The Effect of Experimentally Induced Anxiety on the Experience of Pressure Pain

by

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ABSTRACT

This study compared two theories which address the relationship between anxiety and pain: 1) the attribution theory that relevant but not irrelevant anxiety intensifies pain, and 2) the modified perceptual disruption theory that anxiety in general disrupts the ability to process nociceptive information and thus influences pain reports. Three types of instructions were presented to male university subjects immediately before nociception: 1) a standard set of instructions, 2) the standard instructions plus a pain warning (relevant anxiety condition), and 3) the standard instructions plus a warning that a stressful interview would immediately follow nociception (irrelevant anxiety condition). Pain and stress intensity ratings, heart rate, electromyographic activity, and facial expressions were recorded continuously and pain threshold and tolerance were recorded once. The anxiety-evoking effects of the instructions were confirmed by analyses of the stress measures obtained during a waiting period. The results indicated that both sets of anxiety-evoking instructions increased pain and stress intensity ratings compared to the control instructions when painful pressure was applied to the skin. In addition, the relevant but not the irrelevant anxiety condition increased physiological arousal and facial grimaces and appeared to influence the report of tolerance. These results were interpreted as supporting the modified perceptual disruption theory.

RÉSUMÉ

La présente étude compare deux théories traitent du lien entre l'anxiété et la douleur: 1) la théorie de l'attribution, selon laquelle la douleur est plus intense si elle s'accompagne d'anxiété relativement à la douleur, mais non si l'anxiété est sans rapport avec cette dernière; 2) la théorie modifiée de l'interruption perceptuelle, selon laquelle l'anxiété générale entrave la capacité de traiter l'information sur les stimulus douloureux et influence donc les témoignages de douleur. Trois consignes différentes ont été transmises à des étudiants universitaires masculins, juste avant l'administration des stimulus douloureux: 1) la consigne type; 2) la consigne type suivie d'un avertissement concernant les stimulus douloureux (conditions d'anxiété relative à la douleur); 3) la consigne type accompagnée d'un commentaire indiquant qu'une entrevue stressante suivrait immédiatement les stimulus douloureux (conditions d'anxiété non relative à la douleur). L'évaluation de la douleur et du stress, le rythme cardiaque, l'activité électromyographique frontale et les expressions faciales ont été enregistrées de façon continue; les seuils de tolérance et de détection de la douleur ont été relevés une fois. L'examen des mesures du stress prises pendant une période d'attente a démontré l'effet anxiogène des consignes. Les résultats indiquent que l'intensité de la douleur et du stress est plus élevée, selon les évaluations, chez les deux groupes ayant reçu une consigne anxiogène que chez le groupe témoin, lorsqu'une pression douloureuse est appliquée sur la peau. En

outre, l'attention physiologique est plus grande, les grimaces faciales sont plus nombreuses, et le témoignage de tolérance semble être influencé, dans les conditions d'anxiété relative à la douleur; ces résultats n'apparaissent pas dans les conditions d'anxiété non relative à la douleur. L'interprétation des résultats confirme la théorie modifiée de l'interruption perceptuelle.

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INTRODUCTION

The social and economic costs of pain are enormous. Bonica (1980) estimates that the direct and indirect costs created each year through lost work days, lower productivity, over-the-counter and prescription medicines, and visits to physicians and clinics from people reporting pain are $58 billion in the United States. Over-the-counter analgesics account for over $900 million of this total (Turk, Meichenbaum, & Genest, 1983).

The prevalence of pain complaints within the general population is also striking. Approximately 25 million Americans experience migraine headaches, 7 million report low back pain, and from 20 to 50 million have arthritis (Turk et al., 1983). Crook, Rideout, and Browne (1984) conducted an epidemiological study of 827 Canadians within 500 households randomly selected from a family practice roster. They reported that 16% of this sample reported experiencing pain within the last two weeks; almost 70% of these individuals reported persistent pain. These figures lend strong support to Bonica's (1980) claim that pain is one of society's most pressing problems.

Lewis (1942, in Hayward, 1975) stated that pain is "known to us by experience and described by illustration" (p. 13). Efforts to define pain have generally been unsatisfactory (Melzack & Wall, 1982). Recently, the Task Force on Taxonomy set up by the International Association for the Study of Pain (IASP) defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP, 1979, p. 250).

Melzack and Wall (1982) believe this definition has advantages over its predecessors since it includes both the emotional and sensory aspects of the pain experience and it recognizes the complex relationship between injury and pain. However, they view the word 'unpleasant' as too limiting. Pain experiences can range from mild to excruciating. In addition, this definition is similar to definitions of anxiety and fear. Both can be defined as unpleasant sensory and emotional experiences associated with potential tissue damage.

Defining pain in a truly satisfactory fashion will remain problematic until pain mechanisms are more fully understood. Melzack and Wall's (1982) work on the language of pain has led them to conclude that "the word 'pain' represents a category of experiences, signifying a multitude of different, unique experiences having different causes, and characterized by different qualities varying along a number of sensory and affective dimensions" (p. 71).

Turk et al. (1983) present a typology of pain experiences consisting of five classes:

1) Acute pain; pain that is self-limiting and lasts less than six months (e.g., accidental injury and burn pain).

2) Chronic, periodic pain; acute pain that is intermittent (e.g., migraine headaches).

3) Chronic, intractible pain; pain that is present most of the time yet its intensity varies (e.g., low back pain).

4) Chronic, progressive pain; pain that increases over time (e.g., terminal cancer related pain).

5) Experimentally induced pain; pain produced by nociceptive stimulation in a laboratory setting,

Determining the mechanisms that create the pain experience remains an elusive scientific problem. Numerous theories have been proposed yet no one theory has been completely accepted (see Schneider & Karoly, 1983). Since it is becoming apparent that the mechanisms responsible for acute and chronic pain differ (Bonica, 1977; Melzack & Wall, 1982; Sternbach, 1974), this review will discuss the most recent perspectives concerning the experience of acute and experimentally induced pain.

Theories of acute pain mechanisms

Cassem (1983) describes the standard medical approach to pain in accord with the Seattle model (Loeser, 1980) that conceptualizes pain as a complex process consisting of nociception, pain, suffering, and pain behavior. Each step involves increasingly complex neurophysiologic mechanisms to explain the increasing variation among individuals.

Nociception is defined by Cassem (1983) as activation of "pain" fibers (delta A and type C) that receive stimulation from "pain" receptors (the free nerve endings that specialize as mechanosensitive, thermosensitive, and chemosensitive "pain" receptors) in the skin and other tissue. The neurophysiologic mechanisms producing nociception have been described in detail elsewhere (Cassem, 1983; Guyton, 1981; Melzack & Wall, 1982). According to the modern medical approach, pain arises when nociceptive input is perceived (Cassem, 1983; Guyton, 1981). Therefore, medical models have discarded specificity theories which state that pain is caused by nociception. A person can receive nociceptive input without experiencing pain (see Beecher, 1959; Melzack & Wall, 1982).

However, others (such as Bonica, 1977; Melzack & Wall,

1982; and Merskey, 1980) argue that this account is in error since it does not include the negative emotional experience that is present during pain and therefore this view does not allow pain perception to be different from other types of sensory perception. These researchers and others (Weisenberg, 1977) have argued that pain is an experience that involves interacting sensory, perceptual, and affective processes. In addition, the medical perspective is not consistent with the IASP's definition of pain. On a practical level, Sternbach (1968) stated that studying the type of "pain" that arises without the presence of a negative emotional experience is not relevant to the pain experiences that physicians must assess and treat. People who are not suffering are not likely to seek aid.

Nevertheless, the distinction made between pain and its negative affective quality has allowed many investigators to continue to separate physical from mental constructs (see Engel, 1980; Schwartz, 1983). This may be due to a misconception that perception can be studied without reference to psychological processes.

The medical explanation of the cause of pain when it arises in the presence of nociceptive input is convincing (see Cassem, 1983; Guyton, 1981). However, its utility breaks down when attempting to explain how nociception can occur without pain being experienced. In brief, Cassem (1983) explains that pain is not perceived if "pain" impulses are interrupted at lower levels of nervous system processing. This is due to a descending "inhibitory" system that can modulate "pain" impulses before higher level cerebral centers have received this afferent input. Ultimately, activation of this descending system can only be

explained by reference to mental concepts (Degenaar, 1979). Therefore, the afferent input itself is modified by cognitive, attentional, and emotional processes (Craig, 1984). Melzack and Wall (1982) describe several factors that are related to this perceptual process. Culture, past experience, the type of situation, and attention are some of the factors that can prevent awareness of nociceptive input for a period of time.

Cassem (1983) and Guyton (1981) describe the suffering component of the pain process as the negative affective reaction that usually accompanies pain. Pain behaviors are all the personal and social activities a person undertakes to express and reduce suffering (Degenaar, 1979). Even though suffering is the major determinant of pain behavior (Cassel, 1982; Degenaar, 1979) and if suffering were not present, pain would cease to have significant economic and social repercussions, medical textbooks (see Cassem, 1983; Guyton, 1981) view this component as a source of experimental error in the study of pain processes. However, these textbook authors admit the suffering component is the most distressing to the patient and needs to be assessed by the physician to ensure rapport with him or her.

The mechanisms involved with the experience of suffering (or any other emotion) are difficult to elucidate in a strictly neurophysiological fashion. In brief, Cassem (1983) states that the neurophysiologic connections of "pain" pathways with the hypothalamic and limbic systems (the theoretical neural substrate for emotional states) can help explain why suffering can become the most salient feature of the pain process. Pain evokes a suffering reaction via a reflexive pattern of impulses channeled to these areas. This reflexive system can be

modulated by activation of pain inhibitory systems such as the endorphins and enkephalins (the brain's opiate system). Once again, to ultimately explain the activation of inhibitory systems reference must be made to mental concepts. Thus, the explanatory value of a strictly neurophysiological approach is questionable. The mechanisms behind the occurrence of pain behaviors are described by Guyton (1981) and others (e.g., Cassem, 1983) in psychological terms often with psychodynamic determinants.

Pain production and assessment

A variety of methods are used to produce and assess the acute pain experience in human subjects. There are two avenues of scientific inquiry; the utilization of naturally occurring pain states and the production of pain in a laboratory setting.

Naturally occurring acute pain states include the pain associated with accidental injuries, burns, labor, and post surgical recovery. Assessment of the clinical pain experience includes behavioral measurements, observational data, self-report of behaviour, and subjective pain reports (Chapman, Casey, Dubner, Foley, Gracely, & Reading, 1985).

Behavioral measurements and observational data involve monitoring the frequency of occurrence of various activities, medication requests and intake, and pain complaints. Self report of these measures has also been employed. Chapman et al. (1985) believe that these measures are useful for the assessment of pain relief and treatment effects even though they do not assess the pain experience directly. These behaviors can change for reasons other than the presence or absence of pain.

According to Chapman et al. (1985), the most useful subjective report measures in a clinical setting are the visual analog scale (VAS), the McGill Pain Questionnaire (MPQ), and multiple descriptor scales. The VAS requires that the subject indicate the intensity of the pain by making a mark on a ten centimeter line labelled "no pain" at one end and "the worst possible pain" at the other. As a simple method that can be used to assess pain intensity over time, the VAS is a standard assessment tool in both clinical and laboratory research. It is reliable and for statistical purposes, equal intervals can be assumed (Stewart, 1977). It is more accurate than category scales that require the subject to choose a word from a list to describe the pain experience (Chapman et al., 1985). However, the VAS does not take into account the richness of the pain experience.

The MPQ (Melzack, 1975) asks the subject to select the most appropriate word (or to omit if not relevant) from 20 sets of descriptors to describe the pain. Between two and six words in each set are listed in ascending order. Sensory (ten sets), affective (five sets), evaluative (one set), and miscellaneous (four sets) dimension of the pain experience are presented. A variety of scoring methods can be employed such as counting the number of words chosen or finding the sum of the rank values of all the words chosen. Other questions relating to the pain experience are also asked. Chapman et al.'s (1985) review states the MPQ assesses the multidimensional nature of pain, has wide applicability, and that its factor structure, reliability, and validity have been empirically supported. However, this measure is time consuming and weighs the sensory aspects of the pain

experience more heavily than the affective or evaluative components.

Multiple pain descriptors have been developed by Gracely (1980) to assess both clinical and laboratory pain. Three sets of thirteen words assessing the intensity, unpleasantness, and painfulness are presented in ascending rank order. The subject indicates the most appropriate word in each set. The rank of the verbal descriptors has been quantified by cross-modality matching procedures (Gracely, 1979) in which an increase in handgrip force during noxious electric shock was associated with increases in rank value of the category words.

Turk et al. (1983) describe the most frequently employed nociceptive stimuli in laboratory research; cold pressor, radiant heat, pressure, electric shock, and muscle ischemia. The cold pressor task requires that the subject immerse her or his hand and part of the forearm in ice water maintained at a steady temperature such as 2°C. This procedure is generally considered very painful producing a rapidly increasing set of noxious sensations reaching a point of numbness in three to four minutes. Constancy of immersion is difficult to monitor without observing the subject. In addition, immersion of a part of the body into cold water produces metabolic and physiological changes that may be more related to the experience of cold than to pain (Guyton, 1981).

Radiant heat pain is induced by focussing a high intensity light against a skin surface blackened with india ink. The ceiling for this measure is usually reached within two minutes and in tolerant subjects, tissue damage may occur (Turk et al., 1983).

Pressure pain is produced by a Forgione-Barber (1971) strain gauge with a lucite wedge placed on the skin over a bone (usually the index finger). This stimulus produces a continuously building aching pain that has a ceiling between two and ten minutes depending on the amount of pressure employed. Variations in pressure can be monitored through the use of a polygraph or other recording device.

Electric shock produces noxious stimulation by presenting a series of nondamaging electric shocks of varying amperage. The pain produced is episodic in nature and does not closely resemble clinical pain (Turk et al., 1983). However, the possibility of delivering many trials at varying intensity levels allows the implementation of complex psychophysical measurements such as signal detection methods.

Muscle ischemic pain is commonly produced by the sub-maximum tourniquet method (Smith, Egbert, Markowitz, Mosteller, & Beecher, 1966). A blood pressure cuff is inflated to a high level and the subject exercises his or her arm. The pressure from the cuff impedes circulation to the forearm. Exercise reduces the amount of oxygen reaching the arm and produces a steadily increasing aching pain. Tolerance time is variable, ranging from three to 55 minutes (Turk et al., 1983). The amount of force exerted during exercise can determine the degree of ischemia and must be monitored (Sternbach, 1983). In addition, removal of the cuff following tolerance does not terminate the pain experience. As blood surges into the forearm and hand, an additional source of nociception is created. Finally, individuals must be screened for capillary strength prior to ischemic pain production because the resurgence of blood flow can damage

capillary walls (Feuerstein, personal communication).

It is not known if the subjective experience of these stimuli is equivalent. Scott and Barber (1977, reported that subjects tolerated cold pressor and pressure pain for similar lengths of time and rated the two kinds of pain as equally severe. Hilgard and Hilgard (1975) reported that the intensity of the pain produced by ischemia increases more slowly than cold pressor pain.

There is also little agreement to the extent to which each of these stimuli matches clinical pain. Zwetnow (1979) states that clinical pain differs from experimental pain in that it is "more severe, more prolonged, less predictable in duration, less subject to voluntary termination, and usually more anxiety provoking" (p. 213-214). Zwetnow believes that methods that produce continuously building aching pain have the closest resemblance to the types of pain found in clinical settings. Furthermore, Sternbach (1968) reports that fairly intense physical stimulation coupled with a moderate degree of anxiety can produce pain responses that are not distinguishable from those obtained in clinical settings. In addition, laboratory methods allow controlled manipulation of variables that is not ethically acceptable in clinical studies. Exact monitoring of multiple response systems is only possible in laboratory research (Gracely, 1983).

Chapman et al. (1985) discuss the assessment of the pain experience in laboratory research. The methodologies include basic and advanced psychophysics, rating scales, and task performance. Physiological and facial expression responses associated with the pain experience are often used in conjunction

with one or more of these methods.

Basic psychophysical methods include the determination of pain threshold, tolerance, and endurance (Chapman et al., 1985). Threshold is determined by requiring the subject to identify when the stimulus is first perceived as painful. Pain tolerance is determined by requiring the subject to identify when the stimulus is no longer endurable or tolerable. The endurance time (or pain sensitivity range) is computed by subtracting the threshold duration from the tolerance duration. Tolerance methods are thought to approximate clinical pain states more closely than threshold methods since the pain experienced over the course of the experiment is more severe (Chapman et al., 1985).

The most useful laboratory rating scales are the VAS, the MPQ and multiple pain descriptors (Chapman et al., 1985). The VAS can be used during nociception to provide an ongoing measure of stimulus intensity. The MPQ and Gracely scales must usually be completed after nociception and can be reworded to reflect this change, yet this may diminish their sensitivity. Klepac, Dowling, and Hauge (1981) used the MPQ to assess cold pressor and electric tooth shock stimulation pain in a laboratory setting. After threshold and after tolerance the MPQ was completed. Higher scores on the MPQ were obtained in reference to cold pressor pain than tooth pain in all categories (sensory, affective, and evaluative). In addition, a higher ranking of words was chosen after tolerance than after threshold. Klepac et al., (1981) conclude that the MPQ can be used after experimentally induced pain to interpret the pain experience.

There is evidence that tolerance and intensity ratings are

loosely related (Scott & Barber, 1977). Therefore, it is useful to assess the pain experience using a combination of verbal report measures.

Advanced psychophysical methods include magnitude estimation, cross-modality matching, and signal detection theory. It is beyond the scope of this review to discuss each of these methods in detail (see Chapman et al., 1985). Each method requires repeated brief exposures to a stimulus (usually electric shock) in ascending and descending magnitude in an attempt to quantify pain perception processes. Signal detection methodologies yield two types of data representing the ability of the subject to discriminate among stimulus intensities and the tendency of the subject to rate the stimulus on a conservative or liberal basis (Chapman et al., 1985).

Measures of task performance include reaction time tests and error rates in learning new material. These measures are employed to determine the extent to which nociception interferes with ongoing behavior. Chapman et al. (1985) report that these measures fail to directly measure the pain experience, and suggest that results from studies using this methodology must be interpreted with caution.

Chapman et al. (1985) state that physiological correlates of the pain experience are assessed in laboratory settings to confirm the accuracy of verbal reports, to provide additional information for hypothesis testing, and to assess the emotional aspects of the pain experience. Electromyographic (EMG) recordings provide a method for determining muscle tension levels. Since EMG measures the activity of the nerves innervating the muscle in question, it is an indirect measure of the actual

pressure exerted by muscle contraction (Everly & Rosenfeld, 1981).

Fluctuations in autonomic indices (such as heart rate) and in EMG activity are loosely associated with verbal reports of pain. Familiarity with the nociceptive stimulus can produce decrements in these measures, and increases in these measures are also associated with increases in the report of negative emotional states (such as anxiety and fear; Greenfield & Sternbach, 1972).

Recently, electroencephalography (EEG) measures have become popular as adjuncts to verbal reports (see Chudler & Dong, 1983). However, there is no solid evidence that this or any other physiological correlate is less susceptible to psychological mechanisms than subjective reports (Chapman et al., 1985). Furthermore, when no physiological correlates are present (as in hypnotic analgesia or relaxation) pain intensity ratings still increase over time during nociception (Shor, 1962; Hilgard & Hilgard, 1975).

Videotapes of facial expression are also used as adjuncts to other pain assessment methods. Craig and Prkachin (1983) point out that verbal reports are easier to modify than facial expression. In addition, in naturally occurring settings, facial expressions usually precede verbal reports of experience (Craig & Prkachin, 1983). However, these authors report rapid habituation and suppression of facial indicants of pain in laboratory settings. Thus, monitoring should start prior to the initial phases of nociception. These authors have also reported that one prototypical pain expression has emerged from the research data. This expression consists of lowered eyebrows, eyes tightly shut, and a horizontally stretched open mouth.

Until recently, most experiments recorded only one subjective measure in combination with one or two other measures. This creates difficulty in interpreting and comparing the results of different studies. Because all of these measures vary in the aspect of the pain experience that is being measured, the ideal experiment records multiple measures of subjective, behavioral, and physiological responses.

Social and cognitive influences on the pain experience

Although acute pain is usually perceived in response to peripheral input, the perceptual process can be modified by a number of social and cognitive factors.

Social factors. Social variables that affect the expression of pain in both laboratory and clinical settings include the cultural background of the subject (Melzack & Wall, 1982; Weisenberg, 1977), the family in which the subject was raised (Craig, 1984), and the sex and race of the experimenter (Weisenberg, 1977).

In clinical settings, the patient's belief that the expression of pain will enlist others in reducing his suffering influences pain behavior (Cassel, 1982). Hospital staff are more responsive to the pain behaviors of female rather than male patients (Weisenberg, 1977) serving to reinforce sex differences in pain expressiveness. The degree of rapport between the afflicted individual and other people within the social context also contributes to the expression of pain (Cassel, 1982).

Craig and Prkachin (1978) reported that subjects exposed to a model reporting higher pain tolerance reported less discomfort associated with preselected levels of electric shock.

In addition, the subjects exposed to the tolerant model manifested lower heart rate and forearm skin potentials during electric shock stimulation and a decreased ability to determine differences between the intensities of adjacent pairs of shocks (as assessed by signal detection theory). The authors concluded that the pain experience of the subjects exposed to a tolerant model was altered in addition to the verbal reports of the discomfort associated with this experience.

The effects of placebos, hypnosis, and analgesic suggestions are usually considered under the topic area of cognitive mediators of the pain experience. However, since the efficacy of these manipulations is dependent upon the social context in which they are employed (Barber, 1981), their influence is discussed here.

Beecher (1959) reported that 35% of acute pain patients obtain relief from placebos. The combination of an analgesic suggestion from a perceived authority figure and the presence of an inert substance contributes to this process (Pollack, 1966). The efficacy of placebos is also a function of the enthusiasm of the prescribing physician, the degree of anxiety and stress, and the severity of the pain (Melzack & Wall, 1982).

In laboratory studies, analgesic suggestions and hypnotic analgesia have produced similar reductions in pain intensity, distress, and physiological indicants of arousal (Barber, 1963, 1981). Barber (1981) states that the critical variables are the suggestions of pain relief which are given in a close interpersonal context and not to the induction of a "hypnotic trance state". However, there is considerable controversy concerning this view (Hilgard & Hilgard, 1975). Melzack and

Wall (1982) propose that the combination of relaxation, suggestions for pain relief given by an authority, and focussed attention away from physical sensations produces the effect of hypnotic analgesia. However, no one study has contrasted each of these factors.

In conclusion, although a great deal of the pain research assessing social influences has focussed upon pain behaviors, there is evidence indicating that developmental and social factors modify pain experiences at a perceptual and evaluative level.

Cognitive factors. Cognitive mediators of pain perception and experience include cognitive dissonance, attribution of arousal, control over the stimulus, and the utilization of coping strategies.

Festinger (1957) proposed that perceived incongruity (or cognitive dissonance) between an individual's behavior and beliefs motivates that person to change either his behavior or his beliefs. Zimbardo, Cohen, Weisenberg, Dworkin, and Firestone (1966) tested the hypothesis that subjects would experience less pain when choosing to undergo further electric shock stimulation with little justification (high dissonance) compared to subjects who were provided strong justification (low dissonance). True to prediction, the results indicated that the high dissonance group (but not the low dissonance group) showed reductions in pain intensity, error rate on a serial anticipation task, and galvanic skin response.

Attribution theory states that people seek explanations for observed events. Nisbett and Schachter (1966) manipulated attributions concerning the source of bodily arousal and measured

pain tolerance to electric shock. The state of arousal was either attributed to a drug (a placebo) or to the shock under high or low fear conditions (presence or absence of a pain warning). For the low fear group only, subjects who attributed their increased arousal to the drug tolerated over four times the level of shock than subjects who attributed their arousal to the shock tolerated. Post-experimental questionnaires indicated that subjects receiving a pain warning attributed their arousal to the shock regardless of the instructions.

Melzack and Wall (1982) state that nociceptive input is evaluated before the perceptual experience of pain is produced. Dissonance and attribution of arousal may effect the pain experience by altering this evaluative process.

Thompson (1981) defined control as "the belief that one has at one's disposal a response that can influence the aversiveness of an event" (p. 89). She identified three types of control related to the pain experience; behavioral, cognitive, and informational.

Thompson (1981) reports that behavioral control (the belief that a behavioral response is available that can influence the aversiveness of the nociceptive stimulus) increases pain tolerance time, reduces arousal and anxiety prior to nociception, and increases task performance during nociception. However, it has inconsistent effects on self-report of pain and distress and physiological measures during nociception.

Cognitive control (the belief that a cognitive strategy can be employed to influence the aversiveness of a stimulus) has had inconsistent effects upon the pain experience. Turk (1978) and Tan (1982) concluded that cognitive strategies do

not appear to affect pain intensity or threshold. However, cognitive strategies reduce arousal and anxiety prior to and during nociception and may increase pain tolerance (Thompson, 1981). Assessing the efficacy of cognitive strategies is hampered by the fact that most subjects use strategies on their own and thus, the control groups in most of these studies have been inappropriate (Barber & Cooper, 1972). Barber and Cooper (1972) reported that cognitive strategies exert the greatest effects on pain intensity during the initial stages of nociception. Therefore, the results from studies that have used an average pain intensity rating across the entire nociceptive period may be misleading.

Thompson (1981) reports that in general, information that consists of a warning signal prior to electric shock increases tension and arousal when the subject is unable to influence its intensity, yet a warning will decrease tension and arousal when the subject can influence its intensity. The effect that a warning signal has upon pain intensity and other aspects of the pain experience is not known (Thompson, 1981).

Sensory and procedural information have had inconsistent effects upon the pain experience (Thompson, 1981). Sensory but not procedural information reduced the distress associated with ischemic pain (Johnson, 1973). Providing sensory information without using the word "pain" reduced the anxiety associated with cold pressor pain when compared to procedural information (Leventhal & Everhart, 1979). Including the word "pain" in the sensory information instructions reduced the effectiveness of providing sensory information only.

Thompson (1981) speculates that the underlying theme of

all types of control is the meaning individuals attach to stressful events. This meaning depends upon whether or not the individual views the event as endurable, as a means to a desired end, and as a planned event. Having the ability to control the stimulus within one's repertoire may alter one or more of these dimensions thereby decreasing the effect of nociception.

Emotional influences on pain

The numerous social and cognitive variables that modify pain perception and experience suggest that the central processing of nociceptive information can be altered by psychological mechanisms (Melzack & Wall, 1982). Furthermore, these social and cognitive factors affect the emotional impact of nociceptive stimuli as measured by reduced distress, anxiety, and physiological reactivity. On the other hand, there is a widespread assumption that emotional experience, specifically the experiences of anxiety, stress, or fear, can also modify the central processing of nociceptive input (see Craig, 1984; Melzack & Wall, 1982; Merskey, 1980; Weisenberg, 1977).

Defining anxiety, stress, and fear have proven as problematic as defining pain. Anxiety appears to be the most general concept of the three since it encompasses a wide variety of feelings, thoughts, and sensations. Hayward (1975) states that a common theme in all definitions of anxiety is the associated experience of fear. Fear has been defined by Rachman (1978) as the "feeling of apprehension about tangible and predominantly realistic dangers" (p. 6). Lazarus and Averill (1972) state that anxiety can be distinguished from fear by its anticipatory, symbolic, and uncertain nature. Anxiety usually involves an

imagined threat and this threat can be idiosyncratic and undescribable. Because of the ambiguous nature of the threat, useful coping strategies are difficult for the individual to envision. Lazarus and Averill (1972) believe that anxiety involves a threat to cognitive and psychological integrity whereas fear is related to relatively more tangible dangers.

Anxiety is not a unitary concept (Spielberger, 1972). Two forms have been empirically established; trait and state anxiety. Trait anxiety refers to a relatively permanent feature of an individual's personality that predisposes him or her to perceive nondangerous situations as threatening and to overreact to these situations with a state anxiety response (Spielberger, 1972). State anxiety is transitory and usually experienced in response to specific situations. It is characterized by feelings of fear and tension and is often accompanied by activation of the sympathetic and parasympathetic nervous systems, the endocrine systems and other physiological systems (Everly & Rosenfeld, 1981). In addition, the work of Endler and his colleagues (Endler, 1975; Endler & Magnusson, 1976; Endler & Okada, 1975; Flood & Endler, 1980) indicates that there is a third type of anxiety which reflects the individual's predisposition to respond in specific situations (such as being alone) with a state anxiety response.

