone that accounted for severe localized pain at the
thoracic level. After unsuccessful attempts to treat the
pain with morphine, tubo-curare and a laminectomy, a
prefrontal lobotomy was performed six months following
admission. Two weeks later, the patient's condition was
described by the medical house-officer as:

"Clinically Thomas is, of course, no better, . . .

Mentally, however, he is indeed a new man. He
realizes that he is sick and that he is in pain from
time to time, but it does not bother him one whit. He
tells us that he feels just fine and I'm sure he means
it" (Otenasek, 1948, p. 231).

A similar picture emerges in Hardy, Wolff and Goodell's
(1952) survey of 38 prefrontal leukotomies performed (at
the New York Hospital) for relieving pain of metastatic
cancer, Hodgkins Disease, radiculitis and other illnesses.
Of the 25 patients who underwent unilateral lobotomy, 15
witnessed no alteration in pain while 10 experienced a
reduction in pain. Of the thirteen who underwent bilateral
 lobotomy, two reported no change but 11 were relieved of
pain. In many of the observed instances of pain reduction,
the pain ceased to bother the patient even though its
sensory threshold remained unaltered. The problem however,
was that these patients ceased to be bothered by much else, implying that the reduction in pain complaints was part of a generalized indifference to all stimuli. As Hardy et al. noted,

"These patients exhibited in many ways . . . a flattened affect if not only actual apathy. . . . They failed not only to complain of their spontaneous pain but also of their needs, such as personal nursing care, need of urine bottle, bedpan, or the adjustment of an uncomfortable dressing. When incontinent of feces they were indifferent to the odor it spread about their 'persons and beds' " (1952, p. 317).

The authors point out that the patients were not incapable of reacting to the pain, for when urged to focus attention on it, they frequently acknowledged its presence with stereotypic pain behaviors. In essence, leukotomy does selectively obfuscate the emotional component of pain but this effect is secondary to a generalized affective detachment.

To overcome the cognitive and personality deterioration after large ablations of brain tissue, freehand psychosurgery was superseded by stereotactic operations. The advantage was that the head could be positioned in a fixed plane within a stereotaxic device, and with the aid of three-dimensional maps (stereotaxic atlases) and television monitors, electrodes or other instruments could
be inserted through small holes in the skull into circumscribed areas of the brain. The first application of this method was reported by Spiegel, Wycis and Freed (1949) in which the dorsomedial thalamus of patients was destroyed by electrolysis. This ameliorated pain with a minimum of deleterious effects. A review of similar thalamotomy studies (Bouckoms, 1984) concluded that this surgery ameliorates cancer pain in at least 60 percent of cancer patients although the likelihood of relapse, operative motility, dysphasia and dysaesthesias makes the procedure uncommon today.

In contrast, streotactically-guided removal of the cingulate gyrus has attracted more clinical interest. Foltz and White (1962) indicated that this procedure might modify the emotional response to pain. Consistent with this, they observed the largest decrements of pain in patients with a significant anxiety-depressive element. Gutierrez-Lara (1973) also established that significant reduction in anxiety accounted for much of the reduced pain among 390 cingulotomy patients; no psychological deficits were apparent. A variety of psychosurgical techniques (including electric current, proton beams, ultrasound and radio frequency waves) have been used to eliminate specific brain regions, with the objective of relieving pain. In 60 to 90 % of the cases, a reduction in suffering and anxiety associated with pain has been witnessed (Bouckoms, 1984).
The practice of psychosurgery has attracted trenchant criticism however. Salient among these is the already-mentioned fact that (especially in the case of leukotomy), global psychological deficits arise sometimes leading to depersonalization of the patient. Risks of brain infection and other iatrogenic complications have also been a serious problem. Yet, one must be mindful that psychosurgery often served as a desperate intervention when all else failed (Valenstein, 1986). In the course of its clinical application, the significant impression emerged that pain is both sensory and affective in nature, and this paved the way for more controlled research on these components of pain.

**Psychotropic medication.** The gradual demise of psychosurgery for pain, seemed to coincide with the development of psychotropic drugs. These are affect-altering drugs traditionally employed in the treatment of psychiatric disorders and affective disturbances. Their potential for alleviating pain was soon realized and explained in terms of a reduction in anxiety or elevation of mood (Budd, 1978). Unlike narcotics such as morphine that impact largely on the sensory properties of pain, the psychotropic drug acts on and thereby reflects an affective component of pain.
A class of mood-elevating drugs often prescribed for pain are the antidepressants which range from monoamine oxidase inhibitors like isocarboxasid to tricyclic antidepressants like imipramine. To study these drugs closely, Bradley (1963) recruited 35 subjects with pain and depression. In those experiencing the two conditions simultaneously, administration of tricyclic antidepressants and monoamine oxidase inhibitors had a remedial effect on both sensory and affective aspects of pain; however, in patients who experienced depression as an accompaniment of pain, the same antidepressants only ameliorated the depressive features but not the sensory properties of pain. This seemed to suggest that nociceptive stimulation produces negative affective consequences that are susceptible to the influence of antidepressants.

Several other studies have followed in which those with chronic pain have obtained relief from pain as a function of the attenuation of depressive symptoms. Lascelles (1966) found that depressive features in patients with atypical facial pain were reduced by narail (a Monoamine Oxidase Inhibitor), while severe depression associated with the same condition responded to imipramine. Okasha, Ghalet and Sadek (1973) found that remediation of psychogenic headache was correlated with the relief of depressive symptoms upon the administration of doxepin, a tricyclic antidepressant. Other studies have been summarized by
Monks and Merskey (1984). Feinmann (1985) however has noted several studies in which antidepressants produced an actual analgesic effect. As Whitlock (1982) has suggested, this may be because antidepressants promote levels of brain serotonin thereby elevating mood, but furthermore boosting the action of endogenous opiates so as to reduce the sensory intensity of pain as well. In any case, a conspicuous affective component of pain has been implied by the early studies in which certain antidepressants reduced pain primarily by attenuating depression.

Tranquilizers are another class of psychotropic drugs that once attracted much zeal among pharmacotherapists working on pain. Well known for their anxiolytic effect, minor tranquilizers like benzodiazepines were observed to diminish acute pain with a significant anxiety ingredient, as in the case of gastrointestinal disorders (Lasagna, 1977). However, on the whole, they have been a disappointing group of agents for treating chronic pain (Budd, 1978). Nevertheless, the fact that a sedative was shown to reduce pain (albeit minimally), is important since it attests to a veritable affective component in pain.

In closing, it must also be added that all the drugs in this category pose concerns when used as agents for pain control. The tricyclic antidepressants produce allergies and hypersensitivity, untoward outcomes in the central nervous and cardiovascular systems, and anticholinergic
autonomic effects, while monoamine oxidase inhibitors may cause urinary retention, orthostatic hypotension and severe hepatotoxic reactions (Monks & Merskey, 1984). In addition to these side effects, there is the problem of tolerance and addiction which especially applies to the minor tranquilizers. What remains important for purposes of the present discussion, is that the use of psychotropic drugs led to growing appreciation of the affective variable underlying pain.

**Multivariate Statistics**

The language of pain is made up of words describing sensory properties of pain (e.g., "sharp pain", "throbbing pain") and words alluding to affective qualities (e.g., "nagging pain", "terrifying pain"). This, it might be argued, implies that pain has sensory and affective components, particularly if the pain vocabulary can be reliably divided into two such categories. Multivariate statistics have therefore been introduced to ascertain if in fact pain descriptors coalesce along superordinate dimensions best labelled as "sensory" and "affective". The methodology employed has mainly involved Factor Analysis, and to a lesser extent, Multidimensional Scaling and Cluster Analysis. Common to all these procedures, is the simultaneous analysis of a number of variables without designating them as independent or dependent, so as to
uncover the latent structure underlying these variables (Kerlinger, 1986).

Proceeding from Dallenbach's (1939) list of 44 words describing various qualities of pain, Melzack and Torgerson (1971) obtained additional pain descriptors from the clinical literature and from patients. A final collection of 102 words was then presented to physicians and graduate students to categorize. Data revealed 16 subclasses organizable into three main groups: words describing pain sensation in terms of temporal, spatial, thermal, pressure and other variables, words referring to affective qualities of pain such as tension and fear, and words reflecting the subjective overall intensity of pain. The offshoot of all this was the McGill Pain Questionnaire (MPQ) of Melzack (1975) which incorporated the pain descriptors so that a person's sensory versus affective levels of pain could be assessed according to the particular words he/she endorsed. Of considerable interest to the present discussion is the trail of studies that followed in pursuit of the factor structure of pain descriptors. These hold as much import for the sensory-affective distinction in pain, as they do for the construct validity of the MPQ.

Sensory factor. The first study in this vein was by Bailey and Davidson (1976). Factor analysis was employed to test the hypothesis that intensity is the major
dimension differentiating MPQ pain descriptors, and to uncover other possible representations of pain vocabulary. Likert-scale ratings for 39 descriptor items were obtained from two samples of nursing and medical students. The authors used a principal-axis routine on the data from the first sample extracting each factor with an eigenvalue greater than one and performing a varimax (orthogonal) rotation. Similar procedures were then applied to data from the more heterogeneous second sample, in order to test the stability of the factor structure. Of the six factors that emerged, only the first two were interpretable. The larger of these was pain intensity on which were loaded affective and evaluative terms; the second factor comprised items from the sensory domain. No evidence of a distinctively affective dimension was obtained. The reason may lie in the fact that only half of the pain descriptors listed by Melzack and Torgerson (1971) were used in this instance.

In contrast, Crockett, Prkachin and Craig (1977) administered the MPQ (with all its 78 descriptors) to a large and diverse sample actually experiencing pain. One group of subjects was required to rate increasingly intense electric shocks on a 10-point scale until pain threshold was reached; another group rated the series of shocks on a 100-point scale until tolerance was reached. Both groups were then given the MPQ with instructions to pick the word
that best described the most intense shock received. A third group of back pain patients chose words on the MPQ that most accurately described their average clinical pain. Principal components factor analysis was calculated on intensity values of words selected. Kaiser-Guttman rotation and scree tests suggested that five factors be retained: "immediate anxiety", "perception of harm", "somesthetic pressure", "cutaneous sensitivity" and "sensory information". Although the first two of these hint at the affective and the last three are apparently sensory, there is serious confounding of both sensory and affective items within the first factor and of the sensory and evaluative items within the third and fourth factors. The clarity of affective constituents of pain is hence diminished, although sensory factors are conspicuous.

Instead of the MPQ, Jensen, Karoly, Riordan, Bland and Burns (1989) compiled a set of intensity and affect descriptors from Tursky (1976) and Gracely, McGrath and Dubner (1978a). They instructed 69 postoperative patients to use these verbal descriptors in rating their pain. To the astonishment of the authors, factor analysis indicated the presence of only a single (sensory) factor, based on the very large first and very small second eigen value. It is possible however that this result may have been influenced by the six measures of intensity which were used in combination with the intensity descriptors; this may
have created a response set among subjects that was weighted more on the intensity variable than the affective variable.

Affective and sensory factors. Marginally greater support for the affective construct comes from a study by Leavitt, Garron, Whisler and Sheinkop (1978). They used a sample of 131 back pain patients, the reasoning being that sufficient variety exists in back pain as to allow the reflection of multidimensional features of pain. Patients were given a random list of 87 pain descriptors from Melzack and Torgerson's (1971) list and asked to check those typifying their pain. The 74 words that were endorsed by more than five percent of the sample were intercorrelated and a principal components analysis with varimax rotation applied. Seven factors with eigenvalues greater than one were extracted: the first, "severe emotional discomfort", the second, a mixed factor comprising emotional and sensory items, and the remaining five factors pertaining to the sensory. The first factor is clearly an affective one accounting for 38 percent of the variance. Notwithstanding some confounding within the other factors each of which explains no more than 10 percent of the variance, this study provides critical evidence of a salient affective category across the pain descriptors used by people in pain.
A series of studies was conducted by Reading and colleagues to re-examine the total factor composition of pain descriptors. In the first of these (Reading, 1979), MPQ data from 166 dysmenorrhoea patients was factor analyzed. A varimax rotation was applied and all factors with eigenvalues greater than unity were isolated. Altogether accounting for 80 percent of the variance, the four factors reflected reactive aspects of pain, traction pressure, sensory properties and emotional distress, respectively. Of these, the second and third were sensory, the last was affective, while the first encompassed both affective and evaluative responses. For the first time, clear separation obtained between sensory and affective items.

In a later study, Reading (1982) administered the MPQ to 95 women of comparable demographics from the same hospital as the preceding study, with one difference -- the present patients were in acute (postepisiotomy) pain. As before, principal components analysis of the data was employed, with extraction of all factors having eigenvalues greater than one. Six factors emerged accounting for 65 percent of the variance. Three of these were purely sensory factors, but two other factors exhibited a mixture of the sensory and affective, while one showed a mixture of the evaluative and sensory. These findings were explained in terms of the difference between acute and chronic pain,
acute pain possibly permitting less time for cognitive
differentiation between sensory and affective input.

In a more elaborate study (Reading, Everitt & Sledmore,
1982), maximally dissimilar statistical methodology was
introduced to address the construct validity of the MPQ.
This entailed the use of multidimensional scaling (MDS) and
cluster analysis in an attempt to replicate the main
classes and subclasses of pain descriptors described by
Melzack (1975). Because of high "F-stress" values
pertaining to the MDS, results were reported for the
cluster analysis alone. They revealed considerable
similarity of composition between subclasses of the MPQ and
those of the Reading team. However, a plot of dendogram
linkage level against number of groups sugested that a
16-group solution was parsimonious. These 16 groups were
classifiable as either sensory (groups one to 13) or
reactive (groups 14 to 16). Unlike previous work, separate
sensory and affective categories were now discerned in one
and the same study.

Prieto et al. (1980) however suggested the need for
methodological refinements in investigating the factor
structure of pain vocabulary. They obtained a
heterogeneous sample of 198 back pain patients. Rather
than using individual pain descriptors as units of analysis
forming the correlation matrix, 20 descriptor subclasses
were used. Communality estimates were derived using 14
computer iterations in order to achieve factor stability. Further, the principal factor method was used to extract all factors with eigenvalues exceeding zero, so as to minimize distortion of the factor solutions. Scree tests pointed to a four-factor solution explaining 51 percent of the total variance, each factor carrying an eigenvalue greater than unity; the four factors were first rotated obliquely to derive higher-order factors and then rotated by varimax to determine which rotation provided the more appropriate solution. Items coalesced into groups labelled "sensory pressure", "evaluative", "affective-sensory" and "punishing affect". This represented the first report of a purely "evaluative" category, along with separate sensory and affective categories. However, it must be pointed out that labelling the second factor which accounts for 10 percent of the variance as "evaluative" is a subjective decision open to challenge, especially since the anchor words for the evaluative group are "annoying" and "unbearable" (allusions to affect). This labeling issue is a recurrent one in some of the accompanying studies reviewed.

To replicate the foregoing results, Byrne et al. (1982) repeated the above methods using as data the responses of 98 back patients to 20 subclasses of the MPQ. A comparable set of four factors arose. Coefficients of congruence applied to the factor pattern matrices of the two studies
generally confirmed the replication. However, this time, factor two was not exclusively "evaluative" but also included all six affective subclasses of the MPQ. This is consonant with the earlier conjecture that the evaluative dimension may be too vague or mixed to deserve a separate label; on the other hand, sensory and affective factors continue to be distinct.

Another study of back pain patients (McCreary, Turner & Dawson, 1981) used factor analysis to arrive at four dimensions underlying the language of pain: "fear of bodily harm", an "intermittent sensory dimension", "annoying-unbearable" and "nagging-torturing" dimensions and two "sensory" subgroups. Again, separate sensory and affective categories were evident, but the third factor labelled "evaluative" is questionable for reasons outlined above. It must also be noted that this study was deficient because of a neglect of oblique rotations and the omission of critical information regarding type of analyses and criteria for factor retention.

Burckhardt (1984) performed a principal components factor analysis with oblique rotation on combined MPQ data from inpatient and outpatient arthritics. Six factors with eigenvalues greater than one were extracted, the criteria being that item loadings of each subclass on each factor had to exceed 0.49. Four of the factors were purely "sensory", one (accounting for most of the variance) was
heavily weighted by affective subclasses, and another was a mixed affective-evaluative factor as in the Byrne et al. (1982) and Reading (1979) investigations. In all these studies, the affective-evaluative factor was actually most heavily loaded upon by MPQ subclasses 16 and 20 which refer to "annoying-unbearable" and "nagging-torturing" dimensions, respectively. Despite Melzack's labelling of these subclasses as "evaluative", a strong case can be made for treating them as part of one and the same affective factor.

Recent attempts at multidimensional scaling of pain descriptors are also suggestive of a sensory-affective distinction in pain. Morley (1989) drew 23 verbal descriptors from the intensity and affect scales of Tursky (1976). Eleven headache patients and 9 normals were then asked to sort these descriptors into categories and to make similarity ratings for all possible pairs of words. Subjects did not spontaneously group the words according to intensity and affect, but simply arranged them in order of increasing painfulness. MDS analysis of the similarity ratings however revealed six classes of descriptors that could be located in two-dimensional space described by intensity and affective distress. This is interesting since it suggests that multivariate statistics can uncover latent dimensions that are not part of the conscious self report of subjects.
Clark, Ferrer-Brechner, Janal, Carroll and Yang (1989) carried out an MDS of similarity judgements of all possible pairs of nine pain descriptors. This data obtained from 24 cancer patients and 24 pain-free volunteers yielded a three-dimensional solution with pain intensity accounting for 19% of the variance, emotional quality accounting for 17% of the variance and somatosensory quality explaining 9% of the variance. Mean subject weights for the first two dimensions were significantly greater than for the third dimension. The words, "burning", "shooting" and "cramping" placed on the somatosensory dimension are sensory descriptors (as in the MPQ) while the words "annoying", "intense" and "unbearable" on the intensity dimension are ordinarily regarded as affective descriptors. At best, the evidence supports a distinction between sensation and affect. The tripartite distinction of pain descriptors originally proposed by Melzack has not held up clearly in these multivariate studies.

**Correlation between sensory and affective factors.** No matter how many dimensions/factors are derived, there is the disquieting problem of multicollinearity as illustrated by Turk, Rudy and Salovey (1985). These authors chose confirmatory factor analysis (with the aid of LISREL V) to test the three constructs supposedly represented in the MPQ. Cross-validation of results across samples was
attempted; the samples were 70 patients at a pain management program and 98 orthopaedic hospital patients previously studied by Byrne et al. (1982), both groups differing in homogeneity of pain complaints. Results were in agreement with the three-way classification of pain descriptors, and were replicated in the second sample of different patients. However, it was discovered that the three factors were also highly intercorrelated and therefore not distinct. A minimum criterion for factorial distinctiveness is that the crossconstruct correlations must be less than the within-construct correlations (Campbell & Fiske, 1959). In both samples researched by the Turk team, average correlation between subclasses was 0.71 and exceeded that within subclasses (0.58). While these studies militate slightly against MPQ construct validity, they help explain the frequent confounding of certain components of the pain experience within one or more factors. The instability of the evaluative factor is particularly understandable.

A rebuttal has been issued by Melzack (1985) to the effect that the Turk et al. (1985) findings are not incompatible with a tripartite conceptualization of pain. High intercorrelations among factors, Melzack argues, need not imply lack of distinctiveness among them. He entrenches his position in examples from classical psychophysics: increasing light intensity enhances the
capacity to discriminate color, contours, texture and distance; similarly, in audition, increases of volume lead to enhanced discrimination of timbre, pitch and spatial location. In these cases, Melzack asserts, high intercorrelations exist among the variables of each modality but that does not mean we ignore differences between colour and texture or between timbre and pitch. Perhaps, the findings of Turk et al. (1985) do not detract from the evidence for sensory and affective contributions to pain as much as raise questions about the independence of these categories.

**Closing comments.** To sum up, the multivariate statistics have not permitted a tidy separation of sensory and affective components of pain, although they have clearly alluded to the existence of these components. The reasons are data-based (as in the study by Turk and colleagues), or else methodological. To expand on the latter, it must first be pointed out that many of the factor-analytic investigations reviewed failed to conform to the following basic standards stipulated by Comrey (1978): the need for large samples with no less than an N of 200, inclusion of at least five times as many variables as the number of expected factors, judicious labeling of factors, and the incorporation of a programmatic series of studies in which factor structure is progressively refined.
There are also inherent limitations in multivariate statistics that impose constraints on studying the problem at hand. As already explained, the particular stimuli selected and task assigned influences subjects' responses. Post hoc problems of factor analysis and MDS are cogently summarized by what Rudy (1988) calls the illusion of transparency, that is, the tendency to regard one's interpretive account of the results as an accurate reproduction of the data. Moreover, as illustrated earlier, the broad arrangement of (pain descriptor) items into categories based on similarity judgments by groups of subjects says little about the separability of sensation from affect in the conscious experience of pain — an issue perhaps better addressed by experimental designs.

