RELATIONSHIP OF MMPI PROFILE CLUSTERS TO PAIN BEHAVIORS

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The purpose of this study is to replicate and extend
earlier work involving cluster analysis of MMPI profiles
among persons with chronic low back pain. There are two
specific goals. The first goal is to demonstrate the exis-
tence in a new sample of four distinct and homogenous profile
clusters that have been found in previous research. The
second goal is to investigate the relationship of the four
profiles to the subjects, self-reported pain history and
response to treatment.

Subjects were 46 male and 46 female patients in a multi-
modal inpatient low back pain treatment program. All subjects
participated in an intensive inpatient treatment program
involving reduction and supervision of drug intake, physical
therapy, occupational therapy, biofeedback, behavior modifi-
cation, and individual and group therapy. All subjects
reported low-back pain at the L1 level or below.

On admission, all subjects were given the MMPI, Form R,
and the MMPI and the MMPI profiles were subjected to a cluster
analysis technique. In this procedure, each subject's pro-
file (using K-corrected T scores for the 10 basic and 3 validity
scales) was treated as a subgroup, and subgroups were

Successively combined so as to produce the fewest possible groups with a minimal estimate of within-group variance.

A visual examination of the within-group variance at each step clearly showed four distinct MMPI profile clusters. When the resultant profiles were visually matched to profiles demonstrated in previous research, and pattern similarity coefficients (r_p) obtained, the two sets were found to be significantly repeated ($p < .02$).

The subgroups found by the cluster analysis for the combined sample were then compared in terms of reported pain histories as measured by: (a) body pain drawings, (b) months in pain, (c) months of disability, (d) presence of a reported pain precipitant, (e) number of hospitalizations for pain, (f) number of back surgeries, and (g) whether previous treatments helped or failed. These variables were used to discriminate between the four groups with a multiple discriminant analysis procedure. One significant discriminant function was found ($p = .026$). This function loaded most strongly on months in pain and the presence of a reported pain precipitant. It was possible to correctly classify 40% of the subjects by predicting group membership with this discriminant function.

The four groups identified in the clustering procedure were then compared in terms of response to treatment. Changes in the pretreatment versus posttreatment measures were taken of: (a) range of motion, (b) pain estimate, (c) analgesic intake, and (d) time spent out of bed. At posttreatment, an estimation was made of each patient's proportion of goals

accomplished and percentage of physical improvement in treatment. These variables were used to discriminate between the four groups in a multiple discriminant analysis procedure. None of the three possible discriminant functions were significant.

This study concludes that four distinct MMPI profiles can be identified among chronic low back pain patients. Further, these profiles are the same for males and females, and are the same profiles found in previous research. These profiles are significantly related to subjects' history of behaviors in dealing with pain. However, no relationship to treatment response was found. It was inferred that the MMPI is of value in understanding the nature of patients' pain coping behaviors, but that further research is needed before any statements can be made regarding the utility of the MMPI in understanding their response to treatment.

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RELATIONSHIP OF MMPI PROFILE CLUSTERS TO PAIN BEHAVIORS

Of all possible forms of human suffering, none is so universal as that of pain. Pain is experienced at some time by almost all people, in almost all illnesses (Maltbie, Cavenar, Hammett, & Sullivan, 1978). Since the time of Aristotle, writers and thinkers have pondered the nature and significance of pain. Yet, despite progress in understanding other physiological and psychological mechanisms, pain remains a poorly understood phenomenon.

Although writers speak knowingly about pain, and patients report their pain symptoms with conviction, there is still no common understanding of pain that can be clearly communicated from one human to another. Pain remains a unique, personal experience that is as difficult to communicate as one's feelings on hearing a symphony or viewing a painting (Sternbach, 1968). Even so, its importance in daily life is such that there is a pressing need to subject pain to objective scrutiny.

Pain, occurring acutely, has great benefit. It serves to warn the person of disease, injury, or impending injury. It provides a source of motivation to avoid the danger or to seek medical help. A careful description of the onset, duration, location, time of occurrence, and provoking and and reliving factors of pain is a major diagnostic tool

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(Maltbie, Cavenar, Hammett, & Sullivan, 1978). This value is most dramatically seen by observing the consequences of not experiencing pain. People born without a sense of pain have been known to bite their tongues, to accidentally push out their own eyeballs, and to even face death by ignoring a serious wound (Melzack, 1973).

Pain, occurring chronically, looses its survival value and becomes a pathologic process in its own right (Bonica, 1977a). Chronic pain produces a loss of ability to function physically. Chronic pain places increased stress on both personal emotional adjustment and the patient's social and family systems. It can become a malevolent self-pertuating process leading to iatrongenic complications, repeated mutilating surgeries, depleted finances, and personal desperation.

Of all forms of pain known to man, none is more pervasive in modern society or more costly in both economic and human terms than low back pain. It has been estimated that 90% of the population over 50 years of age has had at least one episode of low back pain. One in 10 of these seeks medical care (Tio & Moya, 1978). In one pain clinic, 70% of all referrals were for treatment of low back pain (Sternbach, Wolf, Murphy, & Akeson, 1973a).

While human suffering cannot be measured in monetary terms, some idea of the importance of low back pain can be gained by looking at the available cost data. In a survey of inpatient facilities in California, Pheasant (1977) found

72,645 patients were admitted for backache in 1 year.
Based on these data, he estimated the national in-hospital
treatment cost to be $1.38 billion. Sixty percent of that
was spent on the 25% of the patients who had surgical treat-
ment. It has been estimated that the cost for all complica-
tions of pain, including outpatient care, loss of work produc-
tivity, and compensation payments could be as high as $50
billion annually (Bonica, 1977a).

The psychological and social cost of low back pain is
hard to evaluate, but it is possible to look at some of the
variables related to chronic low back pain. Patients with
low back pain are known to experience high levels of anxiety,
depression, and feelings of hopelessness and despair (Bonica,
1977b). Both patients and physicians express inward feelings
of anger and frustration as the pain continues (Maltbie,
Cavenar, Hammett, & Sulivan, 1978). Two thirds of all
chronic pain patients report decreased libido, and one-third
report deterioration in their marriages (Maruta & Osborne,
1978). The situation is especially telling when both the
therapist and the patient come to believe there is no end
to the pain. As one writer stated, "the longer I have been
involved in low back dysfunction, the more hesitant I am to
employ the term 'cure' in connection with the condition,"
(Finneson, 1977).

While pain is an extremely difficult concept to define,
perhaps the best effort so far is that of Sternbach (1968).

Pain is an abstract concept which refers to (1) a personal, private sensation of hurt; (2) a harmful stimulus which signals current or impending tissue damage; (3) a pattern of responses which operate to protect the organism from harm. These responses can be described in terms which reflect certain concepts, i.e., in neurological, physiological, behavioral, and affective "languages." (p. 12)

This definition has gained wide acceptance, and was used as a basis for this study. However, neither this definition nor any other currently available definition is fully adequate (Weisenberg, 1977).

Pain is both a sensation and an emotional-motivational concept. As a sensation, pain can be viewed as a warning system of actual or impending tissue damage. Such an approach to pain is used extensively in animal and laboratory studies. If pain is primarily sensation, it should vary in a positive relationship to the amount of tissue damage. However, in his classic study of men in combat, Beecher (1956) found that men wounded seriously in battle asked for far less narcotics for pain relief than did civilians with similar wounds. Further, men who showed little pain reaction to battle wounds greatly feared the pain of an injection at the aid station. There is also pain for which no apparent stimuli can be demonstrated. Some pain, such as causalgia, may persist long after the tissue wound has healed. It is clear that both psychological and physiological aspects

be considered as essential in the study of pain, but neither alone is sufficient to explain the nature of pain.

One of the most important tasks in the study of pain is to obtain some kind of measurement procedure. Unfortunately, this has proven nearly as difficult as the definition of pain. Numerous procedures have been developed using both analogue and clinical pain, but none has yet been found to be totally satisfactory.

Experimental pain has been created by a number of procedures falling under four general headings: mechanical, chemical, electrical, and thermal. Mechanical methods include the use of a pressure algometer or sphygmomanometer cuff to induce pressure pain. The pain is produced either by a sharp metal object or by pressure against a bony surface (Wolff, 1978). This procedure is not used much in current research. Chemical pain is most often induced using the Canthardian blister method or the ischemic method. In the blister method, a blister is developed by use of a plaster, and then various chemicals are applied to the sensitive area (Wolff, 1978). The ischemic procedure, introduced by Smith and Beecher (1969), is still in popular use. In this procedure, venous blood is drained from the extended nondominant arm by use of an Emarch bandage. A tourniquet is then placed on the arm and inflated to 250 mm Hg. The subject then waits 60 seconds, and performs 20 squeezes with a hand-spring. At this point, pain will begin to build

up slowly in the arm. This procedure is very commonly used (Sternbach, Deems, Timmermans, & Huey, 1977; Wolff, 1978).

Electrical stimulation has been used to induce pain. This has the advantage of being applicable to any body part. It is convenient and easily controllable. While muscle spasm can occur, tissue damage can be completely avoided (Weisenberg, 1977). However, subjects often describe the sensation produced in terms of "discomfort" rather than "pain."

The fourth approach to experimental pain is the thermal approach. Thermal pain can be induced either by the use of radiant heat produced by a lamp or laser (Wolff, 1978), or by cold. The former carries the risk of burn. The latter, along with electrical stimulation, is among the most widely used at present. Cold pressor pain is usually obtained by first stabilizing the skin temperature of the hand in a bath of water at body temperature, and then placing it into an ice water bath. This method has been found to produce relatively variable results (Wolff, 1978).

Several approaches have been devised to assess the "amount" of pain produced by the various techniques. Most, however, are a variation of three basic themes. The most common approach is to establish a threshold level at which pain is first experienced. The stimulus can then be continued until a level is found at which the subject cannot accept further stimulation, or the stimulus has reached maximum intensity (Weisenberg, 1977). A third measure commonly

used is simply to take the arithmetical difference between the threshold and tolerance levels. The latter is of particular interest to the clinician since some reserach (Wolff, 1978) has demonstrated it to have a high correlation with postoperative success.

While measurement of experimental pain is made fairly straightforward by the experienter's ability to control the nature, intensity, and duration of the stimulus, clincial pain requires a different approach. In a clinical setting, the clinician or researcher does not know the "true" stimulus intensity and may be uncertain as to the nature of the original stimulus.

Clinical pain can be measured by making use of a pain analogue as a comparison, asking the subject to match his subjective pain to an applied noxious stimulus (Wolff, 1978). This approach is rather cumbersome, however. Beecher (1959) introduced the use of direct scaling as a more economical alternative. In this approach, the subject rates his pain verbally along a numerical scale. Alternatively, the subject can rate the pain visually on what is termed a visual analogue scale. Rather than give a numerical value, the pain is rated by marking a straight line fixed by the extreme limits of possible pain. The visual analogue scale has been found to be more sensitive to changes following analgesic intake than verbal scales (Woodforde & Merskey, 1972).

Another avenue for clinical pain measurement that has come under recent scrutiny is the use of verbal pain

descriptors. The best known example of this is the McGill Pain Questionnaire developed by Melzack (1975) which contains 102 descriptors divided into three major categories of sensory, affective, and evaluative pain. The words are analyzed according to the numerical values assigned to the word, and the number of words chosen. While of recent development, this approach shows a lot of promise as a stable and sensitive measure of pain (Leavitt, Garron, Whisler, & Sheinkop, 1978; Wolff, 1978).

The procedure of matching clinical to experimental pain and the use of visual analogue scales are both examples of cross-modality techniques. More recently, though, an effort has been made to match verbal descriptors like those used by Melzack (1975) to responses in other nonverbal modalities. Gracely, McGrath, and Dubner (1978a, 1978b) matched verbal descriptors to handgrip force and to 7 line lengths varying in equal log steps. Through these procedures they were able to obtain ratio scales that showed very high correlations (.89 to .98) between groups and over time. This provides indirect support for both visual scaling procedures and the use of verbal descriptors, but suggests that even better results are possible with more sophisticated use of cross-modality procedures.

Numerous theoretical models have been put forward to explain pain. Some, such as the quale theory of Marshall (Melzack & Wall, 1965), have been abandoned. But two theories proposed at the turn of the century, the

specificity theory and pattern theory, are still being used in research. Recently two new theories, the gate-control theory and the biochemical theory, have been proposed.

Specificity theory, proposed in 1894 by von Frey, is based on the observation that there are two sets of free nerve endings associated with two pain qualities (Mountcastle, 1974). The smaller A-delta fibers are associated with short-latency pricking pain, while the larger C fibers are associated with long-latency burning pain. The major tenet is that there is a direct connection between the specific nerve ending and a pain center in the brain. Afferent impulses in these fibers are seen as necessary and sufficient to evoke a painful sensation. Pricking impulses are viewed as entering the dorsal cord, where they ascend through the anterolateral system to the thalamic centers and are then projected onto the somato-sensory areas of the cortex. Burning pain is conceptualized as being projected to different thalamic, hypothalamic, and cortical areas. The latter are seen as responsible for autonomic, affective pain reactions.

Specificity theory has been a major force in pain treatment (Clark & Hunt, 1971). However, it has also been subjected to considerable criticism. While there clearly are highly specialized receptors in the sensory system, there is no evidence that there is a special class of receptor-fiber units that are exclusively devoted to pain (Melzack & Wall, 1975). Likewise, while there are some specialized central nervous system functions in pain, it is not clear

that this represents a specific pain system. Lesions in the lateral spinothalamic tract or thalamus may abolish pain, but pain can also recur after the successful cordotomy (Nathan, 1963). Further specificity theory does not provide an explanatory mechanism for the role of cognitive factors in pain. The evidence suggests that there are many psychological variables present in the perception of pain (Hill, Kornetsky, Flanary, & Wilder, 1952). For example, specificity theory does not account for Beecher's (1959) classic findings of reduced or nonexistant pain sensations in a combat setting.

Goldscheider (Melzack & Wall, 1975) proposed the pattern theory of pain largely as a reaction against the specificity model. He rejected the idea of specific pain receptors, and instead proposed that stimulus intensity and central summation are the critical determinants. Pain is not seen as a specific sensory modality. Pain is simply a function of the number of nerve endings stimulated and the intensity of the stimulation. Modifications of the pattern theory in recent years have accepted the idea of a modulation of the impulse pattern during transmission of central nervous system input such as affect, attention, and past experience (Fordyce, 1978).