State anxiety is difficult to distinguish from psychological stress and the two terms are often used interchangeably. It is generally agreed that the experience of psychological stress or state anxiety is evoked by the perception that a particular stimulus is threatening to the individual's psychological or physical integrity (Everly & Rosenfeld, 1981). This perception

evokes the stress response or state anxiety response which includes increased reports of fear, tension, and anxiety usually accompanied by increased physiological arousal. The interpretative process leading to the perception of a threat is not always a conscious one nor is it always reportable (Plutchik, 1977). Numerous situational and predispositional variables influence this perceptual process (Lazarus & Averill, 1972). Thus, the evaluation of incoming stimuli does not always lead to an emotional response (Plutchik, 1977). The interpretative process is continually reshaped as new sensory information, emotional experience, and physiological responses feed back to cognitive mediators of this experience.

Considerable controversy exists as to whether the perceptual interpretative antecedents of emotional experience are primarily cognitive in nature (Lazarus & Averill, 1972; Spielberger, 1972) or are an integration of cognitive and affective processes (Mandler, 1984). Mandler (1984) presents a model in which emotions are evoked through the coaction of autonomic arousal and cognitive interpretation. A discrepancy (a perception that does not fit into perceptual expectations or cognitive schemas) interrupts ongoing actions and cognitive schemas and produces general arousal. This arousal serves as a signal for cognitive evaluation. The degree of interruption determines the emotional intensity of the experience, and the ease through which the individual can assimilate or accommodate the discrepancy determines the qualitative aspects of the emotion. When no action or thought is available that can terminate the interruption, anxiety, distress, or fear will arise.

Once anxiety is experienced, cognitive mediators reappraise

the significance of the stressor that initiates the emotional experience (Plutchik, 1977). This reappraisal can lead to coping, avoidance, or defensive behaviors.

Production and assessment of psychological stress

Due to ethical constraints, there is no way to produce experimental conditions that will be perceived as threatening by all individuals (Patkai, 1974). Three classes of stressors are used in laboratory work (McGrath, 1970; cited in Patkai, 1974). Physical stimuli that involve actual or anticipated pain have been employed to produce threats to the individual's physical integrity. Related to this methodology, Lazarus, Spiesman, Mordkoff, and Davison (1963) developed filmed events of physical trauma to evoke a stress response. The second class of stressors employs social psychological stimuli that imply a threat to the psychological integrity of the individual. Evaluative threats such as criticism, interpersonal conflict, or evaluation of some aspect of the individual's personality have been used to induce stress responses. The third class of stressors involves the presentation of complex cognitive tasks which evoke performance anxiety through fear of failure. The difficulty of the task can be manipulated, the task can be ambiguous, or the subject can be required to work under time pressure.

Multimodal assessment of the stress response is necessary due to the large individual differences in response to different stressors (Patkai, 1974). Subjective reports of emotional experience include the VAS which can be anchored by the extremes of "no stress" and "the worst possible stress". Questionnaires assessing anxiety to obtain more detailed information include

the State-Trait Anxiety Inventory (STAI; Spielberber, Gorsuch, & Lushene, 1970) which provides measures of present and general anxiety levels, the Taylor Manifest Anxiety Scale (TMAS; Taylor, 1953) which provides a measure of trait anxiety, and the Stimulus-Response Inventory of General Trait Anxiety - Revised (GTAR; Endler & Okada, 1975) which provides a measure of anxiety in specific situations.

The STAI consists of two scales to assess state and trait anxiety. Each scale has twenty items with four possible responses ("almost never", "sometimes", "often", and "almost always") and can be administered separately or together. This inventory has generated considerable reliability and validity data (Spielberger & Gorsuch, 1966; Spielberger et al., 1970) and is considered a sound measure of state and trait anxiety (Everly & Rosenfeld, 1981).

The TMAS consists of 28 items, to be marked true or false, drawn from the Minnesota Mulitphasic Personality Inventory. Although the validity and reliability of this measure has not been extensively assessed, the TMAS is a popular measure of trait anxiety. Taylor (1953) presents data indicating that this instrument has high test-retest reliability (r = 0.88) and can discriminate between normal and psychiatric patients.

The GTAR is a multidimensional measure of trait anxiety which asks the subject to rate, on a one to five scale, fifteen items in reference to specific situations (evaluation, physical danger, strange environments, meeting people, and daily routines). Inter-item reliability is high, the situations are relatively independent of each other, and neurotic individuals report more anxiety than normal subjects on all situations except physical

danger (Endler, 1975; Flood & Endler, 1980). Normal and neurotic individuals report the same level of anxiety on the physical danger scale and this scale usually has the highest anxiety score (Endler, 1975).

Subjective reports of anxiety and psychological stress are not always reliable or accurate since the subject may not want to admit these feelings or may not be aware of them (Patkai, 1974). A subject may also respond to the 'demand characteristics' of the experiment and do what she or he thinks the experimenter wants (Orne, 1970). Therefore, additional self-report measures have been developed to assess the subject's defensiveness and desire for approval.

The Social Desirability Scale (SDS; Crowne & Marlowe, 1964) provides a valid measure of the individual's defensiveness and inclination to respond in a socially appropriate fashion. It is a 33 item, true or false, scale that has high test-retest and split-half reliability (Crowne & Marlowe, 1964).

Weinberger, Schwartz, and Davidson (1979) suggest using both the TMAS and SDS scores to determine differences in reporting anxiety. A two by two matrix can be tabulated consisting of low TMAS and low SDS subjects (true low anxious), low TMAS and high SDS subjects (repressor), high TMAS and low SDS subjects (high anxious), and high TMAS and high SDS subjects (defensive high anxious). Verbal reports obtained from repressor and defensive high anxious subjects should be assessed with caution since these subjects are less likely to respond honestly to subjective questionnaires related to anxiety than other subjects.

The individual experiencing psychological stress exhibits facial expressions similar to those of pain such as facial

grimaces, knotted brows, and downcast eyes (Izard, 1977). Performance on cognitive tasks during psychological stress has tended to support an inverted U-shaped relationship between stress and performance (Patkai, 1974).

Physiological indicants of state anxiety are similar to those associated with the pain response. These include measures of muscle tension, autonomic activity (such as heart rate), and endocrine reactions. With respect to EMG, the frontalis muscle group has been singled out as an area specifically sensitive to anxiety and arousal and can serve as a useful indicator of the general activity of the striate musculature (Everly & Rosenfeld, 1981).

In general, during psychological stress, measures of autonomic activity tend to increase. The heart rate measure is sensitive to differences in cognitive orientation to stressors (Barrell & Price, 1977) and to differences in ability to control a stressor (Obrist et al., 1978). Levels of endocrine activity are determined either by blood or urine analysis. The secretion of epinephrine is reported to be a sensitive indicator of the stress response (Patkai, 1974).

Physiological arousal can be generated by many different stimuli such as smoking, exercise, and intake of caffeine. Furthermore, correlations between physiological indices of stress tend to be low, suggesting that people differ in the pattern of activation of physiological systems (Patkai, 1974). The existence of low correlations between physiological measures also necessitates the assessment of more than one physiological variable to determine is physiological arousal occurred in response to a stressor (Patkai, 1974).

The confound in pain and anxiety assessment. This review of assessment issues in anxiety research reveals that anxiety and pain are assessed in a similar fashion. Sternbach (1984) reports that the overall pattern of the acute pain experience is an emergency response that is also seen in individuals experiencing anxiety attacks. Gross and Collins (1981) state that both acute pain and state anxiety share common adjective descriptors on self-report measures, that physiological data generally reflect activation of identical systems, that facial expressions in both are indicative of distress, and that complex task performance in both is usually impeded.

Unique patterns of arousal and behavior that discriminate between anxiety and pain have not been found (Gross & Collins, 1981). In addition, correlations between anxiety and pain intensity ratings during nociception tend to be high. Gross and Collins (1981) compared individuals with pain states to individuals with anxiety states on a variety of self-report measures. When responding to questions which measured anxiety, subjects with pain did not differ from subjects with anxiety. However, individuals with pain endorsed significantly more pain symptoms than individuals with anxiety and the severity of these symptoms was greater. Therefore, self-report measures remain the most useful discriminators between the experience of anxiety and pain.

Gross and Collins (1981) point out that research manipulating pain states and assessing changes in distress levels, behaviors, and physiological systems is measuring the anxiety component of the pain experience. It is possible that anxiety can be modified with little or no influence on direct measures of the pain

experience. Direct measures of pain include pain intensity, threshold, tolerance, and other subjective report and psychophysical methodologies that require the subject to report on some aspect of the pain being experienced.

The relationship between anxiety and pain

Nociception is a physical stressor that may evoke anxiety and psychological stress. When anxiety is associated with tissue damage, it is generally assumed that the severity of the pain is increased (Beecher, 1966; Merskey, 1980) as well as the frequency of pain complaints and other related behaviors (Melzack & Chapman, 1973). Prolonged psychosocial stress of a more general nature has been implicated as a factor in the exacerbation of acute pain states and length of recovery time after surgical and other medical procedures (Sternbach, 1974; Volicer, 1978a). Furthermore, the level of anxiety due to psychosocial factors or personality disposition that is present prior to nociception is thought to increase the subsequent pain experience (Sternbach, 1968). However, Gross and Collins (1981) caution "since anxiety and pain share identical features both in terms of assessment and treatment, statements concerning the role of anxiety in pain or pain in anxiety are generally confounded by the interrelationship of these two states" (p. 376).

It is important to acknowledge Cassel's (1982) criticism related to the separation of pain from the distress associated with it. The ethical and humane practitioner is devoted to reducing the suffering that patients experience in addition to determining and alleviating the cause of that suffering. Treatment studies and research aimed at finding ways to reduce anxiety and suffering are essential to improve the physician's

capacity to aid the patient's recovery. Ample evidence exists that reducing the anxiety associated with acute pain reduces drug requests and other pain behaviors, increases compliance with medical procedures, and speeds up recovery time (Hayward, 1975; Leventhal & Everhart, 1979; Melzack & Wall, 1982). Thus, reducing suffering must be considered an essential (yet seldom directly administered) part of the treatment process.

However, for purposes of brevity and clarity, this review of the relationship between anxiety and pain will consider only those studies which report a direct measure of pain.

Clinical studies. Volicer (1978a) measured the stress due to hospitalization in 535 medical and surgical patients. She correlated stress ratings with pain ratings taken both pre and post discharge, contfolling for age, seriousness of illness, and other relevant variables. Patients who scored high on hospital stress reported more pain, lower physical status during hospitalization, and less improvement after discharge than patients low in hospital stress. In addition, level of life stress prior to hospitalization was positively correlated with self-report of pain intensity during hospitalization.

Melzack, Taenzer, Feldman, and Kinch (1981) assessed the efficacy of prepared childbirth training in reducing labor pain as measured by the MPQ in primiparous and multiparous women. Prepared childbirth training usually consists of classes that instruct the mother in obstetrics, breathing, and relaxation exercises. Primiparas who received childbirth training showed significantly lower pain scores on both sensory and affective dimensions compared to primiparas who had not received childbirth training. Multiparas reported significantly less

pain on the MPQ than primiparas, but there were no differences between multiparas who had received childbirth training and those who did not. Melzack et al. (1981) emphasize that with or without training, labor pain is one of the severest forms of pain recorded by the MPQ.

Anecdotal evidence suggests that people who experience extremely high arousal (on the battlefield, in sports events, or dangerous situations) do not experience pain until these ongoing activities cease (Craig, 1984; Melzack & Wall, 1982; Merskey, 1980). In conclusion, the clinical evidence suggests that low levels of anxiety may reduce sensory and affective aspects of pain yet not abolish it, moderate to high levels of stress may increase pain intensity, and extremely high levels of arousal can abolish pain perception for a period of time.

Trait anxiety and pain. The individual's predisposition to be anxious has been implicated as a factor that may increase the severity of the pain experience. For instance, Spear (1967) noted that pain complaints in psychiatric wards are highest in patients suffering from depression, anxiety, and hysterical neuroses.

Dougher (1979) chose subjects who were high or low in anxiety on the basis of their TMAS scores using (approximately) the highest and lowest deciles. High trait anxious subjects had significantly lower pain thresholds than low trait anxious subjects when pain was produced by pressure stimulation. Six pain threshold determinations with two different weights were made for signal detection analysis. This analysis found that both of the groups were able to discriminate between the two weights in a similar fashion. However, the high

trait anxious subjects were more likely than low trait anxious subjects to label the different stimuli as painful.

In contrast, Von Graffenried, Adler, Abt, Nuesch, and Spiegel (1978) found no relationship between scores on the TMAS and VAS ratings of pain intensity during ischemic arm pain.

To conclude, inconsistent results have been reported concerning the relationship between trait anxiety and pain. When subjects at extreme ends of the trait are being compared, high trait anxious subjects appear to be more reactive to nociceptive stimuli than low trait anxious subjects. In addition, Weisenberg, Wolf, Mittvoch, Mikulincer, and Aviram (1985) reported that trait anxiety can interact with the experimental situation to produce different effects on the pain experience. In this study, individuals with high trait anxiety scores on the STAI reported higher pain intensity ratings on a VAS than low trait anxious subjects when the shocks were predictable but not when the shocks were unpredictable.

State anxiety and pain. Three different research strategies have been employed to study the effects of state anxiety on pain perception. The first involves assessing the level of state anxiety through psychometric instruments and then determining the relationship between anxiety scores and different pain parameters. The second strategy employs experimental manipulations that increase anxiety, then assesses the subsequent pain experience. The third approach uses techniques that reduce anxiety or arousal and then assesses the pain experience.

Unde, Slever, Post, Jimerson, Boulenger, and Buchsbaum (1982) assessed state anxiety using the STAI and applied signal detection theory (SDT) analysis to shock intensity ratings

that centered about the pain threshold level. They reported that the higher the state anxiety scores, the lower the ability of subjects to discriminate between shock intensities.

Brown, Fader, and Barber (1973) obtained a state anxiety scale from the Multiple Affect Adjective Checklist - Today Form. They reported no correlation between anxiety scores and pain threshold, tolerance, or intensity ratings in either pressure or cold pressor pain conditions. In addition, Von Graffenried et al. (1978) found no correlation between state anxiety (the questionnaire is not named) and the time to reach 75 on a zero to 100 VAS during ischemic arm pain. However, the sooner the subjects reached 75, the more likely they were to report higher anxiety over the second and third trials of ischemic pain.

This research indicates that scores on state anxiety questionnaires are not particularly useful to predict pain threshold, tolerance, or intensity ratings.

Of the two types of experimental manipulations employed to increase state anxiety, the most commonly used stressor involves creating conditions that threaten the subject's physical integrity. Threats to psychological integrity have not been frequently employed.

Threats to physical integrity can be manipulated by providing instructions that emphasize the strength of the stimulus or the painfulness of the upcoming stimulus. Hall and Stride (1954; cited in Melzack & Wall, 1982) reported that using the word "pain" in the instructions given to psychiatric patients who received electric shock stimulation was associated with reports of higher pain intensity levels than when the word "pain"

was absent. Sternbach (1968) found that magnitude estimates of electric shock intensity were significantly higher in subjects told to expect an increase in shock intensity level compared to subjects who were told to expect the same shock intensity.

Haslam (1966) emphasized the importance of assessing the subject's appraisal of these instructions to determine if they were actually stress-provoking. He reports that only subjects who stated that they were anxious about the probability of receiving a strong shock during radiant heat nociception revealed a significantly lower radiant heat pain threshold. However, Malow (1981) produced anxiety (as measured by verbal, physiological, and combined indices) by threat of shock during pressure pain — and found no differences in pressure pain threshold between threat and no threat conditions. SDT analyses found that the threat impaired the ability of anxious subjects to discriminate between different pressure weights. The subjects who showed both verbal and physiological indices of anxiety also showed an increased tendency to report pain.

In a related study, Nisbett and Schachter (1966) reported that subjects given attribution of arousal instructions and a pain warning did not show the same increase in pain tolerance as the subjects given only the attribution instructions.

In conclusion, there is relatively strong evidence that when anxiety associated with physical integrity is manipulated through instructions about the strength or painfulness of a future nociceptive stimulus, pain perception and experience are intensified. The mechanisms that may be responsible for this effect will be discussed in the next section.

A paucuty of studies have manipulated threats to psychological

integrity to assess the relationship between this type of anxiety and pain. Weisenberg, Aviram, Wolf, and Raphaeli (1984) compared two types of anxiety in a study using four electric shocks. All subjects received a serial anticipation learning task and were instructed to watch two rows of lights during the experiment. Subjects received one of five instructions: 1) High anxiety pain focus; subjects were instructed to pay attention to the lights and for safety purposes to immediately report if the red light went on; 2) High anxiety task focus; subjects were provided instructions that the learning task was a dynamic measure of intelligence; 3) Combined high anxiety focus; subjects received both sets of instructions; 4) Low anxiety pain focus; subjects received instructions about the shock's safety and were asked to focus on the lights to answer questions about them; and 5) Low anxiety task focus; subjects were told to do well on the task. Anxiety intensity was rated on a VAS after the first and third shocks and pain intensity was rated on the VAS after the second and fourth shocks. Heart rate (HR) and skin conductance (SR) were monitored as well as task performance.

In general, the results from the postexperimental questionnaires indicated that the combined and high anxiety pain focus groups reported the highest fear of shock and the high anxiety task focus reported the lowest fear of shock. The VAS anxiety ratings indicated that the three high anxiety groups reported significantly more anxiety than the low anxiety groups. The anxiety task focus group made the most errors compared to other groups on the task. These measures suggest that the anxiety manipulations were effective yet HR did not differ

between groups and SR was lowest in the high anxiety task focus. There were no differences between groups on the pain intensity ratings obtained after the shocks yet there was a trend for the combined group to report the highest pain.

Therefore, moderate levels of self-reported anxiety were created in the anxiety condition yet this anxiety did not appear to influence pain intensity. It is important to note that subjects reported the shock intensity as relatively mild, averaging from 20 to 40 on the zero to 100 VAS. A floor effect may have been operating in which relatively mild nociception is not susceptible to these types of anxiety manipulations.

Mayerson and Rhodewalt (1984) reported that subjects given negative performance feedback on verbal intelligence tests reported higher pain intensity ratings during cold pressor pain than subjects given positive performance feedback. However, these authors were not investigating the relationship between anxiety and pain and thus, the presence of increased anxiety in the negative feedback group compared to the positive feedback group was not adequately assessed.

In conclusion, the two studies that manipulated threats to psychological integrity and obtained pain intensity ratings reported different results. It is possible that these manipulations affect the perception of strong nociceptive stimuli (such as cold pressor) but not milder nociceptive stimuli. In addition, the effects that these manipulations have upon other direct measures of the pain experience such as tolerance or the MPQ has not been assessed.

The last area of research assessing the relationship between

anxiety and pain involves reducing anxiety through the use of anxiolytics, relaxation, and other arousal reduction techniques. Anxiolytics have been used to reduce anxiety in acute pain states and include diazepam, chlordiazepoxide, and meprobamate (Pert, 1980).

Chapman and Feather (1973) reported that diazepam reduced state anxiety (on a five word category scale) scores over time during ischemic arm pain and increased tolerance time compared to a placebo. In a second study, they reported that diazepam increased pain tolerance to ischemic pain compared to aspirin, but there were no differences in state anxiety scores. In a third study, SDT analysis indicated that neither placebo nor diazepam affected the ability of the subjects to discriminate between radiant heat stimuli of varying intensities or the willingness of subjects to label various intensities as painful. Stern, Brown, Ulett, and Sletten (1977) reported that the pain ratings obtained on a zero to four category scale were not affected by diazepam, placebos, or aspirin during either cold pressor or cuff pain (pressure produced by inflating a blood pressure cuff to 300 mG).

Relaxation training and transcendental meditation reduce anxiety and physiological arousal (Hoffman et al., 1982; Shapiro, Schwartz, Ferguson, Redmond, and Weiss, 1977). Bobey and Davidson (1970) reported that presenting a relaxation tape prior to radiant heat or pressure nociception produced the highest tolerance level during both stimuli in comparison with a cognitive rehearsal tape, a 'stress' tape of women in labor, and a control tape.

Mills and Farrow (1981) compared the responses to cold

pressor pain of advanced meditators with control subjects. Pain intensity ratings did not differ between groups but pain tolerance was significantly greater and distress ratings were significantly lower in the meditators compared to the control group.

Clum, Luscomb, and Scott (1982) selected subjects for moderate to high levels of anxiety associated with painful stimulation. Subjects were given relaxation training, relaxation instructions, or a cognitive strategies package to use during ischemic pain after establishing a baseline level of anxiety and obtaining a cross-modality matching estimate of ischemic pain. Relaxation training reduced distress ratings during ischemic pain and the measure of pain intensity obtained after nociception. Cognitive strategies reduced pain as measured by cross-modality matching. Relaxation instructions did not produce any significant effects on these measures.

In conclusion, anxiolytics, relaxation, and meditation increase pain tolerance relative to control conditions yet have inconsistent effects on pain intensity ratings.

Conclusions. This section reviewed the influence that anxiety and psychological stress have upon the pain experience. It was argued that anxiety and pain share many identical assessment features and much of the research data has inferred pain reduction when in fact, anxiety was being influenced. However, reducing the suffering and anxiety associated with pain is an essential factor in improving patient care. It was also stated that stress from sources other than nociception can increase the distress associated with it. Nevertheless, this review focussed on the research that directly assessed the

pain experience.

Clinical and anecdotal evidence indicates that low levels of anxiety can reduce the sensory and affective aspects of pain, moderate to high levels can increase pain, and extremely high levels of anxiety and arousal can prevent the perception of pain for a period of time. Trait anxiety, as assessed by psychiatric diagnosis or psychometric methods appears to affect pain perception if extremely high and low trait anxious individuals are being compared. There is not enough evidence to state that scores on state anxiety questionnaires are related to the subsequent pain experience.

When levels of state anxiety and psychological stress are manipulated, the effect on the pain experience depends on both the nature of the manipulation and the pain assessment measures. Increasing the anticipatory stress associated with nociception increases pain on a number of response parameters. When creating state anxiety by threatening the psychological integrity of the subject, inconsistent effects upon pain intensity have been reported. The effect that threats to psychological integrity have upon pain threshold, tolerance, the MPQ, and other assessment methods has not been investigated. Anxiety reduction techniques increase pain tolerance but there is no strong evidence that this type of manipulation influences other pain parameters.

Theories of anxiety's effects on the pain experience

Melzack and Wall (1982) discuss two ways that anxiety can affect the pain experience. First, it can open the "gate" at the level of the first transmission cell; that is, anxiety can facilitate firing of the transmission cells at the spinal cord.

Second, within higher cortical structures, the "motivational affective" system interacts with central control processes so that 1) the "sensory-discriminative" system is influenced, or 2) the gate mechanism at the spinal cord level is affected. This is a more sophisticated and accurate neurophysiological model than traditional medical ones (see Guyton, 1981; Cassem, 1983) which assume that anxiety is a reflexive response evoked by "pain" impulses. However, this proposal does not offer a means of predicting when anxiety will affect pain at a perceptual level. It offers a useful explanation of how the pain experience was modified yet cannot predict when this will happen. To predict this effect, reference must be made to social-psychological mechansisms.

The variety of experiences subsumed under the label "anxiety" interact in different ways with the variety of experiences subsumed under the label "pain". For clarity, measures of the pain experience will be divided into two categories; direct and indirect. Direct measures include pain threshold, tolerance, and intensity ratings, signal detection methodologies, and multiple adjective checklists such as the MPQ. Indirect measures are usually referred to as pain behaviors and include pain complaints, distress, suffering, medication requests, length of recovery time, activity level, and task performance.

Anxiety can also be classified into two categories; anxiety that is directly related to the pain experience and anxiety that is not. Weisenberg et al. (1984) referred to these two types as relevant and irrelevant anxiety. Relevant anxiety includes the anticipatory psychological stress associated with tissue damage, hospitalization stress, and general fears of physical

trauma. These factors are present prior to nociception. Once nociception occurs, relevant anxiety refers to the perception that the stimulus constitutes a threat to the individual's physical and psychological integrity. Irrelevant anxiety includes trait anxiety, interpersonal conflicts, occupational stress, performance anxiety, and other types of anxiety that are evoked from sources not related to the nociceptive input.

Cassel (1982) discusses the relationship between relevant anxiety and pain behavior. Pain behavior is evoked by the perception that the nociceptive stimulus produces a threat to both physical and psychological integrity. This perception can arise from a host of factors that include when the person feels that there is nothing he or she can do to cope with the experience, when the pain is overwhelming, when the source of the pain is not known, when the meaning of it is dire, and when the pain is chronic. The threat to physical and psychological integrity creates suffering and suffering creates a strong motivational drive to obtain relief. Cassel (1982) believes that the presence of relevant anxiety predicts the occurrence of suffering and other pain related behaviors.

Weisenberg et al. (1984) have proposed a more general theory of the relationship between anxiety and pain. They believe that relevant anxiety increases the pain experience in general but irrelevant anxiety does not. These authors propose that pain is influenced by relevant anxiety (but not irrelevant) because individuals attribute their anxiety to the nociceptive stimulus when it is relevant but do not make this attribution when the anxiety is irrelevant.

These theories have intuitive appeal since it is apparent

that "knowledge of anxiety level per se is not adequate to predict the reaction to pain" (Weisenberg et al., 1984, p. 372). In addition, a number of research investigations discussed earlier support the hypothesis that relevant anxiety increases the pain experience when assessed by both direct and indirect measures.

However, irrelevant anxiety also increases direct and indirect assessment measures. Volicer (1978a) found the level of life stress prior to hospitalization was positively correlated with pain intensity ratings during hospitalization as well as being correlated with several pain behaviors. Extremely high trait anxious individuals tend to give higher pain intensity ratings and show more pain behaviors than extremely low trait anxious individuals. There is also some evidence that evaluation threats can increase the pain experience. Furthermore, it is difficult to argue that the efficacy of anxiety reduction techniques is due to reducing relevant yet not irrelevant anxiety.

To conclude, there is a good deal of evidence to propose that when relevant anxiety is increased or decreased, the pain experience is also increased or decreased. However, the pain experience can also be influenced by irrelevant anxiety. Less research has been conducted to investigate this effect and, therefore, no further conclusions can be drawn.

Chapman (1978) states that anxiety becomes associated with pain when "ongoing perceptual routines are disrupted by the occurrence of pain" (p. 199). If the disruptive effects that nociception has upon attentional mechanisms can be reduced, the anxiety and distress associated with pain will also be reduced. He gives evidence that virtually all manipulations to date that

have modified the pain experience act by either increasing or decreasing the disruptive and disorganizing effect that nociception has upon other ongoing cognitive activities. Mandler (1984) states that when no response is available to terminate this interruption, anxiety, distress, and fear will arise.

The reverse of this theory may also be useful to explain the influence that anxiety has upon pain. That is, the pain experience is modified when the perceptual routine of nociceptive stimuli is disrupted by the experience of anxiety. The experience of anxiety (relevant and irrelevant) prior to and concurrent with nociception can disrupt the individual's ability to process nociceptive input accurately and reliably. The disruptive effects of anxiety impede the person's attempts to evaluate incoming stimuli in reference to its salience, sensory qualities, harmfulness, and so on. Mandler (1984) postulates that psychological stress gives rise to internal autonomic signals. These signals require some conscious capacity and thus, they interfere with ongoing perceptual and cognitive routines. While this interruption continues, attention and processing of certain aspects of the situation is reduced. The aspects to which less focal attention is paid are those that initially attracted a lesser degree of attentional focus. If no available thought or action is available to handle the situation, anxiety will arise maintaining a high level of arousal and reduced ability to process perceptual information. In brief, anxiety disrupts the individual's ability to effectively analyze nociceptive information.

This modified version of Chapman's (1978) and Mandler's

(1984) "perceptual disruption" hypotheses predicts that if low levels of anxiety and stress are present, the evaluation of sensory events is unaffected; if moderate to high levels of anxiety are present, this analysis becomes disrupted and the scores from pain assessment measures would become more variable. Also under these circumstances, individuals would tend to experience more pain and distress and become more suggestible while they were sorting out different sensations and emotions. Extremely high anxiety could totally disrupt the experience and processing of sensory information since cognitive processes would be absorbed elsewhere. This effect would become more pronounced as the strength of the nociceptive stimulus increased. As this input increases, there would be more disruption of ongoing perceptual routines creating new sources of anxiety which would further impede evaluative processes.

Attribution theory can help explain how individuals sort out the different sensory and emotional experiences that are co-occurring during the combined experiences of anxiety and pain. When the individual attributes the source of anxiety to the nociceptive input, a motivational drive to stop this input would be created and pain behaviors would ensue. When the individual attributes the feeling of anxiety to a different source, pain behaviors may be attenuated. However, the direct measures of pain would remain more variable and indicate a higher severity due to the disorganizing effects of the experience of anxiety. Furthermore, if the anxiety is reduced by the attribution, the individual's ability to effectively evaluate incoming stimuli would be restored.

There have not been any direct empirical investigations of

the present author's modified version of the perceptual disruption hypothesis. SDT analyses of the relationship between anxiety and pain indicate that as anxiety increases, the ability of the subject to discriminate between different intensities becomes impaired (Unde et al., 1982; Malow, 1981). Low levels of anxiety produced by relaxation and other anxiety reducing techniques do not impair this ability (Chapman & Feather, 1973). Dougher (1979) did not find this effect when comparing high and low trait anxious subjects yet this finding may be due to the fact that only one stimulus pair was employed in his study. Schumacher and Velden (1984) report anecdotal evidence that one extremely anxious subject was virtually unable to discriminate between electric shock intensities.