**Signal Detection Theory Applications**

Signal detection theory was incorporated into pain research because of the considerable promise it offered for the separation of affective from sensory components of pain (Clark and Yang, 1983). With roots in electrical engineering, its relevance to psychology was first illustrated by Tanner and Swets (1954) and Green and Swets (1966). Soon afterwards, its first application to the study of pain was witnessed (Clark, 1969).

Sensory decision theory (SDT) as it has come to be known, recognizes that the report of a signal amidst a
background of noise is determined by neural sensitivity plus a host of psychological variables governing the willingness to report. In the case of pain, the emotional state of the organism is deemed as an especially important influence on the willingness to report. Random presentations of a noxious stimulus against a background of no stimulation will hence lead to two corresponding Gaussian distributions: the distance between the means of these distributions indexes sensitivity (d'); the ratio of the ordinate of the signal distribution to the ordinate of the noise distribution (at that point on the X axis which represents the criterion for a response) reflects response bias (β). These measures can also be derived from the relative frequencies of subjects' "hits", "false alarms", "correct rejections" and "misses" in detecting the stimulus. In any case, the advantage, as Clark explains, is that, "at a descriptive, or qualitative level, the sensory and emotional components of pain have long been recognized. SDT now permits the quantification of these two components into indices of discriminability and pain report criterion" (1983, p.23).

**Psychological intervention.** Pioneering this approach, Clark (1969) set out to determine if a medication placebo would produce the expected increases in β, relative to d' -- since a placebo is biochemically inert and presumably
reduces pain by only altering expectations. A sample of college students received different levels of radiant heat stimulation of the forearm under two conditions: a control condition in which they rated the stimuli using a category scale ranging from faint warmth to extreme pain, and a placebo condition in which they performed the same task after ingesting a bitter white (lactic) powder described as a narcotic. Each condition was run on a separate day with counterbalancing for order. The results showed that given a placebo, subjects soon reported numbness, confusion and even euphoria, in conjunction with a decrease in the proportion of pain responses as compared to control conditions. SDT analysis revealed this decrement as associated with an increase in $\beta$ from 1.04 to 1.76, although $d'$ remained virtually unchanged between conditions. This represented a significant advancement upon other psychophysical procedures (e.g., the method of constant stimuli) in which only sensory threshold measures were obtainable.

In an attempted replication of the above study, Feather, Chapman and Fisher (1972) randomly presented 50 trials for each of two levels of radiant heat to the forearm of student volunteers. The subjects were required to rate the pain under control conditions or medication placebo, as in the previous study. Again, no significant change in $d'$ was obtained across conditions but a
significant increase in $\beta$ from control to placebo conditions was observed, along with reports of reduced pain and perceived side effects of the supposed drug. This confirms that placebos exert a very selective influence on pain, by reducing the willingness to report pain as a result of expectancy effects. A corollary is that pain embodies both sensory and non-sensory (including affective) variables, each of which responds differentially to treatment intervention.

Mere instructions have been known to influence pain (e.g., Blitz & Dinnerstein, 1968, 1971; Wolff, Krasnegor & Farr, 1965). In view of that, Clark and Goodman (1974) employed SDT to examine the disparate effects of suggestion, on sensitivity versus response bias to pain. Four groups comprising equal numbers of males and females received radiant heat stimuli first without any suggestion and then under one of four suggestions (to raise or lower pain detection criterion or raise or lower pain tolerance criterion). Subjects rated their pain with reference to the same categorical scale as that used by Clark (1969). In all cases, the suggestion of pain alteration with reference to threshold or tolerance, led to changes in the suggested direction. Computations of $d'$ and $\beta$ revealed that these changes were hardly associated with any variation in discriminability of stimuli but instead were attributable to changes in subjects' criteria for reporting
minimal or maximal pain. Like placebos, suggestion may produce certain expectations that comfort or warn the subject, thereby altering his/her emotional component of pain.

Another psychological approach to pain management that has been subjected to SDT analysis, is social modeling. Craig and Cohen (1975) randomly administered electric shocks of five different intensities to subjects who rated them on a 10-category scale. Subjects were also paired with a confederate who either modelled pain tolerance or pain intolerance. The outcome was a greater sensitivity to pain among those assigned an intolerant model than those viewing a tolerant one. Unfortunately, no inferences can be made regarding the relative contributions of sensitivity versus response bias, for $\beta$ was not computed. This was rectified in a later study by Craig and Prkachin (1978) in which female volunteers were either exposed to a tolerant model or an inactive observer, while receiving a random series of electric shocks. The subjects gave verbal ratings of shock intensity which were transformed into numeric values to allow SDT analysis. The analysis revealed significantly lower $d'$ for those exposed to a tolerant model and a nonsignificant difference in $\beta$ between both groups. Limited credence may be ascribed to this finding however, for two reasons: (a) the procedure adopted to transform verbal responses into numerical
equivalents, distorts the picture of individual differences, (b) electric shock is not appropriate for studying affective aspects of pain, because it produces brief and discrete sensations without the characteristic anxiety that attends clinical pain; this is more so in the present study where levels of shock delivered were extremely mild. In short, social modelling may impact on response bias to pain, but the studies by Craig and colleagues have failed to detect this because of methodological inadequacies.

Medical intervention. The possibility of selectively modifying sensory and affective components of pain has also been raised in relation to medical treatments for pain, particularly drugs and acupuncture. Drugs differ in their mechanism of action, some impacting on physiological systems while others modifying psychological functions. Diazepam for instance, has been long used for the management of anxiety inherent in pain (Lasagna, 1977). Chapman and Feather (1973) reasoned that this selective effect of the drug could be examined from an SDT perspective. Subjects received either a placebo or 10mg. of diazepam, before being administered a brief, random series of heat stimuli presumed to be non anxiety-arousing. Computations of $d'$ and $\beta$ showed no significant difference in either sensitivity or response bias between diazepam and
placebo conditions. This was deemed consistent with the hypothesis that diazepam would have an appreciable effect only in cases where the pain has a salient anxiety component. Two further experiments, employing the (anxiety-arousing) tourniquet pain technique of Smith, Egbert, Markowitz, Mosteller and Beecher (1966) vindicated this hypothesis: in one, diazepam extended pain tolerance when compared to a placebo, this extension correlating positively with reduced anxiety; in the other experiment, diazepam was more effective than aspirin in increasing pain tolerance and decreasing anxiety. In conjunction with the SDT results, this demonstrates that the variable effects of diazepam on pain are a function of the degree of affective contribution to that pain.

By contrast, nitrous oxide shows a broader range of effects. In a contemporaneous study (Chapman, Murphy and Butler, 1973), male subjects received 200 presentations of radiant heat stimuli while breathing either nitrous oxide or room air through an anaesthesia mask. A six-category scale was used by subjects to rate anything from thermal properties to strong pain. It was found that B was significantly greater during nitrous oxide than under control conditions, although the willingness to label a stimulus as hot did not change. Additionally, nitrous oxide decreased sensitivity to nonzero stimulation without altering discriminability among adjacent stimuli. Thus,
nitrous oxide reduced pain by affecting both sensory and non-sensory components of pain.

A subsequent study by Chapman, Gehrig and Wilson (1975a) replicated the above finding in the context of electrical stimulation of the tooth pulp. In a sample of 42 healthy men, the inhalation of nitrous oxide produced statistically significant reductions in both d' and β when compared to control conditions.

SDT applications to acupuncture have led to more equivocal results though they remain instructive for the present discussion. The first study in this area was conducted by Clark and Yang (1974) in which 12 subjects each received 24 presentations of radiant heat stimuli to one forearm, and were inserted with electrically-driven acupuncture needles in either the right or left arm. The participants rated the continuum between sensation and pain on a 12-category scale. The outcome was a reduction in pain responses for the acupunctured arm relative to the "control" arm. Further analysis however indicated that this was mainly due to a raised response criterion rather than an alteration in d'. It was concluded that acupuncture does not reduce pain through a sensory-physiological mechanism as previously supposed. However, this report has been widely challenged for a variety of reasons (Capman, Gehrig & Wilson, 1975b; Hayes, Bennett & Mayer, 1975; McBurney, 1975). Many of the
criticisms pertain to procedural details such as the contention that Clark and Yang had not allowed sufficient time for an analgesic effect to develop, that they had picked a difficult site to demonstrate acupuncture analgesia and had presented rather few trials of noxious stimuli.

Consequently, Chapman, Gehrig and Wilson (1975a) re-examined acupuncture analgesia by delivering high, medium, low and zero intensity electrical stimuli to the teeth, with 75 trials at each level. Electrical stimulation of the needles for acupuncture commenced 20 minutes prior to testing and continued for the rest of the session, thus allowing adequate time for the effects to stabilize. Subjects verbalized or signaled their responses with reference to a seven-category scale ranging from "nothing" to "strong pain". The authors found that acupuncture led to significant decreases in d' between baseline and test sessions, while B was significantly greater in the treatment group than in the control group. The effects of acupuncture are hence not restricted to the sensory component of pain.

A subsequent study by Lloyd and Wagner (1976) employed a binary procedure in which subjects decided which of two consecutive radiant heat stimuli was more intense. Each of three levels of the stimulus (plus a zero level) was presented 50 times. It was found that d' decreased
significantly between the zero stimulus and the first-level stimulus, but changes in sensitivity between other pairs of stimuli were not significant. On the other hand, β declined between the zero stimulus and the first-level stimulus. Acupuncture seemed to make subjects less physiologically sensitive to low levels of stimulation for which subjects were also more inclined to report a difference in intensity. This is discordant with earlier findings but may be a unique outcome of a defensible methodology adopted. Until a standard experimental paradigm is developed to study acupuncture, discrepant results may continue. Meanwhile however, it stands that the sensory-affective division can be carved in various ways, depending on the particular intervention used.

Organismic variables. Other studies have been carried out to show that the boundary between sensory and affective pain also differs according to certain organismic variables. Clark and Mehl (1971) found that at almost all intensities of radiant heat pain, older people set a very high pain criterion (β) although in the case of older women this is coupled with a decrease in sensitivity. This was supported by Harkins and Chapman (1977) who found that older females exhibit a higher pain report criterion and a reduced ability to discriminate among noxious intensities when compared to younger members of the same sex.
The Clark and Mehl (1971) study also addressed sex differences and found that averaged over all stimulus intensities women set a higher response criterion than did men, although at noxious intensities younger females increased in propensity to report pain. This finding must be qualified by the fact that experimenters were of the opposite sex for females but not for males; an opposite sex experimenter for the latter might have facilitated greater increase in response criterion for males. The Clark and Goodman (1974) study in fact supported this argument, in finding that males faced with a female experimenter displayed greater response bias than females although the two groups did not differ in sensitivity to pain.

Cultural variables have also accounted for differential effects upon d' and B in pain studies. Clark and Clark (1980) found no differences in discriminability of electric stimuli between the Sherpas of Nepal and people of Western countries. However, the former had a much higher pain threshold owing to what the authors surmised as a culturally/environmentally dictated stoical pain report criterion.

Finally, personality variables may also illuminate the sensitivity and response bias properties of pain reports. Dougher (1979) found that students with trait anxiety revealed a much smaller latency to pressure pain in comparison to control subjects. This was shown to result
from a lower criterion for reporting pain among the anxious. State anxiety, on the other hand, increased sensitivity particularly to high intensities of noxious stimulation (Schumacher & Velden, 1984).

**Closing comments.** Having covered the gamut of SDT applications to pain, it is appropriate to make some general comments about this research body as a whole. In contrast to the multivariate statistical analysis of pain vocabulary, signal detection theory has frequently permitted an experimental demonstration of the components of pain. The identification of these components has not been unambiguous. Crawford Clark has repeatedly argued that when applied to pain, SDT measures a neurosensory parameter in \( d' \) and an emotional one in \( B \) (e.g., Clark & Yang, 1983). This is disputable. The response criterion, \( B \), represents the degree of willingness to report pain, and this likelihood ratio is a product of multifarious psychological variables (such as attitude, payoff, demand characteristics and personality) of which emotion is but one ingredient. This has been a major contention of Rollman (1977). Some interventions like tranquilizers and expectancy manipulations (through suggestion or placebo) clearly testify to an affective component distinct from sensory properties of pain. In the strict sense however, SDT makes very broad distinctions between sensory and
Another concern is the characterization of $d'$. Commonly, it is referred to as an index of sensitivity to the presence or absence of the stimulus i.e., signal detectability. In pain research however, it has come to represent discriminability between various proximate levels of a noxious stimulus, bearing in mind that it is virtually impossible to present a stimulus of zero intensity without evoking sensations of touch or warmth as the case may be. Consequently, McBurney (1975) may be right in pointing out that $d'$ measures differential sensitivity rather than absolute sensitivity to pain.

In this context then, $d'$ may best be characterized as pain discriminability and $\beta$ may be regarded as a measure of both cognitive and affective contributions to pain. It must also be underscored that these two indices need not be independent. Sensation may influence response criterion and vice versa, as noted by Chapman (1980). Despite statistical separation, the two may be functionally related.

Besides the conceptual difficulties inherent in SDT, there are also methodological limitations that preclude unqualified acceptance of the findings reviewed earlier. As Rollman (1977) has observed, these include the occasional paucity of signal and noise trials and the lack of use of carefully trained subjects. Furthermore, all the
SDT studies in the area have employed laboratory methods of pain induction; these are imperative for the experimental control that SDT requires, but they hinder the generalizability of results to clinical pain. A solution may be to incorporate more of the clinically valid experimental pain techniques (namely, the cold pressor and the tourniquet pain technique) which may facilitate greater separation of affective from sensory components of pain.

**Paired Scaling**

Perhaps the most common method of pain measurement is the rating scale. In order to measure sensory in relation to affective components of pain, the approach has been to incorporate a pair of scales, one for quantifying each component. For convenience, this is referred to here as paired scaling. To the extent that differential ratings are obtained on each scale, the sensory-affective distinction is supported. The scales generally conform to two principal types used in psychophysics: category scales and ratio scales (D'Amato, 1970). However, for the purposes of the present review, visual analogue scales (which are really a subset of ratio scales) will be distinguished as a third type, because of their peculiar improvisation to pain measurement that deviates from the essential tenets of ratio scaling. Each class of scales will be described along with a survey of their applications.
to the issue at hand.

**Category scales.** These consist of a fixed set of equally-spaced categories labeled by numbers or less often by adjectives, and they require subjects to assign each intensity of a stimulus singly into one of the available categories. The level of measurement achieved is hence interval or ordinal, depending on whether the categories are numeric or non-numeric, respectively. In pain research, category scales have commonly assumed the form of 10-point scales interspersed by certain adjectives at various points on the scale.

One of the first studies to use paired category scaling for sensory and affective components of pain, is that by Johnson and Rice (1974). The tourniquet pain technique was selected to induce ischemic pain, and subjects were given different degrees of information regarding sensations to be expected from the procedure: one group received no information, another received a description of unlikely sensations, a third group was told of two probable sensations arising from the procedure. Subjects rated sensory intensity on a zero to 100 scale while distress was rated with reference to equally-spaced intervals labelled "slightly distressing", "moderately distressing", "very distressing", and "just bearable", successively. The experimental manipulation was found to have little effect
on sensation but it did lead to a significant reduction in distress ratings among those given partial or complete information about sensations to expect. The authors viewed this as evidence for sensory and affective components of pain and their selective modifiability by informational variables.

This was taken up recently by Wallace (1985) in a study of females undergoing laparoscopic surgery. These patients rated "pain intensity" on a 10-point scale and "upset associated with pain" on another continuum ranging from "none" to "extreme upset". Correlation coefficients suggested a positive relationship between expected pain and post-surgical distress. Also, patients provided with accurate procedural information showed reduced pain and distress upon return from surgery, compared to control groups not given such information. The authors did not provide data on the precise relationship between pain and distress, but considered it meaningful to measure them separately.

Knox, Morgan and Hilgard (1974) used novel hypnotic procedures to derive category scales for sensory and affective pain. Noxious stimulation was generated by the tourniquet pain technique. At various intervals during the procedure, subjects were solicited for ratings of "pain intensity" and "suffering" each made in relation to a category scale ranging from 0 to 10 and beyond. An initial
baseline session yielded lower ratings for suffering than sensory pain — until tolerance was reported, whereupon the two approached equivalence. It was surmised that suffering would have overtaken sensory pain had the experiment continued further. The authors interpreted this as "important evidence on the possibility of making the distinction between sensory pain and suffering" (Knox et al., 1974, p. 843). The subsequent hypnotically-suggested anaesthesia led to a common 90 percent attenuation of both pain parameters. However, Hilgard's technique of communicating with the "hidden observer" by "automatic talking" suggested that at some remote level both sensory pain and suffering were being processed as they would be under non-anaesthetic conditions — separately and undiminished. The relevance of this study to the present treatise is its claim that sensory and affective properties of pain may be altered to a degree in hypnosis, but by and large they remain quite distinct. It must be pointed out however that inasmuch as the ratings for sensory pain and suffering (albeit discrepant) were positively correlated, they detract from the notion of separate components; in other words, if when one increases the other also increases (and vice versa), then it is not unlikely that they are measuring the same thing. [Turk et al., (1985) had expressed a similar kind of skepticism with respect to the factor distinctiveness of sensory and affective descriptors
of pain, because the two were highly correlated). By itself, the Knox et al. study is not convincing of the separability between sensation and affect in pain, although in conjunction with other studies (which demonstrate that one component of pain can be altered without affecting the other), it provides some clues about the variety of conditions in which sensory-affective separation can occur.

Paired category scaling of pain components has also been used to examine the effects of social modelling (Craig, Best & Best, 1978). These authors reported that both sensory and affective responses to nociception were reduced in the presence of a tolerant model, but the reduction of the affective component surpassed that of the sensory component. Hence, some divergence is seen between the two components.

There are problems however, with category scaling. Firstly, because of the fixed number of categories by which stimuli are judged, the method is prone to bias. Parducci and associates have progressively demonstrated that category scales are especially sensitive to context effects such as range of categories and frequency of stimuli (Parducci & Perrett, 1971; Parducci & Wedell, 1986). In pain measurement, a major source of error has been ceiling effects arising from the imposition of an arbitrary upper anchor in the scale (Fernandez, in press; Moore, Duncan, Scott, Gregg & Ghia, 1979). This necessitates caution in
interpreting paired category ratings at the upper end of the pain continuum. There is the additional limitation that category scales do not permit statements about the ratio of difference between obtained pain measures: it is meaningful to state which measure is greater than another or subtract one from the other, but it is not possible to deduce how many times one measure is greater or less than another.

**Visual analogue scales.** In an effort to overcome some of the limitations of category scales, visual analogue scales (VAS) have attracted considerable interest in pain measurement. Variations are possible (Sriwatanakul, Kelvie, Lasagna, Calimlim, Weiss & Mehta, 1983), but the commonly preferred version of the VAS is a line 10cm in length, the extremes of which designate bounds of the pain experience i.e., "no pain" and "unbearable pain". The subject's task is to mark the line at a point corresponding to the severity of his/her pain, the distance of the mark from the end of the scale being treated as a measure of pain. Such a method has been reported to be readily understood by most people (Huskisson, 1974; Scott, Ansell & Huskisson, 1977), reliable (Boeckstyns & Backer, 1989; Revill, Robinson, Rosen & Hogg, 1976), sensitive (Joyce, Zutshi, Hrubes & Mason, 1975) and concurrently valid with other methods (Downie, Leatham, Rhind, Wright, Branco &

The use of a pair of VAS to procure separate measures for sensory and affective pain, has been repeatedly explored by Price and his colleagues. In the first of their studies, Price, McGrath, Rafii and Buckingham (1983) used 150 mm. lines, one with endpoints labelled "no sensation" and "the most intense sensation imaginable" and the other with endpoints labeled "not bad at all" and "the most intense bad feeling for me" -- to quantify sensation and affect respectively. The participants were chronic pain patients and healthy volunteers each subjected to six noxious heat stimuli applied for a five-second duration to the forearm. Subjects' ratings of these stimuli revealed power functions, the exponent for the sensory function being 2.1 and that for the affective function being 3.8. Differentiation between the two components was further implied by the observation that sensation and affect both declined as a function of duration of treatment by Transcutaneous Electrical Nerve Stimulation and acupuncture, but the ratio of affective to sensory change varied markedly as a function of the progress of treatment. This contrasts with the "parallel covariation" of sensation and affect under hypnosis, (Knox et al., 1974). An ancillary finding was that the above VAS power functions were predictive of ratio judgements of sensation intensity and affective magnitude; this was deemed to be indicative
of the ratio properties of VAS data; however, this conclusion is somewhat tenuous because it is based on only two ratio judgements per dimension scaled.

It has already been shown in an earlier section that certain drugs influence the affective features of pain. In the case of morphine, this depends on the dosage of the drug, as witnessed in a study by Price, von der Gruen, Miller, Rafii and Price (1985). As in the previous study, thermal pain was induced and paired VAS measurements were procured. Subjects were intravenously given saline or a dose of morphine sulphate ranging from 0 mg/kg to 0.08 mg/kg. It was found that a dose between 0.04 and 0.06 mg/kg was accompanied by statistically reliable reductions in affective but not sensory VAS ratings, whereas 0.08 mg/kg of morphine reduced both sensory and affective responses to the noxious heat. Hence, at lower doses, morphine selectively influences affective pain, but at higher doses it does not distinguish between the sensory and the affective.