While the pattern approach has proved of great value in its ability to handle specific clinical phenomena, it does not account for all the known data. Particularly, it does not incorporate the known data regarding receptor-fiber specialization (Barber, 1959; Melzack & Wall, 1975).

The biochemical theory of pain is a new porposal that places emphsis on the importance of metabolic factors (Lindahl, 1974a, 1974b). This theory emphasizes the stimulus rather than the receptor. Pain is seen as caused by an elevated hydrogen ion concentration bathing a nerve ending. Lindahl found that the pain from an absess could be augmented or reduced by the injection of an acid or alkaline saline solution. It was also noted that ulcers, painful tumors, and ischemic pain are all associated with the presence of an acid pH. In one study, Lindahl (1974b) was able to get a 60% improvement of arthritis-like pain by changing the pH level in joints.

While the biochemical theory shows great promise, it is a new theory that has not yet been adequately tested. Further, the theory says nothing about the central processing factors in pain, nor about the relationship between pain and affect. Finally, the theory does not yet encompass other known biochemical factors in pain. It is known that the level of endorphins (opioid peptides) in the cerebral spinal fluid varies in relation to the presence of pain and its chronicity (Almay, Johnsson, von Knorring, Terenius, & Whalstrom, 1978). It is also known that serotonin, histamines, and prostiglandin E play a role in pain perception (Cannon, Leibeskind, & Frenk, 1978).

The gate-control model proposed by Melzack and Wall (Melzack, 1973; Melzack & Wall, 1975) is another theory that has come into prominence recently. This approach is of

particular interest to psychologists, because it provides a clear and specific role for psychological factors. Melzack (1973) rejects the specificity theory, but accepts the idea of some special functions. Specialization is present at the receptor sites, with fibers such as the A-delta and C fibers, that react to specific types and degrees of energy. However, the approach does not imply that stimulation of the fibers always produce pain, as in the specificity model, because other factors also enter into the sensation of pain.

Melzack and Wall (1975) postulated that the transmission of nerve impulses from afferent fibers to spinal cord transmission (T) cells is modulated by a spinal gating mechanism in the substantia gelitanosa. In addition to impulses from different types of afferent fibers, the gating mechanism receives input from descending fibers. A specialized system of fibers, the Central Control Trigger, activates selective cognitive processes that influence the modulating properties of the spinal gating mechanism.

According to this model large and small afferent fibers interact at the gate-control. Large diameter fibers tend to inhibit transmission, while small diameter fibers have a facilitative effect at the gate mechanism. Thus, the large fibers will initially fire the T-cells but will then be inhibited. The small fibers reduce presynaptic inhibition and thus result in the exaggeration effect on subsequent input.

In addition, to the afferent barrage, descending impulses from the brain also modulate the gating system,

either increasing or reducing the activity in spinothalamic pain pathways. Finally, the perception/action cycle is triggered when the T-cell output reaches a critical level. Thus, the central cells experience a temporal summation which finally results in pain perception.

Melzack and Wall (1975) also propose the presence of three elements to the pain experience at the central processing level. The three factors believed to be present are the sensory-discriminative, motivation-affective, and cognitive-evaluative processes. The sensory processing occurs with the selection and modulation of the sensory input through projection to the ventrobasal thalamus and somatosensory cortex. Fibers projecting to the reticular formation, medial intralaminar thalamus and the limbic system are believed to be the basis of the motivation and affect that trigger the organism to act. Finally, neocortical and higher central nervous system processes are thought to exert evaluative control over both the sensory and motivational systems. All the systems interacting together could then influence both the gating mechanism and the motor mechanisms, leading to an overt pain response.

The gate-control theory has received some criticism on neurophysiological grounds. Dyck, Lambert, and O'Brien (1976) found evidence that fiber size does not bear a relationship to the facilitation or inhibition of pain perception. For example, patients with Frederick's ataxia have a reduction in large-diameter fibers, but do not experience pain. Further,

both large and small fibers have been found to produce depolarization, which is inconsistent with the presence of the presynaptic gating mechanism (Franz & Iggo, 1968; Vyklicky, Rudomin, Zajal, & Burke, 1969). From the psychological point of view, however, these apparent theoretical problems only place additional emphasis on the possible importance of central processing mechanisms, a stance which has been taken by Melzack (Melzack, & Wall, 1975) in response to his critics.

Considerable research has been performed that appears to support Melzack's contention that cognitive and affective factors are important mediators of pain perception and response. Supporting evidence comes not only from traditional personality measures but from studies of cognitive and social factors as well.

Age has been found to significantly affect pain perception. While some studies found no age-related differences in threshold (Hardy, Wolff, & Goodell, 1952; Woodrow, Friedman, Siegelaub, & Collen, 1975), most research seems to support the idea that threshold increases with age (Chapman & Jones, 1944; Clark & Mehl, 1971, Procacci, Rozza, Buzzelli, & Della Corte, 1970). Sex has also been found to be a significant variable, with most studies reporting that women have a lower tolerance for pain. However, no consistent evidence of sex differences in pain threshold has been found (Della Corte, Procacci, Bozza, & Buzzelli, 1965; Hardy, Wolff, & Goodell, 1952; Merskey & Spear, 1964; Notermans & Tophoff, 1975; Woodrow, Friedman, Siegelaub, & Collen, 1975).

At a social level, a number of variables have been found
to influence the pain experience. Schachter (1959) discovered
that first-born and older children were less tolerant of pain
that later-born children, although Gelfand (1963) could not
confirm this. A number of researchers have found a relation-
ship between family size and pain response. Sweeney and Fine
(1975) found that subjects from smaller families are more
reactive to experimental pain than subjects from large
families. However, Merskey (1965b) and Gonda (1962), in
looking at patients who already had clinical pain, found a
reverse relationship, with patients from large families more
likely to complain of persistent pain. In addition to
pointing to the importance of social factors in pain, the
latter studies also suggest that pain tolerance as measured
in a laboratory and pain behavior as observed in the hospital
may not be equivalent behaviors.

Race and culture have both been found to be significant
covariates of pain perception. Woodrow, Friedman, Siegelaub,
and Collen (1975) performed a pain tolerance test of 41,119
Kaiser health plan patients using pressure to the Achilles'
tendon. They found that there was a significant racial
difference, with whites tolerating the most pain. Orientals
were found to have the least pain tolerance and blacks an
intermediate degree of pain tolerance.

These data do not make it clear, however, whether the
racial differences are related to some underlying biological
difference or are a function of social and cultural variations.

As pointed out by Melzack (1973), it is known that there is a wide variation in culturally based responses to pain. While childbirth is accepted as very painful in Western society, other cultures have been observed where women show virtually no distress in childbirth. Such a view is also supported by experimental data. Earlier studies, such as that of Chapman and Jones (1944), have been criticized for poor methodology, and more recent studies (Lambert, Libman, & Poser, 1960; Merskey & Spear, 1964; Winsberg & Greenlick, 1967) did not show cultural differences in pain tolerance. However, Zborowski (1952) did find consistent cultural differences in how various groups reacted to pain. Tursky and Sternbach (1967) and Sternbach and Tursky (1965) found similar results, and also showed that cultural attitudes were significantly related to the degree of pain tolerance. Thus, it appears that ethnicity by itself might not be a determinant of pain, but that culturally induced attitudes and response styles may influence pain perception and pain behaviors.

Such a view was supported by Weisenberg, Kreindler, Schachat, and Werboff (1975) in a study of black, white, and Puerto Rican dental patients. In a multiple-discriminant analysis, two anxiety and two attitudinal measures mediated differences in pain tolerance among these ethnic groups. Trait anxiety and dental (situational) anxiety were the two anxiety measures, while willingness to deny or avoid pain and willingness to get rid of the pain were the attitudinal variables.

Depression and anxiety appear to be important factors in pain experience. Pain has often, but not always, been found to be correlated with depression (Merskey, 1965a, 1965b; Spear, 1967). When both depression and pain are present, pain has been thought to act as a substitute for both depression and anxiety (Pilling, Branhick, & Swenson, 1967). In a recent study of differential effects of acupuncture on pain (Toomey, Ghia, Mao, & Gregg, 1977), 40 pain patients were divided into responders and nonresponders. Responders were found to be not only less passive and to have shorter pain duration, but to be less depressed. Thus, while depression has not been demonstrated to be a "causative" factor in pain perception, it does appear to be a major factor in pain tolerance and recovery from a pain state when both pain and depression are present.

Anxiety, like depression, may be handled by using pain as a substitute. In addition, anxiety appears to be a critical factor in the initial pain response.

Anxiety has been repeatedly implicated as a factor in pain (Sternbach, 1968). Anxiety has been primarly viewed from one of two frameworks. One view is that anxiety occurs when the organism is aroused by a sudden aversive nociceptive sensory barrage. Pain represents such an event. Then, even if the pain continues, there may be a reduction in anxiety and arousal as the organism adapts to the situation (Chapman, 1978). Such a view would explain reserach demonstrating

relatively low anxiety in chronic pain patients (Sternbach, 1974).

Most pain theorists think of anxiety as an intrinsic response within the limbic system or autonomic nervous system. Within that framework, however, anxiety has been defined and researched in widely varying ways by different researchers.

Another approach for viewing pain that is of particular interest is the distinction between trait and state anxiety. Trait anxiety has been defined as an enduring characteristic response tendency, i.e., a self-description of restlessness, tendency to sweat easily, or to worry frequently. State anxiety is defined as a response to a particular stimulus situation. Cattell and Scheier (1958) found these types of anxiety to be relatively independent.

Most research relating pain to anxiety has centered on the expression of trait anxiety. Such research has consistently shown a tie between anxiety and pain. Schalling and Levander (1964) compared high and low anxiety delinquents, finding that high anxiety delinquents were significantly more sensitive to pain. Hare (1965) found similar results.

In research with psychiatric patients with chronic problems with anxiety, high rates of pain reports have been found. Merskey (1965a, 1965b) found persistent pain to be especially common in the neuroses, but to be rare in the psychoses. In particular, it appears that persons in whom anxiety is associated with anger and resentment have problems with pain (Funkenstein, King, & Drolette, 1957).

In viewing anxiety as a trait, Lynn and Eysenck (1961) related Eysenck's theory of personality and pain tolerance. They predicted that, since anxiety was thought to be a conditioned fear response, pain tolerance should be positively correlated with extraversion and negatively correlated with neuroticism. They had found in other research that extraverts do not condition as well as introverts, and neurotics were found to have high levels of autonomic lability and anxiety. The research results were consistent with the theoretical perspective, as was another study of pregnant women by Eysenck (1961) and other research by Halsam (1967). However, some other researchers were unable to confirm these findings (Leon, 1974; Levine, Tursky, & Nichols, 1966).

Attempts have also been made to identify specific response styles that might be related to pain reactions. Petrie (1967) introduced the idea of augmentors-reducers. Augmentors were defined as those who have a tendency to see a given stimulus as greater, reducers as those who tend to perceive less stimulation. Dinnerstein, Lowenthal, Marion, and Olivo (1962) found support for this distinction with electric shock. A somewhat similar distinction was made between sensitizers and reducers by Goldstein (1973). Some investigators found that reducers had higher initial pain tolerance but lost tolerance with the passage of time (Davidson & Bobey, 1970; Neufeld & Davidson, 1971).

Another concept that has received scrutiny is that between field-dependence and field-independence. Field-independent persons tend to judge a stimulus by disregarding its background, while field-dependent persons make judgements in comparison to the stimulus background. Sweeney and Fine (1975) found high pain reactivity associated with field independence. This was supported by Adler, Gervasi, and Holzer (1973) in situations of low anxiety, but was not confirmed in a study by Adler and Lomazi (1973).

A number of cognitive variables have been investigated and found to influence the pain response. These include the subject's attention, perception of control, and how the subject interprets the pain. As noted by Melzack (1973), attention to the stimulus adds to the perceived intensity of the pain stimulus. When attention is reduced or shifted from the stimulus, as in hypnosis, subjects can be cut or burned and not report pain (Barber, 1959). In contrast, Hall and Stride (1954) found that even the appearance of the word pain can augment a pain response when the person has focused his attention on a potentially painful experience.

In addition to shifted attention, hypnosis makes use of suggestion. Beecher (1959) found that severe pain can be relieved by placebo when the subject is given instructions to expect pain relief. Barber (1963) supported the idea that suggestion was a critical aspect of pain relief by hypnosis. Orne (1974) found that hypnotic suggestion works directly on the patient's verbal report of pain. Both McGlashen,

Evans, and Orne (1969) and Sachs (1971) found that hypnotic suggestion alters the subjective pain reports without necessarily altering the physiological response. It appears, then, that hypnotic suggestion operates at the cognitive-affective level to alter the way the patient perceives and responds to the pain stimulus.

Another cognitive factor that seems to play an important role in pain perception is the subject's perception of control. If the subject feels he can predict or control the stimulus, he may "decide" to tolerate a higher level of noxious stimulation than if he felt he could not control or predict the stimulus. Such a view has been supported in terms of both pain threshold and pain tolerance by a number of researchers (Bandler, Mandaras, & Bem, 1968; Geer, Davison, & Gatchell, 1970; Pervin, 1963; Staub, Tursky, & Schwartz, 1971). Likewise, in experiments in which subjects were given prior information to anticipate severe pain, it was found that such information resulted in a reduced level of reported pain (Jones, Bentler, & Petry, 1966; Kanfer & Goldfoot). Jones, Bentler, and Petry (1966) suggested that the presence of uncertainty about the anticipated pain provokes anxiety, which leads to an augmentation of the pain experience.

One particularly useful way to look at control is the internal-external distinction of Rotter (1966). He held that a person can attribute control to either an internal event or to something external to himself, and that this would be a major determinant of how a subject responds to a given

task. Weiner (1974) took a similar view, but added the
dimension of stability (i.e., probability of a predictable
outcome) to the concept of locus of control. Both authors
viewed these as situation-specific factors. For example, a
person might make one kind of attribution in relation to his
pain, but another in relation to his job situation. As a
result Wallston, Wallston, Kaplan, and Maides (1976) made
a specific adaptation of Rotter's Locus of Control scale to
the medical environment, labeled the Health Locus of Control
scale. Craig and Best (1977) found that internals could
tolerate more pain than could persons with an external rating.
The only other research known to this author applying this
concept to pain is that of McKinlay (1978). He subjected 48
females to a cold pressor stimulus after he administered the
Health Locus of Control scale. No relationship was found
between pain response and the Health Locus of Control scores.
However, he questioned whether the negative results were due
to sampling error or to failure of the scale. Levinson (1973)
may have one possible explanation for the negative findings.
In a factor-analytic approach, she found that the external
dimension actually splits into two attributions--that of
chance and that of control by powerful others. It may be
that to speak only of external versus internal control is an
over-simplication of the control attribution problem.