Numerous social and psychological factors other than the experience of anxiety affect the pain experience. Only a handful of studies have controlled for these variables. In addition, very few studies have made the distinction between relevant and irrelevant anxiety and only one study has made the distinction between direct and indirect measures of pain (Gross & Collins, 1981). Therefore, it is not possible to evaluate this theory or any other theory which addresses relationship between pain and anxiety on the basis of the existing evidence.

Design and goals of the present study

The present study was designed to assess the relationship between anxiety and pain by comparing the effects of relevant and irrelevant anxiety on a number of direct and indirect measures of pain. In addition, it was designed to control for a number of social and psychological variables that can influence

the pain experience.

The major objective of this study was to compare Weisenberg et al.'s (1984) attribution theory with the present author's modification of Chapman's (1978) and Mandler's (1984) perceptual disruption hypothesis. Both theories agree that the experience of anxiety during pain is not a reflexive response of the nervous system. The attribution theory proposes that relevant anxiety will increase the pain experience whereas irrelevant anxiety will not. The perceptual disruption theory predicts that as anxiety of any type increases, direct measures of the pain experience will increase particularly if the nociceptive stimulus is relatively intense. Attributions of anxiety to or away from the nociceptive input would cause an increase or decrease on indirect pain measures.

Sixty-four male university students were paid to participate in the present study. Immediately before the experiment, each subject completed questionnaires that assessed relevant demographic data, reporting style, and levels of state, trait, and situational anxiety. After the experiment, each subject was interviewed to measure the qualitative aspects of the experience. Throughout the experiment, during Baseline, Pain, and Recovery periods, continuous measures of heart rate (HR), frontalis electromyographic activity (EMG), and pain and stress intensity ratings (using a VAS) were recorded. One pain threshold and one tolerance rating was obtained during the Pain period. Facial expressions were videotaped from the seventh and eighth minutes of Baseline, the first and last minutes of the Pain period, and the third and fourth minutes of the Recovery period.

Psychological stress was manipulated by using different instructions prior to the application of the nociceptive stimulus (pressure pain applied to the index finger). Sixteen subjects were told that the subsequent experience could be dangerous (Pain Plus Pain Warning; P+PW). Sixteen subjects were told that their behavior was being monitored and that immediately after the pressure they would receive a stressful interview related to this evaluation (Pain Plus Stress Interview; P+SI). The Pain Only (PO) group consisted of sixteen subjects who were not given an explicit warning. A fourth group of sixteen subjects received the stress interview instructions without receiving pressure pain to verify the stress-producing effect of these instructions. Numerous research projects have indicated that a pain warning increases self-report of anxiety and arousal (see previous section). Therefore, there was no comparison group employed for this condition.

Direct measures of the pain experience that were obtained in this study included the VAS of pain intensity which was recorded at 60 second intervals, pain threshold, tolerance and endurance, the MPQ, and the Gracely descriptor scales. These two detailed scales were completed immediately after the recovery period. Indirect measures of the pain experience included the VAS of stress intensity which was recorded at 60 second intervals, HR, EMG, and facial expression. These indirect measures of the pain experience were considered to be indicative of the experience of stress and anxiety that was occurring during the experiment and, therefore, could not be considered to tap only the experience of pain.

It was hypothesized that all groups that received pressure pain would report that the stimulus was painful. However, only the P+PW and P+SI groups would show increased anxiety and arousal during the initial stages of nociception. This would be due to the stress-provoking effects of the instructions.

If the attribution theory is correct, relevant anxiety (but not irrelevant) affects the pain experience. Therefore, the P+PW subjects (and not the P+SI subjects) would show increased responding on the direct and indirect measures compared to the PO subjects.

If the modified perceptual disruption theory is correct, both the P+PW and P+SI groups would show increases on the direct pain measures relative to the PO group. Relevant anxiety would influence the indirect measures of pain. Thus, the P+PW subjects would manifest increased HR, EMG, and stress intensity ratings throughout the pain period and increased facial expressions of distress when compared to the PO subjects. The responses from the indirect pain measures obtained from the P+SI subjects would show a decrease from the beginning to the end of the pain period since the source of the anxiety is not related to the nociceptive stimulus.

This study was also designed to address several subsidiary goals. A comparison of subjects who reported tolerance during pressure pain to subjects who did not was conducted. Finally, since few studies have employed a multimodal assessment of pain and anxiety, the relationships between the measures were also examined.

METHOD

Subjects

Sixty-four male English speaking McGill University students ranging in age from 18 to 34 years (mean = 21 years) participated in the present study. They were recruited through notices placed in the University Student Center, Engineering Faculty, and Psychology Department. The notices briefly described the nature of the experiment and offered $10 for participation (see Appendix A). Seventy individuals contacted the experimenter and received a participant's manual that gave a detailed rationale for the study and described what was expected of them (see Appendix A). Sixty-five students agreed to participate after reading the manual. Three could not due to scheduling conflicts; two would not due to the nature of the experiment. Potential subjects were screened for the existence of medical problems. One individual was excluded due to high blood pressure. Prior to the study, each subject was asked to read and sign an informed consent agreement (see Appendix A).

The first 48 subjects were randomly divided into the three groups entitled Pain Only, Pain Plus Pain Warning, and Pain Plus Stress Interview Warning. The last 16 subjects were assigned to the fourth group, Stress Interview Warning Only. The mean ages for each group were 21, 20, 21, and 21 years respectively.

Apparatus

The experiment was conducted in the Clinical Psychophysiology Laboratory of the McGill University Department of Psychology.

This laboratory consists of two adjoining rooms; an Experimenter room and a Subject room, connected by a door and a one-way mirror.

Each subject remained in the Subject room for the duration of the experiment. This room was 3.05 m by 4.27 m, sound attenuated and lit by fluorescent overhead lights. The brightness of the lights was controlled by the experimenter using a rheostat set at five eighths of full position for all subjects (35 cd/ft^2).

The temperature of both rooms was set by the experimenter at 24°C. The amount of electrical interference or 'noise' was assessed using the procedure recommended in Section 6.8 of the Grass Instruments Polygraph Manual for Polygraphs 7 and 78 (Grass Instruments Corporation, Quincy, Mass.) for detection of artifacts and microphonics. This test indicated an absence of perceptible electrical interference in the Subject room.

The subject room (see Appendix A) was furnished with tables and chairs placed against opposite walls and a file cabinet in the corner of the room. On top of the file cabinet was a painted wooden box which housed the video camera. An electrovoice 635A Dynamic Omnidirectional microphone (impedance = 150 ohms) was suspended from the wall flush with the one-way mirror. This microphone was used to allow subjects to communicate with the experimenter during each session.

Each subject used the table and chair opposite the one-way mirror before and after the experiment to complete questionnaires. During the experiment, each subject was seated in a padded chair facing the file cabinet in a position so that the aperture of the video camera was 3.05 m from the top of the back of the chair.

The upper third of the subject's body could be videotaped via the camera and the subject's entire body could be seen in profile from the one-way mirror.

Binding posts, consisting of two Grass Model 7P511 input cables (Grass Instruments Corp., Quincy, Mass.), were attached to the back of the padded chair to obtain the chest electrocardiogram (EKG) and frontalis electromyographic activity (EMG), and for the placement of a ground electrode. The cables (and all others leading from the Subject to the Experimenter room) ran through a 20 cm by 20 cm hole at the bottom of the wall behind the padded chair. These two cables were attached to the polygraph in the Experimenter room.

The padded chair included a desk top which contained the pain/stress rating box on the right side and the strain gauge pain stimulator on its left. The surface of the rating box and bottom half of the pain stimulator were flush with the desk surface and at a distance that allowed the subject to rest each arm on the desk top.

The metal rating box (see Appendix A) consisted of a modified telephone touch pad (numbered from one to ten with the push buttons flush with the box top and the letters removed from the buttons) and two light emitting diodes (LEDs). The left LED was marked 'PAIN' and the right LED was marked 'STRESS'. During the rating procedure, both LEDs flashed on and off, after which the LED marked 'PAIN' remained lit until the subject rated his level of pain on a one to ten scale. Following this rating, the second LED marked 'STRESS' turned on and remained lit until the subject has rated his level of stress on a one to ten scale. The rating box was connected to a rating/tone generator control

unit in the Experimenter room by way of a 16 pin connector cable. The timing of the LEDs was set to one-minute intervals by the control unit.

At the beginning of each pain and stress rating period, the subject also heard one 0.5 second duration tone which was delivered through Sony Stereo Headphones Model D5-5A (8 ohm impedance, 1 mV input for right and left ears) at 50 dB. These headphones were also used for delivering taped instructions throughout the experiment.

The left side of the desk contained the strain gauge pressure pain stimulator. This apparatus was specially constructed for the study following the procedure detailed by Forgione and Barber (1971). This apparatus was composed of a bridge transducer (FT-10, Grass Instruments Corporation., Quincy, Mass.) mounted in a solid aluminum handle. The handle was attached to a base housing a space for the subject's finger. This space could be adjusted for finger size and length to allow exact placement of a wedge midway between the end of the fingernail bed and the first knuckle of the left index finger. A lucite wedge (1.5 cm wide and 6 mm thick tapering at the tip to 0.25 mm thick) was attached to the top of the handle at the transducer output and connected to the polygraph. The apparatus was designed to produce a pressure of 2000 g at the focal point when a 40 g weight was attached to the end of the handle. This particular apparatus and application was chosen through a series of pilot studies ($\underline{n}$ = 6, $\underline{n}$ = 5, and $\underline{n}$ = 10) with subjects recruited in the same fashion. Different placement sites and weights were applied until a procedure was found in which 60% of all the subjects reported pain tolerance during a ten minute exposure

period.

The Experimenter room contained the polygraph, videorecorder and video monitor, reel-to-reel tape recorder, cassette recorder, and control unit. The experimenter, sitting in the Experimenter room, could see the subject at all times.

During the experiment, psychophysiological responses were monitored continuously using a Grass Instruments Model 7D Polygraph (Grass Instruments Corp., Quincy, Mass.). A record of physiological responses was recorded on standard polygraph chart paper. The chart speed was 3 mm per second. Electrodes for monitoring heart rate (HR) and EMG, and for grounding were Medi-Trace 1801 Ag/AgCl pre-gelled disposible Pellet Electrodes (Graphic Controls Canada Ltd., Gananoque, Ontario). These electrodes were attached to 24 inch (63.5 cm) snap connectors (5524S; Graphic Controls Canada Ltd., Gananoque, Ontario) and connected to the cables attached to the binding posts of the chair. The recording sites were cleaned with isopropyl alcohol to reduce interelectrode resistance prior to electrode application. The ground electrode was attached to the back of the right elbow above the joint.

To monitor HR, two electrodes were applied in a modified Lead II configuration. One electrode was applied one cm above the subject's right clavicle and the second electrode was applied to the left lateral side of the chest one cm below the sixth rib. Signals from the chest leads were recorded on the polygraph using a Grass Polygraph DC Driver Ampilfier 7DA and a Grass Polygraph EKG Preamplifier and Tachograph 7P4D (Grass Instruments Corp., Quincy, Mass.). The driver amplifier was calibrated by the standard procedure; 100 mV = 2 cm at 75 Hz half amplitude

high frequency and with 60 Hz signals filtered out. The preamplifier was calibrated at 1 mV = 1 cm and with 0.03 Hz half amplitude low frequency filter setting. This was set to 1 Hz half amplitude low frequency filter for recording as recommended by the Grass polygraph manual for the 7P4D (Grass Instruments Corp., Quincy, Mass.). This procedure produced a record of each heart beat. Pulse rate was recorded using the tachograph curcuit of the EKG preamplifier attached to a second Grass Polygraph DC Driver Amplifier (Grass Instruments Corp., Quincy, Mass.) with input from the tachograph leading to the J1-J2 jack of the driver amplifier. The tachograph was triggered by the EKG 'R' wave as it was processed by the amplifier unit of the 7P4D. The trigger mechanism was set in the AC - fast position and the trigger threshold was adjusted to filter out low frequency activity of the signal and to allow only high frequencies (that is, the 'R' wave) to trigger the tachograph. The measurement range was set to 40 - 120 beats per minute. The tachograph provided beat-to-beat changes of rate. Using two channels of the polygraph allowed simultaneous recording of both EKG and pulse rate.

Raw and integrated EMG were recorded from the frontalis region. Two electrodes were applied to the subject's forehead midway between each eyebrow and the hairline as described by Basmajian and Blumenstein (1980). Signals from this area were recorded on the polygraph using a Grass Polygraph DC Driver Amplifier 7DA and a Grass Polygraph Wide Band AC Preamplifier and Integrator 7P3BC (Grass Instruments Corp., Quincy, Mass.). Standard calibration (100 mV = 2 cm) was used on the driver amplifier at 75 Hz half amplitude high frequency setting and

with 60 Hz signals filtered out. Raw EMG was recorded using the preamplifier calibrated at 200 uV = 2 cm and 0.03 Hz half amplitude low frequency setting. This was set at 3 Hz half amplitude low frequency for recording as recommended by the Grass Polygraph manual for the 7P3BC (Grass Instruments Corp., Quincy, Mass.). The integrator was employed to display integrated EMG (an average level of the raw EMG) and calibrated at 200 uV = 2 cm. Full wave rectification was used and signals were integrated over a 0.2 second time period. Output from the integrator was recorded on a separate channel of the polygraph using a second Grass Polygraph DC Driver Amplifier 7DAF (Grass Instruments Corp., Quincy, Mass.) with standard calibration, 75 Hz half amplitude high frequency filter setting and with 60 Hz signals filtered out. Thus, a record of both raw and integrated EMG was obtained simultaneously.

To provide an exact record of the time course of pressure stimulation and of any variations in pressure, a Grass Low-Level DC Preamplifier 7P1 for DC Potentials and Transducers and a Grass Polygraph DC Driver Amplifier 7DAF were used (Grass Instruments Corp., Quincy, Mass.). The shielded cable attached to the transducer of the nociceptive apparatus led into the input of the preamplifier. The driver amplifier was calibrated in the standard fashion using 75 Hz half amplitude high frequency and with 60 Hz signals filtered out. The preamplifier was calibrated following the procedure recommended by the Grass polygraph manual for 7P1 - strain gauge amplifiers - at an impedance of 2K ohms (Grass Instruments Corp., Quincy, Mass.). The baseline was set to equal 2000 g and scaled to print a 1 mm excursion per 10 g - change in pressure. Thus a recording range of 1800 to 2000 g

was possible.

The pain/stress rating box was connected to the control unit in the Experimenter room which controlled the timing and delivery of both the LED and tone prompts to each subject. In addition, it gave an LED display of the subject's ratings on two separate one to ten scales. This display remained visible for 20 seconds to allow the experimenter to record the ratings. The control unit was connected to one channel of the polygraph. The input cable was plugged into the J6A jack of a Grass Polygraph DC Driver Amplifier 7DAF (Grass Instruments Corp., Quincy, Mass.). Each time a tone was generated, a downward deflection appeared on the polygraph record providing a means of correlating subjective and physiological responses over time.

The subject's verbal pain threshold and tolerance responses and his responses to taped questions were monitored from the microphone in the Subject room via a videorecorder and a television monitor. A videocamera was housed in a specially constructed compartment on top of the file cabinet so that only the aperture was visible. This camera was used to create a videotape of each subject's upper body and facial movements during the experiment. Each tape was synchronized by shining a flashlight through the one-way mirror immediately after the seventh baseline tone was delivered as well as immediately after the third minute of recovery (application and removal of the pain apparatus was automatically recorded on the videotape).

The equipment used to deliver the taped instructions included a cassette recorder and a four track reel-to-reel stereo tape recorder. The cassette recorder was used to play the taped instructions which had been recorded by a male university student.

Connections were made between the cassette recorder, reel-to-reel tape recorder, and rating control unit to allow the experimenter to hear the instructions and tone prompts as they were being delivered. A digital stopwatch was used to provide an additional measure of pain threshold and tolerance.

Questionnaires

Subjects completed questionnaires before and after the experiment. Copies of all original questionnaires are included in Appendix B. Preexperimental questionnaires were designed to assess demographic variables, presence of state and trait anxiety, types of responses elicited by anxiety provoking situations, and the importance of socially desirable behavior. All subjects received these preexperimental questionnaires in the following order:

1) Demographic questionnaire: Age, citizenship, language, religion, father's and mother's occupation, height, and weight.

2) State-Trait Anxiety Inventory - State measures (Spielberger, Gorsuch, & Luschene, 1970).

3) Taylor Manifest Anxiety Scale (Taylor, 1953).

4) Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960).

5) Stimulus-Response Inventory of General Trait Anxiety - Revised; two subtests named Physical Danger and Evaluation Anxiety (Endler & Okada, 1975; Flood & Endler, 1980).

6) Present stress - 10 cm visual analog scale anchored by the terms 'No stress', 'Moderate stress', and 'Extreme stress'.

Postexperimental questionnaires were designed to obtain the subject's level of stress, a description of the pain experience, and other comments about the study. Subjects in

~~the three~~ groups receiving pressure pain completed these questionnaires:

1) Gracely's Pain Descriptors (Gracely, 1980).

2) McGill Pain Questionnaire - Part 2 (Melzack, 1975) with modified instructions to describe the pain experienced during the experiment.

3) State-Trait Anxiety Inventory - State measures (Spielberger, Gorsuch, & Luschene, 1970).

4) Present stress - visual analog scale.

5) Post Laboratory Phase Interview - questions about the representativeness of the pain and the worst pain experienced in the past.

6) Subjective Impressions Questionnaire - questions asking about strategies employed to cope with the pain, belief that the stimulus would be painful, possible hypotheses, and whether or not friends had participated.

Subjects in the group receiving the stress interview warning but not pressure pain completed these postexperimental questionnaires:

1) State-Trait Anxiety Inventory - State measures (Spielberger, Gorsuch, & Luschene, 1970).

2) Present stress - visual analog scale.

3) Subjective Impressions Questionnaire - questions asking about how they felt before the stress interview, belief that the interview would be stressful, possible hypotheses, and whether or not friends had participated.

Behavioral response data

The data obtained from the videotapes (referred to as the behavioral response data) were analyzed in the following manner.

Two independent raters, blind to group membership and to the experiment's hypotheses, viewed six tapes selected at random from those subjects who had received pressure pain. The raters recorded the subjects' behaviors (see Appendix A). From this list, four general categories of behaviors were specified; head movements, mouth movements, body movements, and facial grimaces. These categories were divided into 14 specific behaviors and were operationally defined (see Appendix C).

The behavioral response data were divided into six 60 second intervals. These intervals were chosen prior to conducting the study as the most relevant for subsequent analyses and the remaining videotape data was not rated. The measurement periods consisted of the seventh and eighth minute of baseline, the first and last minute of pressure pain, and the third and fourth minute of recovery. The tapes were rated in terms of frequency of each movement per 30 second interval.

A trained rater, blind to group membership and experimental hypotheses, rated 43 videotapes (only the tapes from subjects receiving pressure pain were analyzed). Five tapes were unusable due to equipment failure. The first rater-trained a second blind rater to record frequency of movement. The second rater viewed six tapes selected at random and rated each for frequency of movement. The percentage agreement between these two raters in terms of the occurrence and nonoccurrence of the 14 behaviors ranger from 89% to 95% on the six tapes with a mean percentage agreement of 91%. Cohen's kappa coefficient of agreement (1960) was also calculated to provide a more conservative estimate of the agreement between the two raters. This statistic allows a researcher to partial out the amount of agreement due to chance

(Cohen, 1960). The kappa coefficients ranged from .78 to .94, with a mean of .83.

Instructions

The instructions used in the present study to produce a stressful experience were selected through a series of pilot studies (n = 4, n = 4, and n = 18) utilizing various types of instructions. The criteria employed to choose the final versions of these instructions were increases in HR and self report of stress intensity when compared to control subjects. Given the importance of the instructions in this experiment, most of the instructions are reproduced in full for each group in the next section.

Procedure

Table 1 presents a chronology of the experimental procedure indicating the similarities and differences among groups. Subjects participated in the experiment between the hours of 10 a.m. and 6 p.m. from August, 1983 to April, 1984. One female experimenter (the author) operated the equipment and recorded the responses of all subjects. The experimenter's interaction with each subject was kept to the minimum required to give each subject information and instructions regarding the experiment.

In order to reduce physiological variability, each subject was given the following instructions prior to participating in the study:

1) No consumption of coffee or other caffeinated beverages, nor cigarettes for one hour prior to the experiment;

2) No drugs or alcohol for 24 hours prior to the study;

3) No heavy exercise or heavy meals during the day of the experiment; and,

Table 1

Chronology of the experiment

PAIN ONLY

PM	ss	GA	Qs	I	Baseline	I2	AP	Pain	Recovery	ss	Qs	Debriefing
		pain only			10 min.	pain only	1 min.	0 - 10 min.	10 min.			

PAIN + PAIN WARNING

PM	ss	GA	Qs	I	Baseline	I2	AP	Pain	Recovery	ss	Qs	Debriefing
		pain only			10 min.	pain warning	1 min.	0 - 10 min.	10 min.			

PAIN + STRESS INTERVIEW WARNING

PM	ss	GA	Qs	I	Baseline	I2	AP	Pain	AP	Recovery	ss	Qs	Debriefing
		pain + SI			10 min.	pain then SI	1 min.	0 - 10 min.	2 min.	10 min.			

STRESS INTERVIEW WARNING

PM	ss	GA	Qs	I	Baseline	I2	AP	Recovery	ss	Qs	Debriefing
		SI			10 min.	SI	2 min.	10 min.			

Key

PM = Participant's manual
GA = Group assignment
SI = Stress Interview
Qs = Questionnaires
I = Instructions re: task demands
I2 = Instructions re: pressure pain and stress interview
AP = Anticipation period

4) To wear loose and comfortable clothing on the day of the experiment.

Before participation in the study, each subject was questionned to check if he had followed these instructions. Two subjects had consumed alcoholic beverages the previous evening and were rescheduled. All other subjects reported that they had complied with the instructions.

The subject was met by the experimenter at the laboratory and briefly shown the Experimenter room. The subject was then led into the Subject room and was told that he would remain there for the duration of the study. The subject was given a brief explanation corresponding to the group to which he had been assigned; the pressure pain, pressure pain plus stress interview, or stress interview only. The experimenter explained that he would first fill out questionnaires for about 15 minutes, then be asked to sit in the padded chair and have electrodes attached to monitor HR and muscle tension. He was also told that he would be asked to sit quietly for ten minutes then would receive the pressure pain or the stress interview depending upon his group membership. Subjects in the combined group were told that following the pressure pain they would receive a stress interview. The subject was told that he would be asked to sit quietly for another ten minutes after which he would complete a second series of questionnaires relating to the experiment. The video camera and microphone were pointed out to the subject. At this point, the subject was asked if he wanted to proceed with the experiment and was assured that he could terminate his participation at any time. He was given an appropriate consent form. All subjects chose to

continue at this time. The subject was given the questionnaires to fill out, was told that the experimenter would be in the adjoining room if he had any questions, and was asked to inform the experimenter when he was finished.

The experimenter returned to the Experimenter room closing the door connecting the two rooms. While the subject completed the questionnaires, the experimenter calibrated the polygraph driver amplifiers and preamplifiers as described in the Apparatus section. She also turned on the rating/tone generator control unit, the video equipment, and the tape recorders.

When the subject had completed the questionnaires, the experimenter entered the Subject room, collected them, and put the questionnaires in the other room. The experimenter instructed the subject to sit in the padded chair.

The experimenter cleaned the electrode sites using a sterile cotton ball moistened with isopropyl alcohol. The experimenter explained that the electrodes were monitoring heart rate and muscle activity, in order to reduce any anxiety that the subject may have had concerning the equipment. The subject was also told that it was important to move as little as possible once the study began since movement interfered with data collection. The electrodes were attached to the subject's left shoulder and right side, right elbow, and forehead as described in the Apparatus section. The desk top housing the pain apparatus and rating box was attached to the chair. The subject was told that he would hear further instructions on the headphones and to remember that he could communicate in a normal conversational voice with the experimenter via the microphone.

The experimenter returned to the Experimenter room and

turned on the physiological recording equipment and started the taped instructions. Subjects in the Pain Only group heard these instructions:

> This experiment is designed to evaluate your bodily responses to pain and stress. Pain is defined as a state of physical discomfort or hurt. Stress is defined as a state of mental tension; feelings or thoughts of constraint, worry, or anxiety.
>
> The experiment is divided into three phases. The first phase is a baseline period where you will be asked to sit quietly and adjust to this new environment. This phase will be followed by a period in which you will be asked to place the index finger of your left hand into the apparatus in front of you and a heavy plexiglass wedge will be applied to the first knuckle of your index finger. This will produce an uncomfortable sensation at first that will increase over time. You can ask to have the pressure removed when you feel that you can no longer tolerate it. For the last phase of the experiment, we will again ask you to sit quietly for ten minutes.
>
> During the entire 40 minute period, a tone will sound once each minute. Corresponding to the tone, both lights on the rating box in front of you individually marked 'Pain' and 'Stress' will briefly light up. Then the light marked 'Pain' will light up. This tone and light will alert you to press with your right index finger one of the pushbuttons in front of you and to indicate on a scale from one to ten, the intensity of pain you feel right at that moment. Pain refers to a state of physical discomfort or hurt. A rating of one indicates the absence of pain and a rating of ten indicates that the pain is as bad as it could be.
>
> Please place your right hand near the rating box and keep it close to the rating box throughout the entire session so that minimal movement is necessary to press one of the keys.
>
> Immediately after you have rated your level of pain, the light on the rating box marked 'Stress' will light up. At this time, please press a pushbutton on the recorder in front of you to indicate the degree of overall stress you are experiencing at that moment. Stress refers to a state of mental tension; feelings or thoughts of constraint, worry, or anxiety. Similarly, a rating of one indicates the <u>absence</u> of stress while a rating of ten indicates that you are experiencing an extreme degree of stress. Use the entire scale from one to ten if it applies to your experience.
>
> To make certain that you understand the procedure, we will ask you to complete two practice trials. For

> both trials, first press the button for a pain rating upon hearing the tone and seeing the light marked 'Pain' and then give a stress rating when seeing the light marked 'Stress'. Remember to press with your right index finger and to keep your right hand near the rating box. Please note that it is not necessary to press the buttons heavily; a light press is sufficient to register your response. Also, please place your left hand in a comfortable position in front of you on the board.

Throughout these instructions, the experimenter observed the subject through the one-way mirror to ensure compliance with the instructions. After these instructions, the experimenter turned off the tape and started the pain/stress - tone generator control unit which delivered the tone and light prompts at 60 second intervals. Following two practice trials, the control unit was turned off and the subject was asked if he had any questions via the taped instructions. If he did, the experimenter answered them and then played the following taped message:

> Please sit back and rest quietly with your eyes opened for a ten minute period to allow your biological responses to adjust to this environment. However, during this period, please provide us with both pain and stress ratings when you hear the tone and see the corresponding signal lights. Remember to rate your pain first and your stress second and to press the pushbuttons lightly.

The taped instructions were turned off and the control unit turned on to deliver tone and light prompts at 60 second intervals. The subject's physiological responses were recorded continuously throughout the rest of the experiment. During this phase, the Baseline period, the subject was asked to rate his pain and stress for ten trials. At the end of this period, the control unit was turned off and the subject heard the following instructions:

> You will now be asked to place your left hand into the apparatus in front of you. The technician will assist you with this shortly so do not attempt to do so by yourself. Once the apparatus is in place, please continue to indicate your levels of pain and stress when you hear the tone and see the corresponding light. In addition,

please tell us when the pressure turns to pain, i.e., when it first begins to hurt, by saying PAIN aloud. After this, please continue to indicate your pain and stress ratings when you hear the tone and indicate how you are feeling at that moment. You are to keep the apparatus on for as long as you can tolerate it. When you reach the point at which you feel you can no longer tolerate the pressure say STOP and very carefully withdraw your hand from the apparatus and gently place your hand beside it. Please do not touch your left hand with your right one as it will upset the recording equipment. You may squeeze your left hand gently and move the fingers. Please remain seated as motionless as possible throughout this part of the experiment. Do you have any questions?

Remember to report your pain and stress ratings when you hear the tone and see the light. When you first feel pain, say PAIN aloud and when you can no longer tolerate the pressure say STOP and gently withdraw your hand. The technician will assist you soon.