Relaxation has been shown to reduce both sensory and affective ratings of pain. Using a sample of oncology patients, Graffam and Johnson (1987) reported significant reductions in pain ratings from a mean of 4.06 before listening to a progressive muscle relaxation tape to 1.49 afterwards, and significant reductions in distress ratings from a mean of 4.31 to 1.56 before and after the same
relaxation intervention. Philips (1988) also found significant and sizeable reductions in these pain variables in an experimental group of chronic pain patients receiving relaxation training as compared to a no-treatment control group of patients in chronic pain. Houle, McGrath, Moran and Garrett (1988) also observed significant reductions in strength and unpleasantness of tooth pulp stimulation following relaxation or hypnosis; the same intervention for cold pressor pain however attenuated only unpleasantness. The separability of sensation and affect in pain hence varies with the type of pain.

Price, Harkins and Baker (1987) observed that among back pain, causalgia and cancer pain patients, the affective VAS ratings were significantly greater than their sensory counterparts, except when overall pain was minimal. On the other hand, myofascial pain dysfunction was characterized by equivalent levels of sensory and affective VAS ratings, and experimental (heat) pain led to lower ratings of affective distress than of sensory intensity. Interestingly, it was also observed that the successive stages of labour were associated with VAS ratings of sensation exceeding those for affect, until the final stage of "pushing" during which pain sensation stabilized at a high level while pain affect plummeted dramatically. Additionally, patients who focused attention on the pain reported affective pain levels twice those of patients who
actively anticipated the delivery of the baby. Once again, unlike the Knox et al. (1974) study, a clear divergence of the affective from the sensory is seen in certain types of pain and in particular cognitive states.

In another study (Price & Harkins, 1987), the power functions for sensory and affective pain as initially noted by Price et al. (1983) were confirmed. The VAS ratings of seven randomly presented heat stimuli yielded psychophysical functions with an exponent of 2.1 for pain sensation and 2.4 to 2.7 for pain affect. This, the authors explained, "provides further support that these pain measurement methods can separately measure two dimensions of pain experience" (p. 7). Consistent results have been reported in a recent study (Harkins, Price & Braith, 1989) in which power function exponents for VAS ratings of heat stimuli were between 2.0 and 2.2 for pain sensation and between 2.6 and 3.0 for pain affect.

However, a few caveats are in order. VAS measures are subject to variability depending on the length of the scale and the way the extremities of the scale are labeled (Seymour, Simpson, Charlton & Phillips, 1985). More research is hence called for, before the above findings (especially those pertaining to exponents) can be regarded as robust.

Secondly, even though the VAS is a method of cross-modality matching (to be discussed in the next
subsection), its particular adaptation for pain measurement denies it ratio properties. The pain VAS is anchored at the top unlike ratio scales. Furthermore, it carries an element of category scaling since descriptors are commonly used to demarcate various points on the scale and these have been found to distort the distribution of ratings (Scott & Huskisson, 1976). The distortion is probably less than that witnessed for category scales. Nevertheless, the combined effect of these improvisations is to cast doubt on the ratio properties of the VAS. This also means that in the strict sense, VAS data are not suited for the algebraic operations of multiplication and division which they have often been incorrectly subjected to.

**Ratio scales.** The ratio scale is the highest level of measurement (Stevens, 1951). It requires a non-arbitrary zero or origin and equal intervals between scales. Such a scale is commonly obtained by magnitude estimation in which a subject assigns an easily remembered number like 10 to a standard stimulus and then assigns numbers to subsequent stimuli so that the ratio between the assigned numbers and the number 10 reflect the ratios between sensation produced by the variable and sensation produced by the standard; alternatively, the subject may adjust the length of a line or squeeze a dynamometer to match proportionate changes in the magnitude of the stimulus -- a procedure called
cross-modality matching. This has been the predominant method of choice in the derivation of ratio scales for sensory and affective pain.

The typical items that have been cross-modally matched are verbal descriptors of pain. This commenced with Tursky (1976) who obtained cross modal matches for descriptors of pain intensity, unpleasantness and subjective quality (e.g., "piercing" or "shooting"). A sample of undergraduate students matched these descriptors to magnitude estimates of line length, adjustments of sound amplitude and squeezes of a handgrip dynamometer. The results which were corrected for regression bias, permitted the computation of a ratio scale value for each descriptor within the three classes e.g., "agonizing" received a value of 98. An examination of the range of values within each class showed that in the intensity scale the ratio of the highest to the lowest value was 30:1 while that for the reaction scale and the subjective quality scale was 7:1. As Tursky inferred, this alludes to the distinctiveness of the sensory dimension of pain.

The quest for scale values of sensory and affective pain descriptors has been actively taken up by Gracely and colleagues. Gracely et al. (1978a) instructed subjects to evaluate the lengths of lines and the magnitudes of sensory and affective descriptors by means of both numerical magnitude estimation and cross-modality matching to
handgrip force. The responses to the verbal stimuli were then converted into bias-free relative magnitudes by using line lengths as a reference continuum and by adjusting a common response measure to both line and verbal stimuli. The correlation between relative magnitudes derived by magnitude estimation and those derived by handgrip reached 0.97 for sensory descriptors and 0.98 for affective descriptors. Therefore, the two response measures were combined into a mean relative magnitude scale for each of the descriptor sets. A range difference between sensory and affective descriptor values was found, in the same direction as that reported by Tursky (1976). A second experiment in which line lengths and verbal descriptors were cross-modally matched to time duration and handgrip force, confirmed the range difference in ratio scale values for sensory and affective descriptors of pain.

In a separate publication, Gracely, McGrath and Dubner (1978b) reported on the sensitivity of the above descriptor scales to actual pain and a drug intervention. First, subjects who had served in the previous study were administered ten electrical stimuli. The sensory intensity and unpleasantness thus produced were proportionally matched to time duration and handgrip responses; in addition, subjects judged the two components of their pain experience by selecting items from randomized lists of sensory and affective pain descriptors derived earlier.
The descriptors chosen were then substituted by their scale values for the purpose of data reduction. Results showed that cross modality matching did not produce significantly different psychophysical functions for sensation and affect, whereas the descriptor selection method did. More convincing data on the applicability of paired verbal descriptor scales for differentiating sensory and affective pain, comes from the second experiment in which diazepam was intravenously administered to subjects. Pre-to post-drug comparisons of response to electrocutaneous stimuli indicated a significant reduction in the scaled values of affective descriptors but no such change in the scaled values of sensory descriptors -- a finding consistent with the conception of diazepam as an anxiety-reducing agent. This was interpreted by the authors as evidence for the sensitivity of ratio-based verbal descriptor scales in discriminating between pain sensation and affect.

Gracely and Dubner (1987) presented results aggregated with data from two other studies (Gracely, Dubner & McGrath, 1982; Gracely, McGrath & Dubner, 1979) on the selective effects of pharmacological manipulations of pain. Altogether, verbal descriptors of pain sensation, pain affect and "painfulness" had been chosen to characterize electrical tooth pulp stimulation under conditions of saline (placebo), fentanyl (a narcotic) or diazepam
(tranquilizer). (The "painfulness" dimension essentially conformed to an overall intensity scale). The emergent picture was that painfulness declined significantly in response to all pharmacological manipulations except the saline. Sensory intensity was reduced significantly by fentanyl and not by saline, the addition of diazepam not altering these effects. On the other hand, unpleasantness judgements were reduced significantly by saline, marginally by diazepam and negligibly by fentanyl. The authors discussed these findings in terms of the importance of measuring individual components of overall pain, and the possibility of doing so by using ratio-scaled verbal descriptors.

Although the Gracely team has employed elegant research designs to develop separate scales for pain sensation and affect, the reliability of ratings thus obtained has not been encouraging. Urban, Keefe and France (1984) reported that whereas between 73% and 85% of chronic pain patients could (ratio) scale intensity descriptors with an internal consistency of $\varphi > .90$, only 18% could achieve this criterion when scaling unpleasantness. Even with three repetitions, this figure did not exceed 35%, suggesting considerable intersubject variability in ratio scaling of affective descriptors. Morley (1988) found that 94% of a student sample could scale the intensity variable in an internally consistent manner but only 42% could do so for the
unpleasantness variable. The temporal stability of cross modally matched pain descriptors also depends on the variable being rated. According to Morley and Hassard (1989), 55% of chronic pain patients met the internal consistency criterion ($r>.90$) when scaling intensity of pain two to three weeks apart. On the other hand, for unpleasantness, this percentage was 40%.

There has also been vigorous debate over whether the ratings obtained in this set of studies conform to ratio scales. In a penetrating critique of Gracely's work, Hall (1981) rejected the assumption of ratio properties in these scales. He pointed out that the requirement of a rational, non-arbitrary origin for ratio-level measurement is rarely satisfied even in the natural sciences and probably unattainable in psychology. That subjects can make cross-modal adjustments proportionate to the ratio of differences between two stimuli, is also a source of doubt. Stevens (1975) had asserted that this is a reasonable assumption because in many cases, a ratio scale of judgements predicted a power function relating judgements to stimulus intensity. Others including Anderson (1974) however maintain that fitting a power function to a plot of judgements against stimulus intensity merely illustrates the flexibility of the power function to fit the kind of monotonically rising curves describing the relationship between numerical judgments and stimulus intensity. Other
assertions of Stevens (on which Gracely predicates his claims of ratio properties in verbal descriptor scales for pain) have also been challenged by Hall (1981). Gracely and Dubner (1981) replied to these criticisms by referring to a diverse body of research purportedly obtaining ratio scales through cross-modality matching. More recently, Gracely and Dubner (1987) have conceded that whether or not these methods produced ratio-level measures of sensory and affective pain, is a moot issue.

**Closing comments.** In concluding this section on paired scaling, some general comments may be made concerning the way in which the different scales have been used to separate sensory and affective aspects of pain. First of all, the instructions to subjects to rate sensation and affect often carry with them certain demand characteristics that may generate and accentuate a distinction between the two (Fernandez, 1988). This is especially directed to the category scaling studies that frequently overlooked necessary controls for this source of artifact, although studies using the VAS have also faltered in this regard (e.g., Harkins et al. 1989). Secondly, the observation of separate functions for sensory versus affective pain is inadmissible as evidence for the two components, as was pointed out in relation to the Knox et al. (1974) study. A divergence between the two functions or a difference in
their slope, intercept or range is more crucial for claims of differentiation to be upheld, and these admittedly have been found in several cross-modality matching studies. However, the data from cross modality matching (unlike VAS) has been lacking in intersubject and test-retest reliability. Contrary to early assumptions, they also do not yield ratio scales. Instead, they represent an interval level of measurement which is already obtainable through the VAS.

Within a proper research design, the VAS may be the most effective and convenient tool for separating sensory and affective features of pain. Such a design should use instructions that minimize demand characteristics, and it should employ an intervention that is known to modify selective features of pain. In addition, a third variable, overall pain should be included, as implicit in the work of Gracely and Dubner (1987) and Philips (1988). By comparing the variations in sensory and affective measures as a function of this overall index, a determination may be made as to whether there is a simple additive relationship or else some other mathematically describable relationship in the way sensation and emotion make up pain.

Finally, now that a distinction between sensory and affective pain components seems justified on the basis of the findings of individual studies, it is appropriate to
examine how precisely these two components of pain combine to constitute the experience of pain as a whole. Once a decomposition of the pain construct is thus achieved, the affective variable must in turn be related to the individual emotions that pain patients are likely to experience. To date, no study has attempted this although several have related pain to depression and a recent study (Harkins et al., 1989) has considered syndromes such as anxiety, depression and frustration in relation to pain.

To proceed from the sensory-affective separation of overall pain to an explication of the affective variable, one must next turn to the literature on emotions.

Emotional Aspects of Pain

By definition, pain is aversive, and empirical research supports the existence of an affective constituent in pain, labeled variously as unpleasantness or distress. Beyond that, little seems to have been done to delineate the variety of emotions involved in pain. In this second portion of the paper, effort will be directed to extracting some key ideas from the literature on "emotions", that may help illuminate the emotional complexity of pain.

The topic of emotions has only recently earned a place of importance in psychology. Prior to that, measurement difficulties and the demise of Wundtian introspectionism, led to a neglect of this field of study even culminating in
predictions that the term emotion would eventually disappear from psychology (Duffy, 1934; Meyer, 1933). Fortunately, as Campos and Barrett (1984) recount, there has been a change of Zeitgeist leading to a renaissance of interest in emotion, especially in the 1980's; this has been facilitated by the recognition among scholars of perception that emotional expressions produce resonant reactions in both the perceiver and the expressor (Gibson, 1979; Hoffman, 1978), an appreciation of the role of emotion in information-processing (Bower, 1981; Norman, 1980), the growing possibilities of tracing the roots of language to forms of nonverbal interchange (Bullowa, 1979, Ziajka, 1981), and the continued efforts to explain emotional deficits in the neurologically impaired (Kolb & Taylor, 1981).

**Definition of emotion.** The definition of emotion is a task that has occupied several scholars. It is generally agreed that emotions are phenomenologically discriminable from two other areas of psychological functioning, "cognition" and "behavior" (Izard, Kagan & Zajonc, 1984a). It is further held that emotions differ from moods in that the latter are tonic instead of phasic and often occur in a fleeting manner without necessarily centering around a specific object or event (Frijda, 1986). Emotions are also differentiated from traits which represent more enduring
patterns of response (personality) commonly derived from a combination of emotions, cognition and behavior (Plutchik, 1980). Conceptually, mood, temperament and emotional traits have been subsumed under the general class of affective phenomena within which emotion is one subset (Ekman, 1984).

Emotions denote a number of features variously emphasized by different scholars. Plutchik takes an evolutionary view, that "emotions are total body reactions to the various survival-related problems created by the environment" (1984, p. 214). Frijda defines emotion in terms of "action readiness", either in the form of tendencies to establish, maintain, or disrupt a relationship in the environment or in the form of mode of relational readiness as such" (1986, p. 71). Along similar lines, Tomkins emphasizes the motivational aspect of emotion; he views emotion as the "primary motivational system because without its amplification, nothing else matters, and with its amplification, anything else can matter. . . . It lends its power to memory, to perception, to thought, and to action no less than to the drives (1984, p. 164). Lazarus however assigns special recognition to the cognitive antecedents of emotion: "If, as I do, one regards emotion as a result of an anticipated, experienced, or imagined outcome of an adaptationally relevant transaction between organism and environment, cognitive
processes are always crucial in the elicitation of an emotion" (1984, p. 255). Scherer (1984) concurs with this but interjects elements of the motivational view too: "emotion consists of the constant evaluation of external and internal stimuli in terms of their relevance for the organism and the preparation of behavioral reactions which may be required as a response to those stimuli (1984, p. 296). Whether they highlight antecedents or consequences, all the above definitions are in agreement regarding the functional nature of emotions. Emotions have considerable information value about the ongoing state of the organism, and thus serve as a springboard for motivation towards specific behaviors that assist the organism in its adaptation to the environment e.g., fear implies danger and may thus propel the organism to flee in order to survive.

To this functional definition must be added certain properties that constitute the essence of emotion. As most scholars agree, emotion is a subjective state of mind, associated with patterns of central and peripheral physiological activity and typically expressed as facial, vocal and other motor behaviors (e.g., Izard, Kagan & Zajonc, 1984b; Lewis, Sullivan & Michalson, 1984).

**Typology of emotions.** The purpose of a typology of emotions in the present treatise is to extend the study of pain from a mere recognition of its general affective
component to an appreciation of the individual emotions associated with pain. Classifying emotions has been notoriously difficult however. In the English language alone, well over 500 words are used to label emotional states (Averill, 1975; Davitz, 1969; de Rivera, 1977), and the list varies across languages (Levy, 1984). Besides the cultural uniqueness of any vocabulary of emotions, there is the problem of imprecision due to overlap in meaning and variability in meanings across contexts. As Scherer laments, "one major hindrance to progress in theory and research on emotion has been the difficulty of getting beyond the semantics of emotion terms (1984, p. 304).

Several scholars have derived lists of emotion based on certain theoretical constructs. Arieti (1970) for instance, has distinguished among first, second and third order emotions. The first of these embodies experiences that involve the muscular and hormonal systems with an immediacy so that cognitive mediation is minimal e.g., tension, appetite, fear, rage and satisfaction. Second-order emotions are elicited by cognitive processes like imagery, and they include anxiety, anger, wishing and security. Third-order emotions are largely the product of language, and include depression, hate, love and joy. The suggestion of a cognitive continuum underlying emotions is acceptable although it is doubtful that a clear distinction can be made between second-order emotions like "anger" and
third-order emotions like "hate" purely on the basis of whether imagery or language is involved; it is not inconceivable that both these cognitive processes operate simultaneously in the production of certain emotions.

Izard (1977) arrived at ten fundamental emotions based on what he regards as distinct experiential, expressive and physiological characteristics. The emotions are also described in functional terms: "interest" provides the motivation for learning and creative endeavour; "joy" is associated with a sense of confidence and love; "surprise" clears the nervous system of ongoing emotion and cognition so that the individual can react to sudden change; "distress" makes one responsive to one's own problems and the problems of the wider milieu; "anger" may motivate destructive behavior but may also serve as a source of strength and courage; "disgust" may help in the maintenance of personal and ecological standards such as hygiene; "contempt" can lead to prejudice or be directed against an enemy; "fear" motivates the avoidance of danger; "shame" can produce feelings of ineptness and isolation but shame avoidance can promote self-improvement; "guilt" can torment the mind but the anticipation and avoidance of guilt can foster social responsibility. As mentioned, each of these emotions is distinguished by a unique set of neural, experiential and expressive responses which the author has expounded upon at length; furthermore, cognitive,
behavioral and affective antecedents and consequences are enumerated for each of these primary emotions. Izard (1977) also refers to the combination of the above emotions and their interactions with cognitions, in order to produce such complexes as anxiety, love and hostility. These may be termed "secondary emotions".

The distinction between primary and secondary emotions is also salient in the work of Plutchik (1980). He identifies eight basic emotions which are similar to those described by Izard (1977). "Shame", "guilt" and "contempt" are omitted, while "acceptance" is included. This is somewhat questionable especially since expressive, experiential or physiological correlates are not supplied for "acceptance". Plutchik must be credited however for highlighting an intensity dimension, so that stronger or milder levels of each emotion are possible, sometimes deserving different terms -- an example being anger, which is intermediate in intensity between annoyance and rage. Plutchik also elucidates the production of secondary emotions from a combination of primary emotions -- in much the same way as secondary colours are derived from primary colours, or mixtures and compounds obtained from chemical elements. He hastens to add that certain combinations of primary emotions at specific levels of intensity may be unlikely (just as certain chemical compounds are not found naturally) while others may occur but have not been
semantically labeled yet -- in which case new words are warranted.

Tomkins (1984) has arrived at a total of nine innate emotions. Using neural firing gradients as the criterion, all but one of Izard's listed emotions (guilt) are identified. Three classes of emotions are distinguished: those associated with an increase in the density of neural firing, namely, startle, fear and interest correspond to sudden, moderate and gradual gradients of increase (respectively); those associated with constant neural firing e.g., anger and distress which correspond to high and moderately elevated but unchanging gradients (respectively); and those associated with a decrease in neural firing e.g., joy which corresponds to a gradual decrease in the gradient of neural firing. Unfortunately, the gradients are not clearly specified for some emotions like contempt, disgust and shame. It is likely that neural firing gradients are not defining features but one among many characteristics of neural function related to emotions.

Recently, Johnson-Laird and Oatley (1989) proposed that there are five basic emotions: happiness, sadness, anger, fear and disgust. They base this on a theory that emotions are signals with communicative function within the brain and within the social group. Words describing the five basic emotions have no syntactic structure but their
meanings are immediately recognized. The authors' semantic analysis of 590 emotion-related words in the English language in fact revealed a set of terms (corresponding to the above emotions) with no internal structure that could be analyzed semantically.

Whether arrived at by semantic analysis, behavioral observations or physiological measures, it appears that most of the basic emotions identified are included under one label or another within Izard's (1977) taxonomy. There is a need to incorporate an intensity dimension as proposed by Plutchik (1980) but otherwise the taxonomy seems quite exhaustive. Particularly valuable for present purposes, is the detailed information it provides on the expressive and experiential antecedents and consequences of each emotion. This will hopefully simplify the task undertaken here of relating emotions to pain.

Pain-related emotions: physiological correlates. Of the ten basic emotions characterized by Izard, seven possess a negative valence i.e., they are intrinsically aversive. As pain is by definition aversive, it is associated with the negative emotions, typically, distress, fear and anger. Shame and guilt may coexist with pain, and under certain circumstances disgust and contempt may also be evoked, but these often involve other intervening variables such as personal responsibility and social norms.
Therefore, the present discussion will be mainly confined to three basic, negative emotions (distress, fear and anger) as they relate to pain; moreover, these are the emotions most reliably substantiated by physiological measures.