Another aspect of cognitive control of pain is the
patient's control or perception of control over his own
bodily processes. Imagery of one's internal body processes

has been recognized as important to physiological responses
(McMahon, 1977). White (1978) found that with positive
imagery of self-control, a patient could exercise control
over his rate of salivation. Similar imagery has been demon-
strated in research with primary dysmenhorrea, dental pain,
and chronic pain, to enable the patient to experience control
over the pain and to have a reduction in perceived pain
(Drummond, White, & Ahston, 1978; Horan, Layng, & Pursell,
1976; Levenduski & Pankratz, 1975; Tasto & Chesney, 1974).
It appears that the successful use of relaxation, biofeedback,
meditation, hypnosis, and autogenic training (Barber & Hahn,
1962; Budzynski, Stoyva, & Adler, 1970, Budzynski, Stoyva,
& Mallaney, 1973; Chaves & Barber, 1974; Gannon & Sternbach,
1971; Gessel & Alderman, 1971; Lehrer, 1972; Melzack & Perry,
1975; Orne, 1974; Sargent, Green, & Walters, 1973) may have
as an underlying denominator the presence of positive imagery
by the patient and a cognitive perception of self-control
over bodily function (Frumkin, Nathan, Prout, & Cohen, 1978).

Medical Aspects of Low Back Pain

Low back pain is generally defined as pain originating
at or below the lumbar area, with possible referred pain to
this area. The back is actually a quite complex structure,
and there are a number of factors which can contribute
physiologically to the experience of pain in that region.
The entire upper torso is balanced in a small (7 cm. at the
L1 level) "stack" of bones interspersed with softer fibrous

tissues. These are balanced by a network of ligaments and muscles which must maintain the spine in a proper structural and mechanical relationship. This system permits man to walk erect, while allowing movement of the torso over a wide range. In addition, the spine encloses the spinal cord and a bundle of nerves communicating between the brain and the lower portions of the body, and between the spinal cord and the body. Thus, the spine serves not only a mechanical support, but as housing and protection for the spinal cord.

The nerve supply from the lumbar area is of particular interest, since it is through this sytem that pain impulses travel. The spinal cord itself extends from the first cervical vertebra to the second lumbar vertebra, terminating in the conus medullaris and the filum terminale, which extends to the first segment of the coccyx (Chusid, 1976).

There are five pairs of spinal nerves in the lumbar region and five pairs in the sacral region. These involve sensation and control from the gluteal region down. In addition, there are parasympathetic nerves routed from the sacral region to the colon, kidney, bladder, and sex organs. The lower portion of the sympathetic chain also has white communicating rami to the lumbar region and gray communicating rami to all the spinal nerves.

Thus, the low back must be seen not only as the structural support for the torso and head, but as the main pathway for communication with the rest of the body (Chusid, 1976; Fisk, 1977). As a result of the complexity of the low back

structure, the task of medical diagnosis is quite difficult and complex, even without taking into account psychological variables. A great number of factors can contribute to the perception of pain. Further, because so many nerves pass through this region, the patient may experience pain in other places (referred pain) due to disorders present in the lower back (Anderson, 1977; Gross, 1977; Stotz, 1977).

Brown (1975) identified six general classes of nonsocio-psychological types of pain syndromes of the spine. Spondylogenic pain includes pain due to some disorder of the spinal column. Among the causes of spondylogenic pain is discogenic pain, or pain due to a disorder of the disc. This can represent an acute disc infection or a mechanical and inflammatory irritation of nerve endings in degenerative disc disease. There can also be irritations of the attaching ligaments or an inflammatory condition. Unlike discogenic pain which is worsened by exercise, osteoarthritic pain is relieved. Mechanical insufficiency occurs when there is undue stress on the relaxed ligaments or their attachments.

Neurogenic pain can occur either due to nerve root compression within the spinal canal or entrapment within the foramen. Such pain can also be related to the presence of a tumor, which is quite difficult to distinguish from other conditions.

Osteogenic pain may also be related to the presence of tumors, but can also be associated with the inflammation of

rheumatoid spondylitis. In elderly patients, this type of pain is often seen in disuse osteoporosis.

Vascular pain is due to impairment of the blood supply to the lower back and can represent the presence of an aneurysm or vascular insufficiency. The insufficiency can also be associated with a variety of metabolic disorders.

Viscerogenic pain is actually not due to a disorder of the back but because of the structure of the nervous system, may be perceived as in the back by the patient. Plueral disorders, peritoneal disorders, and problems in the genitourinary system fall in this category.

Finally, pain may be iatrogenic, or arising secondary to some previous medical treatment. Arachnoiditis, spinal stenosis, pseudoarthrosis, and neural adhesions all may have an iatrogenic basis, and may in some instances account for the failure of the technically successful treatment to bring relief of pain.

Treatment of low back pain is as varied as the possible causes. Treatment can range from simple massage and physical therapy to facet injections or surgery. While it is beyond the scope of this paper to discuss the various treatments in detail, it would be worthwhile to review the literature on treatment outcome, for this provides one of the rationales for inlcuding psychological factors among the causes of pain to be assessed clincially.

Whether intervention by traditional psychological methods is viewed as successful depends upon how the data are evaluated.

In the case of lumbar disc surgery, it has been found that 75% or more of the eligible patients have significant pain relief, and that when reoperation is performed on those who failed to find relief, 28% of the remaining portion experience pain relief (Law, Lehman, & Kirsch, 1978). Thomalske, Galow, and Ploke (1977) emphasize that this type of treatment is consistently successful provided that the physician is experienced and is meticulous in removing diseased material.

However, Rothman and Booth (1975) point out that while relief does occur in successful spinal fusions, it may also occur in unsuccessful fusions (pseudoarthrosis). They also found that there is not consistent pain relief in some successful surgeries, which may reflect the presence of other back disorders (especially in older patients).

When pain is measured only by initial outcome, or by self-reports, when, data strongly supports the value of surgical intervention. When the patient's observed behavior is considered, however, less positive results are seen. Surin (1977) studied 116 patients treated for lumbar disc prolapse over a period from 10 years before surgery to 10 years postsurgery. No difference was found in the number of days of sick leave. In fact, the number of sick-days increased steadily over the entire period studied. The author suggested that low back pain must be treated as both a physiological and psychological phenomenon. Ignelzi, Sternbach, and Timmermans (1977) conducted a 3-year follow-up on 54 patients treated in a pain unit. A comparison was made

between surgical patients and nonsurgical patients in terms
of estimated pain activity level and analgesic intake.
Study results revealed that both groups did equally well,
showing lower levels of pain and drug use, with increased
activity. However, surgical patients were more likely to be
readmitted. While this result certainly does not mean surgery
should be abandoned as a treatment, it does suggest that
appropriately selected patients respond as well to psycho-
logical treatment of their pain as do patients treated for
an identified underlying physical cause.

Psychological Aspects of Low Back Pain

Akeson and Murphy (1977) and other writers have criti-
cized work in low back pain as being based on an inadequate
fund of research data. This allegation is applicable to
the psychological and medical aspects of low back pain.
However, in the last few years there has been much published
regarding low back pain patients. While the data base is
still weak and the clinical applications uncertain, some
research trends have emerged reflecting consistencies among
personality variables seen in low back pain patients.

Gentry, Shows, and Thomas (1974) studied demographic
and personality factors among 56 low back pain patients.
Their impressions were that low back pain patients tend to
experience initial symptoms at a relatively young age (33.5
years), and about 15% could not identify a definite precipita-
ting event. The patients tended to have long, steady work
histories, but at low-paying blue collar or clerical jobs.

They started work at the average age of only 16. They
tended to be married, to have less than a high school educa-
tion, and to have several children (2.7 per patient). In
turn, they had also come from large families (5 children)
and were usually later born. Half had some kind of financial
compensation available at the onset. Most had a history of
other physical problems or trauma, and/or had at least one
other family member with a major physical disability, often
(23%) low back pain. The authors inferred from these data
that low back pain patients were "set up" in the sense of
having unmet dependency needs early in life and no opportunity
for emotional or economic gratification until the pain onset.
At the same time they had a model for learning the pain
behaviors.

While the incidence of low back pain is high in psychia-
tric patients, not all emotionally disturbed individuals are
equally susceptible to pain symptoms. Delaplaine, Ifabumayir,
Merskey, and Zarfas (1978) found that female psychiatric
patients were more likely to have pain symptoms than men,
and such symptoms were most often associated with anxiety
or personality disorders rather than schizophrenia, organic
brain syndrome, or situational disturbance.

Although the data are limited, some information is
also available pertaining to observed patient behaviors in
a treatment setting. Most obviously, chronic pain patients
rarely obtain relief from medication, although they frequently
demand it. Addiction and depression are often present, and

the patients often succeed in getting the physician to operate in ill-advised situations because of the extreme degree of "public suffering" (DeVaul & Zisook, 1978). The most difficult to treat medically are often the ones who most resist psychological approaches, removal from medications, and even discharge from the hospital (Swanson, Swenson, Maruto, & Floreen, 1978). The chronic low back pain patient is often certain that he cannot be cured, but behaves as though the more firm this conviction, the less willing he is to forego further medical intervention.

There seems to be a systematic pattern of descriptors applied to pain by low back pain sufferers. The McGill Pain Questionnaire was developed on the assumption that patients would systematically evalaute pain on three dimensions: sensory, affective, and evaluative. The factor analytic work of Leavitt, Garron, Whisler, and Sheinkop (1978) partially supported this multidimensional model. They found low back pain patients described pain along seven factors, all of which were sensory or affective in nature. It is not clear yet, however, if there is a pattern unique to low back pain patients or common to all types of pain responses, or if there may be unique differences in emphasis on the use of the different descriptor dimensions.

Several attempts have been made to describe the low back pain patient in terms of personality characteristics. The most commonly used device has been the Minnesota Multiphasic Personality Inventory (MMPI). Sternbach, Wolf, Murphy, &

Akeson (1973a; 1973b) accumulated data on 117 admissions to the low back clinic of the University Hospital, San Diego. The composite profile showed a quite-consistent pattern, with striking elevations on scales Hypochondriasis (Hs), Depression (D), and Hysteria (Hy). Further, the configuration of the three scales was not the clear, "psychomatic--V" associated with conversion hysteria. They inferred that the patients are undergoing a psychophysiological reaction with depression, and that the physical symptom of pain does not "bind" the affect as in hysteria. In another study of 56 chronic low back pain patients at Duke University Medical Center, Gentry, Shows, and Thomas (1974) found the same three scales to be elevated, but that the configuration did conform to the conversion--V pattern with Depression lower than the other two scales. In addition, they found that the F and K scales formed a "faking good" picture, reflecting denial of emotional problems. Unlike Sternbach et al., Gentry et al., concluded that denial and repression were used as psychological defenses against the affect.

Thus, while there is some uncertainty as to the exact configuration of the Hypochondriasis, Depression, and Hysteria scales of the MMPI, the research consistently shows that low back pain patients have elevations on these personality scales. It is less clear, however, whether this is unique in some way to low back pain patients as opposed to other medical patients. Spergel, Ehrlich, and Glass (1978) collected MMPI data on 46 patients with rheumatoid arthritis,

and compared the obtained profiles with research data previously gathered on low back pain patients, pulmonary disease patients, and multiple sclerosis patients. All groups had elevations of the first three clinical scales of the MMPI, but the patient groups did not significnatly differ from each other. The authors concluded that instead of a unique trait/ disease match, there may be a chronic disease profile common to all disabled patients.

If there is not a personality profile unique to chronic low back pain patients, there is still a question as to whether all have a homogeneous profile or whether there may be differing personality types associated with the different ways of coping with the disease. Louks, Freeman, and Calsyn (1978) examined 74 MMPI profiles of low back pain patients for the incidence of 6 profile types; "conversion--V" without defensiveness, "conversion--V" with defensiveness, dependent, anxious, psychotic, and normal. They found all six types represented in their sample.

Rather than make an a-priori assumption on the existence of a specific personality type, Bradley, Prokop, Margolis, and Gentry (1978) collected MMPI data on 548 low back pain patients and performed a multivariate clustering analysis in an effort to find replicable, homogeneous profile subgroups. They found three male subgroups and four female subgroups, with three of the four groups common to both sexes. The first subgroup, containing 176 patients, was characterized by elevations on scales Hypochondriasis, Depression, and

Hysteria, similar to that found by Sternbach et al. (1973a, 1973b). The authors felt these patients were more likely to be controlled by respondent rather than operant stimuli in the experience of pain. The second group (N = 232) also had elevations on the same three scales but to a lesser degree, and unaccompanied by any other elevation except scale K. These were seen as more likely to resemble psychiatric patients who are experiencing conflict regarding emotional dependency yet are highly reluctant to admit conflict. The third profile subgroup (N = 63) was characterized by elevations on the first three scales with an added elevation on scale Schizophrenia. The authors saw these patients as depressed, preoccupied with somatic concern, and emotionally isolated. The final subgroup consisted of 77 females, and was charcterized by a classic "conversion--V" (elevation of the first three clinical scales with Depression less elevated than Hypochondriasis or Hysteria). These patients were seen as having learned to live with their pain by deriving satisfaction from their roles as invlaids. However, this research has not yet been replicated or tested to see if these profile subtypes are of significance in understanding the etiology or probable treatment response of low back pain patients.

If, then, chronic low back pain patients do have personality profile patterns distinct from patients without physical disorders, it would be of interest to know if there is any directional relationships between the physical symptom and personality type. Sternbach (Sternbach, 1977;

Sternbach, Wolf, Murphy, & Akeson, 1973b) compared the pro-
files of 19 acute and 98 chronic (6 months or more of pain)
low back pain patients. He found a distinct elevation of
Hypochondriasis, Depression, and Hysteria in chronic as
opposed to acute patients. Sternbach (1977) inferred that
while acute pain is accompanied by the "fight or flight"
reaction of sympathetic system dominance with anxiety, the
chronic pain patient has autonomic habituation with "vege-
tating," depressive symptoms. He felt that the profile
differences reflect a collapse of psychological defense
mechanisms with continuing pain. He also noted that the
presence of litigation issues seems to be associated with
a further augmentation of the psychophysiological components
of the MMPI elevations.