The experimenter answered any questions that the subject had after the taped instructions posed the question. Following the instructions, the control unit was turned on to receive one pain and stress rating prior to the application of the pressure. This period, the Anticipation period, provided a measure of stress after hearing the instructions. The experimenter then entered the room and instructed the subject to place his left index finger into the slot below the pain apparatus. While the slot was being adjusted for finger size to ensure exact placement of the lucite wedge, the experimenter gave the subject an index card (placed between the pain apparatus and the rating box) that stated "Remember - when you first feel pain, say PAIN aloud and when you can no longer tolerate the pressure say STOP". The experimenter returned to the Experimenter room and turned on the control unit. The subject's indication of pain threshold was recorded on the polygraph record and by digital stopwatch. Tolerance was recorded by digital stopwatch and by the transducer polygraph record which showed a deflection once

the apparatus was removed. Several subjects did not remove the apparatus when reporting tolerance, and the experimenter entered the Subject room and removed it if this occurred. The maximum exposure time was limited to ten minutes to prevent the possibility of tissue ischemia following pressure. The subject was not informed about this limit in order to prevent artificially long tolerance times.

Upon completion of this Pain period phase of the experiment, the control unit was turned off and the subject received the following instructions:

> Please remain seated ~~as~~ motionless as possible. Rest quietly for a ten minute period with your eyes open. Please continue to indicate your pain and stress ratings during this period.

Each subject made ten pain and stress ratings during this Recovery period. Then the control unit was turned off and the subject heard this message:

> This completes the evaluation. Please remain seated and the technician will remove the monitoring devices soon and explain the purpose of the experiment and your role in it.

The taped instructions and procedure were the same for the subjects in the Pain Plus Pain Warning group with the exception of the instructions given immediately after the Baseline period and before the Pain period. These subjects were given identical instructions up to the question "Do you have any questions?" at the end of the first paragraph. Then they heard:

> We feel that it is necessary to warn you at this time that the sensations you will experience will be very painful. Since there is an immediate danger of tissue damage due to prolonged exposure to this apparatus, you must be careful. Closely observe the discomfort you are experiencing. Since this pressure can become very painful in a short period of time and can cause some physical

damage, be sure to report when you can no longer tolerate the pressure as soon as you feel that way.

Remember to report your pain and stress ratings when you hear the tone and see the light. When you first feel pain, say PAIN aloud and when you can no longer tolerate the pressure say STOP and gently withdraw your hand. The technician will assist you soon.

The instructions and procedure were the same for the Pain Plus Stress Interview Warning group except for the instructions preceding the Baseline period, the instructions prior to the Pain period, and the instructions immediately after cessation of the pressure. The instructions prior to the Baseline period stated:

This experiment is designed to evaluate and to compare your bodily responses to pressure pain and a stress-inducing interview. In addition, we will be asking you to indicate your level of pain and stress. Pain refers to a state of physical discomfort or hurt. Stress is defined as a state of mental tension; feelings or thoughts of constraint, worry, or anxiety.

The experiment is divided into four phases. The first phase is a baseline period in which you will be asked to sit quietly and adjust to this new environment. This phase will be followed by a period in which you will be asked to place the index finger of your left hand into the apparatus in front of you and a heavy plexiglass wedge will be applied to the first knuckle of your index finger. This will produce an uncomfortable sensation at first that will increase over time. You can ask to have the pressure removed when you feel that you can no longer tolerate it. This phase is followed by a stress-inducing interview designed to evaluate your intelligence and psychological maturity and it may produce some sensations that you may find discomforting. For the last phase, we will again ask you to sit quietly for ten minutes.

The remainder of the instructions before the Baseline period were identical to the instructions given to both the Pain Only and Pain Plus Pain Warning groups. The instructions prior to the Pain period were the same as those given to the Pain Only and to the Pain Plus Pain Warning groups up to the question "Do you have any questions?". The subject's questions

if any, were answered and then he received these additional instructions:

> Following the application of the pressure pain, we will ask for your permission to participate in the stress interview. This interview is designed to measure your intelligence as well as your psychological maturity and the results will be revealed to you at the end of the session. We will also reveal how defensive we feel your behavior has been throughout this experiment based on your answers to the first battery of questionnaires and your behavior during the stress interview. During this interview, we ask that you continue to rate your pain and stress when you hear the tone and indicate how you feel at that moment. You can request to have the interview terminated at any time. For ethical reasons, we want you to be aware of potential effects that this interview may have on the heart and circulatory vasculature which are similar to the effects of psychologically stressful situations you may have experienced outside the lab. These effects are especially pronounced following a previous stressor such as pressure pain and are referred to as Selye's Stress Adaptation Syndrome. We ask you to monitor your heart rate and muscle tension during this interview so that you are able to stop the interview if that turns out to be necessary. Anticipation of this interview can adversely affect your performance so we ask you not to think about it.
>
> Meanwhile during this phase of the experiment in which you will be asked to experience the pressure, we remind you to report your pain and stress ratings when you hear the tone and see the light. When you first feel pain, say PAIN aloud and when you feel you can no longer tolerate the pressure say STOP and gently withdraw your hand. Then, the stress interview will begin. The technician will assist you soon.

Immediately after the pressure was terminated, the subject received the instructions to remain seated as motionless as possible and was told that the stress interview would begin shortly. The experimenter turned on the control unit to obtain two ratings of pain and stress during this waiting period. The subject was then given these instructions:

> Please remain seated as motionless as possible.
>
> Rest quietly for a ten minute period with your eyes open. Please continue to indicate your pain and stress ratings during this period. We will not be asking for your participation in the stress interview.

The taped instructions and procedure for the subjects in the Stress Interview Warning Only group were similar to the Pain Plus Stress Interview Warning group except that these subjects did not receive pressure pain stimulation. In addition, the pain apparatus was removed from the desk top. These instructions stated that the experiment was designed to evaluate bodily responses to a stressful interview and that the interview would produce uncomfortable sensations. Thus, they would be asked to rate their pain as well as their stress level. Two practice trials and a Baseline period were given identical to those given to subjects in the other three groups. Following the Baseline period, subjects heard instructions identical to those given the Pain Plus Stress Interview Warning subjects in regard to the nature of the stress interview. Two pain/stress ratings were elicited after these instructions. The subject was told to rest for ten minutes, to continue indicating pain and stress ratings, and that they would not be participating in the stress interview.

After the Recovery period, all subjects received the following procedure. The experimenter entered the Subject room with the postexperimental questionnaires. The desk top was removed from the chair and the electrodes were removed from the subject. He was seated at the desk to fill out the questionnaires. The subject was told to read the instructions, to complete the questionnaires, and to ask for extra information if necessary. The experimenter returned to the Experimenter room.

These questionnaires took about 15 minutes to complete. After completion, the subject was given $10 and then debriefed. Subjects in the three groups receiving pressure pain were told

that experiment examined the influence of stress, as measured by various questionnaires, HR, EMG, and stress ratings, upon the pain ratings and pain behavior. Subjects in the Stress Interview Warning Only group were told that they were in a comparison group in a study assessing the effects of stress upon the experience of pain. All subjects were shown their physiological responses and any questions that were asked were answered by the experimenter. The subject was asked not to discuss this experiment with other students and thanked for his participation. The subject was informed that he could contact the experimenter if he had additional questions or if he wanted the results of the experiment.

Data reduction

Pain and stress intensity ratings. A maximum of 31 pain and stress intensity ratings were obtained from the Pain Only (PO) and Pain Plus Pain Warning (P+PW) groups; ten from the Baseline and Recovery periods, one from the Anticipation period (AP), and up to ten from the Pain period. Because of the addition of two anticipatory stress interview ratings, a maximum of 33 ratings were obtained from the Pain Plus Stress Interview Warning (P+SI) subjects. Since no pain period was present in the Stress Interview Warning Only (SI) condition, a total of 22 pain and stress ratings were obtained from the SI subjects.

Physiological responses. Physiological data were directly obtained from each subject's polygraph record. The records were coded using a randomly assigned subject number unrelated to group membership and then scored blind. The subject number was then decoded to determine group membership for subsequent analyses.

Heart rate (HR) was scored at 60 second intervals during

the Baseline, AP, Pain, anticipating stress interview (when applicable), and Recovery periods. Heart rate was calculated by counting the number of beats for a ten second interval five seconds prior to the onset of the tone which signalled the subjects to make a pain and stress rating. This was converted to beats per minute (bpm). The five second period prior to the tone was not used in the analyses in order to avoid measurement artifacts such as the orienting response that would artificially increase HR (Germana & Klein, 1968).

Frontalis EMG was calculated by measuring the polygraph pen deflections with vernier calipers accurate to 0.05 mm. This measurement was also taken every 60 seconds at five seconds prior to the tone prompt. EMG was scored in mm deflections from baseline and converted to microvolts (uV); 200 uV = 2 cm.

Nine HR and EMG measurements were obtained for both Baseline and Recovery periods because the measurement process began after the first tone prompt for each period. One HR and one EMG measurement was obtained during the AP and up to ten were obtained during pressure pain stimulation. Two anticipatory stress interview HR and EMG responses were obtained from subjects in the P+SI group. Nine Baseline and Recovery physiological measurements plus two anticipatory stress interview measurements were obtained from subjects in the SI group.

When artifacts occurred, the polygraph record immediately preceding the artifact was used.

Pressure stimulation data. Measurement of the time course of pressure pain stimulation was made by measuring the distance on the polygraph record in which a pressure deflection was present and converting the distance to seconds. A digital stopwatch

provided a redundant measure of pain threshold and tolerance. Variations in pressure over time were recorded using vernier calipers to measure the polygraph deflections. The measurements were scored in mm deflection from baseline (2000 g). This was converted to grams of force (1 cm = 200 g). This datum was collected every ten seconds from onset to termination of pressure pain. The mean pressure from each 60 second period was calculated for subsequent analysis.

Behavioral response data. It was stated earlier that the behavioral response data were divided into six 60 second intervals. The measurement periods consisted of the seventh and eighth minutes of Baseline, the first and last minutes of pressure pain, and the third and fourth minutes of Recovery. Each behavior was rated on the basis of frequency for each 30 second segment throughout the six minutes of observation. The total frequency of each of the 14 behaviors was computed for each of the 60 second periods. The mean frequency of behavior from the Baseline, and from the first and last minutes of pressure pain were used as dependent variables in subsequent analyses.

Missing data

One cause of missing data was equipment failure. The EMG channel of the polygraph failed throughout an entire experimental session with a subject from the P+SI group. The video tape system failed to work on five separate occasions and as a result behavioral response data were unavailable for three subjects in the PO group, one subject in the P+PW group, and one subject in the P+SI group. Cohen and Cohen (1975) suggest that when data is lost in this fashion, it is appropriate to drop the subject from analysis. Thus, when analyzing EMG a total of

47 subjects was used and when analyzing behavioral response data, a total of 43 subjects was used.

The second cause of missing data was due to the effects of the experimental manipulation during the Pain period. A maximum of ten pain intensity, stress intensity, HR, and EMG ratings could be obtained from each subject during this period. However, subjects reporting tolerance before the ten minute time limit gave fewer than ten ratings. This source of missing data was handled in three ways for subsequent analyses. First, group means were used to fill missing data cells. Cohen and Cohen (1975) suggest that using group means is appropriate under these circumstances since the mean score will not affect the regression coefficients associated with group effects. Thus, 144 cells out of a total of 480 during the Pain period were filled with group means.

However, Cohen and Cohen (1975) caution that using a high percentage of substitutions lowers the variance within groups to a degree that spurious significance levels can occur. Therefore, identical analyses were performed using only the subjects who did not report tolerance (N = 23) and who completed all ten measurements in each category. In addition, means for each of the dependent variables across all the recorded measurements were obtained from each subject during the Pain period for analyses.

RESULTS

Overview of the data analysis

The present study generated an enormous amount of data. Therefore, a large number of statistical analyses were conducted to test the hypotheses of this study. However, as the number of statistical tests increases, the probability of obtaining spurious results also increases (Cohen & Cohen, 1975). Cohen and Cohen (1975) suggest several methods to circumvent this problem. The first method involves combining dependent variables of a common construct into a multiple analysis of variance. This approach was not chosen for the following reasons. Since differences among groups on the preexperimental questionnaires could affect the responses obtained from the pain measures (Weisenberg, 1977), these data were analyzed using univariate analyses of variance. This method was employed to increase the likelihood of finding significant covariates to use in testing the major hypotheses (Hummel & Sligo, 1971). In addition, most of the pain measures were not directly comparable. For instance, the behavioral response data were obtained during the first and last minutes of nociception, the threshold and tolerance data were obtained only one time, and up to 31 pain and stress intensity ratings were obtained.

Under these circumstances, Cohen and Cohen (1975) suggest using a hierarchical model of hypothesis testing and inference. In this type of model, statistical analyses are classified into levels of research relevance. Thus, the first set of statistical analyses would be designed to address the major research questions.

The second set of analyses would involve factors of secondary interest. The third set would address hypotheses of a speculative nature. By conducting the analyses in this fashion, significant results that are obtained from testing major hypotheses are more reliable (Cohen & Cohen, 1975).

In reference to these issues, the data analysis was carried out in seven steps:

1) Validity checks were performed on the main measures. Specifically, analyses were conducted to determine if the pressure exerted was constant and equivalent within and between groups; if the pressure produced a painful experience; and if the subjects were reporting their experience honestly.

2) The data obtained from the demographic, personality, and state anxiety questionnaires were analyzed separately using univariate analysis of variance (ANOVA; Roscoe, 1975) to determine if the groups were equivalent on each of these measures. Chi-square statistics were calculated on the nonquantifiable demographic data (Roscoe, 1975).

3) The data composed of the direct pain measures were analyzed. The pain intensity ratings were analyzed using repeated measures analysis of covariance (ANCOVA; Cohen & Cohen, 1975). The variances associated with orthogonal polynomials (Cohen & Cohen, 1975) were calculated using data from Baseline, Pain, and Recovery periods to assess trends over time among groups in each of these periods. A univariate ANCOVA was used to compare groups on the average pain intensity rating reported during the Pain period. The pain threshold, tolerance and endurance data were analyzed using univariate ANCOVAs. An analysis of the rate at which subjects dropped out over time during the Pain period was

conducted using the Kolmogorov-Smirnov test with revised critical values (Kanner, Mohanty, & Lyons, 1980). The postexperimental direct pain questionnaire data were analyzed using univariate ANCOVAs. When appropriate, these analyses were followed by the Scheffé test for all possible comparisons (Roscoe, 1975).

4) The data composed of the indirect pain measures were analyzed. The stress intensity ratings were analyzed using repeated measures ANCOVAs. The variances associated with orthogonal polynomials were calculated using the data from Baseline, Pain, and Recovery periods. A univariate ANCOVA was used to compare groups on the average stress intensity rating reported during the Pain period. Scheffé's tests were employed when appropriate.

The psychophyiological data were analyzed using repeated measures ANCOVAs controlling for differences between subjects in the resting levels of these measures. The first five minutes of Baseline were used as the covariate. Subsequently, these minutes were not included in the analyses so that the covariate would not be confounded with the dependent measures (Cohen & Cohen, 1975). Orthogonal polynomials were calculated and when appropriate, Scheffé's tests were employed. Univariate ANCOVAs were calculated to compare groups on subjects' average HR and EMG obtained during the Pain period.

The behavioral response data obtained from the Pain period were analyzed using univariate ANCOVAs controlling for differences in initial Baseline movement frequency. Specific types of behavioral expression were analyzed using separate ANCOVAs.

The postexperimental indirect pain questionnaires were analyzed using separate univariate ANCOVAs.

5) Subjects who reported tolerance during the Pain period were

compared with subjects who did not on the demographic, personality, and anxiety questionnaire data as well as on the direct and indirect pain measures. Univariate ANCOVAs and Student's *t* tests were used (Roscoe, 1975). In cases where the variances between the two groups were not equal as assessed by the Levene test for unequal variances (Levene, 1960; in Brown & Forsythe, 1974b), the *t* test employing separate variances for each group (rather than a pooled error term) was used (Brown & Forsythe, 1974b).

6) An analysis of the relationships among the direct measures of pain was made by calculating Pearson product moment correlation coefficients (Roscoe, 1975) using data collected from the entire sample.

7) An assessment of the relationships between the questionnaire measures of anxiety and the direct pain measures as well as the indirect pain measures and the direct pain measures was performed by calculating Pearson product moment correlation coefficients using data collected from the entire sample.

In all repeated measures analyses, probability levels for significance were determined using the Huynh-Feldt adjustment for conservative degrees of freedom (Huynh & Feldt, 1976). A probability level of $p < .05$ was set as indicating a statistically significant difference between groups. In addition, trends toward significance ($p < .10$) were reported if a directional prediction had been made prior to the study.

Validity checks on the main measures

Four important questions were answered prior to subsequent data analysis:

1) Was the pressure exerted by the nociceptive stimulus constant

over time and equivalent among groups?

2) Was the stimulus felt to be painful by the subjects?

3) Were the pain warning and the stress interview warning experienced as stressful?

4) Were the subjects reporting their experience honestly?

Appendix D includes the statistical analyses conducted for these validity checks.

Constancy of pressure. A 3 (groups) X 10 (minutes) repeated measures ANOVA was performed using the bridge transducer data. There was no significant effect of group membership or group by minutes interaction. There was a significant minutes effect ($F(9,18) = 4.42$, $p < .05$). The average pressure exerted was 1965 g, ranging from a mean of 1924 g during the first minute to 1963 g during the last minute. Sixty-eight percent of this increase occurred during the first minute of pressure and this increase was significant as measured by Scheffé's test ($F(1,46) = 10.8$, $p < .01$). No other significant differences were found. Appendix D includes means and standard deviations (SDs) of the force (in grams) exerted across the pain minutes.

These results indicate that the pressure significantly increased from the first to the second minute of nociception but did not differ between groups. The actual force exerted was slightly below the 2000 g value originally planned. The increase in pressure during the first minute was most likely due to increased displacement of skin tissue before reaching a constant resting state.

Painfulness of the pressure stimulation. As a group, subjects reported pain threshold at 141 seconds after application of the pressure stimulation, with high variability (SD = 145

seconds). Pain tolerance was reported after an average of 469 seconds (SD = 188 seconds). Fifty-two percent of the subjects reported pain tolerance before the end of the ten minute exposure time. The average of the highest individual pain intensity rating delivered during nociception was 6.9 (SD = 1.99) on the one to ten scale. The mean pain intensity rating over the entire period was 4.99 (SD = 1.83).

On the McGill Pain Questionnaire (MPQ), the average individual pain rating index (PRI-T; the sum of the rank values of the words chosen) equalled 20.8 with high variability (SD = 10.14). This average score is comparable to average scores obtained from individuals reporting arthritic pain and individuals reporting toothache pain (Melzack & Wall, 1982).

Thus, the stimulus chosen was effective in producing pain that was described as moderate to extreme within the present sample. The high variability of the subjects' responses reflects the large individual differences found in the general population (Weisenberg, 1977).

Stressfulness of the pain warning. This series of analyses compared the Baseline and Anticipation (AP) periods of the Pain Only (PO) and Pain Plus Pain Warning (P+PW) groups to determine the effectiveness of the pain warning in producing a stressful experience. Table 2 presents the mean stress intensity ratings, mean adjusted HR, and mean adjusted EMG obtained during these two periods. A 2 (groups) X 11 (minutes) repeated measures ANOVA performed using the stress intensity rating data revealed no group difference or interaction between group and minutes. There was a trend for a significant minutes effect ($F(10,10) = 1.98$, $p < .07$).

Table 2

Mean stress intensity ratings (SI), adjusted HR (bpm), and adjusted EMG (uV) obtained during Baseline and AP periods obtained from the PO and P+PW groups

Measure	SI		HR		EMG	
Period	Baseline	AP	Baseline	AP	Baseline	AP
Group						
PO						
M	1.5	1.7	69	70	70	67
SD	0.7	0.8	8	8	27	28
P+PW						
M	1.8	2.4	70	74	72	86
SD	0.9	1.0	11	12	28	34

Two 2 (groups) X 5 (the last four minutes of Baseline and the AP minute) repeated measures ANCOVAs were conducted using the HR and EMG data (covarying the first five minutes of Baseline). The analysis of the HR data revealed no group effect, a significant effect of the covariate, a significant minutes effect ($F(4,120) = 4.83$, $p < .005$), and a significant group by minutes interaction ($F(4,120) = 4.08$, $p < .02$). Scheffé's tests calculated using the adjusted means revealed no significant differences during the Baseline period. HR was significantly greater in the P+PW group during the AP than in the PO group ($F(1,30) = 15.61$, $p < .01$).

A significant group effect was found by the repeated measures ANCOVA performed using the EMG data ($F(1,29) = 5.46$, $p < .03$). There was a significant interaction between group and minutes ($F(4,120) = 4.16$, $p < .02$) and a significant effect of the covariate. There was no minutes effect. Scheffé's tests revealed no significant differences during Baseline and a significantly greater EMG during the AP in the P+PW subjects compared to the PO subjects ($F(1,30) = 23.15$, $p < .01$).

These results indicate that the pain warning produced increases in HR and EMG yet did not appear to influence stress intensity ratings. Since increases in HR and EMG are reflective of an increase in the stress experience (Greenfield & Sternbach, 1972), it appears that the warning produced an increase in stress that was not captured through obtaining only one stress intensity rating prior to pressure pain. This issue will be considered in the Discussion section.

Stressfulness of the stress interview warning. Table 3 presents the mean Baseline, AP, and Recovery stress intensity

ratings, HR, and EMG, and the corresponding SDs obtained from the Stress Interview Warning Only (SI) group. Three repeated measures ANOVAs were performed using the 22 stress intensity ratings and the 20 HR and EMG measurements. There were significant minutes effects found by the analyses of the stress intensity data ($F(21,315) = 2.95$, $p < .005$) and the HR data ($F(19,285) = 2.79$, $p < .05$). There was no minutes effect found for EMG.

Scheffé's tests were performed to determine at which points differences between minutes occurred using the stress intensity ratings. A significant difference between the first and second minute of Baseline was revealed ($F(1,15) = 4.33$, $p < .05$). No other significant differences were found during Baseline. A significant increase was revealed by the comparison of the last minute of Baseline and the first minute of the AP ($F(1,15) = 33.00$, $p < .01$). A significant decrease in ratings occurred between the last minute of the AP and the first minute of Recovery ($F(1,15) = 15.82$, $p < .01$). No other significant differences were revealed.

Scheffé's tests conducted using the HR data indicated no significant differences during Baseline, a significant increase in HR from the last minute of Baseline to the first minute of the AP ($F(1,15) = 10.29$, $p < .01$), and a significant decrease in HR from the last minute of the AP to the first minute of the Recovery period ($F(1,15) = 12.46$, $p < .01$). No other significant comparisons were found.

Visual comparison of the mean stress intensity ratings, HR, and EMG data obtained during the Baseline and AP periods from the PO, P+PW, and SI groups shows a similar increase in stress

Table 3

Mean stress intensity ratings (SI), HR (bpm), and EMG (uV) obtained during Baseline, Anticipation, and Recovery periods from the SI subjects

Measure	SI	HR	EMG
Period			
Baseline			
M	2.0	73	70
SD	0.9	9	27
Anticipation			
M	2.6	78	67
SD	1.4	13	29
Recovery			
M	1.9	74	67
SD	0.8	10	27

and HR from the Baseline to the AP period (Tables 2 and 3). The PO and SI subjects show a similar decrease in EMG from the Baseline to the AP period. No statistical comparisons were made among the PO, P+PW, and SI groups on these measures because the groups were not directly comparable. Prior to the experiment, the SI group knew that no nociceptive stimulus would be employed yet both the PO and P+PW groups were told that the experiment would involve pressure nociception.

These results indicate that the stress interview warning was moderately effective at producing stress as measured by the stress intensity rating and HR data. EMG did not appear to be influenced by the warning.

Validity of self report. As discussed in the Introduction, the Social Desirability Scale (SDS) provides a means of assessing whether of not subjects are predisposed to 'fake good' and say what they believe is appropriate (Crowne & Marlowe, 1964). A univariate ANOVA revealed no differences between the four groups on this scale ($F(3,60) = 0.59$, ns). Thus, all four groups are similar in their reported interest to appear socially appropriate. The values of this measure were within the normal range (see Appendix E).

Two other sets of analyses were conducted using the questionnaire data collected from the three groups receiving pressure pain. As discussed in the Introduction, Weinberger, Schwartz, and Davidson (1979) reported that a combined Taylor Manifest Anxiety Scale (TMAS) score and SDS score can be used to check the accuracy of self-report of anxiety and stress. In the present study, subjects were divided into 'repressor' and 'true low anxious' in the following fashion. Since SDS scores

were within the normal range, high and low scores on this scale were calculated by a median split of the scale scores. However, the present sample was relatively low trait anxious (TMAS) according to published population norms (Taylor, 1953). Thus, high and low scores on this measure were calculated on the basis of scores that were higher or lower than the published population mean. According the Weinberger et al. (1979), subjects with high SDS scores and low TMAS scores are 'repressors' and are not reporting their level of anxiety accurately or reliably. Subjects who have low scores on both measures are 'true low anxious' and reporting their emotional state accurately. Five subjects did not fit into either category (high on both values of high on the TMAS and low on the SDS) and were excluded from analysis. A univariate ANOVA found no significant differences among groups on this combined index ($F(2,40) = 1.32$, ns). This indicates that each group contained a similar number of subjects who were likely to report their experience either accurately or inaccurately.

Each subject in the groups receiving pressure pain was asked at the end of his participation "Have any friends or acquaintances participated in this experiment?" (see Appendix B). Eleven subjects reported positively; three from the PO group, three from the P+PW group, and five from the Pain Plus Stress Interview Warning (P+SI) group. In response to the question "If yes, did you discuss this experiment with them?", three subjects from this group of eleven responded positively; one from the PO group and two from the P+SI group. All three subjects claimed that they received no extra information from these acquaintances that was not already given in the participant's manual. Given the

small number of subjects who talked to other participants, no attempt was made to analyze differences between them and the rest of the sample.

Conclusions. This section examined the methods employed in the present study. The amount of pressure exerted was slightly less than intended, yet it was equivalent across groups. The pressure increased; primarily during the first minute. Subjects experienced the pressure pain as moderately to extremely painful. The pain warning produced significant increases in HR and EMG relative to the PO group, yet did not produce significant differences in pain intensity ratings. The stress interview warning produced significant increases in stress intensity ratings and HR, yet had no effect on EMG. It was concluded that both warnings were effective at producing a stressful experience. However, the issues raised by the differences in the parameters of this stress response will be considered in the Discussion section. The groups contained similar numbers of subjects reporting an interest in social approval and who were likely to report their experience of stress in an accurate and reliable fashion. Few subjects reported that they talked to other participants of the study, and these few claimed to have received no additional information.

Demographic, personality, and state anxiety questionnaire data

In the Introduction it was stated that a wide variety of demographic, personality, and situational variables influence pain assessment measures. The data obtained from each preexperimental questionnaire from the four groups were analyzed separately using univariate ANOVAs and chi-square statistics to increase the likelihood of finding significant covariates

to use in subsequent analyses (Hummel & Sligo, 1971). Appendix E includes the statistical analyses pertaining to this data.

Demographic characteristics of the sample and of the groups. Univariate ANOVAs found no significant differences in age, in the ratio of height to weight (Ht/Wt), or in socioeconomic status (SES) as measured by Blishen and McRoberts' (1976) socioeconomic index among the four groups. Since SES has been reported to influence pain behavior (Tursky, 1973) and the ANOVA revealed a trend towards significant differences among groups ($F(3,60) = 2.59$, $p < .07$), SES was used as a covariate on all analyses of self-report of pain and stress obtained during and after the experiment. Means obtained from each group for SES were 50.2 from the PO group, 54.9 from the P+PW group; 64.8 from the P+SI group, and 60.2 from the SI group.

Chi-square test for contingency tables (Roscoe, 1975) found no association between group membership and season tested ($\chi^2(6) = 2.39$, ns), first language ($\chi^2(4) = 0.17$, ns), citizenship ($\chi^2(4) = 0.17$, ns), or religion ($\chi^2(12) = 7.33$, ns). Group values for these demographic measures as reported in Appendix E.

Personality questionnaires. There were no significant differences among groups on the level of reported trait anxiety (TMAS), importance of socially appropriate behavior (SDS), fear of situations involving performance evaluation [Stimulus-Response Inventory of General Trait Anxiety - Revised (GTAR), Evaluation Anxiety subtest (EA)] or fear of situations involving physical danger [GTAR, Physical Danger (PD) subtest]. Group values are reported in Appendix E with corresponding population norms. The present sample did not appreciably differ in the values from the SDS, GTAR-EA, or GTAR-PD when compared to the reported

norms. However, the present sample reported relatively low trait anxiety (TMAS) when compared to Taylor's (1953) reported norms. As a group, all subjects reported more stress associated with situations involving physical danger (GTAR-PD) than situations involving performance evaluation (GTAR-EA; $F(1,60) = 68.30$, $p < .001$). This finding replicates Endler and Okada's results (1976). There was no interaction between group and situational anxiety. Therefore, there were no significant differences among groups on the scores obtained from the preexperimental personality questionnaires.