The search for physiological substrates of emotions has been vigorous. The idea of a brain site for emotions was seriously entertained by Papez (1937) and popularized by MacLean (1949) both of whom converged on the limbic system as the site involved. Sandwiched between the phylogenetically new and older parts of the brain, this system comprises the amygdala, hippocampus and septal area, and according to some (e.g., Isaacson, 1974) the cingulate cortex and anterior thalamus too.

Consistent with the notion of positive and negative emotions, the septal region of the limbic system has been regarded as a pleasure centre while the hippocampal formation has been linked to aversion (Heath, 1986). The aversion system has in turn been related to specific emotions. For example, in a survey of clinical reports of psychomotor epilepsy in which there is scarring of the limbic cortex, MacLean (1986) found that fear, anger and distress were common observations; these emotions which are the hallmarks of pain, seem to involve the amygdalar basal nucleus, the amygdalar central nucleus and the hippocampus, respectively (Henry, 1986). It must be added however, that
the amygdala and the limbic system are really part of a larger rostral-caudal network involved in emotions (Heath, 1986). As Berntson and Micco (1976) have explained, primary emotions and their corresponding adaptive behaviors are patterned at the level of the brainstem which is in a phylogenetically older part of the brain. It is interesting to note that the brainstem is also involved in the perception of aversive stimuli, thus suggesting that certain emotions like fear, anger and distress are pre-wired to occur during pain. These emotions however, undergo refinement at higher levels such as the limbic system where perhaps their intensity is modulated and their combination into secondary emotions takes place. Finally, at the level of the neocortex, emotions may interact with cognitions to produce attitudes, traits etc. In short, the limbic system is part of a hierarchical network involved in the elaboration of emotions.

The physiological picture is incomplete without a consideration of biochemical responses too. The relevant chemicals are directly or indirectly related to activity in the brain sites already reviewed above. In the case of anger (Henry, 1986), ongoing activation of the right and/or left amygdalar central nuclear systems and the associated right and/or left hypothalamic controls of the autonomic system, result in release of norepinephrine, as witnessed in urine analysis. There is an increase in beta
sympathetic outflow to the renin-producing cells of the kidney and to the adrenal medulla where the amount of tyrosine hydroxilase (the norepinephrine-synthesizing enzyme) increases. There is a small increase in epinephrine too, but as Ax (1953) and Funkenstein (1956) had proposed, anger is primarily related to norepinephrine release.

In contrast, fear, which is concurrent with basal amygdalar activation, is related to a predominance of epinephrine and only a moderate increase in norepinephrine -- another tenet of the Ax-Funkenstein hypothesis. If the fear is accompanied by flight, glucose levels also mount and other metabolic changes follow so as to prepare the animal for escape and prolonged avoidance. Should the fear lead to helplessness, then the distress hormone, adrenocorticotropin is secreted in moderate amounts -- the effect being to make the organism learn new patterns of behavior since the existing behavioral repertoire is rejected (DeWeid, Van Delft, Gispen, Weijnen & Van Wimersma, 1972).

Distress can have different patterns of endocrine activity depending on its intensity. Of particular interest has been that level of distress that becomes dejection; the word "dejection" is preferred to "depression" since the latter, in psychological parlance, has come to refer to a syndrome of various behaviors,
emotions and neurochemical responses. When dejection sets in, there is an increase in adrenocorticotropicin as a result of hippocampal activation of the pituitary (Wilson, 1985). An increased tolerance for pain may also occur due to the combined action of corticosterone and endorphins (Miczek, Thompson & Shuster, 1982). There is also a significant role played by monoamines in depression. It will be recalled from the earlier discussion on psychotropic drugs, that tricyclic antidepressants and monoamine oxidase inhibitors alleviate the affective component in pain; this is what pointed to the monoaminergic basis of distress. In a recent review (Van Praag, 1986), much research has been cited as demonstrating that brain levels of serotonin, noradrenaline and dopamine are low during dejection. Tricyclic antidepressants therefore restore emotional equilibrium by inhibiting the reuptake of these monoamines, while monoamine oxidase inhibitors do so by inhibiting their degradation.

Another angle of research deserving mention at this juncture, is that pertaining to autonomic correlates of emotion. This seems to have been founded upon the James-Lange theory which states that perceived bodily changes are what give rise to emotions e.g., we feel sorry because we cry, angry because we strike, afraid because we tremble (James, 1890). This position was soon discredited by Cannon (1929) who among other arguments, pointed out
that removal of the entire sympathetic nervous system (to preclude visceral feedback) failed to abolish emotions like anger and fear.

Nevertheless, the search for autonomic indices of emotion has continued. As recently as the 1950's, Wenger, Jones and Jones (1956) claimed that fear could be defined as sympathetic activation only, anger as both sympathetic and parasympathetic activation, and distress as reduced activity in both sympathetic and parasympathetic systems. This was based on a modicum of empirical evidence.

Subsequent research explored the possibility of relationships between specific autonomic variables and particular emotions. For example, anger was observed to correlate with increases in diastolic blood pressure (Ax, 1953), although Lacey and Lacey (1958) could not replicate this. Schwartz, Weinberger and Singer (1981) found that blood pressure increases while recollecting angering events, were evident only when subjects were sitting still as opposed to moving about. Elevated skin temperature has also been reported in association with anger (Ekman, Levenson & Friesen, 1983). Other measures like heart rate and gastrointestinal responses have produced conflicting results.

Autonomic correlates of fear have included higher muscle tension peaks, increased heart rate and more rapid respiration (Ax, 1953) -- findings replicated by Schacter
(1957) and Lewinsohn (1956). However, the expected increase in heart rate during fear, failed to appear in a study by Obrist (1981).

Distress has been associated with increased diastolic blood pressure, as has anger (Schacter, 1957) but no elevation in heart rate (Lewinsohn, 1950). Tremors and salivation may also be present (Sternbach, 1968).

It appears that, autonomic variables have so far turned out to be a somewhat unreliable as predictors of specific emotions. The inconsistent results reported may be largely due to differences in methodology involving the emotion-inducing stimuli and the dependent measures used. As Frijda (1986) has pointed out, there have also been confounding variables such as degree of attention, anticipation and stimulus unfamiliarity that may have varied systematically with the different emotions studied. Also, the control condition was usually an affectively neutral condition, which means that to distinguish among the different emotions would remain difficult. Serious conceptual issues also persist in this arena of investigation. Present technology permits the measurement of physiological processes that are below the threshold of awareness, in which case it is difficult to argue that the physiological correlate defines the emotional state. Finally, many of the physiological measures correlate with more than one emotion as well as with other psychological
states such as fatigue and hunger. The notion of autonomic specificity for individual emotions hence warrants more careful scrutiny.

Nevertheless, the different types of physiological measures can in totality contribute to an understanding of emotions. For anger, fear and distress, neural and biochemical substrates have already been reliably identified, although for other emotions like shame, guilt, disgust and contempt, the picture is far more nebulous. This makes sense, because like all aversive stimuli, nociception would lead to one or more of three basic responses; fight, flight or despair. Recalling the definition of emotions as a "springboard" for motivation towards specific behaviors designed to adapt an organism to its environment, anger, fear and distress/despair then represent the emotions that dictate whether a person will fight, avoid or give up (respectively) in the face of pain. These appear to be the fundamental emotions experienced in pain, and comprehending their biological substrate permits a more holistic way of treating pain.

Pain-related emotions: cognitive antecedents. Cognitions have long been recognized as intimately linked to emotions (e.g., Dember, 1974; Neisser, 1963). One of the seminal investigations of this idea was carried out by Schacter and Singer (1962). They demonstrated that
injections of epinephrine (which generated diffuse autonomic arousal) led to reports of happiness if subjects observed a happy experimental confederate, and to anger if subjects were exposed to an impudent experimental confederate. Other cognitive manipulations have been shown to influence dejection/depression (e.g., Beck, 1971) and the response to stress (Bennett & Holmes, 1975). Based on such findings, it has been argued that emotion is determined by cognition (Oatley & Johnson-Laird, 1987; Ortony, Clore & Collins, 1988). Lazarus who has become the main champion of this position states: "cognitive appraisal is a necessary as well as sufficient condition of emotion" (Lazarus & Folkman, 1984, p. 275). This would mean that each of the pain-related emotions (or any emotion for that matter) can be defined exclusively in terms of its cognitive antecedents.

An opposing stance has been taken by Zajonc. He has articulated the almost Jamesian view that emotions are represented by kinaesthetic feedback from motor responses, without any cognitive mediation (Zajonc & Markus, 1984). The shortcomings of such a position have already been covered in the subsection on autonomic correlates of emotion, the basic problem being one of lack of specificity. Furthermore, it has been observed that patients with spinal cord lesions that render them without sensation, still experience emotion (Buck, 1980). Zajonc
(1984) goes on to dismiss the necessity of cognition for emotion by citing experimental findings of an increase in the liking for a stimulus as a function of repeated exposure to it (e.g., Kunst-Wilson & Zajonc, 1980); this is presumptuous of the absence of cognitive activity, which in fact may be present but imperceptible. Besides, Leventhal (1984) describes a much more elaborate experiment which failed to obtain an increase in preference due to exposure alone.

Zajonc's arguments are somewhat tenuous. Those of Lazarus however do require modification. That emotion is always preceded by cognition is doubtful. First of all, at phylogenetically and ontogenetically lower levels, emotion may be inferred even though the likelihood of cognition is remote (Field, Woodson, Greenberg & Cohen, 1982). In adult humans too, emotion can occur without any evidence for cognitive mediation e.g., in instances where psychoactive drugs are used or facial expression is non-spontaneously altered (Laird, 1974).

In the final analysis, it appears that emotion can occur without cognition. However, cognitive events play a pivotal role in the genesis of emotions (at higher levels of the central nervous system). Structurally, all stages in the acquisition, transformation and storage of information (i.e., attention, perception and memory) are relevant. In the case of pain, the concomitant emotion can
conceivably be influenced in quality or intensity by the amount of attention capacity directed to the noxious stimulus, the schema of past painful experiences, and the perception of physical (temporal and spatial) characteristics of the noxious stimulus. In addition, the cognitive appraisal of the situation is of paramount importance. Lazarus and Folkman have defined this process as "categorizing an encounter, and its various facets, with respect to its significance for well-being" (1984, p. 31); it takes three forms: primary appraisal (what is the trouble?), secondary appraisal (what can be done about it?) and reappraisal (modification of one's perspective based on new information). The authors cite considerable research on the influence of these processes on reactions to stress. Other scholars have emphasized related features of cognitive appraisal. For instance, Roseman (1979) includes probability (likelihood of favourable outcome) and legitimacy (perception of fairness of the situation) as instrumental in emotional outcome. Weiner, Graham and Chandler (1982) draw attention to the salience of causal attributions underlying pity, anger and guilt. Scherer (1984) has organized most of these evaluative processes along the following lines: occurrence of event (time, expectation, probability and predictability), evaluation of outcome (intrinsic pleasantness, goal relevance, conduciveness and equity), attribution of causation (agent,
motive and legitimacy), evaluation of coping potential (ability to control event/consequences), power to change event/outcome, external or internal standard (conformity to cultural norms and consistency with real/ideal self-image). Smith and Ellsworth (1985) have identified six orthogonal dimensions of cognitive appraisal that differentiate emotional experience: pleasantness, anticipated effort, certainty, attentional activity, self/other responsibility and situational control. Ortony, Clore and Collins (1988) have in a landmark book asserted that cognition whether conscious or not governs the production of emotion at an early stage itself. They propose a computationally tractable model which allows the prediction of specific emotions from specific kinds of cognition. At this time however, the model has not been empirically tested.

Specific cognitive antecedents for each of the ten basic emotions have been empirically identified by Izard (1977) on the basis of responses from 130 subjects. For purposes of the present paper, particular attention is directed to those thoughts characterizing the pain-related emotions of anger, fear and distress: anger is associated with thoughts of hatred, dislike, hurt, failure and revenge; fear is linked to thoughts of danger, threat, death, the unknown and inadequacy; distress is associated with thoughts of the specific problem at hand, loss of control and rejection. Images and self-statements that
center around the above, are deemed to produce unique emotional outcomes.

In short, cognition whether at a structural or symbolic level, governs the production of emotions. Undoubtedly, the emotions in turn exert reciprocal effects on learning, memory and other cognitive processes (Campos & Barrett, 1984; Lewis et al., 1984). For the present, it suffices to say that the emotions concomitant upon pain are modulated by specific cognitive events. In the case of anger, fear and distress, the link between pain and emotion may be to some extent prewired at a physiological level; cognitive modulation may serve to amplify or attenuate these emotions or to generate other pain-related emotions such as shame, guilt, contempt and disgust. (Secondary) blends of these primary emotions are probably even more susceptible to cognitive influence. The precise mechanism by which these take place is still a matter of speculation. However, physiological findings suggest that the limbic system which elaborates emotion, indeed receives considerable input from the neocortex which is involved in information-processing (e.g., Turner, Mishkin & Knapp, 1980). This is consistent with the view that emotion (whether pain-related or not) is in a significant way, the product of cognitive antecedents.

Pain-related emotions: behavioral concomitants. The link between emotions and behaviors has been less well
studied than that between emotions and cognitions. The behaviors in question include somewhat reflexive facial responses and purposive goal-directed behaviors. Much information is available on facial behavior associated with emotions. As mentioned earlier, the work of Ekman is particularly well-known in this respect. He has devised a Facial Action Coding System (Ekman & Friesen, 1978) that characterizes individual emotions according to lip position, eyebrows and so on. Izard (1977) made it simpler to distinguish emotions by using a standard set of photographs that had been pan-culturally validated. Whatever system is used, the facial expressions most concurrent with pain will be those of negative emotions — of which the facial expressions of anger, fear and sadness will predominate. This is because anger, fear and sadness are presumed to be physiologically prewired responses to aversive situations. Also, the facial expressions for the other negative emotions are more ambiguous and therefore less likely to be noticed.

As for purposive behaviors accompanying pain, considerable work has been carried out within the context of behavioral treatments of pain, although this has not been linked to the subject of emotions. Again, anger, fear and sadness would be preeminent, and their associated goal-directed behaviors may involve aggression, help-selling behavior and withdrawal respectively. Izard (1977)
provides clues about behaviors peculiar to each basic emotion and these probably generalize to the context of pain too.

**Summary and Rationale for Present Study**

At the outset of this paper, different scholarly opinions were cited on the phenomenology of pain. It was asserted that by definition, pain is an aversive bodily sensation, and therefore it encompasses both sensory and affective phenomena. Clinical impressions gained through the use of psychosurgery and psychotropic medication are consistent with this view, inasmuch as they showed that certain drugs and surgical operations could dramatically alter the emotional concomitant of pain while leaving its sensory counterpart intact. Multivariate analyses typically revealed latent constructs of sensation and affect in pain descriptors but not pain itself; the constructs do not imply direct separation of the two variables. Signal detection theory applications demonstrated that the pain report could be accounted for by an index of physical sensitivity ($d'$) and by response criterion ($\beta$), the latter while related to affect is really a composite of psychological factors such as payoff and personality; experiments also showed that each index could be altered by certain psychological and medical procedures and organismic variables, while leaving the other index
unchanged. Paired scaling approaches for the most part corroborated the independent variation of pain sensation and pain affect under various experimental interventions; it is unclear whether ratio scales are obtainable for these two components, but their separation by visual analogue scales has been encouraging. Demand characteristics however were noted and a need to consider sensory and affective variables in relation to overall pain was emphasized.

In view of the spread of evidence for a sensory-affective distinction in pain, this review was extended to elaborate on specific emotional aspects of pain. Emotion was defined as a subjective experience with physiological and motor-expressive concomitants, and functioning as a motivational springboard for adaptive behaviors. Cognition was found to be integral to the production of emotions. A typology of emotions was selected, and for the ten primary emotions (seven of which are negative and thus associated with pain), cognitive antecedents and behavioral concomitants were identified. The construct of pain is therefore subject to decomposition into sensation and affect, and the affective variable is in turn elaborated by several emotions which are tied to specific cognitions and behaviors.

It is now appropriate to make explicit certain hypotheses that have been taking shape in the course of the
foregoing review. These are the subject of an empirical investigation to be described in accompanying chapters.

It is hypothesized that:
(a) Overall pain can be mathematically represented as a linear combination of sensory pain and affective pain.
(b) Affective pain can be mathematically represented as a linear function of particular emotions.
(c) The emotions accounting for the bulk of variance in affective pain will be anger, fear and sadness. Next in contribution will be the remaining negative emotions, guilt, shame, disgust and contempt. Emotions that are neutral or positive in valence, namely, surprise, interest and joy will account for a negligible part of the variance in affective pain.
(d) The intensity of each emotion measured will be correlated with the extent to which certain cognitions and behaviors apply. The higher a negative emotion is rated, the greater will be the endorsement of certain cognitions and behaviors peculiar to that emotion.
(e) Cognitive/behavioral therapy will attenuate the degree to which the (negative) cognitions and behaviors are endorsed.
(f) Reduction in negative cognitions and behaviors will be associated with reduction in corresponding emotions.
(g) Reduction in intensity of negative emotional responses to pain will be associated with reduction in affective
pain.

(h) Reduction in affective pain will be associated with reduction in overall pain.

A few points of clarification are in order. First of all, overall pain is included as a variable, so that sensation and affect may not only be considered in relation to each other (as in previous studies) but also assessed for their unique contributions to pain as a whole.

Visual analogue scales are used to measure all variables since as explained earlier, they are reliable, valid and simple to use. They have also been the most successful of all tools used to separate sensation from affect in pain.

Instructions to rate the variables will depart sharply from previous studies in order to minimize demand characteristics. Subjects will be presented items relating to overall pain, sensory pain and affective pain without any implicit or explicit distinction among the three.

The emotions to be rated will consist of the 10 emotions from Izard's (1977) taxonomy. They will be rated as responses to chronic pain.

The cognitions to be rated are primarily based on Izard's (1977) specification of cognitive antecedents for each emotion in his taxonomy. In addition, they incorporate information from other cognitive models of
emotion (e.g., Ortony et al., 1988; Smith & Ellsworth, 1988). In the present context, the variety of cognitions are organized into metaphoric attributions about pain (pain cognitions) and a set of propositions regarding perceptions of self (self cognitions). This is in keeping with the general consensus among cognitivist scholars of emotion that the emotional response to an event is influenced by perception of the event itself and perception of one's transaction with the event (Lazarus & Folkman, 1984; Ortony et al., 1988). Each of the cognitions assessed corresponds to one of the emotions.

The behaviors to be rated are selected according to their unique association with each emotion. These are based largely on Izard's (1977) specification of behavioral concomitants of emotion, while also drawing from the vast literature on pain behaviors (e.g., Philips & Jahanshahi, 1986; Vlaeyen, Van Eek, Groenman & Schuerman, 1987). The behaviors are organized into two categories: goal-directed behaviors (such as withdrawal and help-seeking) and reflexive-like facial activation patterns. Each item of behavior corresponds to one of the 10 emotions.

Treatment intervention will entail a four-week multidisciplinary program emphasizing cognitive-behavioral approaches to pain management. This is based largely on principles of operant conditioning of pain (Fordyce, 1976) and cognitive restructuring (Turk, Meichenbaum & Genest,
1983). An in-depth review of this vast background of treatment literature is beyond the scope of the present work. The important ideas are that chronic pain behavior (with no discernible organicity) is an operant maintained by certain rewards and extinguishable by removal of such contingent rewards. Therefore, helping gestures, attention and social responsiveness are withheld in the face of verbal, postural and other forms of pain behavior. Instead, attention and praise are shifted to "well behaviors" such as exercise, assumption of responsibility and abstention from complaints. Furthermore, the analgesic medication schedule is altered from a pain-contingent to a time-contingent schedule so that it no longer serves as a reinforcer of pain. A graduated weaning off medication and promotion of exercise is effected by a system of contracts and quotas. Monitoring of overt pain behaviors is carried out. These principles have been effective in restoring healthy, adaptive behavior to those in chronic pain and improving their daily functioning (Fordyce, Fowler & DeLateur, 1968; Keefe, Gil & Rose, 1986).

The cognitive ingredient in the program includes relaxation training and imagery to facilitate voluntary remediation of stress that exacerbates pain. Attention-diversion is used to train patients to distract themselves when in pain. Maladaptive self-statements are, through a dialectical process, replaced by adaptive cognitions. Such
approaches have been shown to enhance coping with pain (Fernandez & Turk, in press; McCaul & Malott, 1984).

Additionally, the program will offer occupational therapy, physical therapy and the services of a physician, pharmacist, nutritionist, nurses and social worker. This is in keeping with the multidisciplinary approach to pain management that has been instituted across pain clinics (Bonica, 1974). A discussion of the outcome of such multidisciplinary treatment is provided in the final chapter of this study.

The purpose of including treatment intervention in this study is twofold. The primary aim is to see if disparate functions can be obtained for sensory versus affective pain using treatment that targets non-somatic aspects of pain. Secondarily, the efficacy of cognitive behavioral treatment in modifying cognitive and behavioral variables and ultimately emotion and pain itself, is of interest.
CHAPTER II

METHOD

Subjects

The subject population consisted of chronic pain patients admitted into the Pain Management Program at the Department of Physical Medicine (Dodd Hall) of the Ohio State University. Admissions were made after careful multidisciplinary evaluation to exclude those with pronounced psychiatric problems, severe medical complications, or litigation pending as a result of pain.