Most explanations of the "chronic disease" MMPI profile
have centered on discussions about the collapse of psycho-
logical defense mechanisms and speculation about predisposing
developmental problems in handling dependency needs. Caldwell
and Chase (1977) suggested that it was not necessary to look
for unique psychodynamic explanations of the Hypochondriasis,
Depression, Hysteria elevation; that the development of the
profile over time can be explained strictly in terms of
reinforcement mechanisms in the disease experience without
reference to antecedent psychologicl difficulties. They
suggested that any patient experiencing pain is motivated
by two factors; (a) to find relief from the immediate dis-
comfort of pain, and (b) to avoid future discomfort. They

suggested that changes of the MMPI with time represent a progressive increase in the fear of pain and suffering, and body vulnerability. Patients first experience a pain-fear reaction with central nervous system arousal. As time passes, this is augmented by efforts to avoid further injury by holding the body rigid, as well as defensive and conforming behavior, since any type of stress or physical "arousal" adds to the pain experience. With time, the patient's reactions become self-reinforcing, since it represents a type of learned avoidance. The patient's extreme avoidance behavior stance directly blocks recovery, because the patient has become slow and cautious, even immovable, and is reluctant to engage in the physical exercises and strengthening activities necessary for medical recovery.

While several studies have been performed with the MMPI, few efforts have been made to describe low back pain patients by use of other types of personality descriptors. Ransford, Cairns, and Mooney (1976) used a pain drawing similar to that developed by Melzack (1973). They found that chronic pain patients "drew" their pain difficulty differently than acute pain patients, showing a tendency to draw a medically inconsistent pain pattern, to "expand" the body areas in which pain is marked, and to exaggerate the amount of pain experienced. Further, they found a very strong relationship between these trends and elevations on the Hypochondriasis and Hysteria scales of the MMPI.

Another approach that has been tried is the Pain Apperception Test (PAT) developed by Petrovich (1958). This is a projective device consisting of a series of cards similar to the Thematic Apperception Test. According to Zeisat and Gentry (1978), this test has provided inconsistent results and shows poor concurrent validity.

A potentially more promising approach is represented by efforts to look at control attributions. Craig and Best (1977) used Rotter's internal-external model to test pain tolerance to electrical shock. They found that internals manifested greater pain tolerance. However, these findings have not yet been applied to clinical pain, nor have efforts been made to determine if the experience of pain has an influence on a patient's subsequent control attributions.

Efforts have also been made to develop specific pain descriptor scales. Thomas and Lyttle (1976) developed a checklist composed of already known demographic and psychological (MMPI) variables. Research with this scale seems to demonstrate the validity of already known characteristics of chronic low back pain in that the scale was successful in correctly identifying 80% of chronic low back pain patients. Sternbach, Wolf, Murphy, and Akeson (1963) developed a Health Index scale consisting of items borrowed from the Cornell Medical Index, Zung's Self-Rating Depression Scale, and items describing the way patients interact with doctors. They found that low back pain patients were more likely to see

themselves as invalid and dependent than patients with arthritic pain. They also described the pain as having a greater impact on their lives and were more hostile and frustrated with the doctor-patient relationship. They concluded that low back pain patients have allowed pain to make a greater difference to them than arthritic patients, and are more likely to be engaged in a self-perpetuating "pain-manship" game with physicians in which they constantly seek, even demand, medical relief, but expect or even firmly believe that such relief will not be found.

A question that has been repeatedly asked of psychologists by their medical colleagues is whether it is possible to differentiate functional from organic pain by utilizing personality tests. Some medical tools are available such as the Pentothal pain test, Amytal interview, and differential spinal block (Borwn, 1975) and these are used in addition to a careful history and examination. Despite such available diagnostic tools, the surgeon is too often faced with a technically perfect operation which has no impact on the patient's pain complaint.

Merskey and Boyd (1978) examined life history factors and found some significant demographic differences between chornic pain patients with known functional versus known organic pain. They found that the patients with functional pain had more family disturbance in childhood, more premorbid personality problems and more neurotic traits than patients who had an organic cause for the pain. It appears possible

that factors might be found that would enable the clinician to determine to which group a patient belongs.

Several systematic efforts have been made to categorize patients into organic/nonorganic pain groups, primarily using the MMPI. The oldest approach still in use was devised by Hanvik (1951). He compared the MMPI profiles of 30 functional and 30 organic low back pain patients. He found that the profiles of the two groups were different in his sample, and that patients could be differentiated with 75% correct placement. As a result he developed the Lb scale for differentiating organics from nonorganics.

More recently, Pichot, Perse, Lebeaux, Dureau, Perez, and Ryckewaert (1972) studied 84 French patients with functional back pain. By comparing item responses with results from a standard normal group they were able to produce a 63 item scale, labeled the DOR, that has the potential for differentiating organics from normals.

Both the DOR and Lb scales were developed by empirical examination of item differences. Another approach has been to make use of factor analysis. Overall, Hunter, Butcher (1973) performed a factor analysis of the MMPI-168. They found 5 factors: Somatization, Low Morale, Depression, Psychotic Distortion, and Acting Out. Calsyn, Spengler, and Freeman (1977) attempted to use the somatization factor to differentiate a group of 58 veterans with low back pain. They were able to do so successfully 75% of the time. In

another group of 48 patients, they were able to do so 83% of the time.

While the somatization factor scale shows promise, there is not yet much supporting research for its validity. However, several studies have been performed using the DOR and Lb scales. Louks, Freeman, and Calsyn (1978) compared a group of 74 low back pain patients on the Lb and DOR scales. They found that both scales were significnatly different between the functional and mixed versus the organic patients. In attempts to use the scales separately, while significance was found, the "hit" rates have been too low to be of clinical utility. But when the two scales are used together, it has been possible to correctly classify patients at rates from 74% to 83% (Calsyn, Louks, & Freeman, 1976; Pichot, Perse, Leabeaux, Dureau, Perez, & Rykewaert, 1972).

While some authors have been enthusiastic about the potential for being able to identify patients with signif- icant functional pain using the MMPI, other reserachers have been more cautious. Towne and Tsushima (1978) also obtained a 75% correct classification using the Lb and DOR scales. However, they pointed out that this still did not represent statistical significance. In addition, they were unable to distinguish low back patients from other psychosomatic or psychiatric patients. While agreeing that functional and organic low back pain patients have different MMPI profiles, McCreary, Turner, and Dawson (1977) found so much overlap between the two groups that they felt it would be unwise to

attempt to place an individual patient in one group solely on the basis of personality data.

Although several authors found significant if not always meaningful differences between functional and organic low back pain patients on the MMPI, Fordyce, Brena, Halcomb, DeLateur, and Loeser (1978) found no MMPI differences between members of a sample of 100 functional and organic low back pain patients, although the MMPI did correlate with the number of hours spent walking each week. Likewise, Sternbach, Wolf, Murphy, and Akeson (1973b) were unable to find any significant differences between 81 patients with positive organic findings and 36 patients without positive findings.

It appears, then, that while there may be some potential for use of the MMPI to distinguish functional and organic low back pain patients, the data are now inconsistent. A possible explanation for the situation may lie in the failure of most studies to control for length of time in pain. Sternbach, Wolf, Murphy, and Akeson (1973b) found that patients in pain have increasing elevations on the MMPI over time, regardless of etiology. The studies available to this point may not have been comparing equivalent groups in terms of time in pain.

While considerable research effort has been focused on finding personality differences between patients with organic versus fucntional pain, little research has been published relating personality variables to treatment outcome. While not specifically focused on treatment outcome, Fordyce,

Brena, Holcomb, DeLatear, and Loeser (1978) found a relationship between five MMPI scales and hours spent walking per week. Patients who walked less responded to the MMPI as more depressed, having more somatic complaints, and as being angry and hypersensitive.

In a study of postoperative success of 130 candidates for cymopapain injection therapy, Wiltse (1975) administered the MMPI, Cornell Medical Index, and the Quick Test. He found a significant negative correlation between MMPI scales Hypochondriasis and Hysteria, and outcome. Patients with T-scores below 54 had a 90% chance of a good or excellent outcome, while only 10% of patients with scores over 85 were rated as having good or excellent response to treatment. On the other hand, physician ratings of the desirability of intervention bore no relationship to symptom improvement.

It appears that it may be possible to identify psychological variables that relate to how well the patient responds to medical treatment for pain, regardless of the organic or functional nature of the disorder. However, the amount of research available is inadequate to ascertain what personality factors are most important, or to ascertain how reliably such variables can be applied in predicting outcome.

While research on the definition and response of pain as a psychological phenomenon is still underway, there is a rapidly rising interest in the application of psychological apporaches to the treatment of low back pain. Most approaches focus on dealing with the affective response to pain, alteration

of the person's identity and life style as a pain patient,
or the secondary gains associated with the pain symptom
(Sternbach, 1974; Chapman, 1977; Bonica, 1977). The most
successful such approaches utilize a multimodal approach.

Several different multimodal approaches to pain have
been proposed in recent years (Gottlieb, Strite, Koller,
Madorsky, Hockersmith, Kleeman, & Wagner, 1977; Newman, Seres,
Yospe, & Garlington, 1978; Turk & Miechenbaum, 1976). Most
are similar to that of Turk and Meichenbaum (1976) in incor-
porating three phases of treatment, either sequentially or
concurrently. Turk and Meichenbaum worked within the assump-
tion of the gate-control theory and the position that cogni-
tive events affect pain perception. Their model focuses
on education, skills acquisition, and application. In the
educational phase, the patient is given a lay conceptualiza-
tion of the pain experience in which there is a possibility
of control. In the skills acquisition phase, the patient is
trained to use relaxation techniques, attention focusing
exercises, imagery, and self-instruction. In the application
phase, modeling, rehearsal, and role-playing are used to
train the patient to accept an increased tolerance of pain.
Khatami and Rush (1978) also developed a three part model
with an emphasis on both interpersonal and intrapersonal
aspects of pain. Symptom control is accomplished by teaching
the patient to control the pain through biofeedback, relaxation,
and autohypnosis. Stimulus control is accomplished by the
use of cognitive modification to alter the patient's beliefs

about the pain. Finally, the family members are encouraged to change the reinforcers operating to maintain the pain behavior at an interpersonal level. This program is particularly noteworthy since it is performed on an outpatient basis for 1 hour each week, while most multimodal pain programs operate on an inpatient basis.

Most psychologically oriented treatment programs have been successful in reducing the patient's perception of pain and increasing activity levels. Gottlieb, Strite, Koller, Madorsky, Hockersmith, Kleeman, and Wagner (1977) found that 57 patients had improved physical function and 59 patients were able to return to work out of 72 low back pain patients treated in a 45 day inpatient program. Khatami and Rush (1978) were able to get significant change with their 36 hour, weekly outpatient program. Most importantly, in an 80 week follow-up of a similar inpatient multidisciplinary program (Newman, Seres, Yespe, & Garlington, 1978), the authors found that positive changes in physical function, use of analgesics, and vocational activity had been maintained.

Purpose

The major purpose of this study was to replicate and extend the work of Bradley, Prokop, Margolis, and Gentry (1978). The first goal was to demonstrate the existence in a new sample of the four MMPI profile clusters identified by those authors in their sample of low back pain patients.

The second goal was to build on their research by investigating the relationship between these profile clusters

and other measures of patient behavior. This goal was accomplished in two parts. In the first part, the relationship of the profile clusters to body pain drawings and historical and demographic pain factors was explored. In the second part, the relationship of the profile clusters to change in functional correlates of pain during participation in a multimodal treatment program was examined. It was hoped that it would be possible to predict which patients were most likely to respond to a multimodal, psychologically oriented treatment program.

The specific purpose was to determine whether the MMPI profile clusters, if present, bore a significant relationship to measures of pain experience or treatment response. Pain experience variables included the body pain drawing (Ransford, Cairns, & Mooney, 1976), duration of pain, duration of vocational disability, presence of a pain precipitant, number of hospitalizations, number of back surgeries, and response to previous treatments. Treatment response variables included range of motion, pain estimate, medication intake, time out of bed, amount of physical improvement, and goal attainment.

Hypotheses

The hypotheses tested in this research were as follows:

1. The MMPI profile clusters identified by Bradley, Prokop, Margolis, and Gentry (1978) will be replicated based on two criteria: (a) the total sample will be divided into four significantly different MMPI profile clusters by use of a cluster analysis procedure; (b) the four clusters will not

significantly differ from those identified by Bradley et al. (1978) when compared by use of pattern similarity coefficients.

2. Subjects in the four profile clusters will have significantly different pain histories as measured by: (a) body pain drawing scores, (b) duration of pain in months, (c) duration of vocational disability in months, (d) presence or absence of a clear pain precipitant, (d) number of previous hospitalizations for pain, (f) number of previous back surgeries, and (g) the ratio of previous treatments helping pain to treatments exacerbating or not affecting pain.

3. The four profile clusters will be associated with significantly different responses to treatment by a multi-modal pain treatment program, as measured by pre/post changes in: (a) pain estimate in percent, (b) mean range of motion in degrees, (c) medication intake in morphine equivalents, (d) daily time out of bed in hours, as well as (e) goal attainment as estimated by the patient on a 5 point scale, and (f) amount of physical improvement at discharge as measured by the physical therapist.

<div align="center">Method</div>

Subjects

Subjects were 46 female and 46 male patients in a multi-modal low back pain treatment program. Subjects were selected at random from those admitted in the last 12 months.

The subjects were patients in a 2-week intensive in-patient treatment program. The program included 24-hour a day

supervision of medication intake, with daily participation in physical therapy, occupational therapy, biofeedback (EMG), individually tailored behavior modification, and individual and group psychotherapy. The only requirement of participation in the program was the presence of low back pain (pain at the L1 level or below).

Instruments

All subjects were administered the MMPI, Form R, as part of the initial admission procedures, following the standard test instructions. Test analyses were based on the K-corrected T scores for the 10 basic and 3 validity scales. All subjects were given the body-pain drawing of Ransford, Cairns, and Mooney (1976) using standardized written instructions as part of the routine admission procedures. Scoring was performed by the standard method to yield a single index of physiological consistency.

All subjects were asked to complete the Patient History Form as part of the initial admission procedures (Appendix A). Patient History Form questions included how many months the patient had been in pain, how many months the patient had been unable to work, how the pain began (scored for whether the patient reported a specific incident in time), how many times the patient had been hospitalized for pain, how many times the patient had back surgery, what previous treatments had helped the pain, and what previous treatments had not helped the pain. The latter two measures were combined as a ratio of treatments helping to treatments not helping.