State measures of anxiety. Univariate ANOVAs were performed using the data obtained from the State-Trait Anxiety Inventory - State measures (STAI-S) and the report of present stress (one to ten scale). There were no significant differences among groups on the STAI-S. The four groups reported significantly different levels of present stress prior to the experiment ($F(3,60) = 2.80$. $p < .05$) yet Scheffé's tests comparing these groups found no significant differences. Examination of this data suggests that the SI group reported the least amount of present stress prior to the experiment (see Appendix E). It is reasonable that the SI subjects would report the least amount of stress since they knew they would not be receiving pressure pain stimulation. The present sample appears to report lower anxiety on the STAI-S than the general Canadian student population assessed by Pacheri, Bernaber, Bellaterra, and Tartaglione (1976).

Conclusions. This section examined differences among groups in scores obtained from the preexperimental questionnaires assessing demographic, personality, and anxiety characteristics. There was a trend for a significant difference among groups on

SES. Thus, SES was used as a covariate in subsequent analyses of the self-report measures obtained during and after the experiment. There was a significant difference among groups obtained from the rating of present stress; the SI group appeared to report the lowest level. No other significant differences among groups emerged. The present sample tends to be less trait and state anxious than the general population.

The direct pain measures

In this section, the analyses conducted with the pain intensity rating data, pain threshold, tolerance and endurance data, and the postexperimental direct pain questionnaires are presented. Appendix F includes the statistical analyses conducted with this data.

Pain intensity ratings. Three sets of analyses were performed using the pain intensity ratings. First, a 3 (groups) X 31 (minutes) repeated measures ANCOVA was conducted using group means to fill missing data cells. Orthogonal polynomials were calculated separately for each group in order to study differences among groups in trends over time during the three main periods (Baseline, Pain, and Recovery). Scheffé's test was used when appropriate. Second, a 3 (groups) X 31 (minutes) repeated measures ANCOVA was conducted using the pain intensity rating data from subjects who did not report tolerance. Orthogonal polynomials were calculated for each period and Scheffé's test was employed when appropriate. Third, a univariate ANCOVA was performed using the mean pain intensity rating obtained from each subject during the Pain period. In each set of analyses, SES was used as a covariate. The two pain intensity ratings obtained from the P+SI subjects during the

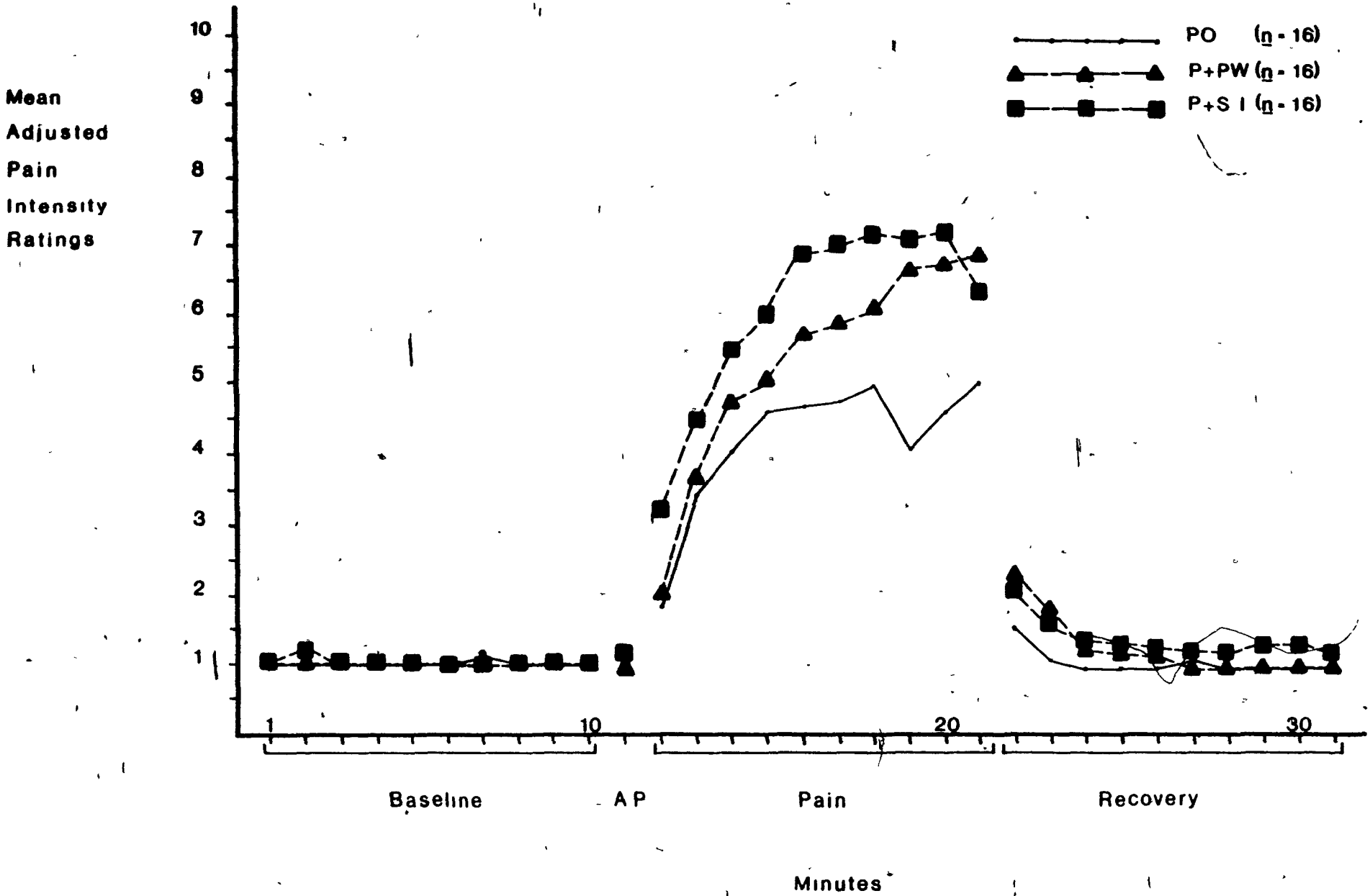
stress interview anticipation period were used as the first two minutes of Recovery data for that group (and the last two minutes of Recovery data was excluded).

Figure 1 depicts the mean adjusted (for SES) pain intensity ratings obtained during each minute of Baseline, AP, Pain, and Recovery periods from the PO, P+PW, and P+SI groups using group means to fill missing data cells. The repeated measures ANCOVA found a significant group effect ($F(2,44) = 8.26$, $p < .001$), a significant effect of the covariate, a significant minutes effect ($F(30,60) = 221.69$, $p < .001$), and a significant group by minutes interaction ($F(60,1350) = 3.17$, $p < .003$).

Orthogonal polynomials calculated from the Baseline period data revealed no significant effects. Orthogonal analyses of the Pain period found a significant effect of group membership ($F(2,44) = 7.21$, $p < .003$), a trend for a significant effect of the covariate, a significant minutes effect ($F(9,18) = 53.75$, $p < .001$), and a significant group by minutes interaction ($F(18,405) = 2.61$, $p < .02$). There were significant linear, quadratic, and cubic trends over time indicating that the rate of increase of the pain intensity ratings decelerated and then accelerated before the end of the Pain period. There was a significant interaction between group and the linear trend ($F(2,45) = 3.71$, $p < .04$) and between group and the cubic trend ($F(2,45) = 3.93$, $p < .03$). Visual inspection of Figure 1 indicates that the pain intensity ratings obtained from the P+PW group follow a linear trend more closely than the pain ratings obtained from the PO and P+SI groups. The pain ratings from the PO group appear to follow a cubic trend more closely than the pain ratings obtained from the P+PW and P+SI groups.

Figure Caption

Figure 1. Mean adjusted (for SES) pain intensity ratings obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups using group means to fill missing data cells.


Mean Adjusted Pain Intensity Ratings
10
9
8
7
6
5
4
3
2
1
PO (n - 16)
P+PW (n - 16)
P+S I (n - 16)
1
10
20
30
Baseline
AP
Pain
Recovery
Minutes

This suggests that the pain ratings reported by the P+PW group increase over time at a constant rate while the rate of increase in pain ratings decreases over time in the P+SI group and the rate of increase decreases and then increases in the PO group.

Orthogonal analyses of the Recovery minutes found a significant minutes effect ($F(9,18) = 19.88$, $p < .001$) due to a decrease in pain intensity ratings over time. Significant linear, quadratic, and cubic trends were found yet there were no group by trend interactions. Visual inspection of Figure 1 suggests that the pain intensity ratings decreased rapidly and remained at a low level for the rest of the period. The cubic trend is barely discernable.

Scheffé's tests were performed to compare groups during the Pain period. Comparisons between the PO and the P+PW groups found no significant differences in pain ratings during the first four pain minutes (abbreviated as P1, P2, P3, and P4) and significant differences during the last six minutes (P5, P6, P7, P8, P9, and P10). The P+PW subjects rated the pain intensity significantly higher in each of the six comparisons.

Scheffé's tests comparing the PO to the P+SI group found significant differences in all comparisons with the exception of P2 in which no significant difference was found. In each comparison, the P+SI subjects reported higher pain ratings than the PO subjects.

Scheffé's tests comparing the P+PW subjects with the P+SI subjects found significant differences in pain ratings during P1, P4, P5, P6, and P7. In each of these comparisons, subjects in the P+SI group reported higher pain intensity than subjects in the P+PW group. No other significant differences were found.

These results indicate that both experimental groups rated their pain as more intense than the control group. The pain ratings obtained during the Pain period increased at different rates depending upon group membership. The rate of increase in these ratings appeared to decrease more quickly in the PO group than in the P+PW or P+SI groups. The experimental subjects did not report significantly higher pain ratings during the Recovery period than the control subjects. Subjects in the P+SI group reported significantly higher pain than the subjects in the P+PW group in five of the ten comparisons.

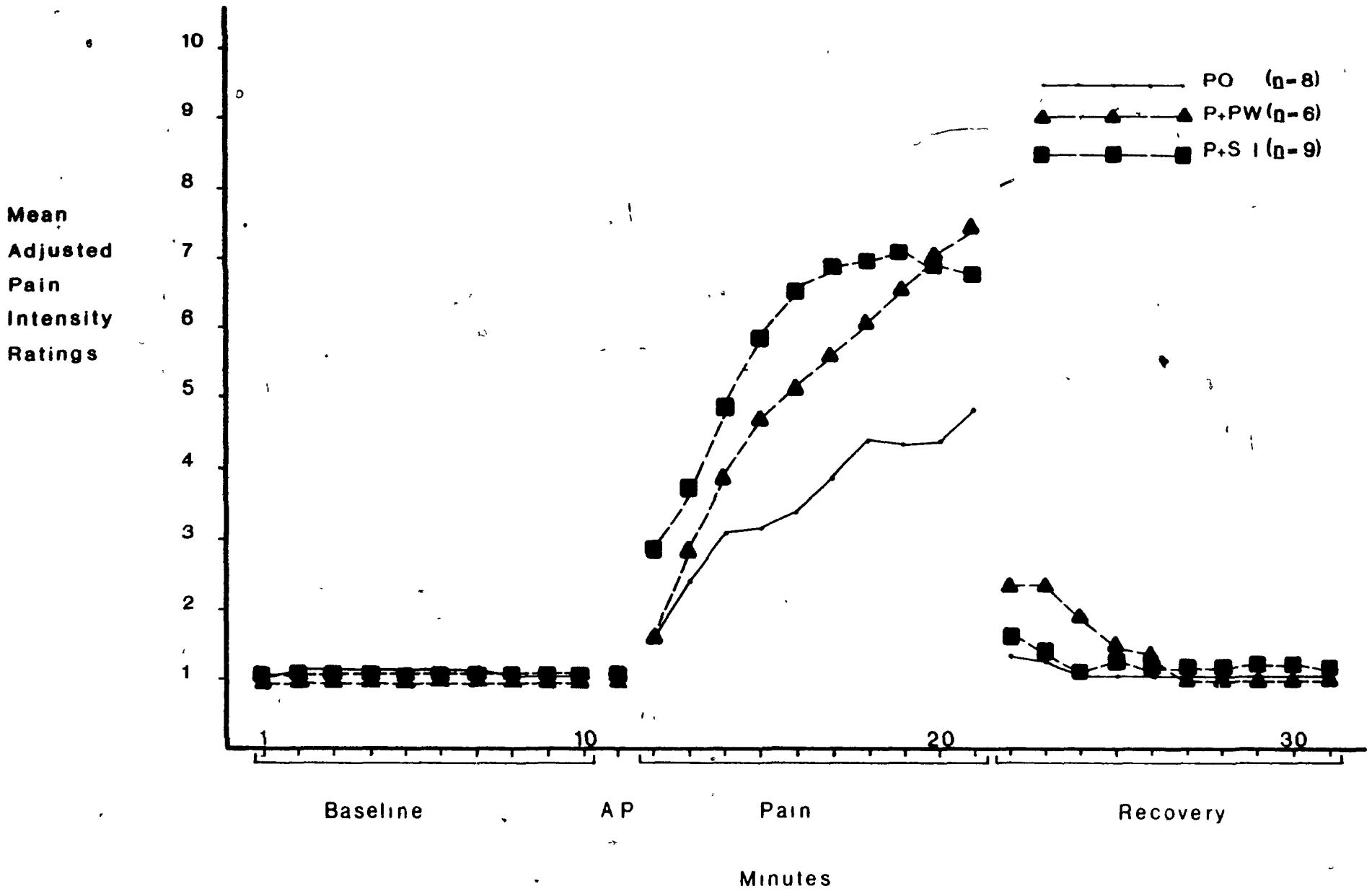
Figure 2 depicts the mean adjusted pain intensity ratings obtained from subjects who did not report tolerance within the PO (n = 8), P+PW (n = 6), and P+SI (n = 9) group during the 31 minutes. The repeated measures ANCOVA revealed a trend for a significant group difference ($F(2,19) = 3.22$, $p < .07$), a significant minutes effect ($F(30,60) = 75.96$, $p < .001$), and a trend for a significant group by minutes interaction ($F(60,1350) = 1.95$, $p < .10$). The covariate was not significant.

Orthogonal polynomials were calculated to determine trends in the pain rating data during each period. No significant effects were found from the analysis of the Baseline minutes. The analysis of the Pain period found a significant group effect ($F(2,19) = 3.53$, $p < .05$) as well as a significant minutes effect ($F(9,18) = 41.96$, $p < .001$). There was a trend for a significant interaction between group and minutes ($F(18,180) = 1.90$, $p < .09$). Significant linear and quadratic trends emerged indicating that the rate of increase of the entire sample's pain ratings decreased over time. There were no interactions between group and trend.

No significant group differences or group by minutes

Figure Caption

Figure 2. Mean adjusted (for SES) pain intensity ratings obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups composed of subjects who did not report pain tolerance.


Mean Adjusted Pain Intensity Ratings
10
9
8
7
6
5
4
3
2
1
PO (n=8)
P+PW (n=6)
P+S I (n=9)
1
10
20
30
Baseline
AP
Pain
Recovery
Minutes

interaction were found from the orthogonal analysis of the Recovery minutes. There was a significant minutes effect ($F(9,18) = 5.20$, $p < .02$) indicating a decrease in pain ratings over time. Significant linear and quadratic trends were found suggesting that the rate of decrease during the Recovery period was faster at the beginning than at the end. There were no interactions between group and trend.

Scheffé's test was conducted to compare groups of subjects who did not report tolerance on their average pain intensity rating delivered during the Pain period. Both experimental groups reported significantly higher average pain ratings during this period when compared to the control group. No significant difference was found by the comparison between the P+PW and P+SI groups. The adjusted group mean pain intensity ratings obtained during the Pain period from these subjects were 3.79 from the PO group, 5.16 from the P+PW group, and 5.96 from the P+SI group.

Therefore, when using subjects who did not report tolerance, both experimental groups rated the pressure pain as significantly more intense than the control group, yet there was no interaction between group membership and the shape of the pain intensity curves. Failure to find this interaction that had been revealed when all subjects were used in the analyses may be due to the small sample sizes of each group. This view is supported by a trend for a significant interaction between group and minutes. In addition, subjects who report tolerance may respond differently to pressure pain stimulation than subjects who do not. This possibility will be examined in the section comparing these two subject groups.

A univariate ANCOVA using the mean individual pain intensity

rating obtained during the Pain period was performed. This ANCOVA revealed a significant group effect ($F(2,44) = 3.73$, $p < .04$) yet no effect of the covariate. Scheffé's tests comparing groups found that the P+SI group reported significantly higher pain intensity than the PO group ($F(2,45) = 4.36$, $p < .05$). No significant difference was found from the comparison of the P+PW to the PO group or from the comparison of the P+PW to the P+SI group. Therefore, when taking an average pain intensity rating from all subjects during the pain period, the P+SI group reported significantly higher pain intensity during the Pain period than the control group but this measure did not differ between the P+PW group and the control group. Table 4 presents the adjusted mean pain intensity rating, pain threshold, pain tolerance, and endurance data reported by subjects from each group.

Pain threshold, tolerance, and endurance data. As described in the Method section, subjects were asked to report when they first felt pain (pain threshold) and when they felt they could no longer tolerate the pain (pain tolerance). Pain endurance was determined by subtracting the threshold value from the tolerance value. Separate univariate ANCOVAs were calculated on each of these measures (covarying SES). There were no significant differences among groups on any of these dependent variables. The variability of each of these measures was very high (see Table 4).

Fourty-eight percent of the subjects did not report tolerance during the Pain period. A second method was employed to determine if there was a difference among groups in the rate at which subjects reported tolerance during the Pain period.

Table 4

Mean adjusted pain intensity ratings (PI), pain threshold, pain tolerance, and pain endurance data (in seconds) obtained from the PO, P+PW, and P+SI groups during the Pain period

Measure	PI	Threshold	Tolerance	Endurance
Group				
PO				
M	4.2	154	478	324
SD	1.7	92	178	191
P+PW				
M	4.9	125	417	292
SD	1.4	178	191	189
P+SI				
M	5.9	114	466	352
SD	1.8	151	189	210

The Kolmogorov-Smirnov test was used with the cumulative number of subjects per group reporting tolerance during the ten minute time period. Using adjusted critical values for Dn maximum (Kaner, Mohanty, & Lyons, 1980), this test found that the P+PW distribution was significantly different from the P+SI distribution ($D = 0.4375$, $p < .05$). There were no significant differences between the PO and P+PW groups or between the PO and P+SI groups. The difference between the P+PW and P+SI distributions was greatest during the latter part of the Pain period indicating that while similar numbers of subjects were reporting tolerance at the beginning of this period, more subjects in the P+PW than the P+SI group reported tolerance toward the end of the Pain period. Figure 3 illustrates the distribution of subjects who did not report tolerance in each group across the ten Pain period minutes.

Postexperimental direct pain questionnaires. As described in the Method section, subjects were asked to complete two questionnaires assessing the pain experience immediately after the end of the Recovery period. These questionnaires were reworded so that the subject would describe the pain that was experienced during the Pain period. Table 5 presents the mean adjusted (for SES) values obtained from the Gracely descriptor scales and the MPQ scales.

The data were analyzed by a series of univariate ANCOVAs. In reference to the Gracely scales, subjects were asked to pick one word out of 12 that best described the intensity of the pressure pain (G-Int), its painfulness (G-Pain), and its unpleasantness (G-Unpl). The ANCOVAs found no significant differences among groups on any of these scales. Sixty-six

Figure Caption

Figure 3. The distribution of subjects in the PO, P+PW, and P+SI groups who did not report pain tolerance across the ten Pain period minutes.

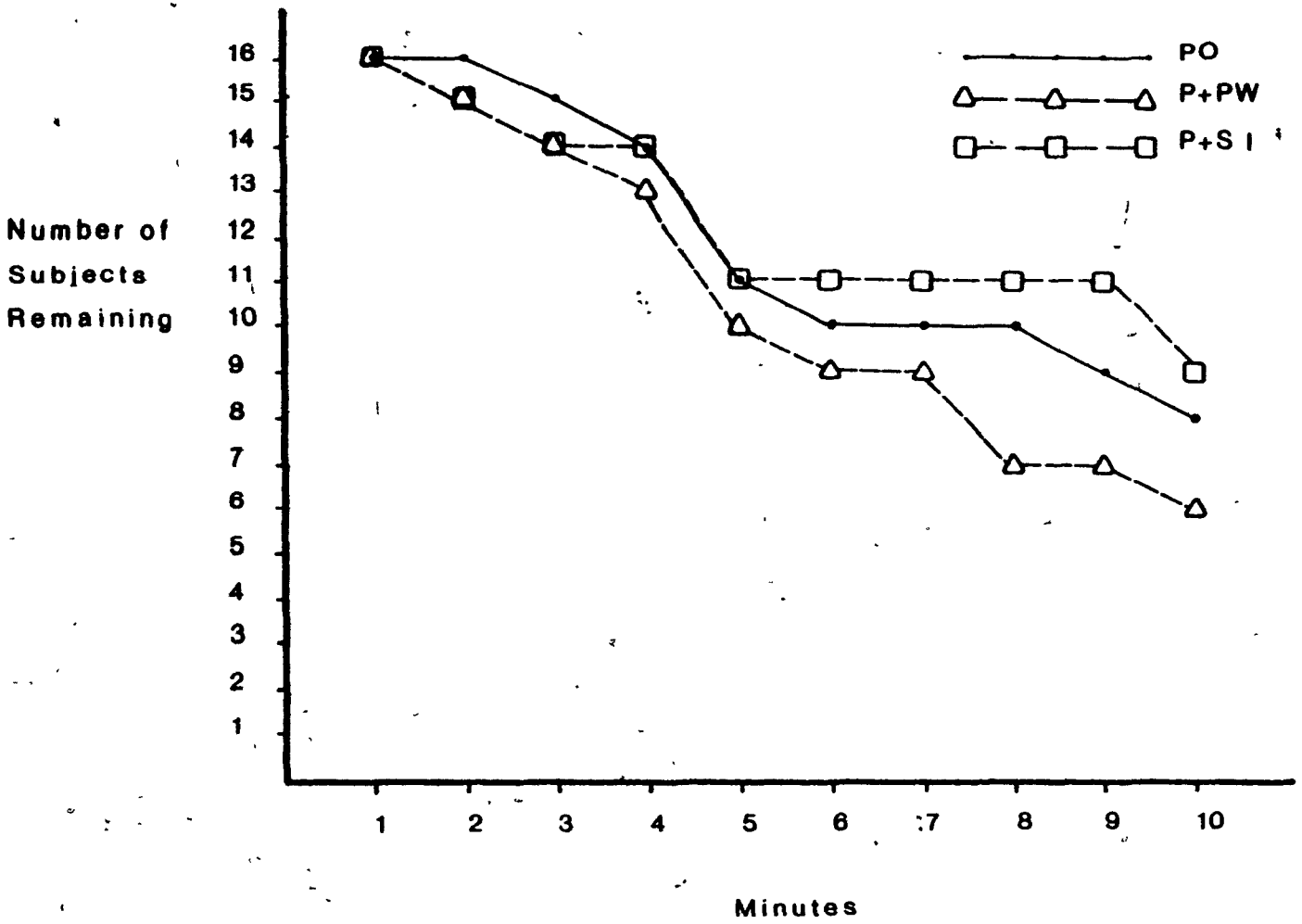

Number of Subjects Remaining
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
1
2
3
4
5
6
7
8
9
10
Minutes
PO
P+PW
P+S I

Table 5

The mean adjusted (for SES) scores obtained from the postexperimental direct pain questionnaires

Measure	G-Int	G-Unpl	G-Pain	PRI-S	PRI-A	PRI-M	PRI-E	PRI-T
Group								
PO								
M	8.1	4.6	5.7	14.7	1.4	4.1	1.6	21.8
SD	2.7	2.3	3.3	6.0	2.4	2.7	1.4	10.5
P+PW								
M	9.1	5.0	6.6	11.2	0.5	4.6	2.0	18.3
SD	1.9	2.4	2.8	7.0	1.1	3.5	1.6	11.7
P+SI								
M	9.4	4.8	6.5	13.7	1.9	4.7	2.1	22.3
SD	1.5	2.9	2.7	5.4	2.1	2.2	1.6	8.1
Grand Mean								
M	8.9	4.8	6.3	13.2	1.3	4.5	1.9	20.8
SD	2.1	2.5	2.9	6.2	2.0	2.8	1.5	10.1

See text for key to abbreviations.

percent of the entire sample reported an intensity level between "barely intense" and "very intense". Sixty-six percent of the sample reported a painfulness level between "mildly painful" and "pretty painful". Sixty-six percent of the subjects reported an unpleasantness level between "annoying" and "distressing".

Means from the four scales of the MPQ were obtained; the sum of the ranks of the sensory words chosen (PRI-S), the sum of the ranks of the affective words chosen (PRI-A), the rank of the evaluative word (PRI-E), and the sum of the ranks of the miscellaneous words chosen (PRI-M). The sum of the ranks of all words chosen (PRI-T) was also obtained. The ANCOVAS found no significant differences among groups on any of these measures. Within the sensory category, more than 75% of the subjects chose the words "pressing", "pulsing", and "hurting" to describe their experience. Very few affective words were chosen. The most common word chosen for the evaluative component was "troublesome"; 66% of the entire sample reported a level between "annoying" and "miserable". Within the miscellaneous category, 75% of the sample chose the words "radiating", "numb", and "drawing" to describe their experience.

Conclusions. This section reported the results obtained from the analyses of the direct pain measures. In general, it appears that both experimental manipulations were effective at increasing pain intensity ratings reported during nociception when compared to the control group. The stress interview warning appeared to have more profound effects on this report than the pain warning. There were no significant differences among groups on pain threshold, tolerance, and endurance times.

However, significantly more subjects from the P+PW group than the P+SI group reported tolerance toward the end of the pain period. There were no significant differences among groups on any of the postexperimental direct pain questionnaire scales. These results are not consistent with the results obtained by asking the subjects to rate their pain experience while it was occurring. Interpretation of these findings and the issues they raise will be presented in the Discussion section.

The indirect pain measures

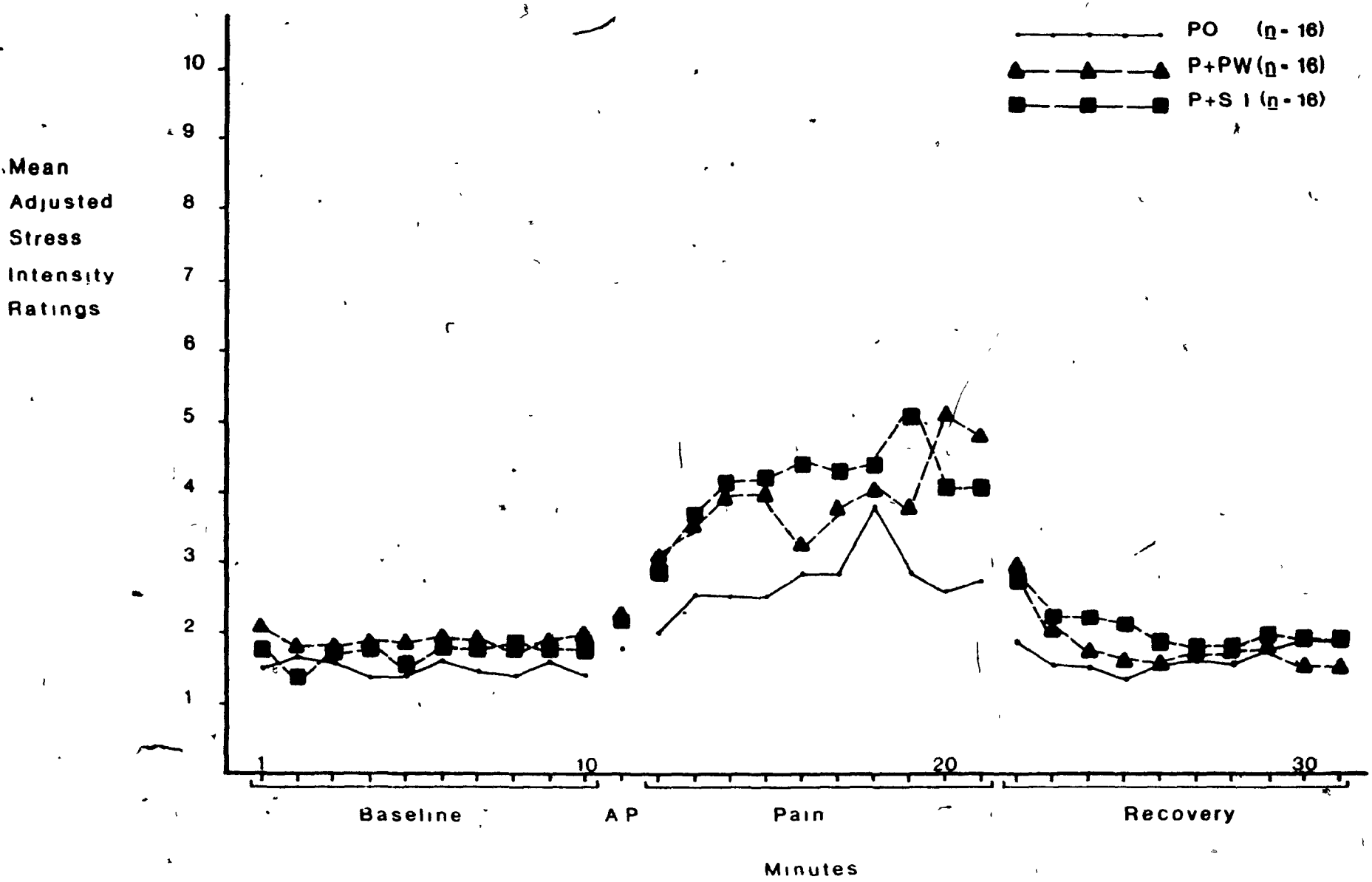
The indirect pain measures employed in the present study included the stress intensity rating data, the psychophysiological measurements, the behavioral response data, and postexperimental questionnaire data.