Rules of informed consent were adopted. All admitted patients were given a brief description of the study (see Appendix A for solicitation script). They were told that participants in the study would complete questionnaires about their pain and related experiences. This posed no discernible risks, but would further knowledge about pain and its treatment. Results of participants would be kept confidential. Participation would be voluntary, without monetary inducements, and with freedom to withdraw from the study at any time. A standard consent form (Appendix B) issued by the Human Subjects Review Committee of this university was signed by subjects and investigators as part
of agreement regarding the conditions of participation.

Of the 46 patients recruited between March and August of 1988, four did not complete the program, one withdrew from the study, and one was excluded due to incomprehension of instructions. The final sample totalled 40 subjects, the demographic breakdown of which is shown in Table 1. As indicated, there were equal numbers of males and females, ranging in age from 20 to 69 years old. The majority of subjects had a high school education, were married and had been employed in skilled labor. They were heterogeneous in pain diagnoses but back pain was the modal category. In all cases, the pain had persisted beyond six months despite medical intervention, and was therefore deemed "chronic". Average chronicity of pain was estimated at 63 months.

Table 1

**Demographic Characteristics of Patient Sample**

<table>
<thead>
<tr>
<th>Age:</th>
<th>M = 44; S.D. = 11.93; range = 20 - 69</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender:</td>
<td>Males = 20; Females = 20</td>
</tr>
<tr>
<td>Education:</td>
<td>&lt;High School = 10; High School= 20; College = 10</td>
</tr>
<tr>
<td>Occupation:</td>
<td>unskilled labor: 11; skilled = 20; misc. = 9</td>
</tr>
<tr>
<td>Diagnoses:</td>
<td>Back Pain = 15; myofascial pain = 13; misc. = 12</td>
</tr>
<tr>
<td>Pain duration in months:</td>
<td>M = 63; S.D. = 62; range = 9 - 240</td>
</tr>
</tbody>
</table>

*Note.* Except for pain duration, all data are frequencies.


**Apparatus**

The research materials consisted solely of paper-and-pencil questionnaires. The assessment questionnaire comprised six pages of 52 items designed to assess aspects of patients' pain, emotional reactions to pain, cognitions about pain, cognitions about self, purposive behaviors and facial behaviors in response to pain (see Appendix C). The last of these sets comprised nine photographs of facial expressions, while the rest of the items were made up of brief statements or words. (The photos of facial expressions were reproductions of those in Izard (1977), and permission to use them for this study was secured from the publisher and author, as documented in Appendix D). Concise written instructions were supplied on how to complete the questionnaire. As mentioned before, each of the randomly sequenced cognitive and behavioral items on the questionnaire corresponded to a particular emotion, and the investigators' key to this correspondence is shown in Appendix E. Also used was a five-page, 25-item proficiency test questionnaire to determine subjects' mastery of the information taught during the program (see Appendix F).

**Design**

The design of the study is depicted in Table 2. As shown, this is a repeated measures design with a single
independent variable, the phase of treatment: pretreatment at the beginning of the first week or posttreatment at the end of the fourth week. At each of these points, multiple dependent measures were collected in the form of questionnaire ratings for pain, emotions, pain cognitions, pain behaviors and facial behaviors. At pretreatment, a validity check was also carried out to determine if subjects saw test items on cognition and behavior as related to items on emotion, along the lines of the a priori assumptions of the investigators. At posttreatment, a reliability check was included to ascertain the degree of consistency in subjects' responses matching cognitions and behaviors with their respective emotions. Finally, upon posttreatment, subjects took a written quiz to determine their level of mastery of material conveyed during the program.

Table 2
Summary of Research Design

<table>
<thead>
<tr>
<th>Week</th>
<th>Treatment</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pre</td>
<td>ratings, validity check</td>
</tr>
<tr>
<td>2</td>
<td>Mid</td>
<td>Nil</td>
</tr>
<tr>
<td>3</td>
<td>Mid</td>
<td>Nil</td>
</tr>
<tr>
<td>4</td>
<td>Post</td>
<td>ratings, reliability check</td>
</tr>
<tr>
<td></td>
<td></td>
<td>proficiency test</td>
</tr>
</tbody>
</table>
**Procedures**

**Treatment.** Subjects were treated by a multidisciplinary team during their four weeks of hospital stay. The team was staffed by a physician, medical resident, psychologist, predoctoral trainee in psychology (also known as the intern), physical therapist, occupational therapist, pharmacist, dietician and two nurses, with social workers available for consultation as needed.

The treatment program was basically cognitive-behavioral in nature. Subjects were informed of this upon acceptance into the program and later given an orientation session on what to expect.

The flow of patients was staggered so that approximately two new admissions and two discharges were made every week, and there was an average of five patients in the program at any point in time. Upon admission, patients began a process of being gradually weaned off analgesic medication. This was achieved by an alteration of medication schedule from a pain-contingent to a time-contingent basis, and a slight reduction in the time interval between medications as the dosage was decreased. All this was facilitated by a system of contracts drawn up collaboratively by patient and pharmacist. Unless patients
showed extreme withdrawal symptoms, these contracts were strictly adhered to in order to effect medication discontinuation by the end of the program.

One feature throughout the program was the monitoring of patient behaviors by staff members. However, these were done sporadically depending on staff availability. Gross estimates were obtained for the frequency and magnitude of pain behaviors, in particular, verbal complaints, subvocalizations (moaning and groaning), grimacing and abnormalities of posture and gait. The change in these over time was noted, along with the frequency and magnitude of well behaviors like physical activity ("up time") and positive interpersonal communications.

In addition to monitoring overt behavior, staff members used behavioral reinforcement principles. Pain behaviors were tendentiously ignored and well behaviors were praised. Social modeling was utilized to introduce patients to a new repertoire of adaptive behaviors and to reinforce the vicarious learning of these behaviors. Both commercially produced videotapes and videotapes from ex-patients of the program were used to this end.

A key component of the program was relaxation training. Four times a week, patients spent half-an-hour practising progressive muscle relaxation under the supervision of the psychologist or psychology intern. This involved alternate tensing and relaxing of major muscle groups of the body.
while maintaining slow and deep breathing. It was followed by 10 minutes of guided imagery to enhance the relaxation response. A separate hourly session was held each week to extend guided imagery to self-directed imagery. Different visual and auditory stimuli (especially music) were also introduced as a means of attention-diversion during pain.

Patients also participated in a problem-solving group every week. This entailed following a prescribed sequence of problem-definition, generating options, choosing the optimal solution (from a cost-benefit perspective), and reviewing the outcome of the selected option. The approach was applied to a set of assigned hypothetical problems and then to problems peculiar to the individual.

An hourly session was held each week to address patients' emotional status, particularly problems of anger, fear (anxiety) and sadness (depression) ensuing from chronic pain. The maladaptive cognitions subserving such emotions were identified and replaced with more adaptive cognitions. The links between cognition, emotion and behavior were emphasized. Role playing and role reversal (Beck, 1985) were used to facilitate behavior change along the lines of the changing cognitive schema. For instance, a patient who reported feelings of hopelessness about his employability because of chronic pain, might be asked to consider vocational rehabilitation which in turn might raise his/her level of hope, and then role play going for a
suitable job interview. Role playing/role reversal were also used to train patients in being assertive rather than passive or aggressive.

In addition to the skills-training component, the program also provided several sessions with an educational component. These encompassed a physician's lecture on medical aspects of pain, a dietician's lecture on proper nutrition and weight control and psychology classes on stress management.

A two-hour session was also conducted one evening every week, that was open to patients and their families. Topics covered in separate sessions were operant conditioning of pain behavior, sexuality for chronic pain patients, physical exercises for rehabilitation, and recreational options for the chronically ill.

All patients received individual therapy at least once a week. This was provided by the licensed psychologist and the pre-doctoral trainee in psychology. The objective here was to address ongoing concerns peculiar to each patient, to consolidate what was taught in groups and to monitor overall progress. Where appropriate, the patient's family members or significant others were also included in these therapy sessions.

Every day, patients were also involved in actual physical therapy. A baseline level of performance was noted for various exercises like "stretching" and
"climbing". Quotas were then laid down for systematic increase in the number of repetitions of each of these exercises by the patient. Swimming in a heated pool was another regular feature of physical therapy.

There was a daily commitment to occupational therapy too. A suitable project (in carpentry or crocheting as the case may be) was developed for each patient. Additionally, the patient's strengths were assessed with a view to vocational rehabilitation if necessary. Matters of time-management and recreational activity were covered in a more didactic fashion.

Finally, a "Family Day" was organized once a month, in which patients and their family/significant others attended a series of lectures on pain management given by members of the treatment team. Those who had completed the program about a month or two before were invited back on this day to speak to participating patients and staff members regarding their experiences in the program and thereafter.

Assessment. Each subject was required to complete the 52-item questionnaire (Appendix C) once upon admission and once upon discharge. The questionnaire was administered by the research investigator who read the instructions aloud and remained available for any questions subjects might have. Subjects were assessed as a group within a 45-minute session set aside every week.
First, the visual analogue scale was presented as the frame of reference for all ratings. The scale ranged from 0 to 10 where 0 represented the total absence of the quantity being rated, 10 represented a maximum level of the quantity being rated, while the numbers between one and 10 were used to denote intermediate levels of the phenomenon in question. Five groups of items were rated, each listed on a separate page of the questionnaire. Subjects were informed that there were no right or wrong answers and that the ratings were supposed to reflect their own individual experiences typical of that week.

Subjects began by rating aspects of their pain. The first of these was a rating of "overall pain" which was described as the magnitude/severity of pain as a whole. Subjects then identified the sensory quality of their pain (be it "throbbing", burning", "piercing" etc.) and rated the degree to which that particular physical sensation was present. This was followed by a rating of the degree to which subjects were distressed by the level of physical sensation described.

Next, subjects rated the degree to which they experienced each of 10 emotions when they were in pain. Borrowed from Izard's (1977) taxonomy of human emotions, these were anger, fear, sadness, guilt, shame, disgust, contempt, surprise, interest and joy. The first seven of these are of negative valence while the others are
positive/neutral in valence.

Subjects then proceeded to rate the extent to which they upheld 10 separate cognitions about pain, each of which corresponds to a particular emotion listed above. For instance, pain may be likened to "an enemy to be fought against" (item 9) which relates most to the emotion of anger (item 4). Similarly, subjects rated the extent to which they professed 10 different perceptions of themselves, each of which also corresponds to a specific emotion listed earlier; for instance, item 30 which reads "I see myself as an outcast because of my pain" relates most to the emotion of shame (item 8). The fourth category consisted of 10 purposive behaviors accompanying pain, each corresponding to a specific emotion e.g., item 35, "when in pain, I do penance/sacrifice to make up for wrongs" relates to the emotion of "guilt". The fifth and final category was made up of photographs of human facial expressions (from Izard, 1977) representing each of the 10 emotions with the exception of guilt (for which no definitive photograph has been available). An example is item 49 which is a photograph of the facial expression for disgust. Subjects rated the extent to which they projected each of the facial expressions when in pain.

Having rated each of the 52 items with reference to a common visual analogue scale anchored at 0 and 10 respectively, subjects returned to items 14 to 52 to match
each of the pain cognitions, self cognitions, pain behaviors and facial expressions with their corresponding emotion (items 4 to 13). This was intended as a validity check of whether or not the cognitions and behaviors were perceived to reflect the emotions they putatively related to. The presumed relationships are summarized in Appendix E where an alphabetic symbol denotes the specific emotion corresponding to each item.

Upon completion of the rating and matching tasks, the questionnaire was checked for completeness. The subjects were thanked and debriefed. Specifically, they were reminded of the purpose of the study, the confidentiality of its results and the opportunity to discuss their own results at the time of discharge.

At discharge four weeks later, each subject underwent the exact assessment procedures outlined above. That is, they provided VAS ratings of overall, sensory and affective pain and each of 10 emotions, 10 pain cognitions, 10 self cognitions, 10 pain behaviors and 10 facial expressions occurring in relation to pain. This was followed by the same task of matching the cognitions and behaviors with their corresponding emotions. This time however, the matching task was designed to yield reliability data rather than validity data.

Furthermore, upon discharge, subjects took a quiz (Appendix F) to broadly ascertain their level of mastery of
educational material imparted during the program. This entailed answering 25 multiple-choice questions spanning psychology, physical therapy, occupational therapy and other medical topics of relevance. Only those passing 75 percent or more of these questions were deemed suitable for the study. However, if a subject scored less, the relevant test material was reviewed with them and they were given a second opportunity to reach criterion.

Data Analyses

Questionnaire ratings from individual subjects were collated into a common data matrix. This was subjected to descriptive and inferential statistics.

The descriptive analysis included calculation of means and standard deviations for all dependent measures at pre-treatment and post-treatment. These were graphed as well as tabulated separately.

A multivariate analysis of variance (MANOVA) was carried out with "treatment phase" as the sole independent variable, to determine changes between admission and discharge. Separate MANOVAS were carried out for each of the five categories of dependent measures: pain, emotions, pain cognitions, self cognitions, pain behaviors and facial behaviors. Within each category, the MANOVA would answer the question of what treatment effects emerged for any of the outcome variables taken singly or collectively. If the
test was significant, then univariate Analyses of Variance (ANOVA) were performed to determine for which specific outcome variable/s the significant effect/s obtained.

Correlational techniques were used to answer questions about the interrelations among the various dependent variables themselves. Firstly, multiple regression was employed to determine whether or not ratings of overall pain could be expressed as a linear function of sensory pain ratings and affective pain ratings. If a linear equation failed to satisfactorily describe the data, non-linear models were to be tried. In the same way, multiple regression was performed with affective pain as the predicted variable and the three sets of emotions as the predictor variables -- negative emotions felt to be physiologically prewired (anger, fear, sadness), the negative emotions felt to be cognitively modulated (guilt, shame, disgust, contempt) and the positive/neutral emotions (surprise, interest, joy).

Furthermore, stepwise regression was used to determine the pain component accounting for significant portions of the variance in overall pain. It was similarly used to identify the sets of emotion variables accounting for significant portions of the variance in affective pain.

To answer questions about the correspondence between emotions and their cognitive and behavioral counterparts, a correlation matrix was derived for each emotion. The
matrix summarized pairwise correlation coefficients (among emotion, pain cognition, self cognition, pain behavior and facial behavior) along with their significance levels. To examine the correspondence between change in one variable and change in another, the same intercorrelations were derived using pre-post treatment difference scores.

Finally, subjects' data from the matching task were also analyzed. Since this data consisted of nominal measures, it was best reduced to frequencies and percentages. The frequency/percentage of subjects giving a particular nominal judgement of the emotion corresponding to each test item, was calculated. The degree of agreement between subjects' modal responses and the investigators' presumed correspondence between items and emotions, served as a validity check for the questionnaire. The degree of consistency in subjects' nominal judgements for the same test between pre and post treatment phases four weeks apart, was an index of reliability of subjects' perceptions of the test.

As alluded to earlier, the proficiency test results were analyzed subject by subject with the aid of percentages. A criterion was set so that only those passing at least 19 out of the 25 test questions were retained in the study.
CHAPTER III
RESULTS

All 40 patients constituting the final sample passed the proficiency test. It is the results of these subjects that are collectively presented hereafter. The order of presentation will begin with regression analysis, followed by correlation coefficients, analysis of variance and frequency data. This roughly parallels the sequence of hypotheses outlined in Chapter I.

Regression

Multiple linear regression was conducted to see if pain is an additive combination of sensation and affect and if affective pain in turn is an additive composite of various emotions. If the linear model failed, non-linear models would be used to fit the data.

A multiple regression analysis was conducted on pretreatment data, with overall pain (OP) as the predicted variable and sensory pain (SP) and affective pain (AP) as predictor variables. It was found that overall pain could in fact be expressed as an additive combination of sensory pain and affective pain as in the linear regression.
equation, \( OP = 3.64 + .18AP + .58SP \). The multiple correlation was .63 and \( R^2 = .40, F(2, 37) = 12.08, p<.0005 \). The correlation between sensory pain and affective pain was significant (\( r = .80 \)), meaning that there was significant association (multicollinearity) between the predictor variables. Correlations between overall pain versus sensory pain and affective pain were .62 and .52, respectively. Stepwise regression revealed that sensory pain by itself accounted for a sizeable portion of the variance in overall pain, \( R^2 = .38, F(1, 38) = 23.71, p<.00005 \), and hence did not proceed to examine the unique contribution of affective pain to variance in overall pain.

Identical (simultaneous and stepwise) multiple regression procedures performed on posttreatment data produced different results. Although overall pain could again be expressed as a linearly additive combination of sensory pain and affective pain, the \( B \) weight was far greater for affective pain than sensory pain: \( OP = 3.07 + .63AP + .09SP \). The multiple correlation was .76 and \( R^2 = .57, F(2, 37) = 24.63, p<.00005 \). Multicollinearity of predictor variables dropped to \( r = .67 \), and the highest pairwise correlation among the three variables was between overall pain and affective pain (\( r = .75 \)) while the correlation between overall pain and sensory pain was .55. Stepwise regression halted after one step when affective
pain alone accounted for most of the variance in overall pain, $R^2 = .57$, $F(1, 38) = 49.84$, $p < .00005$.

Regression techniques were extended to express affective pain as a function of different emotions. Since the ratio of sample size to number of predictor variables is generally supposed to be no less than 10:1, not all the 10 emotions could be used as predictor variables given a sample size of 40. Therefore, the emotions were grouped into three meaningful classes: negative emotions deemed to be largely physiologically-prewired (Anger, Fear, Sadness), negative emotions deemed to be more cognitively modulated (Guilt, Shame, Disgust, Contempt) and emotions of positive or neutral valence (Surprise, Interest, Joy). These were labeled as Set 1, Set 2 and Set 3 respectively.

A linear regression equation fitted the pretreatment data very well: $AP = 3.47 - .72Set3 + .50Set1 + .17Set2$. The multiple correlation was .61 and $R^2 = .37$, $F(3, 36) = 7.18$, $p < .0008$. Table 3 indicates significant correlations between affective pain and Sets 1 and 2 respectively and a marginally significant negative correlation between affective pain and Set 3 — meaning that there was a strong association between affective pain and the experience of negative emotions but an inverse relationship between affective pain and the report of positive/neutral emotions. The positive/neutral versus negative emotion sets were quite uncorrelated while the negative emotion sets were
highly correlated.

Table 3

Intercorrelations Among AP, Set 1, Set 2 and Set 3 at Pretreatment

<table>
<thead>
<tr>
<th>Variable</th>
<th>Set 1</th>
<th>Set 2</th>
<th>Set 3</th>
<th>AP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Set 1</td>
<td>___</td>
<td>.52**</td>
<td>.11</td>
<td>.51**</td>
</tr>
<tr>
<td>Set 2</td>
<td></td>
<td>___</td>
<td>.16</td>
<td>.34*</td>
</tr>
<tr>
<td>Set 3</td>
<td></td>
<td></td>
<td>___</td>
<td>-.25</td>
</tr>
<tr>
<td>AP</td>
<td></td>
<td></td>
<td></td>
<td>___</td>
</tr>
</tbody>
</table>

Note. N = 40. AP= Affective Pain; Set 1 = Anger, Fear, Sadness; Set 2 = Guilt, Shame, Disgust, Contempt; Set 3 = Surprise, Interest, Joy.

* p<.025. ** p<.001.

Table 4 summarizes results of the stepwise regression to ascertain the unique contributions of each of the sets of emotions to variance in affective pain. As shown, Set 1 produced an $R^2 = .26$, $F(1, 38) = 13.53$, $p<.001$, and the addition of Set 3 increased $R^2$ by 38% to a level even more highly significant statistically. However, the procedure stopped without including Set 2. In other words, the variance in affective pain was best explained by anger, fear and sadness. Guilt, Shame, Disgust and Contempt were
extremely correlated with Anger, Fear and Sadness and therefore did not add significantly to the variance explained; however, the positive/neutral emotions of Surprise, Interest and Joy contributed uniquely to the variance explained.