All subjects were given an ischemic pain test as part
of the initial admission procedure. In this procedure, venous
blood was drained from the extended nondominant arm by use of
an Emarch's bandage. A tourniquet was then placed on the arm
and inflated to 250 mm HG. The subjects then performed 20
squeezes with a handspring. The amount of time passing in
seconds was recorded until the patient (a) sensed pain,
(b) felt the pain was equal to his back pain, and (c) could
not tolerate the pain. A pain score was derived as a ratio
of the seconds elapsed until the sensation equaled the back
pain to the seconds elapsed until the pain was unbearable. Each
subject was then asked to make a subjective pain estimate,
in percentage points, using the ischemic pain test ratio as
a reference point. A second pain estimate was made by each
patient at discharge again using the results of the ischemic
pain test as a reference point (Appendices B and C). The
initial pain estimate was then subtracted from the final
pain estimate to yield a change score.

Weekly measures of range of motion, in degrees, were
taken on all subjects by the physical therapist. These were
recorded for hip flexion, knee flexion, knee extension, and
ankle dorsiflexion on each side. The mean initial perform-
ance was then subtracted from the mean performance at discharge
(Appendix D).

Each subject's daily analgesic intake was recorded in
equivalent units of morphine. One equivalent unit of morphine
was defined as 10 mg. of morphine each 4 hours, or 60 mg.

total daily intake (Fordyce, Fowler, Lehmann, DeLateur, Sand, & Trieschmann, 1977). A change score was derived by subtracting the dosage level of the first day from the final dosage level.

Hours spent out of bed daily were recorded for each subject. A change score was derived by subtracting hours up on the first day from hours up on the last day.

Each subject was asked to develop a list of treatment goals in consultation with the staff physician. When the subject was ready to leave, he was then asked to rate his goal achievement in consultation with the staff physician. This was done on a 5-point scale from no goal met (1) to all goals accomplished (5) (Appendix C).

When treatment was completed, the physical therapist estimated the amount of overall improvement (Appendix C). This estimate was recorded on a 5-point scale from 1 (no change) to 5 (no remaining physical limitations).

Procedure

All questionnaires were administered as part of the initial admissions procedures, following the standard instructions for each test. A specific order of test administration was not observed. Physical measures were taken as part of the routine intake and discharge procedures, and the results recorded.

Statistical Analysis

Data for each hypothesis were anlayzed separately. For Hypothesis 1, one marker profile for each of the profile

clusters identified by Bradley, Prokop, Margolis, and Gentry (1978) was added to the subject pool to provide a seeded clustering technique. A cluster analysis of the combined subject pool was performed using the North Texas State University cluster analysis procedure (adapted from Veldman, 1967). This procedure is a hierarchical clustering method which progressively groups profiles so as to maximize the average between group distance while minimizing the average within group distance. This program initially treats each subject as a subgroup. An error potential matrix for all subject profile pairings is developed from the sum of the squared differences between corresponding scores in the profiles, divided by the number of subjects in the potential subgroup. The subgroups are progressively merged until all subjects are members of one of two subgroups. The researcher then examines the total within group variation at each step to find the minimum number of subgroups that can be selected before a large increase in within group variance occurs. This is done visually by selecting the last step at which a minimal increase in error variance is observed, and before a large increase in error variance occurs. The amount of congruence between the resultant MMPI profiles (minus the seeded profiles) and the profiles reported by Bradley et al. (1978) was tested using the pattern similarity (r_p) statistic developed by Cattell (Cattell, 1949; Horn, 1961).

For Hypothesis 2, a multiple discriminant function was performed using the SPSS (Nie, Hull, Jenkins, Steinbrenner,

& Bent, 1975) <u>Discriminant</u> program. A direct analysis of all discriminating variables was obtained. The clusters identified in the first analysis were treated as groups, while the questionnaire items and body drawings were treated as discriminating variables.

For Hypothesis 3, a multiple discrminant function analysis was performed using the SPSS (Nie, Hull, Jenkins, Steinbrunner, & Bent, 1975) <u>Discriminant</u> program. A direct analysis of all discriminating variables was obtained. The clusters identified in the first analysis were treated as groups, while the change variables were treated as discriminating variables.

Results

Following collection of data and preparation for analysis, the 3 hypotheses were tested as described in the section on methodology. Each hypothesis is restated and the results of the statistical testing given in this chapter.

Hypothesis 1

The first Hypothesis has 2 parts. First, Hypothesis 1 predicts that 4 significantly different MMPI profiles will be identified by cluster analysis. Secondly, this hypothesis predicts that these profiles are not significantly different from those found by Bradley, Prokop, Margolis, and Gentry (1978).

The first part of Hypothesis 1 appears to be supported. An examination of the within group variance of the cluster analysis shows that 4 profile clusters are the minimum that can be identified before there is an escalation in the rate

of increase of total within group variance. This is apparent from an examination of Figure 1. It can be seen that there is little change in the amount of error variance as the number of groups is reduced from 10 to 6. There is a slight increase in the amount of change in error variance between 6 groups versus 5 groups, and 5 groups versus 4 groups. However, there is a drastic increase in error variance as the number of groups is reduced from 4 to 3. Thus, the minimum number of groups that can be used while maintaining a minimum distance between the profile scores within each group is 4. This is the same number of clusters identified by Bradley et al. (1978), who found 3 clusters among both male and female cohorts, and a fourth cluster among females. The means for each of the 4 profile clusters on each of the 13 MMPI scales are presented in Appendix E and in tabular form in Table 1. The profiles found in this study are labeled A($\underline{n}$ = 18), B($\underline{n}$ = 25), C($\underline{n}$ = 9), and D($\underline{n}$ = 40).

Results of one-way analyses of variance for each of the 13 scales are presented in Table 1 as an additional check on the stability of the profiles. The univariate $\underline{F}$ ratios are significant for all 13 scales. Levels of significance for the univariate $\underline{F}$ ratios vary between $\underline{p}$ = .02 and $\underline{p}$ = .001. All but one are significant beyond the .01 level. Based on these data, it seems clear that the between group variance is greater than the within group variance for all 13 scales.
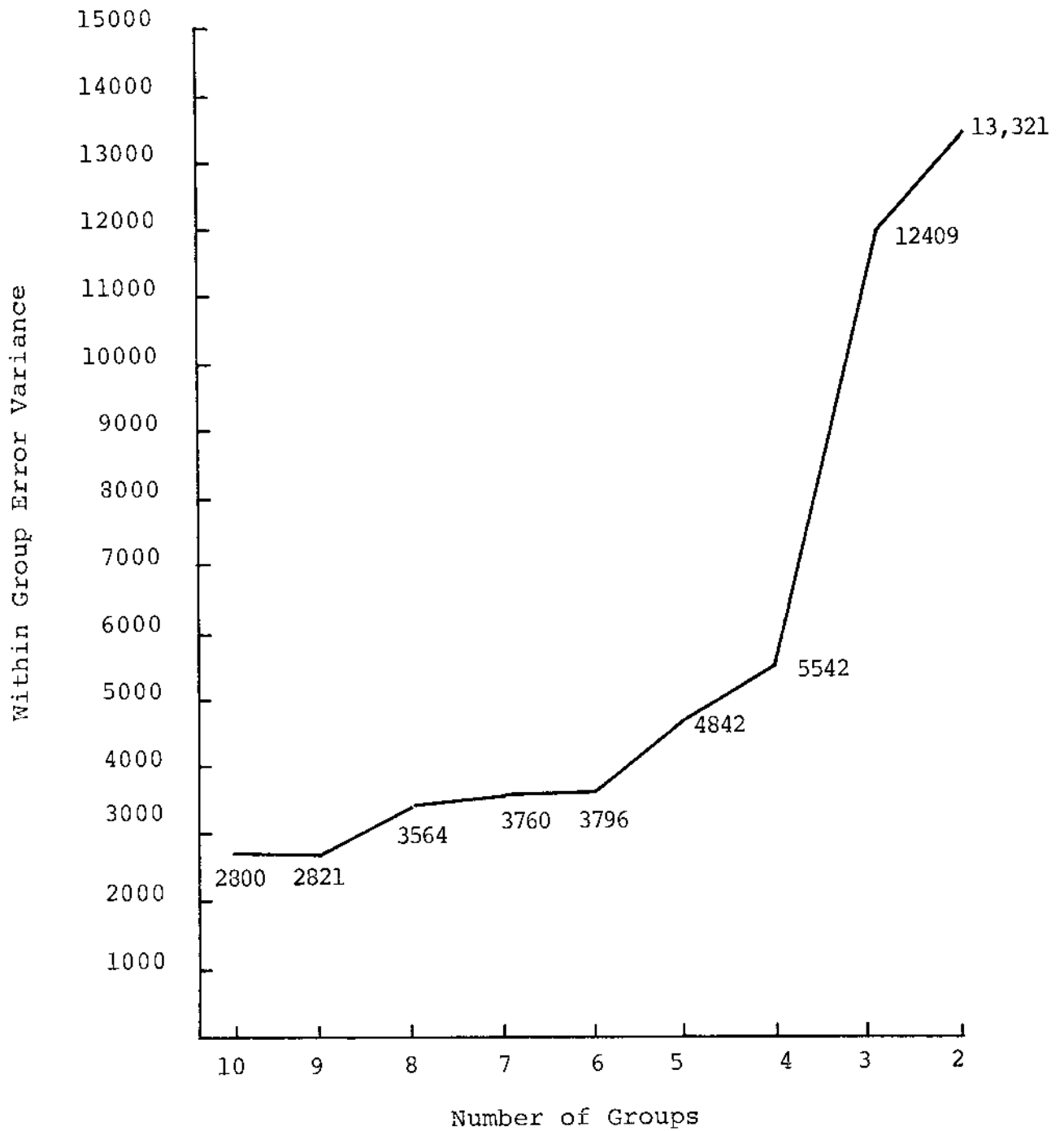
Figure 1. Within group error variance for ten groups to two groups cluster and analysis

Table 1

MMPI T-Score Mean (K Corrected) and Univariate
F Ratios for Combined Study

MMPI Scale	A	B	C	D	$\underline{F}$
Lie	55.78	50.44	46.89	54.40	3.832*
F	60.28	49.12	75.78	54.70	31.986***
K	50.56	54.96	45.78	57.60	6.268***
Hypochondriasis	85.44	60.72	80.33	75.55	33.839***
Depression	83.94	61.52	81.67	65.60	23.693***
Hysteria	79.44	59.76	74.00	73.68	26.076***
Psychopathic Deviate	63.00	52.56	77.89	63.50	15.627***
Masculinity-Femininity	57.61	48.68	60.78	53.88	4.807**
Paranoia	58.67	52.00	76.89	54.38	20.626***
Psychasthenia	73.44	54.00	85.00	59.50	65.631***
Schizophrenia	71.61	51.04	94.33	60.13	73.530***
Hypomania	52.00	53.52	73.89	59.35	12.034***
Social Introversion	65.72	53.96	59.44	47.40	19.369***

$\underline{df}$ = 3.88

*$\underline{p}$ < .02
**$\underline{p}$ < .01
***$\underline{p}$ < .001

The second part of Hypothesis 1 is also supported by the
data. The results of visually matching the profiles from this
study to the combined (male and female) means of those
reported by Bradley et al. (1978) are presented in Appendix E.
Since the profiles from the current study are designated
by the letters A, B, C, and D, their visually matched

counterparts from the Bradley et al. (1978) study are identi-
fied by the same letters, but with a prime mark added (A', B',
C', D'). The visual match between the pairs of profiles
appears to be quite close. The results of testing the
strength of the relationship by the r_p statistic are shown
in Table 2. All of the visually matched profiles correlate

Table 2

Pattern Similarity Coefficients (r_p) for
Bradley et al. (1978) versus Current Profiles

Bradley et al.	Current Study			
	A	B	C	D
A'	.694***	.267	-.106	.810***
B'	-.005	.920***	-.428***	.672***
C'	.665***	-.201	.453**	.191
D'	.229	.598***	-.306*	.909***

$*p < .05$
$**p < .02$
$***p < .01$

significantly. While other significant indices occur in
the table, the visually matched profiles yield the strongest
relationships with the exception of the match between profiles
A' and D, and profiles A and C'. Profile D correlates more
with profile D' than with profile A', even though the relation-
ship with profile A' is strong. Similarly, profile A
correlates better with profile A' than C' even though there is
also a strong correlation with C'.

An additional check on Hypothesis 1 was performed by dividing the sample into independent male and female subsets. The statistical procedure for Hypothesis 1 was independently replicated for each subset. As in the original analysis, four profile clusters are identifiable for both males and females. Bradley et al. (1978) had found only three of the four profiles among males. A one-way analysis of variance of the MMPI scales across groups demonstrates greater between group variance than within group variance on most scales. The only nonsignificant F ratios are for scales Lie and K among males, and Lie and Masculinity-Femininity among females.

Hypothesis 2

Hypothesis 2 predicts that the four profile clusters can be significantly differentiated in terms of pain history as measured by: (a) body pain drawing scores, (b) duration of pain in months, (c) duration of vocational disability in months, (d) presence or absence of a pain precipitant as reported by the subject, (e) number of hospitalizations for pain, (f) number of previous back surgeries, and (g) the ratio of previous treatments helping pain to treatments exacerbating or not affecting pain.

The results of the multiple discriminant analysis, using the discriminating variables to predict group membership, are presented in Table 3. One of the three possible discriminant functions is accompanied by a significant Chi-square. The function accounts for 55.64% of the total variance. If individual subjects were re-assigned to one of the four

Table 3

Standardized Coefficients of Pain History Variables
with Three Discriminant Functions, and Tests of
Significance for Combined Study Groups

Variable	Discriminant Functions		
	I[a]	II[b]	III[c]
Duration of Pain (months)[e]	-.408	.293	.588
Duration of Vocational Disability (months)	-.283	-.291	-.711
Presence of Clear Precipitant (reported)[e]	.779	-.250	-.035
Number of Hospitalizations[e]	.156	-.424	d
Number of Back Surgeries	.157	-.551	d
Help/Hurt Treatment Ratio	-.028	.320	-.101
Pain Drawing Score	.247	-.149	-.081

[a]Chi-square (21) = 35.348, $\underline{p}$ = .026; percentage of variance 55.64; canonical correlation = .470.