Stress intensity ratings. This section examines the analyses conducted with the stress intensity rating data that were performed in an identical fashion as the analyses with the pain intensity rating data. Statistical tables are presented in Appendix G. Figure 4 depicts the mean adjusted (for SES) stress intensity ratings obtained from each minute of Baseline, AP, Pain, and Recovery periods from the PO, P+PW, and P+SI groups. A 3 (groups) X 31 (minutes) repeated measures ANCOVA was conducted using group means to fill missing data cells. This analysis found a significant minutes effect ($F(30,60) = 40.00$, $p < .001$) and a significant group by minutes interaction ($F(60,1350) = 2.15$, $p < .005$). There was no effect of group membership or of the covariate.

Orthogonal polynomials were calculated separately using the Baseline, Pain, and Recovery minutes data to determine differences in the manner in which groups reported stress over

Figure Caption

Figure 4. Mean adjusted (for SES) stress intensity ratings obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups using group means to fill missing data cells.


PO (n - 16)
P+PW (n - 16)
P+S I (n - 16)
Mean Adjusted Stress Intensity Ratings
10
9
8
7
6
5
4
3
2
1
1
10
20
30
Baseline
AP
Pain
Recovery
Minutes

time. There were no significant effects found from the analysis of the Baseline minutes.

Orthogonal analysis of the Pain period revealed a significant group difference ($F(2,44) = 5.64$, $p < .01$), no effect of the covariate, a significant minutes effect ($F(9,18) = 5.65$, $p < .001$), and a significant group by minutes interaction ($F(18,405) = 2.10$, $p < .03$). Significant linear and quadratic trends were found as well as a significant interaction between group and quadratic trend ($F(2,45) = 7.43$, $p < .002$). This indicates that the stress intensity ratings increased more quickly at the beginning of the Pain period and then decreased in the rate of acceleration. In addition, this decrease in acceleration differed among groups. Visual inspection of Figure 4 suggests that the rate of increase of the stress ratings obtained from the PO subjects decreased before the ratings from the other groups decreased.

Orthogonal analysis of the Recovery period revealed no effect of group membership or of the covariate. A significant minutes effect was found ($F(9,18) = 6.90$, $p < .001$) as well as a trend for a significant group by minutes interaction ($F(18,405) = 1.96$, $p < .07$). There were significant linear, quadratic, and cubic trends as well as a significant interaction between group and linear trend ($F(2,45) = 3.57$, $p < .04$). This indicates that the groups differed in the rate at which stress ratings decreased over time. Visual inspection of Figure 4 suggests that these ratings began at higher levels in the P+PW and P+SI groups whereas there is no discernable difference across time in the ratings reported by the PO group.

Scheffé's tests were conducted to determine differences among groups during the Pain period minutes. Comparisons

between the PO and the P+PW groups during the Pain period found that the P+PW subjects reported significantly higher stress during the P3, P4, P8, P9, and P10 minutes. Comparisons between the PO and P+SI groups revealed that the P+SI group reported significantly higher stress during the P2, P3, P4, P5, P6, P8, P9, and P10 minutes. Comparisons between the P+PW and P+SI groups found two significant differences during the P5 and P9 minutes. In the first of these comparisons, the P+SI subjects reported higher stress than the P+PW subjects and in the second comparison, the P+PW subjects reported higher stress. Thus, the experimental groups reported significantly higher stress intensity ratings during the Pain period than the control group.

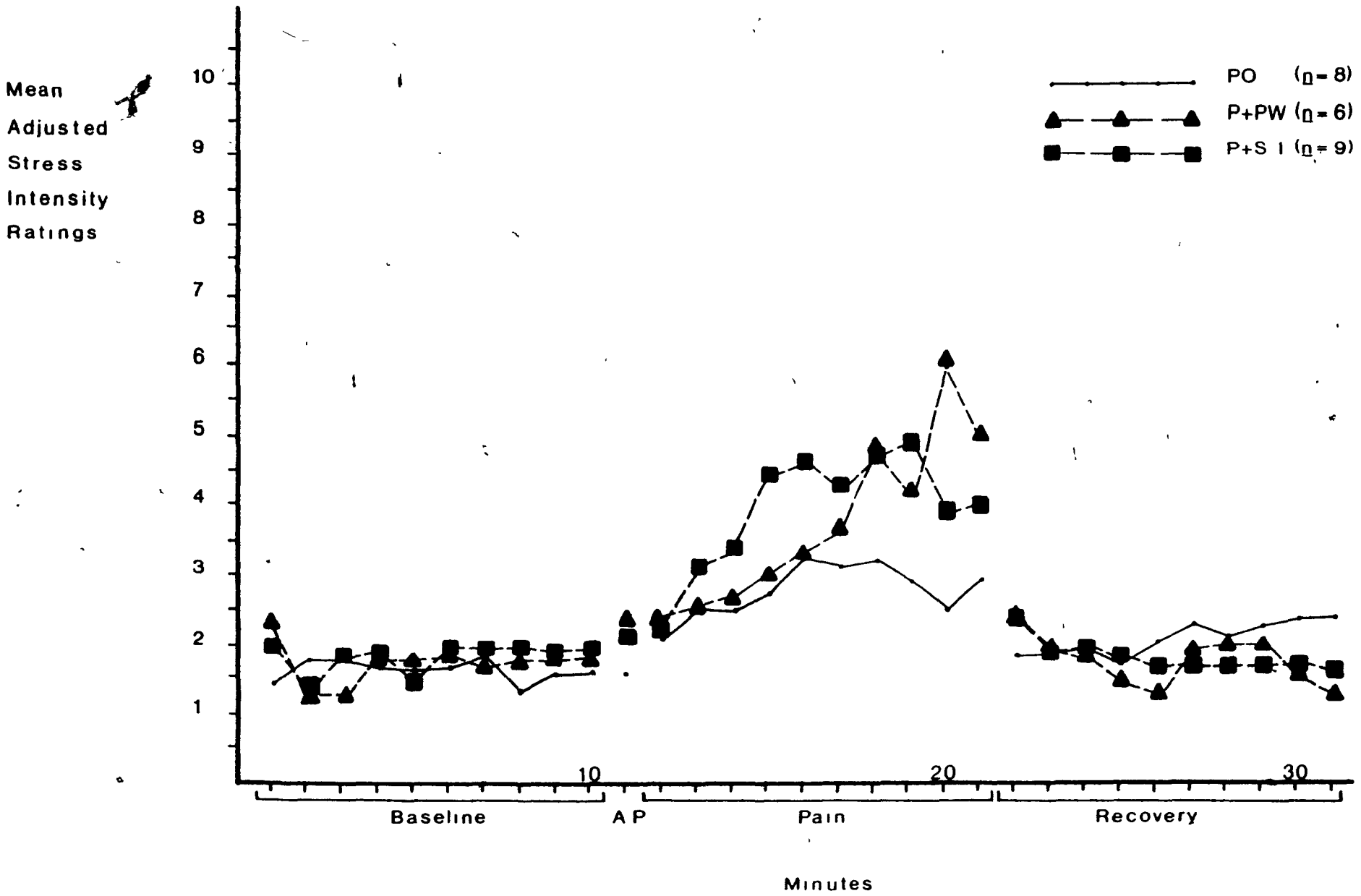
Figure 5 depicts the mean adjusted stress intensity ratings obtained from subjects who did not report tolerance within the PO (n = 8), P+PW (n = 6), and P+SI (n = 9) groups across the 31 minutes. The repeated measures ANCOVA revealed no group effect, no effect of the covariate, and no group by minutes interaction. There was a significant minutes effect ($F(30,60)$ = 15.78, $p < .001$) indicating that the stress ratings changed over time.

Orthogonal polynomials were calculated during the Baseline, Pain, and Recovery periods. No significant effects were found by the analysis of the Baseline minutes.

Orthogonal analysis of the Pain period found no effect of the covariate and no group differences. There was a significant minutes effect ($F(9,18)$ = 6.89, $p < .001$) and a significant interaction between group and minutes ($F(18,180)$ = 2.19, $p < .02$). This interaction was not found by the previous repeated measures ANCOVA of the 31 minutes and appears to be due to an increase

Figure Caption

Figure 5. Mean adjusted (for SES) stress intensity ratings obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups composed of subjects who did not report tolerance.


Mean
Adjusted
Stress
Intensity
Ratings
10
9
8
7
6
5
4
3
2
1
PO (n = 8)
P+PW (n = 6)
P+S I (n = 9)
10
20
30
Baseline
A P
Pain
Recovery
Minutes

in the sum of squares of the interaction component (see Appendix G). Significant linear and quadratic trends were found and there was a trend for a significant interaction between group and trend ($F(2,20) = 3.24$, $p < .07$). In addition, there was a significant interaction between group and quadratic trend ($F(2,20) = 5.34$, $p < .02$). This indicates that the stress ratings increased and decreased over time at different rates depending upon group membership. Visual inspection of Figure 5 suggests that the stress ratings obtained from the P+SI subjects increased faster than the other two groups before decreasing and that the rate of increase of the stress ratings obtained from the P+PW subjects did not decrease in the same fashion as the other two groups.

Orthogonal analysis of the Recovery minutes found no significant effects.

Scheffé's tests were conducted to compare groups during the Pain period minutes. Comparisons between the PO and P+PW groups showed that the P+PW group rated P9 and P10 as significantly more stressful. Comparisons between the P+PW and the P+SI groups showed that the P+SI subjects rated P4 and P8 as significantly more stressful. Comparisons between the P+PW and P+SI subjects found that the P+PW subjects rated P9 as significantly more stressful.

To review, the repeated measures ANCOVA performed using the stress intensity ratings obtained from subjects who did not report tolerance found a significant difference over time in self-report of stress yet these reports did not differ as a function of group membership. However, orthogonal analyses indicated that the groups were significantly different in the

rate at which stress ratings increased and decreased during the Pain minutes. Scheffé's tests showed that the experimental groups rated the Pain period as more stressful than the control group in two out of ten comparisons. There were no differences between groups during the Recovery period. Therefore, these results are similar to those found previously when analyzing data from the entire sample.

A univariate ANCOVA was conducted using the mean adjusted stress intensity rating obtained from each subject during the Pain period. The ANCOVA found a significant group difference ($\underline{F}(2,44) = 5.61$, $\underline{p} < .007$) and a significant effect of the covariate. Scheffé's tests comparing groups found a significant difference between the PO and the P+PW groups ($\underline{F}(2,45) = 4.19$, $\underline{p} < .05$) and between the PO and P+SI groups ($\underline{F}(2,45) = 5.21$, $\underline{p} < .01$). In both of these comparisons, the experimental groups reported significantly higher stress ratings than the control group. There was no significant difference between the P+PW and P+SI groups. Table 6 presents the adjusted (for SES) mean stress intensity rating (SI), and the mean adjusted HR and EMG data from subjects in each group.

Psychophysiological measures. This section examines the HR and EMG data collected from each subject during the experiment. The three analyses conducted with each set of data were similar to those conducted for the pain and stress intensity rating data. First, a 3 (group) X 24 (minutes) repeated measures ANCOVA was conducted using the first five minutes of Baseline physiological data as a covariate and group means to fill missing data cells. These five minutes of Baseline were not included in the analyses thereby reducing the number of minutes

Table 6

Mean adjusted stress intensity ratings (SI), HR (bpm), and EMG (uV) obtained from the PO, P+PW, and P+SI groups during the Anticipation (AP) and Pain periods

Measure	SI		HR		EMG	
Period	AP	Pain	AP	Pain	AP	Pain
Group						
PO						
M	1.5	2.6	70	69	67	66
SD	0.7	1.4	8	8	28	29
P+PW						
M	2.4	4.2	74	76	86	79
SD	1.1	1.8	13	12	34	35
P+SI						
M	2.3	4.3	73	71	76	66
SD	0.8	1.7	9	8	24	18

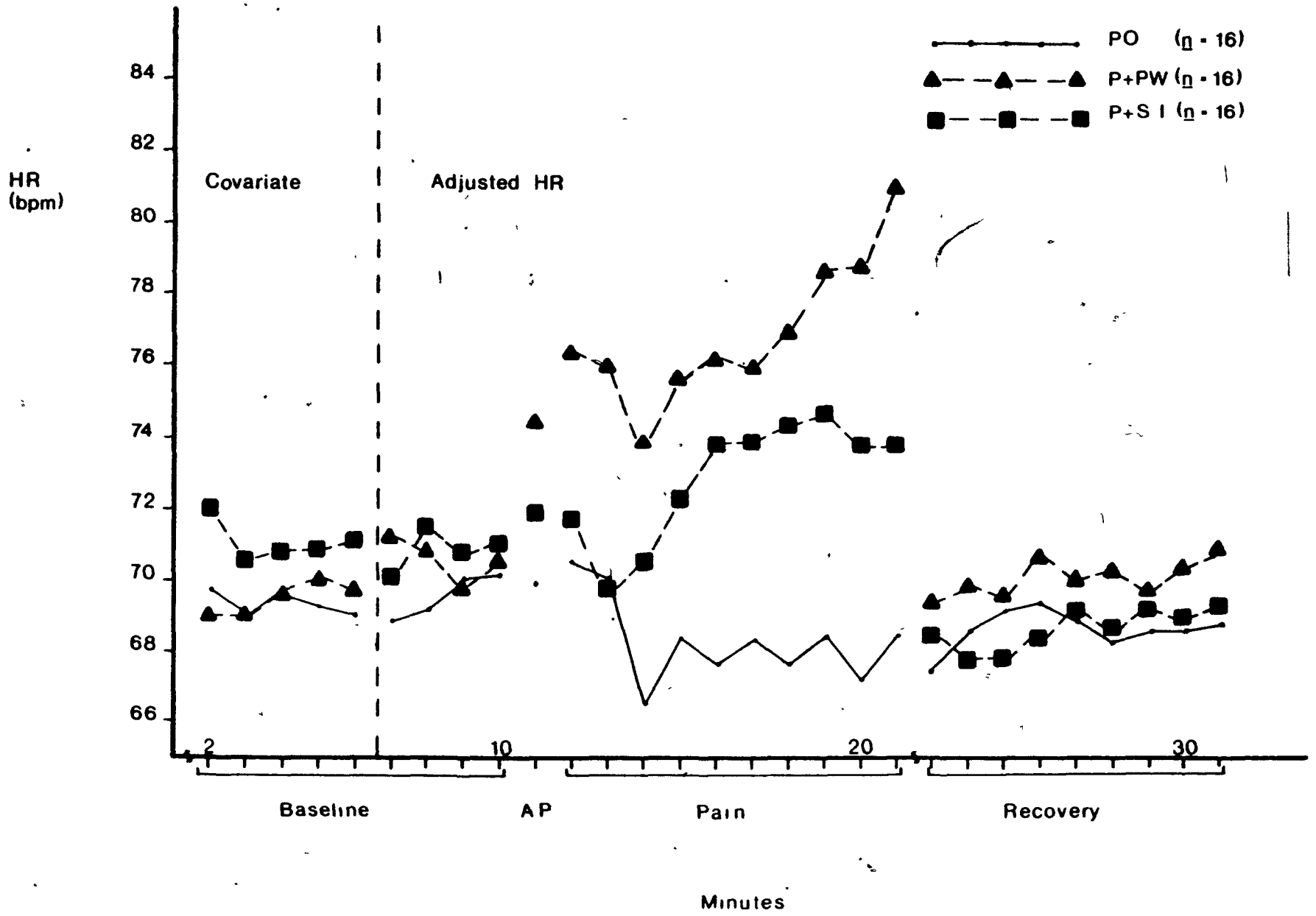
from 29 to 24. Scheffé's tests were conducted when appropriate. Second, a 3 (groups) X 24 (minutes) repeated measures ANCOVA was conducted using Baseline physiological data as a covariate and using only the subjects who did not report tolerance. After each repeated measures ANCOVA, orthogonal polynomials were calculated with the data obtained from the AP minute included in the Pain minutes data. Third, a univariate ANCOVA was performed to determine differences between groups during the Pain period. Consistent with all other repeated measures analyses, significance was determined by using the Huynh-Feldt adjustment for conservative degrees of freedom (Huynh & Feldt, 1976) and the first two minutes of the Recovery period data obtained from the P+SI subjects consisted of the data obtained from the stress interview anticipation period. Due to equipment failure, EMG was not available from one subject in the P+SI group. This subject was dropped from analysis and thus, the sample size for the P+SI group during the EMG analyses was 15. The results of the statistical analyses that are not presented in this section are included in Appendix H.

Figure 6 depicts the mean adjusted HR (in beats per minute; bpm) obtained from the PO, P+PW, and P+SI subjects during the Baseline, AP, Pain, and Recovery period minutes using group means to fill missing data cells. The repeated measures ANCOVA found a significant group effect ($\underline{F}(2,44) = 12.12$, $\underline{p} < .001$), a significant effect of the covariate, a significant minutes effect ($\underline{F}(23,46) = 9.00$, $\underline{p} < .001$), and a significant group by minutes interaction ($\underline{F}(46,1035) = 3.95$, $\underline{p} < .002$).

Orthogonal polynomials calculated using the Baseline minutes data found no significant effects.

Figure Caption

Figure 6. Mean adjusted (for initial values) HR data (in bpm) obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups using group means to fill missing data cells.


PO (n - 16)
P+PW (n - 16)
P+S I (n - 16)
HR (bpm)
84
82
80
78
76
74
72
70
68
66
Covariate
Adjusted HR
2
10
20
30
Baseline
AP
Pain
Recovery
Minutes

Orthogonal polynomials calculated using the AP and Pain period minutes data found a significant group difference ($F(2,44) = 20.22$, $p < .001$), a significant effect of the covariate, a significant minutes effect ($F(10,20) = 3.34$, $p < .03$), and a significant group by minutes interaction ($F(20,450) = 2.23$, $p < .05$). There were significant quadratic, sixth order, and seventh order trends indicating that the HR increased and decreased several times during this period. No significant group by trend interactions were found.

Orthogonal polynomials calculated using the Recovery minutes data found no significant effects with the exception of a significant group by cubic trend interaction ($F(2,45) = 4.21$, $p < .03$). Visual inspection of Figure 6 suggests that the three groups differed in the manner in which HR increased and decreased during the Recovery minutes yet these groups manifested similar HR responses during the initial minutes of this period.

Scheffé's tests were conducted to determine differences in HR during the AP and Pain minutes. All comparisons between the PO and P+PW groups found that the P+PW group manifested significantly higher HR than the PO subjects during this time period. The P+SI subjects showed significantly higher HR in nine of the 11 comparisons with the PO subjects (AP, P3, P4, P5, P6, P7, P8, P9, and P10). The P+PW subjects manifested significantly higher HR than the P+SI subjects in four of the 11 comparisons (P1, P2, P9, and P10).

Therefore, both experimental groups manifested significantly higher HR during the AP and Pain minutes than the control group when using the data from all subjects and group means to fill

missing data cells.

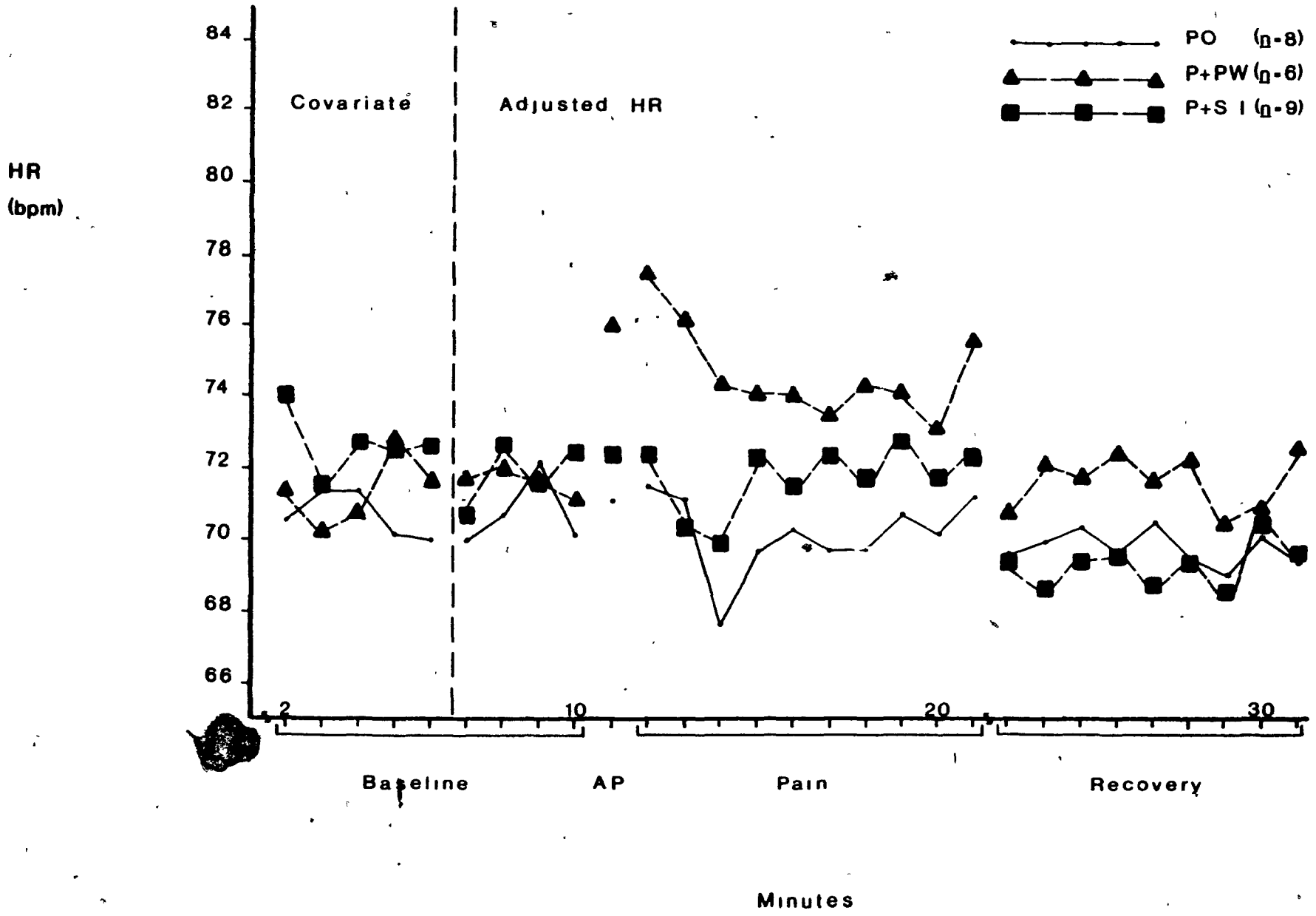
Figure 7 depicts the mean adjusted HR obtained from subjects in each group who did not report tolerance across the four periods. The repeated measures ANCOVA found no effect of group membership, a significant effect of the covariate, a significant minutes effect ($F(23,46) = 2.68$, $p < .02$), and no group by minutes interaction.

Orthogonal analysis of the Baseline minutes found no significant effects. Orthogonal analysis of the AP and Pain period minutes found a trend for a significant group difference ($F(2,19) = 2.77$, $p < .09$), a significant minutes effect, yet no group by minutes interaction. There was a significant quadratic trend indicating a deceleration in the rate of decrease in HR over time. This decrease in HR over time indicates that subjects were habituating to the nociceptive stimulus (Obrist, 1981). Orhtogonal analysis of the Recovery period found no significant effects. Therefore, when using the data obtained from subjects who did not report tolerance during the Pain period, there were no significant differences among groups.

Table 6 presented the mean adjusted stress intensity rating, and the mean adjusted HR and EMG data that was obtained during the Pain period. In reference to HR, the univariate ANCOVA found a significant effect of group membership ($F(2,44) = 6.21$, $p < .001$) and a significant effect of the covariate. Scheffé's tests comparing the PO group with the P+PW group showed that the P+PW subjects manifested a significantly higher HR during this period ($F(2,45) = 7.21$, $p < .01$). The P+PW subjects also manifested significantly higher HR than the P+SI subjects ($F(2,45) = 3.68$, $p < .05$). There was no significant difference between the PO

Figure Caption

Figure 7. Mean adjusted (for initial values) HR data (in bpm) obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups composed of subjects who did not report tolerance.


HR
(bpm)
84
82
80
78
76
74
72
70
68
66
Covariate
Adjusted HR
PO (n-8)
P+PW (n-6)
P+S I (n-9)
2
10
20
30
Baseline
AP
Pain
Recovery
Minutes

and P+SI subjects during the Pain period. These results and possible reasons for their occurrence will be considered in the Discussion section.

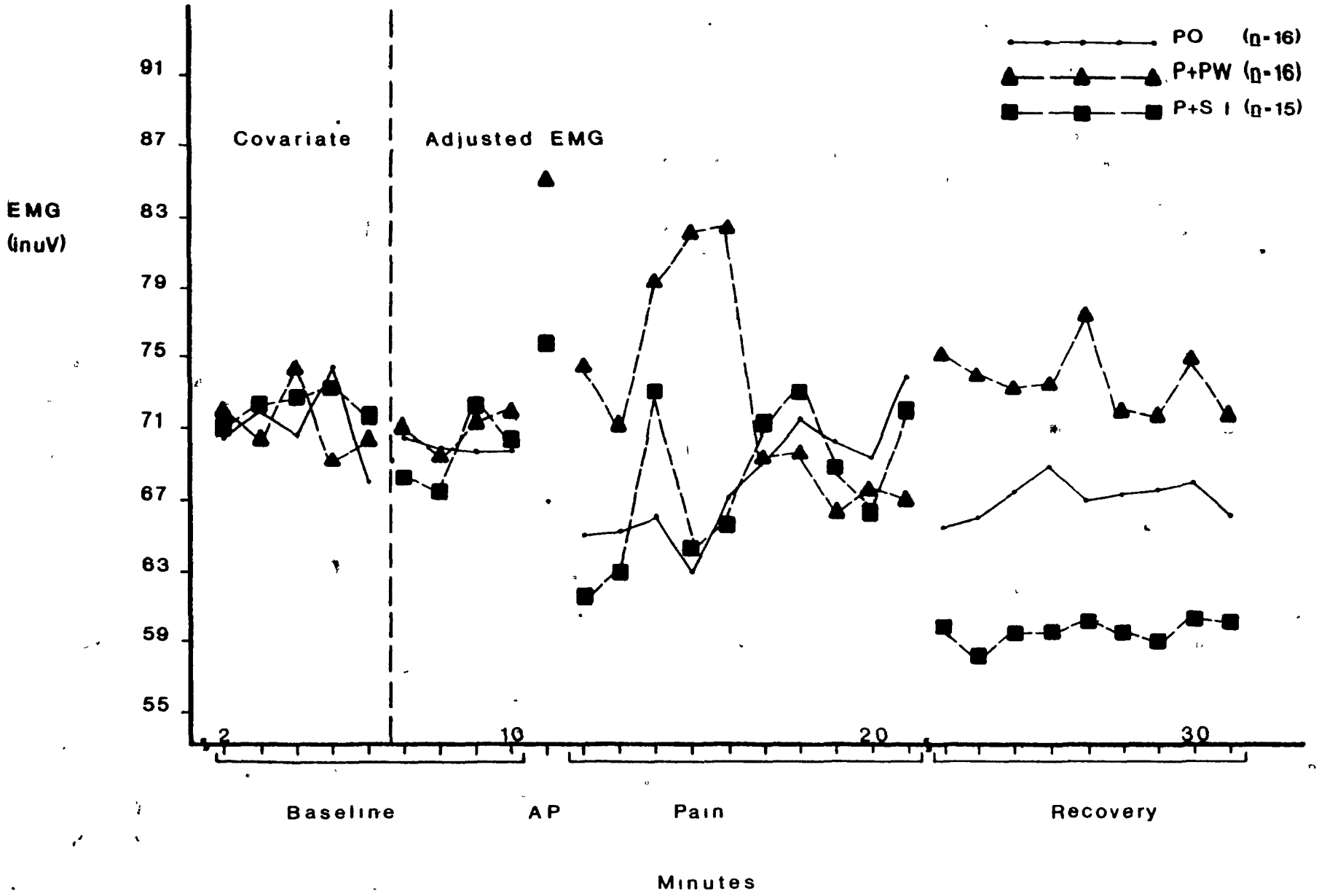
Figure 8 depicts the mean adjusted EMG (in uV) obtained during the Baseline, AP, Pain, Recovery periods using group means to fill missing data cells and covarying the first five minutes of Baseline EMG. The repeated measures ANCOVA found a trend for a significant effect of group membership ($F(2,43) = 3.19$, $p < .06$), a significant effect of the covariate, no minutes effect, and a trend for a significant interaction between group and minutes ($F(46,1012) = 1.78$, $p < .06$).