Table 4

**Stepwise Regression for Predicting AP at Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Multiple</th>
<th>R^2</th>
<th>R^2 change</th>
<th>F</th>
<th>df</th>
<th>change</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Set 1</td>
<td>.51</td>
<td>.26</td>
<td>.26</td>
<td>13.53</td>
<td>13.53</td>
<td>.001</td>
<td></td>
</tr>
<tr>
<td>Set 3</td>
<td>.60</td>
<td>.36</td>
<td>.10</td>
<td>10.41</td>
<td>5.63</td>
<td>.0001</td>
<td></td>
</tr>
</tbody>
</table>

**Note.** N = 40. AP = Affective Pain; Set 1 = Anger, Fear, Sadness; Set 3 = Surprise, Interest, Joy.

The same regression procedures performed on posttreatment data yielded slightly different results. A linear regression equation still fitted the data: AP = .45 + .25Set3 + .86Set1 - .17Set2, but Set 1 was weighted much higher than Set 3 this time. Table 5 indicates that affective pain was most highly correlated with Set 1 but not with Set 2. There was only a modest (albeit significant) correlation between Set 1 and Set 2, and practically no correlation between Set 1 and Set 3.
Table 5
Intercorrelations Among AP, Set 1, Set 2 and Set 3 at Posttreatment

<table>
<thead>
<tr>
<th>Variable</th>
<th>Set 1</th>
<th>Set 2</th>
<th>Set 3</th>
<th>AP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Set 1</td>
<td>_</td>
<td>.29*</td>
<td>-.10</td>
<td>.52**</td>
</tr>
<tr>
<td>Set 2</td>
<td>_</td>
<td>_</td>
<td>-.32*</td>
<td>.08</td>
</tr>
<tr>
<td>Set 3</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>.01</td>
</tr>
<tr>
<td>AP</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
</tbody>
</table>

Note. N = 40. AP = Affective Pain; Set 1 = Anger, Fear, Sadness; Set 2 = Guilt, Shame, Disgust, Contempt; Set 3 = Surprise, Interest, Joy.
* p<.05. ** p<.001.

Stepwise regression showed that Set 1 contributed uniquely to \( R^2 = .27 \), \( F(1, 38) = 14.16, p<.001 \). However, no other sets of emotions provided significant increases in variance accounted for.

There was no need to apply non-linear models to the data. Multiple linear regression fitted the data very satisfactorily in all cases.

Correlation

To elucidate the link between emotions versus cognitions and behaviors reported by the chronic pain
patients, Pearson Product Moment Correlations were computed between each emotion and its associated pain cognition, self cognition, pain behavior and facial behavior. All these five variables were also correlated among one another and the results summarized in Tables 6 to 15 for pretreatment data. (The explanatory note accompanying Table 6 applies to all of Tables 6 to 15).

It was found that emotional responses to pain correlated positively with underlying cognitions and concomitant behaviors. Some emotions correlated more with their cognitive and behavioral counterparts than did other emotions. Table 6 for instance shows that ratings of Anger in response to pain were positively associated (at a highly significant level) with the perception of pain as an enemy to be fought against (pain cognition), the perception of self as a fighter against pain (self cognition), self-reported physical aggressiveness when in pain (pain behavior) and identification with the facial expression of anger when in pain (facial behavior). On the other hand, the emotion of surprise in response to pain was not significantly related to any of its cognitive or behavioral counterparts with the exception of the self cognition variable (Table 13). All other emotions rated were significantly associated with one or more cognitive/behavioral counterparts. A point of note is that the matrix of intercorrelations for the emotion of Joy is
practically empty due to the preponderance of ratings of 0 which made coefficients incomputable.

Table 6

**Intercorrelations of Measures Related to Anger During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
<td></td>
<td>.85***</td>
<td>.88***</td>
<td>.70***</td>
<td>.73***</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td>.81***</td>
<td>.66***</td>
<td>.68***</td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td>.67***</td>
<td>.63***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td>.78***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. N = 40. * p<.01. ** p<.001. *** p<.0005*

Table 7

**Intercorrelations of Measures Related to Fear During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
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<tbody>
<tr>
<td>1. Emotion</td>
<td></td>
<td>.48**</td>
<td>.23</td>
<td>.55**</td>
<td>.01</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td>.68***</td>
<td>.38**</td>
<td>.41*</td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td>.43**</td>
<td>.69***</td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td>.48**</td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 8

**Intercorrelations of Measures Related to Sadness During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
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<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
<td>_</td>
<td>.62***</td>
<td>.72***</td>
<td>.36*</td>
<td>.28</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td>_</td>
<td>_</td>
<td>.55***</td>
<td>.37*</td>
<td>.59***</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>.59***</td>
<td>.47**</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>.50***</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
</tbody>
</table>

Note. There was no variable 5 due to the absence of a facial expression of guilt.

Table 9

**Intercorrelations of Measures Related to Guilt During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>4</th>
</tr>
</thead>
<tbody>
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<td>_</td>
<td>.87***</td>
<td>.82***</td>
<td>.47**</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td>_</td>
<td>_</td>
<td>.86***</td>
<td>.51***</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>.63***</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
</tr>
</tbody>
</table>

Note. There was no variable 5 due to the absence of a facial expression of guilt.
Table 10

*Intercorrelations of Measures Related to Shame During Pretreatment*

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
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<tbody>
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<td>1. Emotion</td>
<td></td>
<td>.64***</td>
<td>.55***</td>
<td>.37*</td>
<td>.10</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td>.74**</td>
<td>.57***</td>
<td>.12</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td></td>
<td>.61***</td>
<td>.31</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.32</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Table 11

*Intercorrelations of Measures Related to Disgust During Pretreatment*

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
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</tr>
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<tbody>
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<td>.40*</td>
<td>.14</td>
<td>.53***</td>
<td>.53***</td>
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<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td>.74***</td>
<td>.34</td>
<td>.52***</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td></td>
<td>.38**</td>
<td>.36*</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.31</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 12

**Intercorrelations of Measures Related to Contempt During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
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</tr>
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<td>.45*</td>
<td>.45*</td>
<td>.44*</td>
<td>.58***</td>
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<td></td>
<td>.74***</td>
<td>-.01</td>
<td>.33</td>
</tr>
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<td>3. Self Cognition</td>
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<td></td>
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<td>.03</td>
<td>.20</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
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<td></td>
<td></td>
<td></td>
<td>.45*</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

Table 13

**Intercorrelations of Measures Related to Surprise During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
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<td>.42*</td>
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<td>.12</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td>.08</td>
<td>.82***</td>
<td>.33</td>
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<td>3. Self Cognition</td>
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<td>.17</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.39*</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 14  
**Intercorrelations of Measures Related to Interest During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
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<td>1. Emotion</td>
<td></td>
<td>.62***</td>
<td>.47**</td>
<td>.39*</td>
<td>.13</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td>.52***</td>
<td>.31</td>
<td>.27</td>
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<tr>
<td>3. Self Cognition</td>
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<td>.04</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.53**</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 15  
**Intercorrelations of Measures Related to Joy During Pretreatment**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.56***</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pain Cognition</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* . = coefficient not computable
In general, ratings of emotional responses to pain were most positively correlated with cognitions about pain. The average correlation between emotion and pain cognition was .57. Self cognitions and pain behaviors were comparable in their correlations with emotions (mean $r = .52$ and .50, respectively). Facial behaviors (though related) were on the whole least correlated with emotion, (mean $r = .34$).

The intercorrelations among cognitions and behaviors showed less of a pattern. A few findings however may be highlighted. For instance, Table 7 shows that the pain cognition (pain as an overpowering danger) was highly associated with the self cognition (about oneself as a weak defendant against pain). Cognitions about pain and self were highly correlated with all emotions except for Surprise (Table 12). Purposive behaviors and facial expressions concomitant upon pain were also positively correlated as in the case of Anger (Table 6), Fear (Table 7), Sadness (Table 8), Contempt (Table 12), Surprise (Table 13) and Interest (Table 14). There were also significant positive associations across cognitions and behaviors as in Sadness (Table 8) where the perception of oneself as a helpless victim of pain (pain cognition) is significantly correlated with crying or withdrawal in response to pain (pain behavior). Similarly, for Anger (Table 6), Fear (Table 7), Guilt (Table 9), Shame (Table 10), Disgust (Table 11) and Surprise (Table 13), significant positive
correlations emerged between cognition and behavior.

For the posttreatment data, the intercorrelations among emotion, pain cognition, self cognition, pain behavior and facial behavior reveal much the same picture. Ratings of emotional responses to pain were most positively related to cognitions about pain (mean $r = .64$) and least to facial behaviors (mean $r = .31$), while correlations of emotion to self cognitions and pain behaviors were intermediate in magnitude (mean $r = .52$ and .48 respectively).

A question of further interest is how the changes in ratings of variables (between pretreatment and posttreatment) were related to one another. Correlation matrices for these difference scores are presented in Tables 16 to 25. As shown, changes in the degree of endorsement of particular cognitions were often associated with changes in corresponding emotions reported, as in Sadness (Table 18), Guilt (Table 19) and Interest (Table 24). Reduced emotional response to pain was also related to reduction in corresponding behavior, as in the case of Fear (Table 17), Disgust (Table 21) and Contempt (Table 22). For Surprise (Table 23), there were significant inverse relationships between emotion and self cognition or facial behavior. The greatest number of significant intercorrelations was found for Anger (Table 16) and Shame (Table 20). Intercorrelations were generally incomputable for Joy (Table 25) due to preponderance of ratings of zero.
### Table 16
**Intercorrelations of Pre-Post Difference Scores Related to Anger**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
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<th>4</th>
<th>5</th>
</tr>
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<tbody>
<tr>
<td>1. Emotion</td>
<td></td>
<td></td>
<td>.57***</td>
<td>.60***</td>
<td>.29</td>
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<td>.59***</td>
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<td>4. Pain Behavior</td>
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<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. N=40. * p<.01. ** p<.001. *** p<.0005*

### Table 17
**Intercorrelations of Pre-Post Difference Scores Related to Fear**

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
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<td>1. Emotion</td>
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<td></td>
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<td>.24</td>
<td>.73***</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>.30</td>
<td>-.45*</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
Table 18

**Intercorrelations of Pre-Post Difference Scores Related to Sadness**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>3</th>
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<tbody>
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<td></td>
<td>.30</td>
<td>.49**</td>
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<td>.23</td>
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<tr>
<td>2. Pain Cognition</td>
<td></td>
<td>.09</td>
<td>.11</td>
<td>.60***</td>
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<tr>
<td>3. Self Cognition</td>
<td></td>
<td>.29</td>
<td>.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
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<tr>
<td>5. Facial Behavior</td>
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<td></td>
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</tr>
</tbody>
</table>

Table 19

**Intercorrelations of Pre-Post Difference Scores Related to Guilt**

<table>
<thead>
<tr>
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</thead>
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</tr>
<tr>
<td>2. Pain Cognition</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td>-.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* There was no variable 5 due to the absence of a facial expression for guilt.
Table 20

**Intercorrelations of Pre-Post Difference Scores Related to Shame**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
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<tr>
<td>1. Emotion</td>
<td>—</td>
<td>.83***</td>
<td>.76***</td>
<td>.66***</td>
<td>-.23</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td>—</td>
<td>.81***</td>
<td>.67***</td>
<td>-.18</td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td>—</td>
<td>—</td>
<td>.61***</td>
<td>-.07</td>
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</tr>
<tr>
<td>4. Pain Behavior</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>-.06</td>
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<tr>
<td>5. Facial Behavior</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<td></td>
</tr>
</tbody>
</table>

Table 21

**Intercorrelations of Pre-Post Difference Scores Related to Disgust**

<table>
<thead>
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</tr>
</thead>
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<td>.30</td>
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<td>.21</td>
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<td>.28</td>
<td>.02</td>
<td>.69***</td>
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</tr>
<tr>
<td>3. Self Cognition</td>
<td>—</td>
<td>—</td>
<td>-.36*</td>
<td>.34</td>
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</tr>
<tr>
<td>4. Pain Behavior</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>-.10</td>
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<td>5. Facial Behavior</td>
<td>—</td>
<td>—</td>
<td>—</td>
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</tr>
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</table>
Table 22

Intercorrelations of Pre-Post Difference Scores Related to Contempt

<table>
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<tr>
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<td>.22</td>
<td>-.02</td>
<td>.81*</td>
<td>-.35</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
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<td>.17</td>
<td>-.02</td>
<td>.08</td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td>-.08</td>
<td>-.23</td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
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<td></td>
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<td>-.38*</td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 23

Intercorrelations of Pre-Post Difference Scores Related to Surprise

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
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<td>.15</td>
<td>.18</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td></td>
<td>-.83***</td>
<td>.62***</td>
<td>-.53***</td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td></td>
<td></td>
<td>.57***</td>
<td>.48**</td>
</tr>
<tr>
<td>4. Pain Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.19</td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>
Table 24

Intercorrelations of Pre-Post Difference Scores Related to Interest

<table>
<thead>
<tr>
<th>Variable</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
<td></td>
<td>.83***</td>
<td>.07</td>
<td>.59***</td>
<td>-.19</td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td>-.11</td>
<td>.46**</td>
<td>-.23</td>
<td></td>
</tr>
<tr>
<td>3. Self Cognition</td>
<td></td>
<td>-.05</td>
<td>.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pain Behavior</td>
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<td>.27</td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 25

Intercorrelations of Pre-Post Difference Scores Related to Joy

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Emotion</td>
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<td>.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Pain Cognition</td>
<td></td>
<td>.</td>
<td>.</td>
<td>.</td>
<td></td>
</tr>
<tr>
<td>5. Facial Behavior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. . = coefficient not computable.
Analyses of Variance

One-way, repeated measures analyses of variance were conducted to determine the change in dependent variables between pre versus post treatment phases. Multivariate Analysis of Variance (MANOVA) explored treatment effects for conceptually related dependent measures taken as a whole; univariate analysis of variance (ANOVA) then examined each variable independently for treatment effects. This was supplemented by graphical representations of treatment outcome.

Table 26 summarizes the results of analysis of variance measures related to pain. Given a highly significant multivariate treatment effect, univariate analyses were conducted. These revealed a significant change in overall pain, sensory pain and affective pain (respectively) between pretreatment and posttreatment. As also illustrated in Figure 1, overall pain declined dramatically from a mean of 8.4 at pretreatment to a mean of 6.03 at posttreatment. This was paralleled by a reduction in affective pain from a mean of 6.10 at pretreatment to a mean of 3.93 at posttreatment. Sensory pain changed to a lesser (though statistically significant) degree from a mean of 6.28 at pretreatment to a mean of 5.58 at posttreatment.
Figure 1 Overall, sensory and affective pain before and after treatment.
Table 26
Effects of Treatment on Pain

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>OP</td>
<td>8.40</td>
<td>1.72</td>
</tr>
<tr>
<td>SP</td>
<td>6.28</td>
<td>1.43</td>
</tr>
<tr>
<td>AP</td>
<td>6.10</td>
<td>1.61</td>
</tr>
</tbody>
</table>

Note. Manova $F(3, 37) = 20.52, p < .00001.$

OP = Overall Pain; SP = Sensory Pain; AP = Affective Pain.

Table 27 summarizes the results of analyses of variance on measures of emotion. As indicated, the highly significant multivariate treatment effect was elaborated on by a set of univariate analyses. These revealed the greatest treatment effects (significant at the .00005 alpha level) for the emotions of Anger, Fear and Sadness. Of the remaining emotions, a marginally significant reduction between pre and post treatment was only observed for Disgust. As illustrated in Figure 2, despite significant reduction, the highest mean ratings emerged for Anger, followed by Sadness and then Fear at both pre and post treatment phases. Lowest mean ratings were for the positive/neutral emotions, with Joy having a negligible mean value during both phases.
Figure 2 Emotional responses to pain before and after treatment.
### Table 27

**Effects of Treatment on Emotional Responses to Pain**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>F</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td>6.83</td>
<td>2.31</td>
<td>5.00</td>
<td>1.52</td>
<td>40.66</td>
</tr>
<tr>
<td>Fear</td>
<td>4.75</td>
<td>1.56</td>
<td>3.60</td>
<td>1.45</td>
<td>25.44</td>
</tr>
<tr>
<td>Sadness</td>
<td>5.78</td>
<td>1.79</td>
<td>4.18</td>
<td>1.32</td>
<td>46.65</td>
</tr>
<tr>
<td>Guilt</td>
<td>2.85</td>
<td>2.05</td>
<td>2.68</td>
<td>1.91</td>
<td>.35</td>
</tr>
<tr>
<td>Shame</td>
<td>2.08</td>
<td>1.46</td>
<td>1.60</td>
<td>1.66</td>
<td>2.59</td>
</tr>
<tr>
<td>Disgust</td>
<td>3.63</td>
<td>1.92</td>
<td>3.30</td>
<td>1.83</td>
<td>3.85</td>
</tr>
<tr>
<td>Contempt</td>
<td>2.70</td>
<td>1.95</td>
<td>2.18</td>
<td>1.57</td>
<td>3.53</td>
</tr>
<tr>
<td>Surprise</td>
<td>1.13</td>
<td>.97</td>
<td>1.18</td>
<td>1.06</td>
<td>.25</td>
</tr>
<tr>
<td>Interest</td>
<td>1.85</td>
<td>1.58</td>
<td>1.38</td>
<td>1.73</td>
<td>2.23</td>
</tr>
<tr>
<td>Joy</td>
<td>.08</td>
<td>.27</td>
<td>.05</td>
<td>.22</td>
<td>.33</td>
</tr>
</tbody>
</table>

*Note.* Manova F(10, 30) = 11.96, p < .00005.

Table 28 summarizes the results of analyses of variance on measures of pain cognition. A highly significant multivariate treatment effect was found. Accompanying univariate analyses revealed significant changes for pain cognitions related to Anger, Fear, Sadness and Surprise. Figure 3 shows that the ordinal differences in mean ratings for these cognitions were maintained before and after treatment, except for Surprise-related cognition which dropped below that for Interest during posttreatment.
Figure 3 Emotion-related pain conditions before and after treatment.
Table 28

Effects of Treatment on Pain Cognitions

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment M</th>
<th>Pretreatment SD</th>
<th>Posttreatment M</th>
<th>Posttreatment SD</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger</td>
<td>6.45</td>
<td>2.28</td>
<td>5.13</td>
<td>2.15</td>
<td>23.86</td>
<td>1,39</td>
<td>.00005</td>
</tr>
<tr>
<td>Fear</td>
<td>4.85</td>
<td>2.26</td>
<td>3.38</td>
<td>1.97</td>
<td>23.25</td>
<td>1,39</td>
<td>.00005</td>
</tr>
<tr>
<td>Sadness</td>
<td>5.65</td>
<td>2.26</td>
<td>4.13</td>
<td>1.90</td>
<td>26.29</td>
<td>1,39</td>
<td>.00005</td>
</tr>
<tr>
<td>Guilt</td>
<td>3.58</td>
<td>2.22</td>
<td>3.30</td>
<td>2.33</td>
<td>.87</td>
<td>1,39</td>
<td>.3573</td>
</tr>
<tr>
<td>Shame</td>
<td>1.98</td>
<td>1.91</td>
<td>1.40</td>
<td>1.97</td>
<td>2.93</td>
<td>1,39</td>
<td>.0947</td>
</tr>
<tr>
<td>Disgust</td>
<td>2.98</td>
<td>2.49</td>
<td>2.60</td>
<td>1.97</td>
<td>1.67</td>
<td>1,39</td>
<td>.2039</td>
</tr>
<tr>
<td>Contempt</td>
<td>2.78</td>
<td>1.89</td>
<td>2.53</td>
<td>1.74</td>
<td>2.91</td>
<td>1,39</td>
<td>.0960</td>
</tr>
<tr>
<td>Surprise</td>
<td>1.70</td>
<td>2.00</td>
<td>.73</td>
<td>1.06</td>
<td>12.06</td>
<td>1,39</td>
<td>.0013</td>
</tr>
<tr>
<td>Interest</td>
<td>1.13</td>
<td>1.56</td>
<td>1.23</td>
<td>1.85</td>
<td>.12</td>
<td>1,39</td>
<td>.7325</td>
</tr>
</tbody>
</table>

Note. Manova F(9, 31) = 12.10, p<.00005. M for Joy = 0.