[b]Chi-square (12) = 16.217, $\underline{p}$ = .182 (not significant); percentage of variance = 33.28; canonical correlation = .381.

[c]Chi-square (5) = 4.208, $\underline{p}$ = .520 (not significant); percentage of variance = 11.07; canonical correlation = .231.

[d]undefined.

[e]Wilk's Lambda (Chi-square test) for variable, $\underline{p}$ < .05.

groups at random, it would be expected that 25% of the subjects would be correctly placed by chance. However, when the coefficients derived from the first discriminant function are used to place subjects into one of the four groups, 40.22% of the subjects are placed in the correct group (Table 4). This discriminant function is more successful in placing

Table 4

Results of Group Prediction from the First Discriminant
Function for Pain History Variables, In Percentages

Actual Group Membership	Predicted Group Membership			
	A	B	C	D
A	11.1	5.6	38.9	44.4
B	0	16.0	56.0	28.0
C	0	11.1	66.7	22.2
D	7.5	5.0	25.0	62.5

Percent of "grouped" cases correctly classified: 40.22.

individuals appropriately into group C and D than groups A
and B.

The results of a one-way analysis of variance of the
discriminating variables in relation to the four profile clus-
ters are presented in Table 5 in order to assist in understand-
ing the relationships among the variables. Four of the seven
univariate F ratios are significant beyond the .05 level. The
number of months of vocational disability, ratio of treatments
helping to treatments not helping, and the body pain drawing are
not significantly different among the four groups. There is
a large absolute difference in means for the number of months
of disability but this is accompanied by a large within-group
variance.

Table 6 includes the results of making pair-wise com-
parisons among the means where the overall F ratio is signif-
icant. Groups A and B differ in the number of hospitalizations

Table 5

Mean Scores of Pain History Variables
and Univariate F Ratios

Variable	A	B	C	D	$\underline{F}$
Duration of pain (months)	34.11	12.76	26.22	44.08	3.097*
Duration of Vocational Disability (months)	50.39	12.16	22.56	32.38	2.409
Presence of Clear Precipitant (reported)[b]	1.67	1.76	1.78	1.40	3.852**
Number of Hospitalizations	5.56	1.89	3.78	3.70	3.568**
Number of Back Surgeries	3.33	.36	2.22	1.58	2.735*
Help/Hurt Treatment Ratio	2.18	1.38	1.00	1.23	.929
Pain Drawing Score	1.50	1.29	1.63	1.30	.553

[a]$\underline{df}$ = 3.88

[b]No = 1, Yes = 2

*$\underline{p} < .02$
**$\underline{p} < .01$

and number of back surgeries reported. Groups B and D differ significantly in the number of months in pain and whether group members reported a clear precipitant to their pain. Groups C and D are also different in terms of whether group members reported a clear pain precipitant.

Hypothesis 3

The third hypothesis predicts that the four profile groups differ in response to treatment as measured by changes in (a) pain estimate in percent, (b) mean range of motion in

Table 6

Pair-Wise Comparisons by Group of Pain History Variables
(Duncan's Multiple Range Test for Significant F Ratios)

Variable	A vs. B	A vs. C	A vs. D	B vs. C	B vs. D	C vs. D
Duration of Pai (months)	21.35	7.89	9.97	13.46	31.32**	17.86
Presence of Clear Precipitant (reported)	.09	.11	.27	.02	.36*	.38*
Number of Hospitalizations	3.67**	1.78	1.86	1.89	1.81	.08
Number of Back Surgeries	2.97*	1.11	1.75	1.86	1.22	.64

*p < .05
**p < .01

degrees, (c) medication intake in morphine equivalents, and
(d) time out of bed each day in hours, as well as (e) amount
of goal attainment as rated on a 5-point scale, and (f) amount
of physical improvement as rated on a 5-point scale.

The results of testing this hypothesis with a multiple
discriminant analysis, using the discriminating variables to
predict group membership, are presented in Table 7. None of
the three possible discriminant functions are accompanied
by a significant Chi-square. Therefore, Hypothesis 3 is not
confirmed. This negative result is further supported by
the results of the one-way analysis of variance. None of the
univariate F ratios are significant (Table 8). Since the
overall F ratios are nonsignificant, no pair-wise mean comparisons are given.

Table 7

Standardized Coefficients of Response to Treatment Variables
with Discriminant Functions, and Tests of Significance
for Combined Study Groups

Variable[d]	Discriminant Functions		
	I[a]	II[b]	III[c]
Change in pain estimate (percent)	-.596	.185	.805
Change in time out of bed daily (hours)	-.522	.029	-.578
Change in Drug Use (morphine equivalents)	.081	.365	-.399
Change in Mean Range of Motion (degrees)	-.283	-.193	.357
Amount of goal attainment (rating)	-.872	-.351	.078
Amount of physical improvement (rating)	.827	-.435	.229

[a]Chi-square (18) = 18,262, p = .438; (not significant) percentage of variance = 62.41; canonical correlation = .356.

[b]Chi-square (10) = 7.022, p = .723; (not significant) percentage of variance = 32.21; canonical correlation = .264.

[c]Chi-square (4) = 1.032, p = .905 (not significant); percentage of covariance = 5.38; canonical correlation = .111.

[d]Wilk's Lambda for all individual variables not significant by Chi-square test.

The results of two further analyses of the data used in Hypotheses 3 are presented here in order to gain more insight into possible explanations for the negative findings. The first analysis involves repeating the procedure for Hypothesis 3 with the four pretreatment measures associated with each of the four change scores. The results of this

Table 8

Mean Scores of Response to Treatment Variables
and Univariate F Ratios

Variable	Groups[ab]				$\underline{F}$
	A	B	C	D	
Change in pain estimate (percent)	-11.22	-18.20	-7.22	-20.35	1.221
Change in time out of bed daily (hours)	4.78	4.56	2.33	3.25	1.480
Change in drug use (morphine equivalent)	- .09	- .15	- .08	- .11	- .725
Change in range of motion (degrees)	8.42	10.14	6.31	7.78	.765
Amount of goal attainment (rating)	3.94	4.42	3.44	4.00	1.493
Amount of physical improvement (rating)	2.83	3.33	2.88	3.28	1.621

[a]$\underline{df}$ = 3, 88.

[b]all are nonsignificant.

multiple discriminant analysis are given in (Table 9, Appendix F). This analysis yields one significant discriminant function, accounting for 67% of the variance. When the coefficients derived from this discriminant function are used to predict group membership, and it is assumed that there is an equal expectancy that a subject could be placed in any one group by chance, it is possible to classify only 29.67% of the subjects (Table 10, Appendix F). This is only slightly better than the 25% correct classification rate expected by chance alone.

The results of a one-way analysis of variance for the four pretreatment measures are presented in Table 11 (Appendix F). Only one of the four pretreatment measures is associated with a significant univariate F ratio, pretreatment pain estimate. When pair-wise comparisons are made for this variable, differences appear with two comparisons (Table 12, Appendix F). Groups A and B are significantly different, and groups B and C are significantly different.

In addition to the analysis of pretreatment variables, data relating to the same variables at posttreatment are included here, along with the two additional variables that were taken only at discharge (amount of goals met and amount of physical improvement). When six posttreatment scores are used and a multiple discriminant analysis is performed, no significant discriminant functions emerge (Table 13, Appendix F). However, when a one-way analysis of variance is performed, there is a significant univariate F ratio for one of the posttreatment measures, posttreatment pain estimate (Table 14, Appendix F). When pair-wise comparisons are made for this variable, differences appear with all but two of the possible comparisons (Table 15, Appendix F). Group A is significantly different in mean posttreatment pain estimate from groups B and D. Group B differs from group C, and group C differs from group D.

Discussion

The same four profiles reported by Bradley et al. (1978) are present in the sample of low back pain patients studied

in the present report. All four profiles are present among
both males and females, while Bradley et al. (1978) only
observed profile D' among females. The four profiles are
also associated with significant differences in time since
the pain started, number of hospitalizations, number of back
surgeries, and whether the subjects reported a clear pain
precipitant. However, no significant differences are
observable in how the four groups respond to treatment.

The current study was successful in replicating the
MMPI profile subgroups delineated by Bradley et al. (1978).
The match was present not only for the total sample, but
when a separate clustering procedure was performed for male
and female subjects, the same four profiles emerged. The only
inconsistencies with this statement appear in two of the
pattern similarity coefficients. The first is a high similarity
coefficient between profiles A' and D, although the coefficient
for D versus D' is stronger than this value. The second is
between profiles A and C', but again, the coefficient for
A versus A' is higher. The reason this might occur is best
seen by a visual examination of the profiles (Appendix E).
While the profiles are visually unique in pattern, all
represent relative elevations above the normative mean for
the test of a $\underline{T}$ score of 50. Further, there are commonalities
between the profiles in terms of which scales are elevated.
Profiles A and D in particular involve elevations of the first
three clinical scales of the MMPI. Further, while C' bears
a significant relationship to profile A, profile C has a

negative relationship to A' (Table 2). This may be because profile C', as with profiles A and D, involves some elevation of the first three clinical scales, and while it shares with profile C additional elevations on scales Paranoia (Pa), Psychasthenia (Pt), Schizophrenia (Sc), and Hypomania (Ma), the elevation is not so pronounced for scale C' as for scale C. While the configuration is different, profile A (but not A') shares with profile C' an elevation on scales Pt and Sc. Thus, it is quite reasonable that some intercorrelations would occur in the pattern similarity matrix. While some mathematical interrelationships may exist, the visual match of the profiles from this study to their counterparts in the Bradley et al. (1978) study is quite striking, and clearly supports the hypothesis that the four profiles are replicable.

Hypothesis 2 is also supported by the research data. The first discriminant function extracted is statistically significant. This function (Table 2) appears to load most heavily on the presence of a clear precipitant (positively) and the duration of pain in months (negatively). The probability of a subject belonging to any one group is a function of the distance in time from the onset of pain and the subject's likelihood of identifying the specific circumstances for the pain onset. It may be that this represents a time function, although some kind of denial or emotional coping function could also be involved in the subject's ability to recall the specific circumstances in which the pain began.

While the first discriminant function is 40% successful in classifying subjects into the appropriate profile clusters, it can be seen from Table 4 that it is much more successful with groups C and D than groups A and B. From an examination of the loadings (Table 3) and mean scores (Table 5), it appears that this discriminant function is a linear relationship that is able to distinguish reliably between groups C and D because patients in group D tend to have the highest scores on the duration of pain variable, while group C subjects have the highest scores on the presence of a clear precipitant variable. Because of the strong loadings of these two variables, groups A and B may tend to be misclassified into groups C or D based on their response to these two items. The failure to find an additional significant discriminant function that would allow a multidimensional distinction between groups A and B versus C and D may suggest that other variables not included in this study need to be reviewed. An insufficient degree of power may also have been available in this study to find all possible relationships among the variables included in the study.

The four profile clusters can also be viewed descriptively in terms of the high and low scores on the one-way analysis of variance (Table 1, 5, 6). Profiles A and D have in common a pronounced elevation on the first three clinical scales of the MMPI (Hs, D, and Hy). While profile B is much less elevated, it also has some elevation of these

scales. By contrast, profile C is elevated on all scales, but with relatively less emphasis on the first three scales and more emphasis on the remaining scales.

In discussing profile A', Bradley et al. (1978) suggested that persons with this profile are more likely to be influenced by respondent conditioning, and that they are more likely to maintain their attention to physical symptoms than other subjects. This is consistent with the information in this study. Profile A subjects experienced a significantly greater number of back surgeries and hospitalizations than profile B subjects, and had a greater absolute mean on these variables than any of the other groups. It is also interesting to note that this group was the only one whose mean period of vocational disability was greater than their mean period in pain. One could speculate that they have either intermittent episodes of pain, or other physical complaints that predate the pain experience. This seems consistent with the profile interpretation given by Bradley et al. (1978).

Profile D is similar to profile A in having major elevations on the first three clinical scales, but differs in configuration, with the first three scales forming a "V" pattern, commonly referred to in clinical use as the "conversion-V." This profile has been historically associated with conversion hysteria, and was characterized by Sternbach (1974) as representing persons who have adapted to their roles as invalids by focusing on a single pain symptom. He believed these persons are more likely to respond to treatment than

persons with the A profile. In the present study, this group
reports a higher duration of pain than the other three groups,
and is least likely to report a clear pain precipitant. This
is in particular contrast to profile B. These findings are not
inconsistent with Sternbach's view, but would appear to be
particularly interesting if related to Caldwell and Chase's
(1977) interpretation of the same profile. They suggested
that elevations on these scales represent a coping reaction
to the pain experience, as the patients attempt to avoid any
autonomic situations of either a physiological or psychological
nature that might exacerbate the pain experience. If this
explanation is correct, then persons with this profile might
be individuals who have been in pain for longer lengths of time,
and who have, as a consequence, developed a strong emotional
defense posture. If this explanation is valid, a longitudinal
study should reveal an increase in the first three scales
as the pain experience continues.

Profile B subjects have a similar configuration to
profiles A and D, but without a major elevation. These
subjects differ from groups A and D in their behavior history
as well. In contrast to profile A, these subjects have had the
least number of back surgeries and hospitalizations. In
contrast to profile B, they have been in pain the least amount
of time. Bradley et al. (1978) suggested that persons with
this profile may be highly defended individuals who are reluc-
tant to acknowledge unmet dependency needs. However, in view
of their lack of pain behaviors over an extended period of

time, this profile group might be related developmentally to the explanation of profile D suggested by Caldwell and Chase (1977). If this explanation is correct, this profile might represent individuals who have been in pain for shorter lengths of item, and who have yet to develop the strong emotional defense posture of profile D, or the somatic preoccupation of profile A. If the Bradley et al. (1978) explanation is correct, these subjects would be likely to hold onto their pain symptoms as an acceptable way of meeting unmet dependency needs. If the latter explanation is correct, subjects with this profile should be relatively responsive to pain treatment.

Profile C is not accompanied by any extreme scores on the discriminating variables. Based on a visual inspection of the means (Table 5), it can be seen that subjects in this cluster have the highest scores on the pain drawing and are least likely to have reported benefit from previous treatment, but neither of these are statistically significant. Group C differs from the others in having what is often termed a "floating" profile, with elevations on most scales. This profile is also the one most likely to be accompanied by deficiencies in reality testing. Bradley et al. (1978) suggested that these persons are emotionally isolated and more severely disturbed than persons in the other groups. This group may have the least predictable pain behaviors of the four presented here.