Orthogonal polynomials calculated during the Baseline period found no significant effects. Orthogonal polynomials calculated during the AP plus Pain minutes found no effect of group, no minutes effect, and a significant group by minutes interaction ($F(20,440) = 2.41$, $p < .03$). There were significant quartic, sixth order and eighth order trends indicating that EMG increased and decreased several times over the AP and Pain minutes. There was a trend for a significant interaction between group and linear trend ($F(2,44) = 2.87$, $p < .07$) and significant group by trend interactions on the quartic and sixth order trends ($F(2,44) = 3.25$, $p < .05$; and $F(2,44) = 6.76$, $p < .003$, respectively). Visual inspection of Figure 8 suggests that the shape of the curves does not appreciably differ between the PO and P+SI groups. The P+PW subjects began the AP and Pain period with higher EMG which decreased and increased before decreasing to a similar level as the other groups.

Orthogonal polynomials calculated on the Recovery data found a significant group difference ($F(2,43) = 6.77$, $p < .003$)

Figure Caption

Figure 8. Mean adjusted (for initial values) EMG data (in uV) obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups using group means to fill missing data cells.


PO (n-16)
P+PW (n-16)
P+S I (n-15)
Covariate
Adjusted EMG
EMG (inuV)
91
87
83
79
75
71
67
63
59
55
2
10
20
30
Baseline
AP
Pain
Recovery
Minutes

and a significant effect of the covariate but no other significant effects.

Scheffé's tests were performed to determine differences between groups across minutes during the AP and Pain minutes. Four of the 11 comparisons between the PO and the P+PW subjects showed that the P+PW subjects had a higher EMG during this time period (AP, P3, P4, and P5). There were no significant differences between the PO and P+SI groups on any of the 11 comparisons. The P+PW group manifested significantly higher EMG than the P+SI group during P4 and P5. The results of these comparisons support the conclusions drawn from the orthogonal analysis of the AP and Pain minutes.

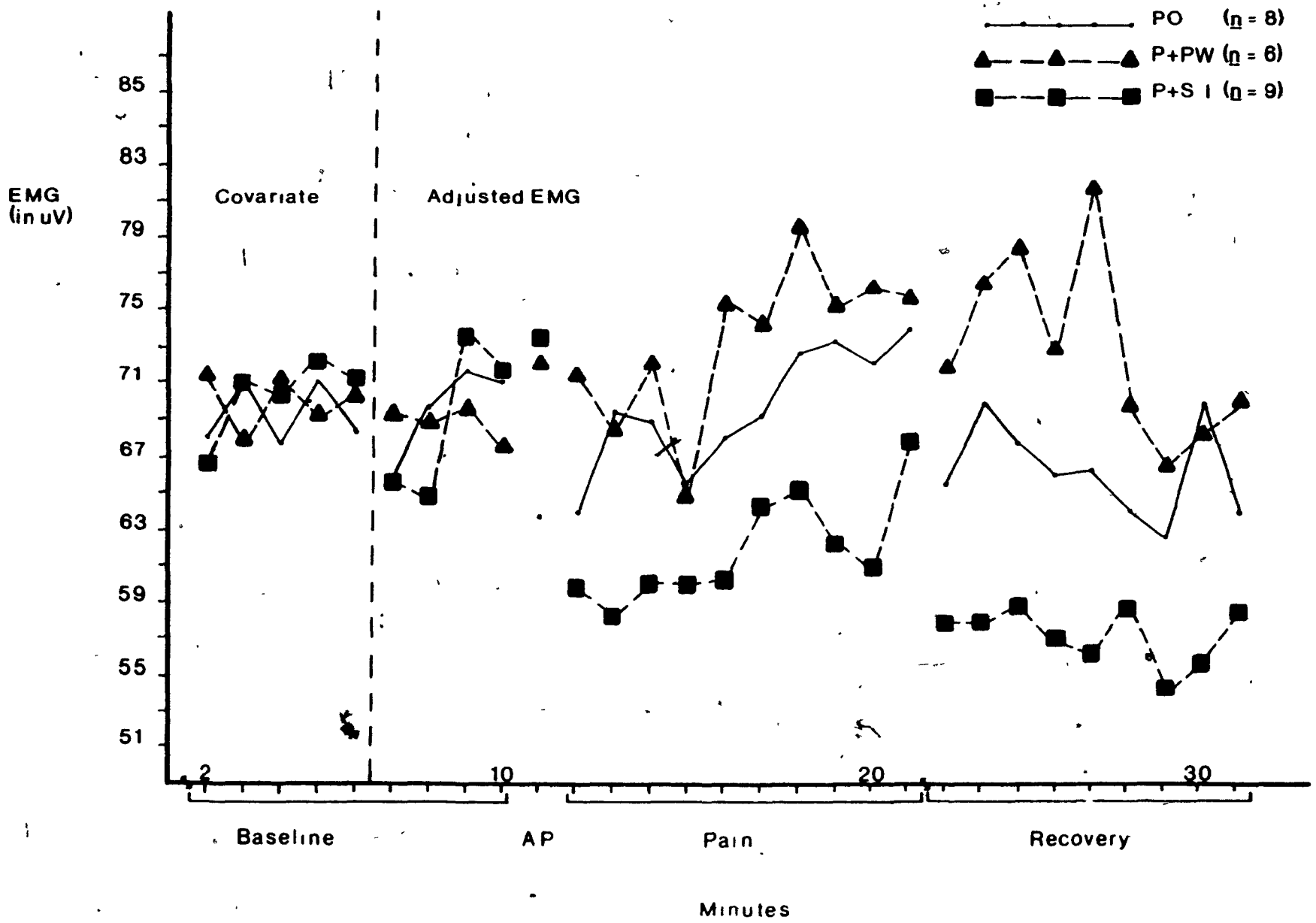
Scheffé's test was used to determine the nature of the group effect during Recovery. The P+PW group manifested a significantly higher EMG than the P+SI group ($\underline{F}(2,44) = 9.42$, $\underline{p} < .01$) yet did not differ significantly from the PO group. The difference between the PO and P+SI groups was not significant.

These results indicate that the P+PW subjects manifested increased EMG after hearing the warning and during the initial part of the Pain period compared to the control group. However, the P+SI subjects did not differ from the control subjects on this measure. In addition, the P+PW subjects manifested sustained increases in EMG during the Recovery period whereas EMG decreased in the P+SI subjects.

Figure 9 depicts the mean adjusted EMG obtained from the subjects who did not report tolerance during the Baseline, AP, Pain, and Recovery periods. The repeated measures ANCOVA found a trend for a significant effect of group membership ($\underline{F}(2,19) = 2.63$, $\underline{p} < .10$) and a significant effect of the covariate. There

Figure Caption

Figure 9. Mean adjusted (for initial values) EMG data (in uV) obtained during the Baseline, Anticipation (AP), Pain, and Recovery periods from the PO, P+PW, and P+SI groups composed of subjects who did not report tolerance.


PO (n = 8)
P+PW (n = 6)
P+S I (n = 9)
EMG (in uV)
85
83
79
75
71
67
63
59
55
51
Covariate
Adjusted EMG
2
10
20
30
Baseline
AP
Pain
Recovery
Minutes

was a trend for a significant minutes effect ($F(23,460) = 1.81$, $p < .06$) but no other significant effects. No further analyses were conducted since none of the major effects were close to significance. Thus, when using the subjects who did not report tolerance, the repeated measures ANCOVA failed to find any significant differences between groups in EMG levels. Since visual inspection of Figure 9 suggests that the data collected were similar to those obtained when using all the subjects' data and group means to fill missing data cells, this result may be due to the small sample size and to the very high variability ($MSw = 117.8$).

Table 6 (p. 107) included the mean adjusted Pain period EMG from each individual within the three groups. The ANCOVA found a trend for a significant effect of group membership ($F(2,43) = 3.05$, $p < .06$). Once again, the variability in measurement was very high ($MSw = 260$) possibly obscuring differences between the groups. The P+PW subjects appeared to manifest a higher average EMG during this period than either the PO or P+SI subjects. Thus, when taking an average EMG during the Pain period, there was a trend for the P+PW group to have higher levels of EMG than the other two groups.

Behavioral response data. This section presents the results obtained from the analyses of the behavioral response data. The frequency of fourteen movements was rated as described in the Method section. Due to equipment failure, data from three subjects in the PO group, and one subject from both the P+PW and P+SI groups were not available for analysis. These subjects were dropped from analysis. Two sets of identical analyses were conducted; one with the overall frequency data and the

second with the grimace frequency data. The frequency of movement during the Baseline period (minutes seven and eight) was used as a covariate. Appendix I includes the statistical analyses conducted with this data.

Table 7 presents the mean adjusted frequency of overall movement (BR) and of grimaces (G) during the first minute of the Pain period (Pain F) and the last minute of the period (Pain L). Four univariate ANCOVAs were performed with this data. There were no significant differences among groups in the frequency of overall movement during either Pain F or Pain L. The analyses of the grimace data found a significant difference among groups on the Pain F grimace data ($F(2,39) = 3.54$, $p < .04$). There were no group differences on the Pain L grimace data. Scheffe's tests found that the P+PW group showed a greater frequency of grimaces than the PO group ($F(2,40) = 3.54$, $p < .05$) but not the P+SI group. There was no significant difference between the PO and P+SI groups. Thus, there were no significant differences among groups in terms of the overall frequency of movements. However, the P+PW subjects showed more grimaces during the first minute of pressure pain than the PO subjects.

Postexperimental indirect pain questionnaires. The postexperimental questionnaire data that indirectly assessed the pain experience were analyzed by a series of univariate ANCOVAs covarying SES (see Appendix J). Appendix J includes the mean adjusted (for SES) values obtained from these questionnaires from each group and from the entire sample receiving pressure nociception.

The ANCOVAs found no significant differences among groups on any of these postexperimental indirect pain measures. As a

Table 7

Mean adjusted (for initial values) frequency of the behavioral response (BR) and the grimace (G) data obtained during the first and last minutes of nociception

Measure	BR-First	BR-Last	G-First	G-Last
Group				
PO				
M	6.3	5.5	0.2	0.2
SD	4.1	2.7	0.3	0.4
P+PW				
M	7.9	6.9	0.6	0.4
SD	2.3	2.6	0.5	0.5
P+SI				
M	7.1	5.6	0.4	0.4
SD	3.3	2.8	0.5	0.6

group, subjects reported the pain experience was moderately representative of other pain experiences outside of the laboratory ($\underline{M}$ = 4.2 on the one to ten visual analog scale). The average rating by the entire sample pertaining to how painful they believed the pressure be on the one to five scale was 3.6. The sample reported experiencing a mild degree of stress prior to pressure pain and while completing the postexperimental questionnaires. The means of the one to ten analog scales were 3.4 and 2.6 respectively.

In response to the question "Did you use any strategies to cope with the pressure pain?"; 14 subjects from the PO group, 10 from the P+PW group, and 13 from the P+SI group responded affirmatively. In response to the question "If yes, what were they?"; six subjects reported relaxation or meditation, 19 subjects reported distraction or trying not to think about it, one subject reported focussing on the pain, eight subjects reported reinterpreting the experience by using self-statements such as "This is not so bad", and three subjects reported using self-control. The chi-square test for contingency tables found no significant difference between the type of strategy employed and group membership ($\chi^2(8) = 7.5$, ns). In response to the question "How effective do you think these strategies were?" on a one to five visual analog scale, the average rating obtained from the entire sample of 37 was 4.1 ($\underline{SD}$ = 0.94) indicating that subjects who used a strategy found it effective. There were no significant differences between groups pertaining to this rating of effectiveness.

These results indicate that the postexperimental questionnaire data is not consistent with results obtained

by asking subjects to rate their experience as it was occurring. Postexperimental questionnaires may be insensitive to differences in the experience of pressure pain. This issue will be considered further in the Discussion section.

Conclusions. This section reported the results obtained from the analyses of the indirect pain measures. In general, both experimental manipulations increased stress intensity ratings reported during nociception when compared to the control group. Both experimental groups manifested higher HR during the AP than the control group. However, it appears that only subjects receiving a pain warning had a higher overall HR during nociception than the control group. Subjects who did not report tolerance showed a decrease in HR over time indicating habituation to the stimulus.

Subjects receiving a pain warning manifested higher EMG than control subjects during the AP and first half of the Pain period whereas there were no signigicant differences between the P+SI subjects and the control group. Subjects receiving a pain warning also had higher sustained levels of EMG during Recovery than subjects who had received a stress interview warning. The frequency of grimaces during the first minute of nociception was higher in the P+PW group than the control group yet no significant difference was found between the P+SI and control subjects. Postexperimental indirect pain questionniares failed to find any significant differences among groups.

A comparison between subjects who reported tolerance and subjects who did not

This section compares subjects who reported tolerance during

pressure nociception (RT) with subjects who did not (NT) on the main measures. Since no directional predictions were made prior to this study, a two-tailed rejection region for significance was employed. Statistical analyses are included in Appendix K.

Table 8 presents the demographic and preexperimental data obtained from subjects divided into the RT and NT groups. Student's t tests were calculated on the quantifiable data and chi-square statistics were calculated with the nominal data. The t tests found two significant differences between these two groups. Subjects who reported tolerance also had significantly lower SES scores ($t(46) = 2.18$, $p < .04$) and significantly higher evaluation anxiety on the GTAR-EA ($t(46) = -2.12$, $p < .04$).

Chi-square statistics were calculated for the season tested, citizenship, language, and religious affiliation data. A significant difference between religious affiliation and group membership was found ($\chi^2(4) = 12.69$, $p < .02$). Table 9 presents the frequency of reported religious affiliation from subjects in the RT and NT groups. Visual inspection of this table indicates that subjects reporting affiliation with the Jewish faith were the most likely to report tolerance during the experiment and subjects who reported no affiliation were the least likely to report tolerance. No other significant differences were found on any of the preexperimental measures.

To assess the possible relationship between SES and religious affiliation, a chi-square test for contingency tables was employed. Subjects were divided into two groups on the basis of a median split of the SES scores (median = 62) and further divided into the five religious affiliations. This test found no association between SES and religious affiliation ($\chi^2(4) =$

Table 8

Demographic and personality characteristics of the subjects who reported tolerance (RT) and of the subjects who did not report tolerance (NT)

Measure	Age	SES	Height/ Weight	TMAS	SDS	GTAR-EA	GTAR-PD	STAI-S	Stress
Group									
RT									
M	21.9	51.5	0.45	8.8	15.0	41.2	50.4	61.8	4.0
SD	3.9	16.3	0.05	5.4	2.6	10.4	13.0	7.7	2.1
NT									
M	20.4	61.1	0.46	6.8	15.0	34.8	52.9	64.7	4.0
SD	2.6	13.4	0.05	3.8	3.1	10.6	12.0	8.2	2.1

Key

TMAS = Taylor Manifest Anxiety Scale
SDS = Social Desirability Scale
GTAR-EA = Inventory of General Trait Anxiety Revised - Evaluation Anxiety
GTAR-PD = Inventory of General Trait Anxiety Revised - Physical Danger
STAI-S = State-Trait Anxiety Inventory - State measures
Stress = Present stress level on a one to ten visual analog scale

Table 9

Frequency of reported religious affiliation within the RT and NT groups

Affiliation Group	Protestant	Catholic	Jewish	Other	None
RT	4	6	10	1	4
NT	5	5	2	0	11

3.34, ns). Therefore, the SES and religious affiliation data appear to be associated with reporting tolerance in two distinct ways.

SES was not used as a covariate in the subsequent analyses of the pain and stress data to prevent reducing the effects of other variables that might have been correlated with it. The relationships between SES and other measures are discussed at the end of this section.

A repeated measures ANOVA was conducted to compare groups on the pressure stimulation data to determine if they differed on the grams of pressure exerted during the Pain period. This analysis found no effect of group membership or group by minutes interaction. There was a significant minutes effect ($F(9,9) = 4.48$, $p < .02$) indicating that the force exerted during the Pain period increased over time. This result was also found by the analysis of the pressure stimulation data comparing the PO, P+PW, and P+SI groups.

Table 10 presents the mean pain intensity rating, and the pain threshold, tolerance, and endurance data obtained from the RT and NT groups. There was no significant difference between groups on the average pain intensity rating obtained during the pain period. There were significant differences between groups on both threshold and endurance data. The RT group had a lower threshold ($t(46) = 3.47$, $p < .001$) and a shorter endurance time ($t(46) = 3.47$, $p < .002$). The Levene test for unequal variances (Brown & Forsythe, 1974b) found that the RT group had a significantly lower within-subjects variance associated with the pain threshold data than the NT group did. Using the t test employing separate error terms (Brown & Forsythe, 1974b),

Table 10

The mean pain intensity rating (PI), and the pain threshold, tolerance, and endurance values obtained from the RT and NT groups

Measure	PI	Threshold	Tolerance	Endurance
Group				
RT				
M	5.1	72	311	238
SD	1.5	59	158	161
NT				
M	4.9	199	600	401
SD	1.8	162	000	162

the RT group had a significantly lower threshold than the NT group ($t(27.6) = 3.49$, $p < .002$).

There were no significant differences between the groups on the mean stress intensity ratings obtained during the AP or Pain periods, the mean adjusted HR or EMG data obtained during the AP or Pain periods, or the frequency of grimaces obtained during the first or last minutes of pressure stimulation. The values associated with these measures are presented in Appendix K.

Two t tests were calculated to determine if these groups differed in the Baseline average level of HR or EMG. No significant differences were found indicating that neither group manifested higher levels of arousal during the Baseline period as assessed by these measures.

Data obtained from the postexperimental direct and indirect pain questionnaires were analyzed using a series of Student's t tests. The values obtained from these measures are presented in Appendix K. There were no significant differences between groups on the Gracely intensity scale or unpleasantness scale. Subjects in the RT group reported significantly more pain on the painfulness scale than subjects in the NT group ($t(46) = -2.36$, $p < .03$). No significant differences were found by the analyses of the MPQ scales.

No significant differences were found between groups by the analyses of the postexperimental indirect pain questionnaires with the following exception. Subjects in the RT group reported that the strategies they employed to help them cope with the pain were significantly less effective than subjects in the NT group ($t(35) = -2.51$, $p < .07$). There was no significant difference

between groups obtained from the chi-square test comparing the two groups on the type of strategy employed ($\chi^2(4) = 7.49$, ns).

Pearson product moment correlation coefficients (Roscoe, 1975) were calculated to determine the relationship between SES and the GTAR-EA, pain threshold and endurance, the Gracely painfulness scale, and the effectiveness of strategies employed. None of these correlation coefficients were significant. Thus, differences between the RT and NT groups do not appear to be due to a relationship between SES and these measures.

Conclusions. Subjects who reported tolerance in the present study also reported significantly lower SES and significantly higher evaluation anxiety than subjects who did not. Subjects who reported tolerance were also more likely to report affiliation with the Jewish faith and subjects who did not report tolerance were more likely to report no religious affiliation. SES and religious affiliation were not related. Subjects who reported tolerance also reported threshold at an earlier time, had shorter endurance times, higher scores on the Gracely painfulness scale, and significantly less effective strategies to help them cope with the pressure pain than subjects who did not report tolerance. SES was not significantly related with any of these measures. There were no other significant differences between the groups.

The relationship between the direct measures of pain

This section provides a preliminary assessment of the relationships between the direct measures of the pain experience. Pearson product moment correlation coefficients were calculated using the data from the sample receiving pressure nociception. The 13 direct pain measures employed were the pain threshold,

tolerance, and endurance data, the average pain intensity rating obtained during the Pain period, the three Gracely scales, the five MPQ scales, and the report of tolerance (1 = yes; 2 = no). A correlation matrix of these measures is presented in Appendix L.

In general, pain measures of the same type (threshold, tolerance and endurance; postexperimental questionnaires; and so on) were more likely to be significantly correlated with each other. The exception to this was the mean pain intensity rating. This measure was significantly correlated with threshold (r = -.43, $p < .01$), endurance (r = .40, $p < .01$), the three Gracely scales (Intensity - r = .49, $p < .01$; Pain - r = .46, $p < .01$; and Unpleasantness - r = .32, $p < .01$), and the evaluative scale of the MPQ (r = .37, $p < .01$). Therefore, the higher the average reported pain intensity rating, the lower the threshold time and the longer the endurance time. In addition, the higher the average pain intensity rating, the higher the scores on the Gracely scales and the evaluative component of the MPQ. However, the average pain intensity rating was not significantly correlated with pain tolerance time or report of tolerance.

<u>The relationship between the measures of anxiety and the direct pain measures</u>

A preliminary assessment of the relationships between the preexperimental measures of anxiety and the direct measures of pain was accomplished by calculating Pearson product moment correlation coefficients from the data obtained from the subjects who received pressure pain stimulation. Correlation coefficients between the six preexperimental questionnires and the 13 direct pain measures are presented in Appendix L. The six preexperimental

questionnaires consisted of the TMAS, SDS, GTAR-EA, GTAR-PD, STAI-S, and present stress level.

In general, very few significant correlations were found. The higher the level of trait anxiety (TMAS), the higher the scores on the Gracely unpleasantness scale and the evaluative component of the MPQ (r = .37, $p < .01$ and r = .30, $p < .05$, respectively). Desire for social approval (SDS) was not significantly related to any of the direct pain measures. The higher the level of evaluation anxiety (GTAR-EA), the higher the score obtained from the Gracely unpleasantness scale (r = .38, $p < .01$) and the more likely the subject to report tolerance (r = -.29, $p < .05$). The fear of physical danger (GTAR-PD) was significantly correlated with the Gracely unpleasantness scale (r = .39, $p < .01$). State anxiety (STAI-S) was not significantly related to any direct pain measure. The rating of present stress level was significantly correlated with the evaluative scale of the MPQ (r = .39, $p < .01$), the miscellaneous scale of the MPQ (r = .35, $p < .01$), and the total sum of ranks of the words chosen on the MPQ (r = .34, $p < .01$).

Therefore, only one significant correlation was found between the anxiety questionnaires and the direct measures of pain that were obtained during nociception. That is, subjects who reported tolerance also reported higher evaluation anxiety than subjects who did not report tolerance. This result was also reported earlier when comparing subjects who did and did not report tolerance. These results suggest that preexperimental anxiety questionnaires are not useful predictors of the pain experience. However, alternative explanations are offered in the Discussion section.

Correlation coefficients were calculated to assess the relationship between the five indirect pain measures that were obtained during the Pain period and the 13 direct pain measures. The five indirect measures were the mean stress intensity rating, mean HR and EMG data, and the frequency of grimaces during the first and last minutes. A correlation matrix is presented in Appendix L.

Few significant correlation coefficients were found. Of major interest, the correlation between the average pain intensity rating and the average stress intensity rating was .53 ($p < .01$). Thus, 28% of the variance in the mean stress intensity rating obtained during the Pain period could be accounted for by the mean pain intensity rating during that period (and vice versa). This was the only indirect pain measure that was significantly correlated with any direct pain measure obtained during the Pain period. The average stress intensity rating was also significantly correlated with the three Gracely scales and four of the MPQ scales (affective, miscellaneous, evaluative, and total sum of ranks scales). The frequency of grimaces during the first minute of nociception was significantly correlated with the MPQ miscellaneous and evaluative scales. No other correlation coefficients were significant. These results indicate that measures of the affective aspects of the pain experience that do not include self-report of stress are poorly correlated with direct pain measures.

DISCUSSION

Two theories have been proposed to account for the influence of anxiety (a threat to physical or emotional well-being) on the experience of acute pain. The attribution theory (Weisenberg et al., 1984) states that relevant anxiety intensifies pain while irrelevant anxiety does not. In other words, if anxiety is associated with the nociceptive stimulus, the pain experience will be increased. Anxiety that is not associated with the nociceptive stimulus will not increase pain. The present author proposed a modified perceptual disruption hypothesis (Chapman, 1978; Mandler, 1984) that states that any type of anxiety disrupts the ability to process nociception information, and will have a measurable effect on the direct measures of the pain experience. In addition, attributing the anxiety to the nociceptive stimulus will increase the responses obtained from the indirect measures of pain.

To contrast these theories, three types of instructions were presented to subjects immediately before the application of the pressure stimulus. There was 1) a standard set of instructions, 2) a standard set of instructions plus a pain warning, and 3) a standard set of instructions plus a stress interview warning. If the attribution theory is correct, the subjects who received a pain warning should show increases in the direct and indirect pain measures relative to the other two groups. If the modified perceptual disruption theory is correct, both groups hearing warnings should react to the instructions. This effect could

manifest itself by either increased variability of the direct pain data obtained from the experimental groups (as reported by Unde et al., 1982; and Malow, 1981; using signal detection analyses of pain) or by an increase in the direct pain measures (as reported by Mayerson & Rhodewalt, 1984). In addition, if this theory is correct, the indirect measures of the pain experience will be influenced by a pain warning but these measures will not be influenced by a stress interview warning.

The results and their bearing on these theories are discussed in the following six sections: 1) the characteristics of the sample; 2) the methods employed; 3) the direct pain measures; 4) the indirect pain measures; 5) comparison between subjects who did and did not report tolerance; and 6) relationships among the measures. In the remainder of this Discussion section, a new theory is proposed to account for the relationship between anxiety and pain. The limitations of the study are considered, and implications for future research are discussed. Finally, clinical implications which may be drawn from the study are presented.

Major findings and issues

The sample. The subjects were young, healthy, male English speaking university students. As a group, they reported relatively low levels of trait and state anxiety when compared to norms collected from the general and college populations. It is reasonable that only students with relatively low levels of anxiety participated, because it was known in advance that the experiment would involve a stressful and a painful experience.

The sample reported relatively higher performance evaluation anxiety than published population norms. This result may be due

to the context in which the questionnaires were being completed. Subjects were told that their responses would be monitored continuously during the experiment. These instructions may have evoked an increase in performance anxiety. The scores on the questionnaires assessing fear of physically dangerous situations and the desire for social approval were similar to published college population norms. Consistent with Endler and Okada's (1975) research, subjects in the present study reported significantly higher anxiety associated with physically dangerous situations than with performance evaluation.

The analyses of the subjects' responses on the preexperimental questionnaires showed no significant differences among groups in age, ratio of height to weight, socioeconomic status (SES), season tested, first languaga, citizenship, religion, trait anxiety, importance of socially appropriate behavior, fear of situations involving physical danger or performance evaluation, or state anxiety. There was a significant difference among groups on the preexperimental rating of present stress intensity; the subjects who received the stress interview warning, but did not receive the nociceptive stimulus, reported the lowest level of stress. As noted earlier, this is reasonable since these subjects knew when they gave their stress rating that they would not be receiving pressure pain. There was a trend for a significant group difference in SES, the group receiving pressure pain and a stress interview warning reported the highest level. The groups receiving pressure pain had similar distributions of a combined social approval and trait anxiety score. The few subjects who reported talking to other participants of the study claimed that they received no additional information.

The present sample was relatively homogeneous. This simplifies interpreting the major findings because a number of demographic and personality factors influence the stress and pain experience. However, this relatively low between-subjects variability may limit generalizability of the research findings. This issue is explored in detail later.

The methods. Continuous monitoring of the time course and force exerted by the nociceptive stimulus showed that the average amount of pressure was slightly less than the 2000 g intended, but was equivalent across groups. The pressure increased, during the first minute of nociception, due to displacement of skin tissue.

The subjects described their experience as moderately to extremely painful. Qualitatively, the pressure was described by most subjects as "pressing", "pulsing", "hurting", "radiating", "numb", and "drawing". These descriptors are appropriate to the stimulus employed; a pressure wedge on the index finger. The stimulus was tolerated for a long period of time (relative to cold pressor or radiant heat nociception) and exerted a continuous pain. Zwetnow (1979) states that stimuli that create a dull aching continuous pain are the most relevant for extrapolation to clinical acute pain states. For these reasons, pressure nociception appears to be a useful analog of acute pain. However, approximately half of the subjects did not report tolerance during the ten minute exposure time and the distribution of the pain tolerance data was negatively skewed. The tolerance data will be discussed again in the following section.

It was concluded (see p. 85) that the pain warning and the stress interview warning each produced psychological stress.