Table 29 summarizes the results of analysis of variance on measures of self cognitions. Again, a highly significant multivariate F was obtained. This was followed by univariate F tests which revealed significant pre versus post treatment differences for self cognitions associated with Anger, Fear, Sadness, Guilt, Shame and Disgust. As Figure 4 illustrates, although parallel, the functions for Sadness-related and Fear-related self cognitions were closer to each other than to the Anger-related function.
Figure 4 Emotion-related Self Cognitions before and after treatment.
### Table 29

**Effects of Treatment on Self Cognitions**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment M</th>
<th>SD</th>
<th>Posttreatment M</th>
<th>SD</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anger</td>
<td>5.98</td>
<td>2.72</td>
<td>4.93</td>
<td>2.19</td>
<td>6.57</td>
<td>1,39</td>
<td>.0144</td>
</tr>
<tr>
<td>Fear</td>
<td>3.63</td>
<td>2.22</td>
<td>2.80</td>
<td>2.34</td>
<td>7.60</td>
<td>1,39</td>
<td>.0088</td>
</tr>
<tr>
<td>Sadness</td>
<td>3.85</td>
<td>2.21</td>
<td>3.08</td>
<td>1.72</td>
<td>10.53</td>
<td>1,39</td>
<td>.0024</td>
</tr>
<tr>
<td>Guilt</td>
<td>2.38</td>
<td>2.05</td>
<td>1.65</td>
<td>1.56</td>
<td>24.13</td>
<td>1,39</td>
<td>.00005</td>
</tr>
<tr>
<td>Shame</td>
<td>1.80</td>
<td>1.87</td>
<td>.85</td>
<td>1.81</td>
<td>6.40</td>
<td>1,39</td>
<td>.0155</td>
</tr>
<tr>
<td>Disgust</td>
<td>1.75</td>
<td>2.10</td>
<td>1.40</td>
<td>2.07</td>
<td>5.77</td>
<td>1,39</td>
<td>.0211</td>
</tr>
<tr>
<td>Contempt</td>
<td>1.65</td>
<td>1.59</td>
<td>1.50</td>
<td>1.50</td>
<td>1.06</td>
<td>1,39</td>
<td>.3095</td>
</tr>
<tr>
<td>Surprise</td>
<td>.30</td>
<td>.60</td>
<td>.73</td>
<td>1.68</td>
<td>2.52</td>
<td>1,39</td>
<td>.1204</td>
</tr>
<tr>
<td>Interest</td>
<td>.63</td>
<td>.95</td>
<td>.68</td>
<td>.92</td>
<td>.33</td>
<td>1,39</td>
<td>.5703</td>
</tr>
</tbody>
</table>

**Note.** Manova F(9,31) = 5.49, p<.00005. M for Joy = 0.

Table 30 summarizes the ANOVA on reports of pain behaviors. The highly significant multivariate treatment effect was followed by univariate analyses. These showed significant differences in pre versus post treatment ratings for those pain behaviors related to Anger, Fear, Guilt, Disgust, Surprise and Interest. As illustrated in Figure 5, the highest mean at both phases was for pain behaviors related to Anger. Means for the other behaviors were crowded within a small range far below that for Anger.
Figure 5 Emotion-related Pain Behaviors before and after treatment.
Table 30

**Effects of Treatment on Reported Pain Behaviors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Anger</td>
<td>5.05</td>
<td>2.11</td>
</tr>
<tr>
<td>Fear</td>
<td>2.78</td>
<td>1.78</td>
</tr>
<tr>
<td>Sadness</td>
<td>1.80</td>
<td>2.15</td>
</tr>
<tr>
<td>Guilt</td>
<td>1.78</td>
<td>1.67</td>
</tr>
<tr>
<td>Shame</td>
<td>2.73</td>
<td>1.75</td>
</tr>
<tr>
<td>Disgust</td>
<td>2.93</td>
<td>1.72</td>
</tr>
<tr>
<td>Contempt</td>
<td>1.90</td>
<td>1.84</td>
</tr>
<tr>
<td>Surprise</td>
<td>2.35</td>
<td>1.85</td>
</tr>
<tr>
<td>Interest</td>
<td>.63</td>
<td>.95</td>
</tr>
</tbody>
</table>

**Note.** Manova F(9,31) = 8.33, p<.00005. M for Joy = 0.

Table 31 summarizes the ANOVA on facial behavior ratings. There was a significant multivariate treatment effect. This was accompanied by univariate analyses which revealed significant changes in facial behaviors related to Anger, Sadness, Guilt, Shame and Surprise between pre versus post treatment. Figure 6 shows that in both phases, mean ratings were highest for the facial expression of Disgust, followed by that for Anger and that for Sadness.
Figure 6 Emotion-related Facial Behaviors before and after treatment.
Table 31

**Effects of Treatment on Reported Facial Behaviors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
<th>F.</th>
<th>df</th>
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*Note.* Manova F(9,31) = 5.80, p<.0005. M for Joy = 0.
The correlation matrices provided earlier have already indicated links between emotions and pain cognitions, emotions and self cognitions, emotions and pain behaviors and emotions and facial behaviors. To see if subjects do see the test items on cognitions and behaviors as corresponding to specific emotions, the frequency and percentage of subjects matching test items with particular emotions was obtained.

Table 32 contains the frequency with which each of the cognitive and behavioral test items were labeled as related to any one of the 10 emotions. The rightmost column gives the percentage of subjects giving responses in agreement with the a priori conceptualization of the items. As many as 100% of subjects agreed on labeling six of the items in accordance with their presumed correspondence to emotions. For the remaining items, percentage agreement with the presumed item-emotion correspondence was no less than 68%. For example, 35 out of 40 subjects recognized item 30 of the questionnaire ("I see myself as an outcast because of my pain") as corresponding to the emotion of Shame -- as had the investigators. The majority of subjects matched test items with specific emotions in a manner that concurred with the investigators. This contributes to the (construct) validity of the test in measuring what it purports to measure.
Table 32

Validity Check

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Note. N = 40.
To assess the temporal consistency of subjects' matching responses, the number of subjects giving identical responses at both administrations of the test (four weeks apart) was tallied. The results are displayed in Table 33. As indicated, between 60 and 100% of subjects assigned the same emotion labels to test items as they had done four weeks earlier. This provides support for the reliability of subjects' perceptions of the test.

The final bit of data concerns subjects' performance on the proficiency quiz. A criterion had been set requiring subjects to pass at least 19 of the 25 questions on the quiz. Thirty-one subjects achieved this on their first attempt, and nine reached criterion after two attempts. No subject scored less than 50% correct on the first attempt. The mean passing score was 22 (or 88% correct) with a standard deviation of 1.75.
Table 33

**Reliability Check**

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**Note.** $N = 40.$
DISCUSSION

A long line of research had suggested that pain can be characterized in terms of sensory and affective variables. The present study is original in its attempt to delineate the relative contributions of each of these variables and their particular combinatorial pattern with respect to pain as a whole.

The first hypothesis considered the suitability of a linear model in quantifying pain as a function of sensory and affective variables. Results provided overwhelming support for this model. It was unequivocally shown that sensory and affective features are combined in a linearly additive fashion to constitute overall pain of the chronic kind. In other words, an equation for a straight line fits the data, so that the level of overall pain is a summation of the magnitude of physical sensation and degree of affective distress (each multiplied by a unique coefficient) plus a constant. The fit is so compelling that non-linear models are abandoned altogether.

This is not surprising since linear models are generally known to outperform other models in decision making. As Dawes and Corrigan (1974) point out, this is
particularly so when the predictor variables have conditionally monotone relationships to the criterion variable. In the present context, that means the predictors (sensory pain and affective pain) can be scaled so that higher values on each are associated with higher values on the criterion (overall pain), independently of the values of remaining variables. Also, the relative weights derived from linear regression are not affected by error in the criterion variable. Such error reduces the expected values of all these weights by the same constant hence reducing the absolute value of the criterion variable by that same amount. Finally, measurement error in the predictor variables tends to make optimal functions more linear. It is unclear to what extent these factors are responsible for the linear pattern obtained in this study, but all the same they must be acknowledged. In the present study, the approximation is exceedingly good so that competing models are less likely to improve on the representation of the same data.

The mathematical disaggregation of chronic pain into sensory and affective constituents should not be taken to mean that the whole is equal to a simple sum of its parts. A sensory pain rating of 6 and an affective pain rating of 7 would not yield an overall pain rating of 13; instead, when substituted in the linear equation for pretreatment data, they predict an overall pain level of 8.38. The
additive relationship is thus qualified by parameters pertaining to the intercept and slope of regression lines.

Gestalt psychology is replete with examples of the non-equivalence of a whole to the sum of its parts. In vision, audition and other modalities, there are numerous cognitive-interpretive processes that moderate the relationship between sensation and perception. As Turk (1989b) reminds us, pain too is a subjective, perceptual response and therefore cannot equal the totality of its parts. Numerically, it may be greater (or for that matter less) than the sum of sensory and affective ratings; experientially, it is far too complex a phenomenon to be reconstituted by a simple juxtaposition of its sensory and affective components.

In short, a decomposition of pain into its sensory and affective components has been accomplished. But the combination of these two parts need not amount to the original whole. There are at least two possible explanations. First of all, there may be yet other parts (albeit less integral) that elude the investigator. Secondly, even if all existing parts were brought together, the whole may remain superordinate to this because individual parts are themselves likely to be transformed in the process of being fused. The linear equations derived in this study are not incompatible with this possibility. They merely provide a decomposition of chronic pain into
its key components, but no claims are made about the reconstitution of these components to form the original whole.

The linear model held both before and after treatment, but the relative contributions of each component varied among subjects and across treatment phases. For some patients, affective distress exceeded sensory intensity, while for others the reverse was true. This is consistent with the findings of Clark et al. (1989) that subjects differed in the weight assigned to the affective dimension of pain. Individual differences in the weighting of each component are also suggested by the observation that some studies (e.g., Chen et al. 1989; Graffam & Johnson, 1987) report greater salience of the affective component while others (e.g., Knox et al. 1974; Price & Harkins, 1987) report greater salience of the sensory component.

As mentioned, the relative contribution of sensory versus affective variables also changed with treatment. Before treatment, sensory pain slightly exceeded affective pain but the two were not markedly disparate. In fact, they were highly correlated. Treatment, however, wedged a gap between the two, so that by discharge four weeks later, patients reported significant reduction in overall pain that was largely related to a comparable reduction in affective distress. Sensory pain on the other hand accounted for a much smaller portion of the variance in
overall pain at this point. This may be explained by the nature of treatment. Patients received no pharmacological or biomedical intervention that might greatly alter their physical sensation of pain, but instead underwent psychosocial treatment designed to improve coping skills. Therefore, distress from nociception was substantially diminished along with a reduction in overall pain. The affective variable thus accounted for the bulk of variance in overall pain after treatment.

It is also possible that treatment sensitized subjects to the difference between sensation and affect in pain. Through the cognitive-behavioral intervention upon reactive aspects of pain, patients may have learned to view pain in terms of its component features and this could have accentuated the differential ratings they gave for the two components. This is however not incompatible with the earlier interpretation of the same finding as a treatment effect. After all, the treatment had an important educational component designed to alter subjects' cognitions and perceptions of pain in addition to modifying their pain behaviors.

The functional relationship between affective pain itself and a corpus of emotions was the subject of the second hypothesis. The emotions were grouped into negative emotions deemed to be physiologically prewired, namely, anger, fear and sadness (Set 1), negative emotions thought
to be more cognitively modulated than prewired, namely, guilt, shame, disgust and contempt (Set 2), and finally, the neutral/positive emotions of surprise, interest and joy (Set 3). Once again, a linear model provided a satisfactory account of the data. Affective pain turned out to be a linear combination of all three sets of emotions; Sets 1 and 2 were positively related to affective distress, while as expected, Set 3 showed an inverse relationship with affective distress. As in the decomposition of overall pain into its sensory and affective components, it cannot be inferred here that the level of affective pain is a simple addition of the magnitude of negative emotions and negation of positive emotions. For reasons already outlined, it must be conceded that the whole (in this case affective pain) is a complex entity that transcends the sum of its parts. Meanwhile, it is significant to note that affective pain can be linearly disaggregated into individual emotions. As much reference as has been made to the emotional aspects of pain, no other study is known to have attempted such a disaggregation before.

The third hypothesis focused on the order of importance of each set of emotions contributing to affective pain. As predicted, the emotions accounting for the bulk of variance in affective pain were anger, fear and sadness. A survey of the emotions literature (in Chapter I) had already
indicated that these are the emotions most likely to arise in the face of aversive stimuli, since they prepare the organism to fight, flee or yield, respectively. They are thus regarded as physiologically prewired. A recent study by Harkins et al. (1989) similarly found a high level of these emotions or related syndromes among those with clinical as well as experimental pain.

The physiologically prewired emotions were however also correlated with the more cognitively-initiated negative emotions. This is to be expected in view of their common negative valence. By posttreatment though, the latter set of emotions failed to add significantly to the variance in affective pain already accounted for by anger, fear and sadness. Positive/neutral emotions accounted for the smallest portion of variance in affective pain upon completion of treatment. This is probably due to the type of treatment which targeted anger, fear and sadness more than any other set of emotions. So even though guilt, shame, disgust and contempt are largely modulated through cognition, they did not change as much, and thus failed to add significantly to the variance in affective pain at posttreatment.

The fourth hypothesis addressed the interrelations among emotions, cognitions and behaviors associated with chronic pain. In this respect, it was found that emotional responses to pain were most related to cognitions about
pain, then to cognitions about self and purposive pain behaviors, and least to facial behaviors. However, within each of these categories, items varied substantially in the strength of correlation with emotion. For example, there was close correspondence between the facial expression for disgust and reported experience of the emotion of disgust, but little correspondence between the facial expression for shame and reported experience of the emotion of shame. There was close correspondence between cognition about oneself as an outcast because of pain and the experience of shame, but little correspondence between the perception of oneself as an unclean person because of pain and the experience of disgust. For each individual emotion too, there was variability in correlations among its corresponding cognitions and behaviors. In short, although cognitions were generally more closely linked to emotions than were behaviors, the correlations tended to be item-specific. Much seems to have depended on the particular item selected to measure pain cognition, self cognition, pain behavior or facial behavior as the case may be.

The findings here do provide a strong case for viewing emotions in conjunction with cognitions. Price's (1988) assertion that emotional responses to pain are influenced by meaning and expectations about pain finds credence here. Some have argued that the cognitions are in fact precursors of emotions (e.g., Lazarus & Folkman, 1984; Ortony et al.
1988). However, as pointed out by Ellsworth and Smith (1988), it is difficult to claim such a causal connection without more complex experimental manipulations and longitudinal studies. Suffice it to say that emotional responses to pain are intimately linked to specific cognitive correlated. Similarly, the purposive behaviors and facial activation patterns are best regarded as behavioral correlates rather than concomitants of emotion.

The intercorrelations among cognitions and behaviors themselves exhibited little in the way of a pattern. They were greatest for the emotion of anger, but much less so for the emotion of shame. Beyond that, they seem to be item-specific. What is more important is whether or not change in one measure (after treatment) was paralleled by change in other correspondent measures. This would be a better index of how closely linked they are. The present study showed that pre-post treatment reductions in emotional responses to pain were indeed matched by comparable reductions in the degree of endorsement for corresponding cognitions as well as reductions in corresponding behaviors reported. This was especially the case for anger and shame, beyond which the pattern is again best described as item-specific.

The remaining hypotheses pertained to treatment outcome. The four-week treatment program produced significant reductions in ratings for cognitive and
behavioral items on the questionnaire. Pain behaviors showed the greatest number of significant effects, although significant reductions were also observed for items of pain cognition, self cognition and facial behavior. The breadth of these effects is largely attributable to the fact that the treatment regimen spanned both cognitive and behavioral types of intervention. It is of further interest that the cognitive and behavioral items receiving the highest ratings were those related to anger, fear and sadness; these were the same items that also witnessed the greatest reductions. This is probably because the treatment program (operating on the premise that anger, fear and sadness are physiologically prewired response options for aversive stimuli such as pain), targeted those very cognitions and behaviors related to this set of emotions. Therefore, as high as the ratings were for these items, they also underwent the greatest reductions. Of the remaining items, those related to disgust and joy are worthy of some note. Cognitions and behaviors related to the latter remained at a low both before and after treatment, since joy is a positively valenced emotion that rarely occurs in response to pain. Cognitions and behaviors related to disgust approached the magnitude of those related to the anger-fear-sadness constellation. This was not expected, but it may be that disgust is intermediate in quality between the physiologically prewired emotions and the cognitively
modulated ones; therefore, its cognitive and behavioral correlates underwent changes nearly comparable to those of anger, fear and sadness.

Treatment produced negligible change in the already low ratings of positive/neutral emotions, but reductions across all of the negative emotions. These reductions reached statistical significance for anger, fear and sadness, and marginal significance in the case of disgust. Guilt, shame and contempt did not decline notably with treatment. This is in part because these emotions do not covary with pain to the same degree that the other negative emotions do. Secondly, as already stated, the treatment program was directed more towards the attenuation of anger, fear and sadness than the cognitively-initiated emotions. In any case, the (already-discussed) correlations between emotions and their cognitive/behavioral counterparts using pre-post difference scores, suggests that the changes in emotion are related to changes in corresponding cognitions and behaviors.

Of the foremost concern in treatment outcome was the impact on pain itself. First, a significant diminution of affective pain was observed between commencement and termination of treatment. Correlational data indicate that this is essentially a product of the significant reductions in certain negative emotional responses to pain, namely, anger, fear, sadness (and disgust, to a lesser extent).
Regression analyses established that these are the emotions accounting for the bulk of variance in affective distress from pain. In other words, affective distress from nociception is markedly reduced as a function of reductions in the physiologically prewired emotions of negative valence.

Interestingly, even though most subjects reported a small decline in sensory pain, it did not approach the magnitude of reduction observed for affective pain. This means that the treatment program had a greater ameliorative impact on affective responses to pain than their sensory counterparts. As mentioned earlier, the explanation may lie in the nature of treatment which was predominantly psychosocial rather than biomedical. These findings are consistent with many other studies utilizing psychological forms of intervention. Johnson and Rice (1974) found that information manipulation reduced distress ratings while exerting only a marginal effect on sensory aspects of experimentally-induced ischemic pain. Houle et al. (1988) found that hypnosis and relaxation during cold pressor stimulation significantly reduced reported unpleasantness without a comparable reduction in sensory pain.

In all fairness, one must not disregard the studies in which psychological intervention produced equivalent reductions in sensory and affective pain (e.g., Graffam & Johnson, 1987; Knox et al. 1974). These slightly divergent
findings may stem from differences in subject samples, type of pain, operationalization of dependent measures, treatment parameters and other aspects of methodology. Yet, broadly speaking, the present findings are in agreement with earlier research in suggesting that at the very least, psychological intervention produces a diminution in the affective variable of pain.

An additional finding was the highly significant reduction in pain as a whole. Regression analysis and correlations based on pre-post difference scores indicated that this was essentially related to the drop in affective pain. The chain of events is now a little clearer: Overall pain declined largely as a function of reduced affective distress that was linked to a reduction in negative emotional responses (particularly, anger, fear and sadness), which in turn was associated with reduced maladaptive cognitions and behaviors that had been the target of a predominantly cognitive-behavioral intervention. Care is taken to avoid inferences of causality, since, with the exception of the pre-post manipulation, the present study is correlational in design. The pre-post variable however does greatly reduce ambiguity about the role of treatment in the changes observed.

The outcome of this treatment program compares favorably with the handful of treatment outcome studies that have been published. However, one must be mindful of
the differences in dependent measures and program ingredients.

Roberts and Reinhardt (1980) reported on the outcome of a six-week program consisting of contingency contracts, inattention to pain behaviors and reinforcement of well behaviors, graduated reduction of pain medication, physical therapy and occupational therapy. They found that 77% of pain patients thus treated met criteria for success. These criteria included discontinuation of medication and resumption of employment or gainful activity.

Guck, Meilman, Skultety and Dowd (1986) researched a four-week program employing the core ingredients of medication-tapering, exercise promotion, relaxation training, counseling and education. Adopting the same criteria of success as Roberts and Reinhardt (1980), they found that 47% of patients improved significantly.

Keefe, Block, Williams and Surwit (1981) subjected chronic low back patients to a comprehensive behavioral program emphasizing relaxation procedures. Their results showed a 29% drop in pain over the course of the program. The 25% of patients revealing the most improvement reported highly significant reduction in pain, while the 25% who succeeded least also reported significant reduction in pain. Both subgroups also showed notable decrease in medication intake, while the top 25% also achieved significant reductions in electromyographic levels and
increases in physical activity. So, there was variability in performance but the vast majority of subjects showed improvement.

The pain program studied by Chapman, Brena and Bradford combined psychological facets of previous programs with routine nerve blocks. With six weeks of intervention at three hours per week, a highly significant reduction in pain intensity was obtained. Given the biomedical plus psychological features of the treatment, it would have been interesting to explore sensory-affective relationships, but this was not done.

Parris, Jamison and Vasterling (1987) also incorporated nerve blocks within a multidisciplinary pain management program. They found that 49% of patients evidenced appreciable decreases in pain, 51% decreased medication usage and 46% significantly increased physical activity.

The foregoing studies are not uniform in their degree of treatment success. This is very likely a result of numerous procedural differences. Some of the studies (e.g., that by Parris et al. 1987) were quite limited in the duration and frequency of treatment provided to patients, and thus obtained lower success rates.

In contrast, the present study yielded slightly more encouraging findings. Part of the explanation resides in the comprehensiveness and intensiveness of the treatment program. Other reasons relate to methodology: the
statistical power of the study was enhanced by a very large, undivided sample of 40 patients. Subjects were quite homogeneous in pain diagnosis and had to demonstrate a minimal level of proficiency to be included in the study. Error variance was also contained by the inclusion of only one independent variable which was a "within subjects" variable.

The proficiency quiz in this study was passed by 31 out of the 40 subjects on the first attempt; all the rest passed on the second attempt. The criterion for a pass entailed correctly answering 75% of the questions on the quiz. The pass rate was high partly because of the low level of difficulty of the questions. The quiz had not been designed with stringent criteria in mind but merely to ensure that all participants shared minimal grasp of the principles of pain management.

A final ancillary part of the present study was to ascertain if the cognitive and behavioral items on the questionnaire that were supposed to correspond to certain emotions were perceived in that manner by subjects. This is important because if independent judges failed to see this correspondence, then attempts to relate emotions, cognitions and behaviors would be thwarted. Results showed that the items pertaining to cognitions and behaviors were matched with specific emotions in the same way as presumed by the investigators, by no less than two-thirds of the
subject sample. For six of the items, there was perfect agreement among all subjects regarding their correspondence with emotions. This suggests that the assessment instrument used here has an acceptable level of face validity and construct validity.