Based on the interpretations suggested by Bradley et al. (1978), it had been predicted that subjects in the four groups would show different responses to the multi-modal pain treatment program. This hypothesis is not supported, either in terms of the discriminant function analysis or the analysis of variance. From a visual inspection of Table 8 it can be seen that, for all the means except percent of physical improvement, profile C subjects were the least responsive to treatment. Profile B subjects showed the best response on all measures but time out of bed and pain estimate. This is consistent with the reasoning presented earlier in this chapter, particularly the suggestion that profile B could be viewed as a precursor to a pain stress reaction, and that persons with this profile would respond rapidly to treatment. However, the variations can be viewed only as suggestive since the means were not significantly different in statistical terms. The nonsignificance is, in fact, rather surprising in view of the clear differences in pain histories, since it would be reasonable to expect that differences in past behavior would be related to differences in present and future behavior.

The subsequent analysis of the response to treatment data at discharge was not particularly helpful. The discriminant function was not statistically significant, although subjects in profile clusters A and C (Table 14) seemed to end treatment with the highest estimated pain. The analysis of pretreatment response to these measures did produce a significant discriminant function (Table 9). This function appears to

load most heavily on the subjects' pretreatment medication intake (negative) and the pretreatment pain estimate (positive). This appears to be related to a tendency to depend more on drugs for pain relief, but it is not clear whether this reflects demand for medication from the subject, or some other factors that might increase the probability the physician would be willing to prescribe analgesics for some persons and not others. The attempt to predict group membership with this function was not very successful, with only 30% of the subjects correctly classified. However, there was a difference in the classification rate between groups, with members of groups B and C placed correctly 54% and 56% of the time, respectively (Table 10). Group C subjects were less likely to be using analgesics, and more likely to report pain. If, as Bradley et al. (1978) suggested, persons in this cluster are more obviously disturbed than persons in the other groups, the physicians may have reacted to this with a reluctance to prescribe analgesics. The only significant variable in the one-way analyses of variance is pain estimate. However, this is consistent with the discriminant function, in that group B reported the lowest mean pain estimate, and group C reported the highest pain estimate.

There appear to be four plausible explanations for the negative findings with Hypothesis 3. First, there may in fact be no differential effect in terms of subjects' response to treatment. This is the simplest explanation but, in this author's view, the least likely. Considerable research and

actuarial data already exist on all four profiles in relation to nonpain subjects (Bradley et al., 1978), demonstrating different behaviors over time. In addition, there clearly are differences among the groups in their pain antecedents, as demonstrated in relation to the second hypothesis.

A second possible explanation is that there are differences in the way the groups respond, with the different groups responding better to some treatment modalities than others. The multimodal treatment program used with this subject population may have resulted in no visible differences at discharge because different subjects benefitted from different aspects of the program. This is possible, but would be difficult to test in a clinical setting, since it would be necessary to withhold treatment from some groups, but not others to produce a controlled design. Such an action would have to be justified ethically before research could be conducted.

The third potential explanation is that all subjects benefitted temporarily from treatment because of the intensity of the multimodal approach, but that there are long-term differences. Some profiles may be associated with lasting benefits, while other groups may tend to return for treatment after a relatively brief interval. This seems plausible, since the pain history differences found in this study also developed over a period of months, rather than the few weeks covered by this project. Further, it has been this researcher's experience that many patients do show a positive response over

a few months, but then return with new pain complaints. Unfortunately, a follow-up study was not included in the present program of research.

A fourth explanation is related to the individual MMPI scales. The individual scales are designed to measure specific traits that have been identified through clinical experience. In contrast, the profile clusters identified in this study are derived from combinations of several scales. Subjects are grouped together by the cluster analysis procedure so that they share particular configurations of the scales. While the predictive and explanatory power of the cluster may be related to the predictive and explanatory power of the individual scales, this may not always be true. It is possible that specific scales have clear predictive value that is masked by high variability on the other scales. This possibility was not explored in the current study, so it is not known whether individual scales on the MMPI are related to treatment response.

The four MMPI profiles identified by Bradley et al. (1978) appear to be stable profiles that are replicable in other samples. Further, the pain behavior histories associated with the four profiles are significantly different. However, it was not possible to establish a relationship between the profile clusters and treatment outcome in this study.

Additional research is needed to explore the possible relationship between the profiles and outcome. First, this

study did not include a follow-up of the subjects' behavior after they left treatment. While no differences were found in terms of change during treatment, persons with some profiles may show more long term benefit from treatment than others. In addition, it was not possible to explore the possibility that persons with one profile might respond better to a specific treatment modality than others. This will be difficult to investigate in a clinical setting since it would be necessary to withhold some treatments from some subjects.

Developmental aspects of the MMPI might also represent a fruitful field for research. In particular, there appears to be a possibility that some profile clusters may not in fact represent different character types that developed prior to the onset of pain, but may evolve from other profile clusters (such as profile B) as the pain experience continues. This possibility will need to be explored through longitudinal studies.

The four MMPI profiles represent stable entities that may have some explanatory value in understanding the pain experience. While their predictive value in a clinical setting is not yet established, they appear to represent important tools for future research in chronic pain.

Appendix A

Patient History Form

Name _____   Date _____

Age _____   Present job _____

Last job _____   How long?_____

1. What date (roughly at least) did your present pain start?

2. How long have you been unable to work or do normal house-work?

3. Did your pain start gradually ___ suddenly ___ injury ___ where ___ .

4. Do you get short of breath or a tight feeling in your chest with your back pain?

5. Do you notice your pain after you exercise or exert yourself?

6. If sudden onset, please describe what happened.

7. My pain is: Check the appropriate box

	Better	Worse	No Different
With cough or sneeze	---	---	---
Sitting in straight chair	---	---	---
Sitting in soft easy chair	---	---	---
Bending forward to brush teeth	---	---	---
Walking up stairs	---	---	---
Walking down stairs	---	---	---
Lying flat on back	---	---	---
Lying flat on stomach	---	---	---
On side with knees bent	---	---	---

	Yes	No

8. My back sometimes gets stuck when I bend forward   ___ ___

    After walking, bending forward relieves my pain   ___ ___

    My back feels like giving way when I bend
        forward   ___ ___

    My pain stops me when I walk a certain
        distance   ___ ___

9. Have you been in a hospital for back, leg, or neck
    pain?

    Number of times ____. Please give dates.

10. Have you had myelograms? Number of times _____.

11. Have you had neck or back surgery?

    Number of times _____. Please give type and dates.

12. Do you have any serious medical problems other than back?

13. What treatments have made your pain better?

    What treatments have made your pain worse?

14. What medicine are you taking?

15. Do you have an attorney helping you?

16. Do other members of your family have significant back
    trouble?   Who?

17. Do you have to change jobs?   To what?

18. Are you under pressure at home? _____at work? _____

    Mild _____ Moderate _____ Severe _____

19. What is the most aggravating thing about your pain?

Appendix B

Pain Estimate Form

Date _____

Tourniquet Test

Name: _____   Arm Used: _____

	Pain Levels	Time: Minutes-Seconds
Pain Estimate:	_____	_____
Clinical Estimate:	_____	_____
Maximum Level:	_____	_____
Percentage:	_____	_____

Comments: _____

_____

_____

_____

Signed: _____

Appendix C

Discharge Data Form

Name: _____

Date: _____

Goal Attainment

1	2	3	4	5
no goals				all goals

Percentage of Pain Estimate

1	2	3	4	5
81-100	61-80	41-60	21-40	0-20

Psychological Adjustment

1	2	3	4	5

Percentage of Physical Improvement

1	2	3	4	5
0-20	21-40	41-60	61-80	81-100

Medication Reduction

1	2	3	4	5
same medication				no medication

Up Time

1	2	3	4	5
0 hours	2 hours	4 hours	6 hours	8 hours

1 - Poor

2 - Minimal

3 - Fair

4 - Good

5 - Excellent

Appendix D

Physical Therapy

Muscle Strength

Iliopsoas (Hip flexor), left   ____   ____

Iliopsoas (Hip flexor), right   ____   ____

Hamstrings (Knee flexor), left   ____   ____

Hamstrings (Knee flexor), right   ____   ____

Quads (Knee extensor), left   ____   ____

Quads (Knee extensor), right   ____   ____

Anterior Tibialis (Ankle dorsi-
    flexors), left   ____   ____

Anterior Tibialis (Ankle dorsi-
    flexors), right   ____   ____

Abdominals   ____   ____

Back extensors   ____   ____

Range of Motion

Hip flexion, left   ____   ____

Hip flexion, right   ____   ____

Knee flexion, left   ____   ____

Knee flexion, right   ____   ____

Knee extension, left   ____   ____

Knee extension, right   ____   ____

Range of Motion

    Ankle dorsiflexion, left   ____   ____

    Ankle dorsiflexion, right   ____   ____

Appendix E

Figure 2. Combined group profiles, Bradley et al. (1978) A' and current study A profiles.

Figure 3. Combined group profiles, Bradley et al. (1978) B' and current study B profiles.
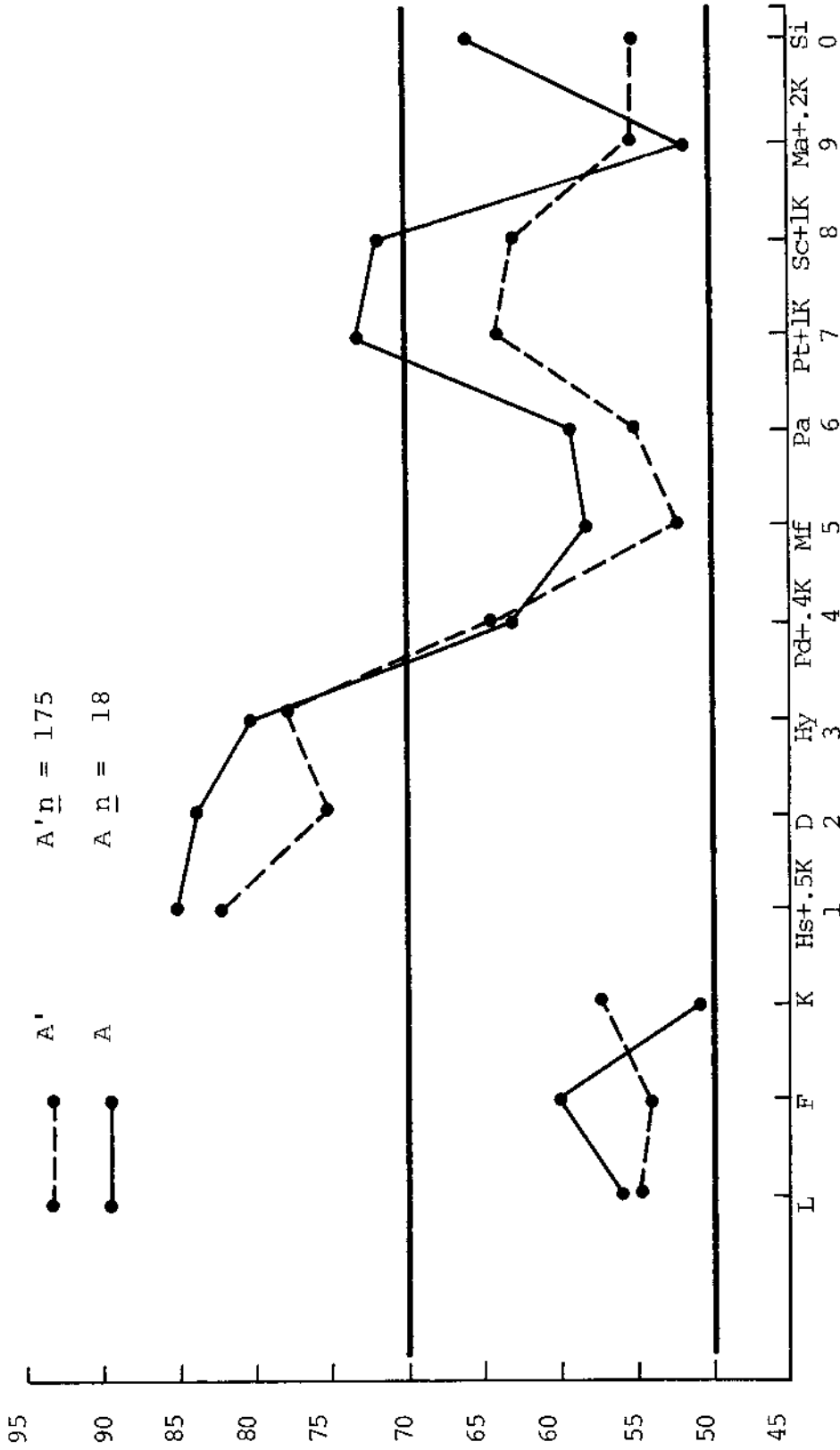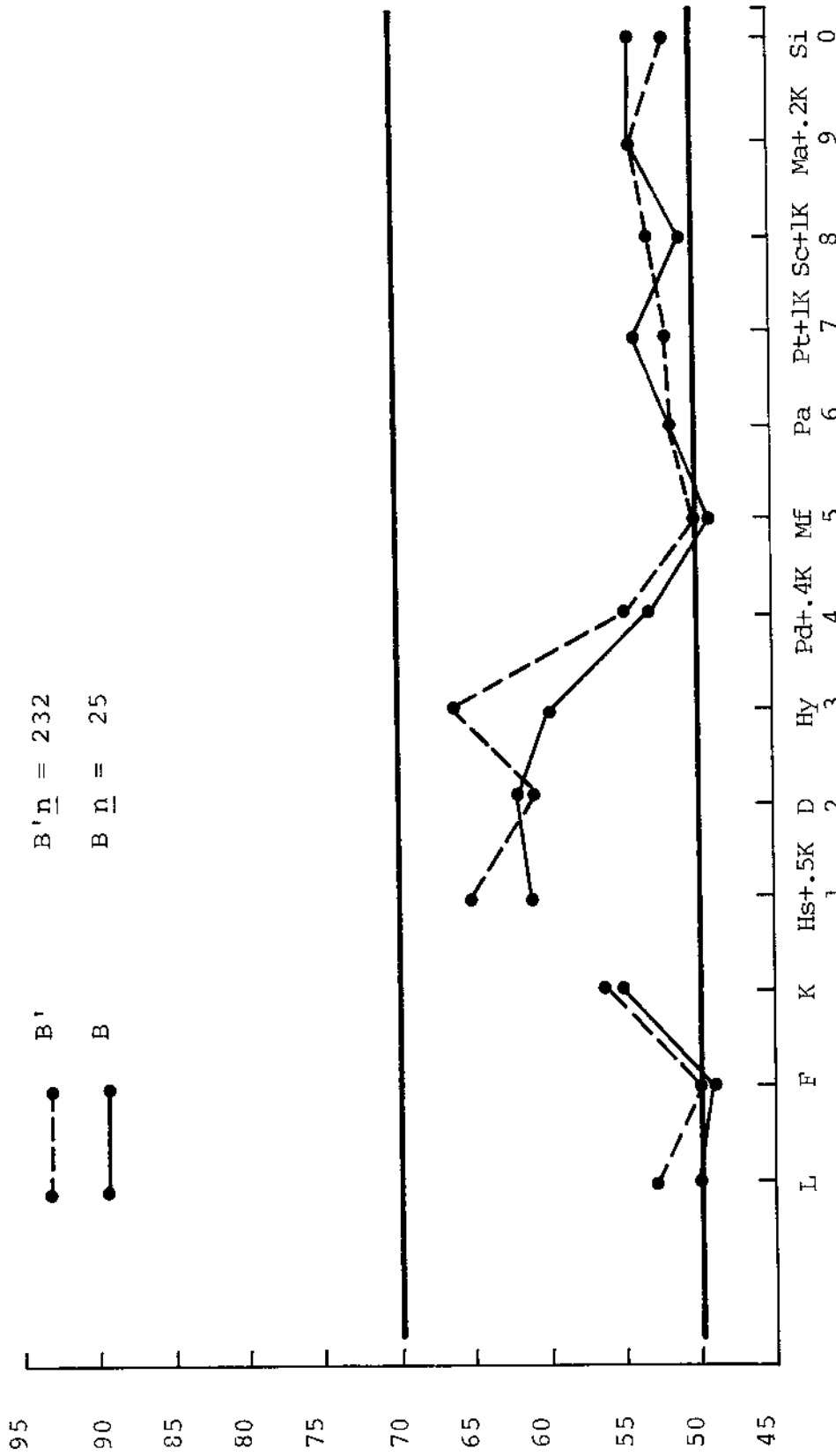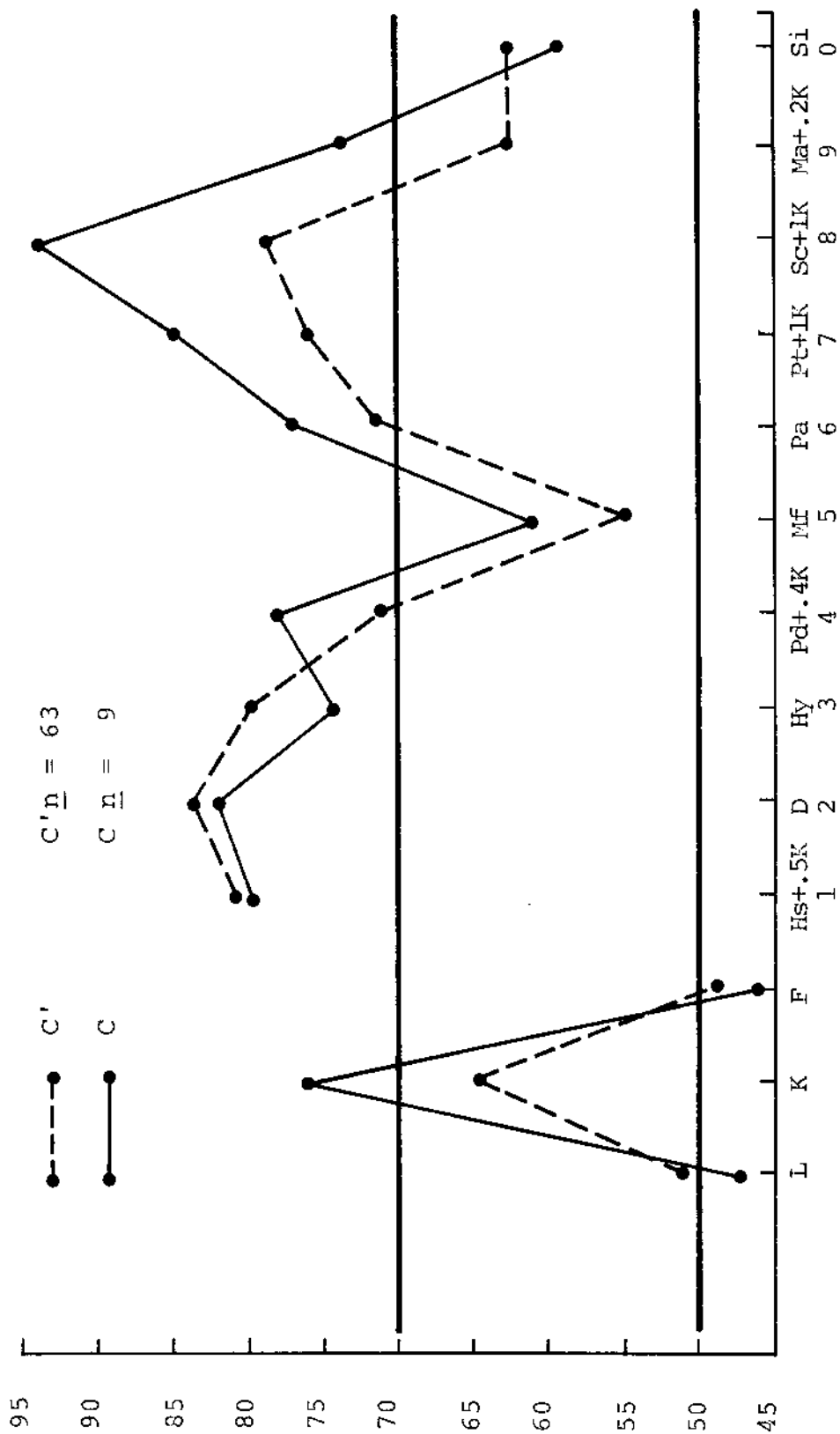
Figure 4. Combined group profiles, Bradley et al. (1978) C' and current study C profiles.
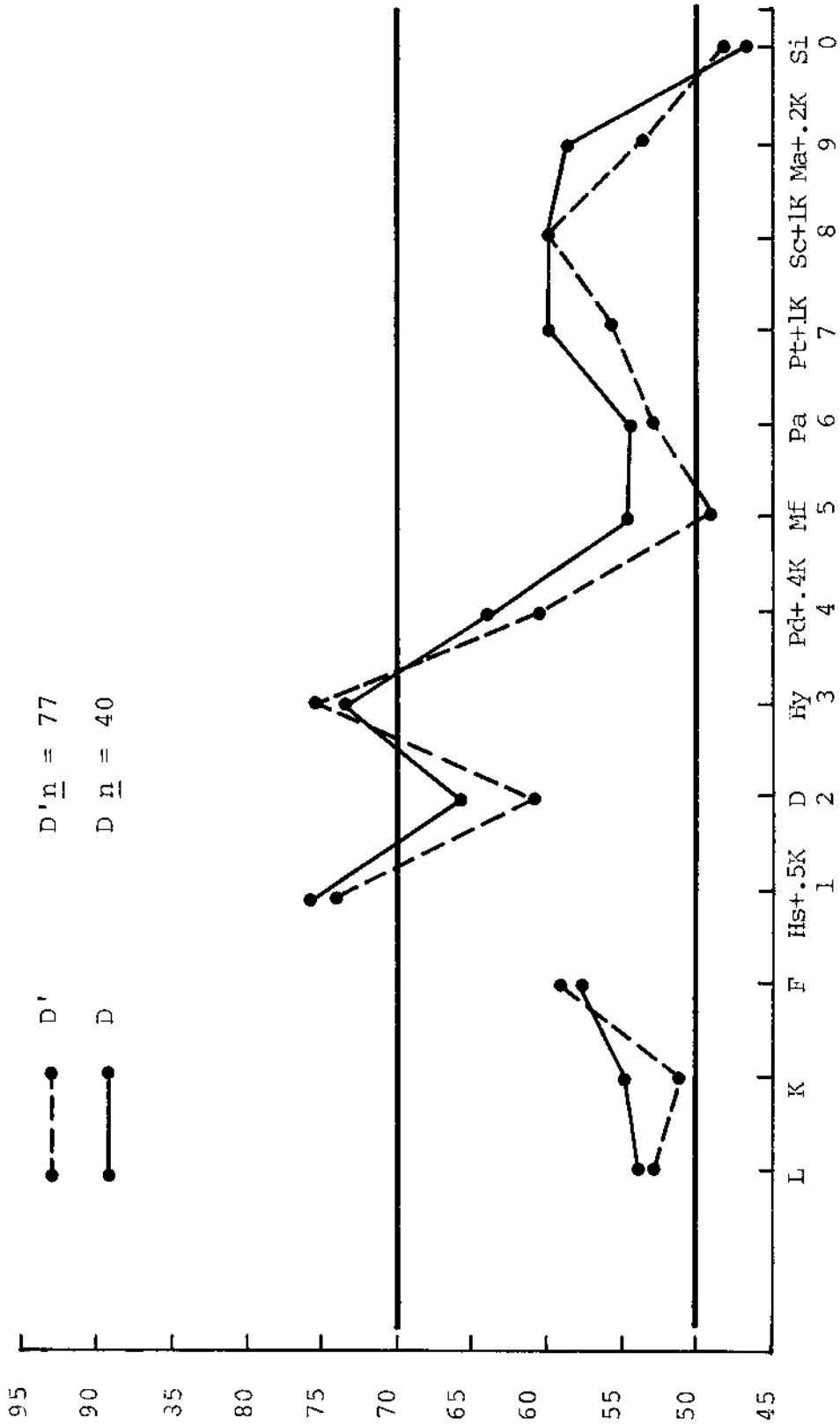
Figure 5.   Combined group profiles, Bradley et al. (1978) D' and current study D profiles.

Appendix F

Table 9

Standardized Coefficients of Pretreatment Variables with
Three Discriminant Functions, and Tests of
Significance for Combined Study Groups

Variable[d]	Discriminant Functions		
	I[a]	II[b]	III[c]
Pretreatment pain estimate (percent)	.859	.282	-.256
Pretreatment hours out of bed daily	.321	-.757	-.638
Pretreatment drug use (morphine equivalents)	-.479	-.250	-.180
Pretreatment mean range of motion (degrees)	.003	-.396	-.903

[a]Chi-square (12) = 20.997, $\underline{p}$ = .05; percentage of variance = 66.82; canonical correlation = .385.

[b]Chi-square (6) = 7.165, $\underline{p}$ = .306 (not significant); percentage of variance = 32.05; canonical correlation = .278.

[c]Chi-square (2) = .253, $\underline{p}$ = .881 (not significant); percentage of vairance = 1.13; canonical correlation = .054.

[d]Wilk's Lambda's for all individual variables not significant by Chi-square test.

Table 10

Results of Group Predictions from the First Discriminant
Function for Pretreatment Variables, In Percentages

Actual Group	Predicted Group Membership			
	A	B	C	D
A	22.2	27.8	33.3	16.7
B	8.3	54.2	20.8	16.7
C	22.2	11.1	55.6	11.1
D	27.5	35.0	25.0	12.5

Percent of "grouped" cases correctly classified: 29.67.

Table 11

Mean Scores of Four Pretreatment Variables
and Univariate F Ratios

Variable	Groups[a]				
	A	B	C	D	F
Pretreatment pain estimate (percent)	68.06	55.42	72.78	62.65	3.279*
Pretreatment hours out of bed daily	6.83	7.40	10.11	9.10	2.463
Pretreatment drug use (morphine equivalents)	.10	.16	.08	.14	.747
Pretreatment mean range of motion (degrees)	79.36	80.70	82.81	83.73	1.014

[a]$\underline{df} = 3.88$.

*$\underline{p} < .05$

Table 12

Pair-Wise Comparisons by Group of Pretreatment Response
Variables (Duncan's Multiple Range Test for
Significant F Ratios)

Variable	Mean Differences					
	A vs. B	A vs. C	A vs. D	B vs. C	B vs. C	C vs. D
Pretreatment pain estimate (percent)	12.64*	4.72	5.41	17.36*	7.23	10.13

*$\underline{p} < .05$

Table 13

Standardized Coefficients of Posttreatment Variables with
Three Discriminant Functions, and Tests of Significance
for Combined Study Groups

Variable	Discriminant Functions		
	I[a]	II[b]	III[c]
Posttreatment pain estimate (percent)	-.883	.089	-.089
Posttreatment hours out of bed daily	-.087	- .250	-.356
Posttreatment drug use (morphine equivalents)	.412	- .009	.744
Posttreatment mean range of motion (degrees)	-.076	- .721	-.013
Amount of goal attainment (rating)[d]	-.016	d	.176

Variable	Discriminant Functions		
	I[a]	II[b]	III[c]
Amount of physical improvement (rating)	.338	− .490	−.500

[a]Chi-square (18) = 28.030, p = .062 (not significant); percentage of variance = 64.75; canonical correlation = .441.

[b]Chi-square (10) = 10.259, p = .418 (not significant); percentage of variance = 31.76; canonical correlation = .326.

[c]Chi-square (4) = 1.061, p = .900 (not significant); percentage of variance = 3.49; canonical correlation = .113.

[d]Undefined.

[e]Wilk's Lambada's for all individual variables not significant by Chi-square test.

Table 14

Mean Scores of Four Posttreatment Variables
and Univariate F Ratios

Variable	Groups[a]				
	A	B	C	D	F
Posttreatment pain estimate (percent)	56.83	36.46	65.56	42.30	5.239**
Posttreatment hours out of bed daily	11.61	11.96	12.44	12.35	.251
Posttreatment drug use (morphine equivalents)	.01	.01	0.0	.03	.496
Posttreatment mean range of motion (degrees)	87.78	90.83	89.11	91.52	.975

[a]df = 3.88

**p < .01

Table 15

Pair-Wise Comparisons by Group of Posttreatment Response
Vairables (Duncan's Multiple Range Test for
Significant F Ratios)

Variable	Mean Differences					
	A vs. B	A vs. C	A vs. D	B vs. C	B vs. D	C vs. D
Posttreatment pain estimate (percent)	20.38**	8.72	14.53*	29.10**	5.84	23.26**

*$\underline{p}$ = .01
**$\underline{p}$ = .05

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