It was further revealed that 60 to 100% of subjects gave the same judgement of correspondence in separate sessions four weeks apart. This attests to the temporal stability of subjects' responses that matched cognitions and behaviors with their respective emotions.

Retrospective Critique of Present Study

The present study addressed several questions about chronic pain, some of which are quite new while others are already familiar to the area. As in most undertakings of this extent, there are both merits and shortcomings.

A central achievement of this study was the partitioning of overall pain into sensory and affective variables, with a minimum of demand characteristics. Previous studies typically clued subjects about the connection and distinction between the two variables before getting them to rate these (e.g., Duncan et al. 1979; Gracely et al. 1978a; Harkins et al. 1989; Knox et al. 1974). Subjects may thus have given disparate ratings for sensation and affect even if they perceived them otherwise. In the present study, questions about the two variables
were presented as separate items on a questionnaire with no discussion of how they might relate to each other. In this way, demand characteristics were more or less circumvented.

Another improvement was the use of numerical ratings on a visual analogue scale. As Turk (1989a) notes, this avoids the ambiguity that attends verbal descriptor scales. A comparison of previous studies (e.g., Gracely et al. 1978a; Tursky, 1976) reveals discrepancies in magnitude assigned to the same word by different subjects. The numerically marked VAS permits the quantification of different phenomena using a standardized scale, whereas a verbal descriptor scale would have required a separate set of words for sensation and affect.

Unlike multidimensional scaling and factor analytic studies, the presented study was not limited to an extraction of latent variables underlying subjects' responses. Instead, subjects responded directly to the constructs being studied. This brings a certain robustness to the findings here.

The criteria for separation of sensory from affective pain were relatively strict. First of all, parallel functions for sensation and affect (as had been reported by other investigators) were not regarded as definitive evidence of dissociation of the two variables. The variables were regarded as distinctive to the extent that their functions diverged, as might be expected when a
particular intervention targets one variable and not the other. Also, for the first time, sensory and affective pain ratings were not only pitted against each other but also considered in relation to pain as a whole. In this way any disparity arising between sensation and affect could be qualified by the relative contributions of these two variables to overall pain. The present study was also original in its attempt to functionally relate affective pain to individual emotions.

Despite the above strengths, teasing apart the sensory and the affective did not always prove to be a tidy matter. At treatment outset, the two variables were significantly correlated though disparate. This problem of multicollinearity is pervasive in the literature on this subject. Much like the correlation of .80 between sensation and affect in this study, confirmatory factor analysis (Turk et al. 1985) had produced a correlation of .82 while paired VAS scaling (Chen et al. 1989) had arrived at a correlation coefficient of .78 for these variables. There are many other studies in which collinearity of sensory and affective pain was not reported but is all the same quite apparent in the data published (Dunbar et al. 1988; Duncan et al. 1989; Houle et al. 1988; Knox et al. 1974; Price et al. 1987). It is encouraging to note that the multicollinearity witnessed in this study declined substantially following treatment, with affective pain
emerging as the main predictor of overall pain at this point. This implies that the treatment intervention (which was predominantly cognitive-behavioral in nature) attenuated affective pain thereby diminishing overall pain while leaving sensory pain relatively invariant. Sensory and affective features of pain are hence separable with the aid of treatment intervention that selectively alters one of the two variables.

A brief comment on terminology is in order here. Although dissociated from its sensory counterpart, affective pain may arguably not represent a component of pain as supposed throughout much of the literature. After all, it frequently arises as a consequence of nociceptive stimulation. (Of course it can also occur in anticipation of noxious stimuli). But the temporal gap between sensation and affect is ordinarily so small and the conjunction between nociception and emotion so reliable, that for all practical purposes the two may be regarded as interwoven. At the very least, they are correlates if not components of pain.

Among the methodological deficiencies of the present study are the absence of objective data such as behavioral observations and physiological measures. This investigation relied exclusively on subjects' self-report of pain, emotions, cognitions and behaviors. There is some support for this approach. Physiological measures have
been criticized as not sharing much specificity with pain. Hilgard for instance asserts: "there is no physiological measure of pain which is either as discriminating of fine differences in stimulus conditions, as reliable upon repetition, or as lawfully related to changed conditions, as the subject's verbal report" (1969, p. 107). As discussed in the Introductory chapter, physiological measures have also been inadequate in differentiating or quantifying emotions. On the other hand, behavioral indices have been widely employed in the study of pain. They encompass measures of gait, posture, verbal complaints and level of physical activity. These can help corroborate self-report data at different stages of treatment. However, due to staffing limitations, it was not possible to procure these measures in a systematic and dependable way.

Another limitation of this study is the absence of follow-up data. It is conceded that the maintenance of treatment gains is important to an appraisal of treatment efficacy (Turk et al. 1989c). Increasingly, pain programs have been gathering information on a common set of dependent variables at various points up to eight years posttreatment (see review by Aronoff, Evans & Enders, 1983). However, the present study was planned so that data collection itself would take approximately six months thus leaving insufficient time for any reasonable follow-up
before writing up the study. Also, treatment outcome was really a secondary part of this study. The primary objective was to ascertain the separability of pain into sensation and affect, along with an elaboration of the affective variable in terms of individual emotions.

Given the major thrust of the study, it was not mandatory to have a comparison group. Subjects served as their own controls by being assessed before and after treatment. However, a no-treatment group of chronic pain patients wait-listed for the same program could have been assessed over a four-week period in order to serve as a between subjects control. It would also be relevant to see how normal subjects administered experimental pain rated their pain, sensation, affect, individual emotions and their corresponding cognitions and behaviors. But this must be set aside for another study altogether.

Considerations for Further Research

The present study broached several questions and attempted to answer them to the extent possible in one sweep. The answers may not be complete, but they provide a basis for further research in this area. Future research can profit from rectifying some of the limitations in this study as well as making extensions to it.

It has already been mentioned that other populations of subjects may be studied in the same manner as those in this
study. The separability of pain into sensation and affect may be posed in the context of experimental pain under psychological intervention. A better comparison group may include those with different types of chronic pain. It is likely that a sample consisting exclusively of headache sufferers may respond differently from one with low back pain patients. Furthermore, comparisons may be made between those in chronic malignant pain (as from cancer) and those with chronic non-malignant pain; this is likely to reveal differences in the emotional variables subserving affective pain. Comparison groups would serve to cross-validate the results of the present study. This is imperative for purposes of generalizability.

Moderator variables not related to pain may also be considered. For example, age, gender, occupational status and other demographic characteristics may be used as predictors of the measures studied herein. It is probable that personality features may be particularly predictive of the weighting a subject accords to affective versus sensory aspects of pain and the potential of that subject to benefit from psychosocial as opposed to biomedical treatments. Also, certain treatment variables such as rate of medication tapering may be related to the magnitude of treatment effects obtained.

As already alluded to, more objective measures based on behavioral observations can serve to corroborate subjects'
self-reports. Dependent measures may be collected beyond treatment so that follow-up information is available. This would reflect on the stability of both treatment outcome and pain analysis data.

At the level of data analysis, innovative techniques have developed to tackle the problem of multicollinearity among predictor variables. Prominent among these is structural equation modeling, which has been recently recommended for studying constructs related to pain (Rudy, in press). The technique reportedly provides more accurate correlation and regression estimates by statistically adjusting for the amount of error associated with predictor variables. To make specific inferences about the composition rules for predictors of overall pain, the method of conjoint measurement (Krantz & Tversky, 1971; Nygren, 1985) may be particularly useful. This could enable comparisons among additive, multiplicative and distributive models with regard to how well they fit the data.

Future research may also look into the psychometric properties of the questionnaire developed for this study. Although there are indications of its face validity and construct validity, its criterion-related validity could be determined. This would entail obtaining other measures (such behavioral observations or the results of other questionnaires) and relating them to the self-report
measures from this questionnaire. The test-retest reliability of the instrument could also be ascertained by administering it at separate points in time without intervening treatment. In the present study, the individual test items were really used as independent measures of different phenomena. It remains for future studies to determine how these items collectively stand up to psychometric standards.

Conclusion

The present study achieved several objectives. First, it defined an area of study that was hitherto made up of pockets of research isolated from one another; in the process, it also bridged the bodies of literature pertaining to pain and emotions, respectively. Findings from this corpus of information were critically integrated to arrive at specific hypotheses for investigation. The research investigation shed considerable light on the questions raised, demonstrating above all that pain is indeed separable into sensory and affective features. The affective variable is a function of individual emotions of negative valence. Treatment of a predominantly cognitive-behavioral kind successfully reduced maladaptive cognitions and behaviors corresponding to the negative emotions, reducing the intensity of these emotions and thus diminishing the intensity of affective distress. Reduction
in affective distress was associated with a comparable reduction in pain as a whole, despite a relatively small change in the sensory properties of pain.

It may be inferred that the future of pain management looks quite encouraging within the province of multidisciplinary pain clinics emphasizing cognitive-behavioral approaches. Particularly if intervention focuses on the unique relations between cognitions and emotions and between behaviors and emotions, it can modify the experience of pain. Since anger, fear and sadness are the main negative emotions triggered by pain, treatment might pay special attention to the behaviors and cognitions (about pain and self) that subserve these emotions. Attenuation of these emotions would diminish affective pain which would in turn reduce overall pain. In short, altering the sensory aspects of pain is not essential for reduction in overall pain. In the absence of biomedical intervention, psychological intervention via its influence on affect can produce an appreciable alteration of pain.

While treatment may be selectively directed at one variable, the assessment of pain must be multifaceted. It is increasingly evident that pain is not a unitary phenomenon, but that it has a sensory as well as affective quality. All these variables must be concurrently measured in order to achieve a fuller appreciation of the complexity of pain as a construct and as an experience.
List of References


Lacey, J. I., & Lacey, B. C. (1958). Verification and extension of the principle of autonomic


Pain Questionnaire reconsidered: Confirming the factor structure and examining appropriate uses. *Pain*, 21, 385-397.


APPENDIX A

SOLICITATION SCRIPT
Solicitation Script for Study on "Primary Emotions in the Affective Contribution to Chronic Pain"

Dr. Steven Beck and myself (Ephrem Fernandez) of the Ohio State University are conducting a study on pain and emotions. It involves each participant filling out a paper-and-pencil questionnaire that takes about 40 minutes to complete.

Your participation in this study is voluntary, that is, you have the right to decline or withdraw from the study at this point or at a later time, without any coercion from others. However, your willing participation will be greatly appreciated since it will further our understanding and treatment of your pain as well as others' in chronic pain. Only your treatment staff and research investigators (Dr. Steven Beck and Ephrem Fernandez) will be allowed to see your results. If you have any questions regarding this study, please contact Dr. Steven Beck at 292-6849 or Ephrem Fernandez at 293-3830.

Should you agree to participate, please sign the attached consent form (HS-027).
APPENDIX B

CONSENT FORM
CONSENT FOR PARTICIPATION IN
SOCIAL AND BEHAVIORAL RESEARCH

I consent to participating in (or my child's participation in) research entitled:

"Primary Emotions in the Affective Contribution to Chronic Pain"

Steven Beck, Ph.D. or his/her authorized representative has
(Principal Investigator)
explained the purpose of the study, the procedures to be followed, and the expected duration of my (my child's) participation. Possible benefits of the study have been described as have alternative procedures, if such procedures are applicable and available.

I acknowledge that I have had the opportunity to obtain additional information regarding the study and that any questions I have raised have been answered to my full satisfaction. Further, I understand that I am (my child is) free to withdraw consent at any time and to discontinue participation in the study without prejudice to me (my child).

Finally, I acknowledge that I have read and fully understand the consent form. I sign it freely and voluntarily. A copy has been given to me.

Date: ____________________  Signed: ____________________ (Participant)

Signed: ____________________  Signed: ____________________
(Principal Investigator or his/her Authorized Representative) (Person Authorized to Consent for Participant - If Required)

Witness: ____________________

HS-027 (Rev. 3/87) — (To be used only in connection with social and behavioral research.)
Assessment

Opening Instructions to subjects:
This is a simple six-page questionnaire that should take about 40 minutes to complete. You will be asked to rate various aspects of your pain, emotions, thoughts and behaviors for the past week. For each item listed, indicate the degree to which you experience it by choosing a number between 0 and 10 where 0 means the total absence of the item from your experience and 10 means the highest level of the item you could possibly experience. (Feel free to use fractions). Please be as honest and accurate as possible.

Your participation in this study is voluntary, that is, you have the right to decline or withdraw from the study at this point or any time later, without coercion from others. However, your willing participation will be greatly appreciated since it will further our understanding and treatment of your pain as well others in chronic pain. Only your treatment staff and research investigators (Dr. Steven Beck and Ephrem Fernandez) will be allowed to see your results. If you have any questions regarding this study, please contact Dr. Steven Beck at 292-6849 or Ephrem Fernandez at 293-3830.

Should you agree to participate, please sign the attached consent form (HS-027) and then proceed with the questionnaire as instructed.
Please rate for the past week

0 1 2 3 4 5 6 7 8 9 10
Nothing Extremely
High

1. Overall Pain: ___

2. physical intensity of throbbing, burning, piercing etc... ___

3. how much you're distressed by the throbbing, burning, piercing... ___
Please rate for the past week:

<table>
<thead>
<tr>
<th>Absent</th>
<th>Extremely High</th>
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<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

1. Anger (A)
2. Fear (F)
3. Sadness (Sd)
4. Guilt (G)
5. Shame (Sh)
6. Disgust (D)
7. Contempt (C)
8. Surprise (Sp)
9. Interest (I)
10. Joy (J)
Please rate for the past week

<table>
<thead>
<tr>
<th>Not at all</th>
<th>Very much so</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>10</td>
</tr>
</tbody>
</table>

14. For me, pain is an overpowering danger

15. For me, pain is a cowardly, low-down pest

16. For me, pain is a discouraging setback

17. For me, pain is a disgrace

18. For me, pain is a fascinating mystery

19. For me, pain is an enemy to be fought against

20. For me, pain is a delightful experience

21. For me, pain is a filthy impurity

22. For me, pain is the unexpected

23. For me, pain is punishment from God/Nature
Please rate for the past week

| __________ | __________ |
| 0 1 2 3 4 5 6 7 8 9 10 |

Not at all | Very much so

24. I see myself as an unclean person because of my pain

25. I see myself as a proud authority with no respect for pain

26. I see myself as a keen explorer of pain

27. I see myself as a helpless victim of pain

28. I see myself as a sinner/wrongdoer deserving pain

29. I see myself as a fighter against pain

30. I see myself as an outcast because of my pain

31. I see myself as a celebrator of pain

32. I see myself as a weak defendant against pain

33. I see myself as someone amazed by pain
Please rate for the past week

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
</table>

Not at all | Very much so

34. When in pain, I panic or plead for help

35. When in pain, I do penance/sacrifice to make up for wrongs

36. When in pain, I rejoice and try to promote it

37. When in pain, I get sick and tired or nauseated

38. When in pain, I become curious about it

39. When in pain, I cry or withdraw

40. When in pain, I get physically aggressive

41. When in pain, I suddenly become aware of it

42. When in pain, I mock it and dismiss it from my concerns

43. When in pain, I conceal it so others don't think less of me
Closing Instructions to subjects:

Now, please go back to the last four pages, and indicate which one of the ten emotions you most associate with each item. Use the following abbreviations: "A" for Anger, "F" for Fear, "Sd" for Sadness, "G" for Guilt, "Sh" for Shame, "D" for Disgust, "C" for Contempt, "Sp" for Surprise, "I" for Interest and "J" for Joy. Write the appropriate abbreviation in the space next to the rating for each item.

Thank you for your participation in this study. I would be happy to discuss your results with you if you wish.
APPENDIX D
COPYRIGHT PERMISSION
Ephrem Fernandez
Psychology Internship Program
Western Psychiatric Inst. & Clinic
University of Pittsburgh
3811 O'Hara Street
Pittsburgh, PA 15213-2593

June 26 1989

Dear Dr. Werner:

I am writing to request permission to reproduce nine photos from Dr. Carroll Izard's 1977 book "Human Emotions" that was published by Plenum Press. I would like to use these photos as part of a dissertation project and have them appear in my dissertation. Dr. Izard has granted me verbal permission to do so, but I would still appreciate a written letter from you granting permission to use and reproduce the photos only in the way mentioned above.

Looking forward to hearing from you as soon as possible. Thank You

sincerely,

Ephrem Fernandez

P.S. I enclose a copy of the nine photos which I would like permission to use.
September 28, 1989

Mr. Ephrem Fernandez
Psychology Internship Program
Western Psychiatric Institute & Clinic
3811 O'Hara Street
Pittsburgh, PA 15213

Dear Mr. Fernandez:

I am happy to grant you permission to use my photographs of facial expressions that appeared in *Human Emotions* in your dissertation.

Sincerely,

[Signature]

Carroll E. Allard
Unidel Professor
APPENDIX E

QUESTIONNAIRE KEY
Key to Correspondence between Test Items and Emotions

<table>
<thead>
<tr>
<th>Item</th>
<th>Emotion</th>
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<tbody>
<tr>
<td>14</td>
<td>F</td>
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A = Anger; F = Fear; Sd = Sadness; G = Guilt; Sh = Shame;
D = Disgust, C = Contempt; Sp = Surprise; I = Interest;
J = Joy.
APPENDIX F

PROFICIENCY QUIZ
Proficiency Quiz

Please select the one (most accurate) answer to each question

1. Medication
   (a) is most beneficial when combined with alcohol
   (b) is alright as long as it clears the symptoms of illness
   (c) can remove symptoms of illness but often produces side-effects

2. Dependence on pain medication (analgesics)
   (a) can never be reduced after six months of use
   (b) can be reduced by a contract to use medication at set times in
      gradually decreasing dosages.
   (c) will increase only if one has used "street drugs" before

3. What are the three common sources of caffeine?
   (a) coffee, tea, chocolates
   (b) zucchini, cucumbers and carrots
   (c) milk, orange juice and brandy

4. Which of the following is not a U.S. Dietary Guideline?
   (a) reduce cholesterol
   (b) reduce salt and sugar
   (c) eat adequate starch and fiber
   (d) stick to a small variety of foods
5. Exercises recommended for those in chronic pain are
   (a) stretching
   (b) strengthening
   (c) endurance
   (d) all of the above

6. Stretching exercises are best performed
   (a) slowly and at least once a day
   (b) rapidly whenever time permits
   (c) once every twelve months but twice in a leap year

7. Which of the following is not an occupational adjustment to injury?
   (a) remaining unemployed
   (b) retraining in some new skill
   (c) becoming self-employed
   (d) doing the same job but under better working conditions

8. To manage time productively, it helps to
   (c) do many different tasks all at the same time
   (b) switch between easy and difficult tasks within a daily routine
   (c) set your wristwatch at least a couple of hours ahead of time

9. The main purpose of leisure education is to:
   (a) be able to understand the sports column of the newspaper
   (b) find out those recreational activities most satisfying to oneself
   (c) get people to give up work
10. Which of the following is not part of leisure education?
   (a) social interaction
   (b) leisure resources
   (c) cardiac pulmonary resuscitation skills

11. What is the most common cause of back pain?
   (a) pinched nerve
   (b) ruptured disc
   (c) soft tissue injury
   (d) broken vertebrae

12. What is the most serious health hazard of smoking?
   (a) heart disease
   (b) lung disease
   (c) cancer
   (d) chronic cough

13. In progressive muscle relaxation, we ______ the muscles before relaxing them.
   (a) elevate
   (b) tense
   (c) squeeze

14. The main aim of progressive muscle relaxation is to
   (a) build muscle strength
   (b) become aware of tension in parts of the body and then release it
   (c) induce sleep
15. Imagery is most useful
   (a) at the onset of pain, to prevent it from getting worse
   (b) after the pain has stopped
   (c) while driving on the highways

16. When guided imagery is not helpful, it is best to:
   (a) give up
   (b) take drugs
   (c) try self-directed imagery

17. The assertive communication style is somewhere in between
   (a) passiveness and aggressiveness
   (b) avoidance and contact
   (c) looking and touching

18. What happens when two assertive persons interact?
   (a) combat
   (b) compromise
   (c) withdrawal

19. Coping with chronic pain requires reducing
   (a) exercise
   (b) pain complaints
   (c) knowledge about pain

20. Worry is
   (a) a necessary step to solving a problem
   (b) relaxing
   (c) prolonged thinking about possible events without acting on them
21. Managing stress involves changing one's
   (a) sex
   (b) neighbors
   (c) thinking, feeling and behaviors

22. What is the first step in problem-solving?
   (a) evaluate options
   (b) list options
   (c) define the problem

23. To decide which is the better of two options, it is best to
   (a) flip a coin
   (b) do a cost-benefit analysis
   (c) choose the option with quick results

24. Those in chronic pain will benefit most if their family members
   (a) are attentive and sympathetic to pain complaints
   (b) reinforce (encourage) "well" (healthy) behaviors
   (c) take over as many responsibilities as possible

25. The statement "I want to get rid of my pain; I can't stand it" is most
    likely the type of response from
    (a) a child
    (b) an adult
    (c) a parent