However, the response parameters representing the stress were different between the two groups. The stress intensity ratings, heart rate (HR) data, and frontalis electromyographic activity (EMG) obtained during the period immediately following the instructions and immediately preceding the application of the nociceptive stimulus (the Anticipation period - AP) were analyzed. The pain warning produced significant increases in HR and EMG, but not stress intensity, compared to the control group. The stress interview warning produced significant increases in stress intensity and HR, but not EMG, during the AP. (The stress interview warning group was not statistically compared to the control group since Baseline instructions given to these two groups differed). These differences between responses to the warnings suggest that although the pain warning and the stress interview warning evoked responses that are similar to those that occur during a stressful experience, they may also have evoked different cognitive orientations toward the experimental situation.

Janis (1982) states that when a warning message evokes anxiety without providing the means to avert the threat, subjects ignore, minimize, or deny the presence of the threat. Self report measures would reflect this denial since there would be no evidence of an increase in self-report of stress while other less subjective response parameters would increase. Obrist (1981) states that under aversive conditions in which the subject perceives that no control is available to help him lessen the aversiveness, EMG and HR will increase. When some control is available, HR, but not EMG will increase.

In the present study, the pain warning may have evoked a cognitive orientation that no control was available to reduce

the aversiveness of the subsequent nociceptive experience. Terminating participation in the study (permitted by the instructions) may not have seemed a viable option. The stress interview warning may have evoked the appraisal that some control was available. Because performance would be judged, the subjects could attempt to perform in a manner that would obtain a positive evaluation. This proposed difference in cognitive orientation was not assessed during the present study and therefore, cannot be empirically evaluated. Further investigation of the cognitive nature of the threat that is produced by each of these warnings is warranted.

To conclude, the nociceptive stimulus produced a painful experience that varied in degree among subjects. Both of the experimental warnings produced stressful experiences that differed in reference to the response parameters associated with each.

Direct measures of pain. The direct measures of pain were the pain intensity ratings obtained after each minute of pressure nociception; the pain threshold, tolerance, and endurance data obtained once during nociception; and the Gracely and McGill Pain Questionnaire (MPQ) word sets completed immediately after the ten minute Recovery period. According to the attribution theory, subjects who received a pain warning, but not a stress interview warning, should show increased pain. According to the modified perceptual disruption theory, both experimental groups should show either an increased variability in these measures or increased pain.

In the present study, there were no significant differences among groups in the variability of these measures. Possible

reasons for this result are discussed at the end of this section.

The pain intensity ratings increased faster in the experimental (warning) groups than in the control group. The increase in ratings slowed down and leveled off over time in all groups. This type of pain intensity curve has been obtained using other continuous nociceptive stimuli such as radiant heat (in Melzack & Wall, 1982), and cold pressor and ischemic pain (Hilgard & Hilgard, 1975).

Using data from all subjects receiving pressure, the subjects receiving a pain warning reported higher pain intensities than the control subjects during the last six minutes of pressure stimulation. Subjects receiving a stress interview warning reported higher pain intensities than control subjects for all but the second minute of the ten minute period. Using data from subjects who did not report tolerance (n = 23), both experimental groups reported higher average pain intensity ratings for the entire Pain period than the control group. However, when comparing the mean individual pain intensity rating obtained from each subject during the Pain period, only the subjects who received a stress interview warning had a higher pain rating than the control group. There were no significant differences among groups during the Recovery period.

Therefore, the stress interview warning produced increased pain intensity ratings whether or not subjects in this group reported tolerance. This result does not support the attribution theory, which claims that only relevant anxiety will influence the pain experience. However, the data obtained from the subjects receiving a pain warning are inconsistent. One reason for this inconsistency could be that subjects in this group who did not

report tolerance experienced a higher pain intensity than the subjects who reported tolerance. This possibility is supported by the analysis of the average pain intensity ratings obtained from the subjects who did not report tolerance. For these subjects, both experimental groups reported higher pain intensity ratings than the control group. When using data from all subjects, there was no significant difference between the pain warning subjects and the control group on this measure. Subjects who received a pain warning and reported tolerance may have dropped out before reaching an intensity level similar to the subjects who did not report tolerance. The instructions stated to monitor the stimulus carefully and to report tolerance as soon as the pressure was too painful. Subjects who ignored this demand would subsequently experience greater intensities due to the anxiety evoking effects of the instructions. This possibility should be explored in further empirical investigations.

There were no significant differences among groups in the pain threshold, tolerance, or endurance data. Although subjects who received a pain warning reported tolerance an average of one minute earlier than control subjects, this difference was not significant. The variability of this measure was very high. A ceiling effect may also have been operating since 48% of the sample did not report tolerance during the Pain period. In order to more carefully examine the effects that anxiety has upon pain tolerance, a stimulus that most subjects find intolerable (before tissue damage ensues) would be appropriate. Electric shock stimulation or muscle ischemic pain, although they have associated methodological problems, may be more appropriate to determine the relationship between anxiety and

pain tolerance.

The rate at which subjects dropped out (reported tolerance) was different across the three groups. A larger proportion of the stress warning subjects completed the full ten minute Pain period than the pain warning subjects. The difference was most pronounced at the end of the Pain period. There is thus some evidence that the pain warning affected tolerance. This possibility requires further empirical investigation, employing nociceptive stimuli that are less tolerable than the pressure pain used in this study.

There were no significant group differences found on the analyses of the Gracely or MPQ scales. This finding is unusual since a number of authors have reported significant correlations between pain intensity ratings and MPQ data when both were obtained during a pain experience. Melzack (1975) reported a correlation of .42 between an overall pain intensity rating and the sum of the rank values on the MPQ. Walsh and Leber (1983) reported a correlation of .57 between pain intensity ratings and the MPQ obtained from chronic pain patients. In the present study, the average pain intensity rating was not significantly correlated with the sum of the rank values on the MPQ. Nor was it significantly correlated with the sum of the rank values of the sensory or affective scales as reported by Melzack (1975) and Walsh and Leber (1983).

These findings suggest that subjects rate their pain experience differently during a pain episode than during a pain-free period. There is little available research to evaluate the extent of this difference. Pakula and Milvidarte (1983) studied memory for cardiac pain two weeks after the pain episode

and reported a decrease in remembered pain intensity when comparing this rating to one obtained immediately after the pain experience. Kent (1985), in a study of dental anxiety, reported that there was a closer association between remembered and expected pain on the MPQ than between remembered and experienced pain. And although Hunter, Phillips, and Rachman (1979) reported a high correlation between actual and remembered headache pain on the MPQ, Kent (1985) points out that this may be due to remembering the words chosen rather than the actual pain experience. Therefore, caution should be exercised in interpreting pain reports obtained during pain-free periods until more data is available to evaluate this type of report.

In conclusion, a number of direct pain measures were employed during the present study. The instructions did not appear to affect the pain threshold, tolerance or endurance data collected during the Pain period or the Gracely and MPQ scales collected after the experiment. However, fewer subjects who received a pain warning completed the Pain period than subjects who received a stress interview warning. The stress interview warning increased pain intensity ratings throughout the Pain period. The pain warning increased these ratings only in subjects who did not report tolerance. After receiving a pain warning, reporting tolerance may serve a protective function against experiencing a higher pain intensity. Further investigation of this possibility is warranted.

These results do not support the attribution theory that relevant but not irrelevant anxiety exacerbates the pain experience. There was some evidence that the pain warning facilitated the report of tolerance compared to the stress

interview warning. As noted, investigations using more painful stimuli are needed to determine if this is the case. The perceptual disruption theory seems to be more useful to explain the data. Both relevant and irrelevant anxiety influenced the self-report of pain intensity.

Anxiety did not influence the variability of the direct pain measures. This may be due to the relatively long period of time between measurement intervals. A signal detection (SDT) approach may be more appropriate to determine if these effects on pain intensity are due to increased difficulty in judging the stimulus as opposed to an alteration in the criteria used for this judgment. Studies employing SDT methodologies to investigate the relationship between anxiety and pain have shown that the ability to discriminate between stimulus intensities is impaired by anxiety while response bias remains unchanged.

Indirect pain measures. The indirect measures of pain employed in the present study included the stress intensity ratings, the HR and EMG activity, and the behavioral response data. Other indirect measures were included in the postexperimental questionnaire package. They assessed the representativeness of the experience, the belief that the stimulus would be painful, the rating of the level of stress remembered before the application of pressure pain, and the rating of stress present during the completion of the questionnaires. They also included the State-Trait Anxiety Inventory - State measures (STAI-S), and the presence, type, and effectiveness of coping strategies.

The modified perceptual disruption theory incorporates the

attribution theory in that it states that relevant but not irrelevant anxiety increases the aversive quality and motivational drive associated with the pain experience. If the modified perceptual disruption theory is correct, the subjects who received a pain warning, but not the subjects who received a stress interview warning, should show increased responding on the indirect measures of pain. The results obtained from all the indirect measures will be reviewed before discussing the appropriateness of this modified theory.

All the analyses of the stress intensity rating data found that both experimental groups reported higher stress than the control group during the Pain period. Generally, this increase was sustained throughout this period. These results are interesting; although the subjects receiving a pain warning reported higher levels of stress throughout the Pain period, the average pain intensity rating obtained during this period was not significantly different from the control group. Subjects who received a pain warning rated their experience as more stressful, but only the pain warning subjects who did not report tolerance also rated this experience as more painful.

For the entire sample, the correlation between the average pain and stress intensity rating was .53. This supports the claims made by Melzack and Wall (1982) and others (e.g., Merskey, 1980; Weisenberg, 1977) that pain and anxiety increase together. However, this correlation accounts for about 28% of the variance, indicating that subjects did not choose a stress rating identical to the selected pain rating. Subjects determined the stress intensity rating by different, perhaps overlapping, criteria.

During the initial minutes of the Recovery period, subjects

in both experimental groups reported higher stress intensity than did the control group. This could have happened for several reasons. Since the stress intensity ratings were higher during the Pain period, the experimental subjects may have needed more time before returning to the Baseline level. In addition, subjects receiving a stress interview warning were told to expect that the interview would begin soon. As reported earlier, these instructions produced increases in stress intensity ratings even without the presentation of the pressure nociception.

Both sets of instructions produced increases in HR during the Anticipation period (AP). In all analyses of HR data, the subjects who received a pain warning showed higher HR during the Pain period than did the control subjects. In addition, subjects from both experimental groups who did not report tolerance showed a decrease over time in HR indicating increased familiarity with the stimulus (Obrist, 1981). There were no significant differences among groups during the Recovery period.

The analyses of the HR data obtained during the Pain period from the stress interview warning subjects were not as conclusive. The average HR for the entire Pain period and the HR data obtained from subjects who did not report tolerance were not significantly different from the control group. However, a significant difference was found when using group means to fill missing data cells. As noted in the Results, these findings appear to be due to the decrease in HR over the Pain period following an initial increase in HR during the AP.

Subjects who received a pain warning but not subjects who received a stress warning showed higher EMG during the AP than the control subjects. They also showed higher EMG than the

stress interview warning group throughout the Recovery period. Subjects in the pain warning group but not the stress warning group showed higher EMG than the control group during the third to fifth minutes of nociception. The average EMG during the Pain period was higher in the pain warning subjects than either other group, but this difference was not significant. The large variability of this measure may have obscured differences among groups. In short, the pain warning produced increased EMG during the Anticipation and Recovery periods and also appeared to increase EMG during the first half of the Pain period.

There were no differences among groups in the overall frequency of movements during the first or last minutes of nociception. However, the frequency of grimaces during the first minute was significantly greater in the group receiving a pain warning than in the control group. The group receiving a stress interview warning did not differ significantly from either of the other two groups. There were no significant differences among groups during the last minute of nociception.

No significant differences among groups were found by the analyses of the postexperimental questionnaire data that included the remaining indirect pain measures. This finding lends further support to the suggestion that postexperimental questionnaires are not as sensitive to group differences as the measures obtained during the experimental periods.

In conclusion, the experimental instructions evoked increases in stress intensity ratings during nociception and the initial minutes of the Recovery period when compared to the control instructions. The instructions evoked increases in HR in both experimental groups yet HR remained elevated only in subjects

who received a pain warning. In general, EMG was elevated during the Anticipation, Pain, and Recovery periods only in the subjects receiving a pain warning. There was a higher frequency of grimaces during the first minute of nociception in subjects receiving a pain warning compared to control subjects. The postexperimental questionnaires did not reflect these differences among the groups.

The combined results of all the indirect pain measures indicate that the subjects receiving a pain warning experienced the nociceptive stimulus as more aversive than subjects receiving a stress interview warning or no explicit warning. Although both experimental groups reported higher levels of stress during the Pain period, subjects receiving a pain warning also showed increased physiological arousal during the experiment and a higher frequency of grimaces during the first minute of nociception than the control subjects. These results support the modified perceptual disruption theory that the overall aversiveness of a nociceptive stimulus is higher when associated with relevant anxiety than when associated with irrelevant anxiety. Before discussing the implications of these findings, two other facets of the research data will be explored; the characteristics of the subjects who did and did not report tolerance and the relationships among the dependent measures.

Comparison of subjects who did and did not report tolerance. Subjects who reported tolerance during the Pain period were compared to subjects who did not in order to explore other related differences between these two groups. The pain threshold and endurance times were significantly shorter in subjects who reported tolerance compared to subjects who did

not. However, no significant differences were found on any other direct or indirect measure obtained during the experimental periods. That is, these two groups did not differ on the average reported levels of pain and stress intensity obtained during nociception or in the average HR, EMG, or frequency of grimace behavior. Baseline levels or HR and EMG were not significantly different between the two groups. There were very few differences between the two groups on the postexperimental questionnaire data; subjects who did not report tolerance reported a lower painfulness rating on the Gracely scale that assessed this factor and these subjects rated the strategies they employed as more effective than subjects who reported pain tolerance.

These two groups did not differ significantly on preexperimental questionnaires that assessed the desire for social approval, state and trait anxiety, or fear of physically dangerous situations. However, subjects who did not report tolerance reported lower performance anxiety than subjects who did. Therefore with the exception of reporting higher performance anxiety, these two groups did not differ on any measure of psychological stress or anxiety obtained in this study. Yet the two groups differed on the responses obtained from two measures of cultural factors. That is, subjects who reported tolerance also reported significantly lower SES scores than subjects who did not report tolerance and they were more likely to report affiliation with the Jewish faith. Subjects who did not report tolerance were more likely to report no religious affiliation.

These findings are unusual since most of the literature

reviewed in the Introduction indicated that tolerance was reduced by the presence of anxiety measured by questionnaires or induced by an experimental manipulation. In addition, Weisenberg (1975) stated that attitude and anxiety are the major sources of cultural differences in pain tolerance. The absence of this relationship in the present study may be due to several factors. With reference to the preexperimental questionnaires, the sample employed in this study could be considered as low anxious. Thus, tolerance may only be related to anxiety questionnaire data when the report of anxiety is high. Second, tolerance may have no relationship to pain intensity, stress intensity, or phasic and tonic differences in physiological arousal. In general, the relationships between these measures are low (Craig, 1984). Third, cultural and social factors such as SES, religious affiliation, fear of being negatively evaluated, and other related variables may have more influence on pain tolerance than the experience of anxiety. It is not possible to determine from this study which of these alternatives (or some combination of all three) is most reasonable. Future research investigations may want to explore the relationship between these factors and pain tolerance.

Relationships among the dependent measures. In general, most of the correlations between dependent measures were not significant. Dependent measures that appear to share common characteristics were more likely to be significantly correlated than measures that did not. For instance, the scales of the MPQ were significantly intercorrelated as were the scales from the Gracely word descriptors. Pain intensity and stress intensity were significantly correlated. Threshold and tolerance were

significantly correlated.

Threshold was significantly negatively correlated with pain intensity indicating that the longer the threshold time, the lower the average pain intensity rating. Threshold was significantly correlated with the Gracely intensity and painfulness scales. However, pain tolerance was not related to either pain intensity or the Gracely scales. The average pain intensity rating was significantly correlated with one MPQ scale (the evaluative word set) and significantly correlated with each Gracely scale.

With reference to the indirect measures, the average stress intensity was not related to any other indirect pain measure or to pain threshold or tolerance. Average stress intensity was significantly correlated with the three Gracely scales and all the MPQ scales (excluding the sensory scale). Neither HR or EMG were significantly correlated with any of the dependent measures.

These findings underscore previous reports that measures of the pain experience are loosely associated and that no one measure can capture the entire process (Chapman et al., 1985; Craig, 1984).

The relationship between anxiety and pain

The present study employed numerous measures to assess different aspects of the anxiety and pain experiences. The findings indicate that not all the measures of "anxiety" were related to the pain experience. Furthermore, when "anxiety" influenced this experience, it affected some but not all of the pain measures.

The self-report questionnaires assessing state, trait,

and situational anxiety were not significantly correlated with any dependent measure obtained during nociception. As noted previously, a reasonable explanation of this finding is that only when the scores on these questionnaires are high will they be related to the pain experience. If the scores reflect low to moderate levels of anxiety, they will not be predictive of pain responses. The validity of this proposal could be easily assessed by research projects in which subjects were selected on the basis of questionnaire scores and exposed to a nociceptive stimulus. Then the responses of low and high anxious subjects to this stimulus could be compared.

In this study, experimentally induced anxiety produced a number of effects upon the pain measures. Both relevant and irrelevant anxiety-evoking instructions produced higher pain and stress intensity ratings, compared to instructions with no explicit warning. This effect was particularly strong in subjects given the irrelevant anxiety instructions. These data indicate that the relationship between anxiety and pain is more complex than the attribution theory proposed by Weisenberg et al. (1984), that relevant but not irrelevant anxiety influences the pain experience. There was some evidence that relevant but not irrelevant anxiety facilitated the report of tolerance. In addition, the relevant anxiety condition produced significant increases in HR, EMG, and grimace frequency during nociception, and the increased level of EMG was sustained after nociception, compared to the other groups.

A theory that combines the perceptual disruption hypothesis with the attribution theory appears to be the most appropriate. The experience of anxiety influences the subjects' experience of

the intensity of the stimulus. In this study, this effect was manifested by increased pain and stress intensity ratings reported by both experimental groups. If the anxiety is related to the nociceptive stimulus, then a number of other elements related to the distress associated with the pain experience will also increase. In this study, relevant anxiety evoked higher physiological arousal and more facial expressions indicative of distress. In addition, the decision to terminate the nociceptive stimulus once it became intolerable may have been influenced by the experience of relevant anxiety.

This theory of the relationship between anxiety and pain is consistent with Melzack and Wall's (1982) comprehensive theory of the mechanisms subserving the pain experience. Pain contains a sensory-discriminative component (how the stimulus feels), a motivational-affective component (the motivational drive and negative affect associated with the stimulus), and a cognitive evaluative component (the overall evaluation of the painfulness of the stimulus). These three components interact with each other. Cognitive-evaluative processes can exert control over the activity in both the discriminative and motivational systems.

In the modified perceptual disruption hypothesis (which incorporates the attribution theory), the individual's cognitive evaluative processes become less accurate in determining the intensity of the nociceptive stimulus when anxiety is experienced. The individual's experience of anxiety competes for focal attention with the nociceptive experience, which makes it difficult for the individual to accurately evaluate the properties of either experience. Cognitive-evaluative processes are also

used to relate the relevance of the anxiety to the co-occurring pain experience. The experience of pain creates its own relevant anxiety that is mediated by the interplay of cognitive and motivational components. Furthermore, the cognitive evaluation that there are additional sources of relevant anxiety increases the motivational and aversive aspects of the pain experience.

Chapman (1978) and Melzack and Wall (1982) state that anxiety is not a reaction to the pain experience, but is a part of that experience. If a nociceptive stimulus disrupts cognitive processes, the disruption will create anxiety (Chapman, 1978; Mandler, 1984). This disruption and associated anxiety will evoke attempts by the individual to terminate the disruptive event. If additional sources of anxiety are present during nociception, additional disruption of cognitive processes will occur. The individual's ability to evaluate information from sensory-discriminative channels will become increasingly impaired as the disruption to cognitive processes increases. Attempts to terminate the disruptive event would include an evaluation of the relevance of the anxiety experience with reference to the nociceptive situation. The evaluation that the associated experience of anxiety is or is not meaningful will further influence the motivational and affective processes associated with the pain experience.

Limitations of the study

Generalizability of the findings. The sample was composed of young, low-anxious male university students. Considerable research data indicates that women report pain tolerance earlier than men, but that pain threshold does not differ between these two groups (Leventhal & Everhart, 1979; Weisenberg, 1977). Since

threshold does not differ between men and women, the difference in pain tolerance has been attributed to a greater willingness on behalf of women to report illness and other stressful experiences in general (Leventhal & Everhart, 1979). It is important to note that Scott and Barber (1977) found no significant difference in pain tolerance between men and women using either pressure or cold pressor pain. In addition, a number of researchers have reported no sex differences in the report of pain intensity, degree of discomfort, or MPQ scores (Jacox, 1980; Taenzer, Melzack, & Jeans, 1986; Volicer, 1978a). Therefore, it is reasonable that the findings from the present study pertaining to pain threshold, pain and stress intensity, and MPQ data would have been similar if women had been subjects.

The research data relating to the differences in the pain responses of older and younger individuals are inconsistent. Sternbach (1968) concluded that pain threshold and tolerance tend to decrease with age. However, Weisenberg's (1977) review reports research findings that suggest the exact opposite; both pain threshold and tolerance tend to increase with age. Chapman's (1985) review reports that age is not related to the reported severity of pain. Taenzer et al. (1986) reported that age was not related to MPQ data. However, Volicer (1978a) reported that younger patients tended to rate their pain higher than older patients. Because of this inconsistency concerning variations in the pain experience over the life span, caution should be exercised when extrapolating the present findings to older populations.

As discussed previously, the individual's predisposition to anxiety may influence the severity of the pain experience

as well as the reports of pain threshold and tolerance. Although it is reasonable to propose that employing high-anxious subjects would have influenced the results obtained, it is not possible to say which measures may have been most affected.

Level of educational attainment has also been implicated as a factor influencing the pain experience. Individuals with less education tend to have lower threshold and tolerance times than individuals with more education (Sternbach, 1968). In addition, Taenzer et al. (1986) reported that education was negatively correlated with MPQ data and with pain intensity. That is, higher educational attainment was correlated with lower scores on both the MPQ and pain intensity scales. Thus, employing subjects with less education may have increased the pain experience.

Due to the lack of conclusive research data relating to the pain experience as it changes over the life span, as it differs between low and high anxious individuals, and how it differs between different levels of educational attainment, caution should be employed when extrapolating the present findings to populations different in these aspects. However, it is reasonable to assume that with the exception of pain tolerance, the data obtained from this study could be extended to similar female populations.

Generalizability of the findings to other nociceptive stimuli. There is not enough data to evaluate the equivalence of the pain experiences produced by different nociceptive stimuli. However, the results obtained in the present study are consistent with those obtained in other research assessing the relationship between anxiety and cold pressor, radiant heat, and electric

shock stimulation pain. For instance, threats to physical integrity have been reported to increase pain intensity ratings, magnitude estimates of the strength of the stimulus, and to decrease threshold and tolerance (Hall & Stride, 1954, in Melzack & Wall, 1982; Haslam, 1966; Nisbett & Schachter, 1966; Sternbach, 1968). Threats to psychological integrity have been reported to increase pain intensity ratings during cold pressor nociception (Mayerson & Rhodewalt, 1984) and to increase anxiety ratings during electric shock (Weisenberg et al., 1984). In addition, the pain intensity curves obtained in this study are similar to those obtained for radiant heat nociception (Melzack & Wall, 1982) and for cold pressor and ischemic pain (Hilgard & Hilgard, 1975).

Pressure stimulation was chosen for this study since Zwetnow (1979) and other researchers (e.g., Merskey, 1973) state that methods that produce continuously building, aching pain have the closest resemblance to the types of pain found in clinical settings. The results obtained in the present study are consistent with Volicer's (1978a) clinical study in which life stress in general was related to increased reports of pain and discomfort. Volicer and Volicer (1978) reported greater physiological changes during hospitalization in patients reporting high life stress than in patients reporting low levels of life stress. Further investigation of the role of relevant and irrelevant anxiety in clinical settings is possible using procedures that assess general and specific levels of anxiety and then relate these measures to the subsequent pain experience.

Because the stimulus employed in the present study was not sufficiently intolerable, it was not possible to determine

the exact relationship between anxiety and pain tolerance. The data suggested that relevant but not irrelevant anxiety decreased pain tolerance. However, this finding was not borne out by examination of the pain tolerance data with reference to the time spent undergoing nociception. Further research is required on the effects of relevant and irrelevant anxiety upon cold pressor or ischemic pain which are eventually experienced as intolerable by most subjects.

Cognitive orientation towards the nociceptive stimulus. The experimental instructions produced effects similar to those found in other stressful situations. However, the nature of the stress response differed between groups. It was suggested that these instructions may have produced differences in cognitive orientation toward the nociceptive experience. The pain warning may have evoked the belief that no control was available to lessen the aversiveness of the event whereas some control was available to subjects receiving a stress interview warning. Although the experimental instructions were designed to evoke two types of anxiety (physical danger and performance anxiety), the present study did not determine what the subjects believed they could do to control the aversiveness of the event. Future investigations exploring the relationship between anxiety and pain could incorporate a means of determining this belief. For instance, the two types of anxiety instructions could be compared by asking questions about the subject's beliefs about the controllability of the upcoming stimulus. Plutchik (1977), however, states that subjects may not be consciously aware of the appraisals they make and thus, may be unable to discuss them. Although it may not be possible to discuss the subjects' beliefs

about the stress experience, they can be inferred from other measures. This is because the instructions affect direct and indirect pain measures in different ways. The nature of these differences, caused by cognitive appraisal, is an issue that warrants future research.

Methodological implications

The distinction between direct and indirect measures. In the past, research designed to determine the relationship between anxiety and pain has failed to operationally distinguish measures that directly assess the pain experience from measures that assess the experience of anxiety (Gross & Collins, 1981). One reason for this may be the strong theoretical influence that Melzack and Wall (1982) and others (Bonica, 1977; Sternbach, 1968) have had upon the research assessing the psychological mechanisms involved with pain perception. According to their views, pain is an experience composed of interacting sensory, perceptual, and affective processes. Pain cannot be said to exist without its aversive emotional quality. The distinction made between pain and its negative affective quality by Cassem (1983) and others who view pain processes from a medical perspective (e.g., Guyton, 1981; Villaverde & MacMillan, 1977) has impeded the development of interdisciplinary approaches aimed at understanding both the psychology and physiology of the pain experience. Nevertheless, when empirical investigations are designed to determine the influence of affective processes upon the interaction of sensory, perceptual, and affective components of pain, it is essential to make a distinction between the affective processes and the sensory experience.

Empirical measures available at this time that attempt to

evaluate each component separately include the MPQ and the Gracely scales. In the present study, these scales were completed during a pain-free period because it was not possible for subjects to complete them during nociception. Therefore, in this study, a distinction was made between the indirect measures and the direct measures of the pain experience. Indirect measures were operationally defined as the measures traditionally used by researchers to assess the anxiety and stress experiences. These measures include reports of anxiety and distress, facial expressions, and physiological indices of arousal. Direct measures were defined as the measures specifically designed to assess the individual's perception and evaluation of the nociceptive stimulus. These measures include pain intensity ratings, threshold, tolerance, and pain questionnaires as well as SDT methodologies.

Due to the methodological confounding of the assessment of pain and anxiety, the distinction between direct and indirect measures is crucial in research projects designed to determine the influence of anxiety upon the pain experience. Manipulations that create changes in physiological indices and distress ratings are not equivalent to manipulations that create changes in pain intensity or MPQ data. Although the reduction of suffering is an essential factor in pain relief (Cassel, 1982), it is theoretically important to determine if the experience of anxiety influences the sensory and perceptual components of pain as well as contributing to the affective processes associated with pain.

In the present study, the division of response measures into direct and indirect allowed an empirical investigation of the influence that relevant and irrelevant anxiety had upon different

aspects of pain. The major findings of this study indicated that experimentally induced anxiety not only influenced the distress associated with nociception, it also influenced the intensity of the pain produced by the stimulus. Furthermore, the two sets of anxiety-evoking instructions produced different effects upon the direct and indirect measures of the pain experience. Therefore, the distinction between direct and indirect measures enhances understanding of the relationship between anxiety and pain.

Assessment of the production of anxiety. Until recently, investigators manipulating anxiety to determine the relationship between anxiety and pain have failed to document whether anxiety was actually produced by the experimental manipulation (e.g., Hall & Stride, 1954; cited in Melzack & Wall, 1982; Nisbett & Schachter, 1966; Mayerson & Rhodewalt, 1984; Sternbach, 1968). In one study, the measures used to determine if the anxiety manipulation was effective were obtained after nociception had been started (Weisenberg et al., 1984). Evidence that the anxiety manipulation actually produced anxiety prior to the application of nociception is necessary before it can be concluded that the results obtained during the experiment were due to the effects of anxiety and not to another psychological or social factor influencing both the anxiety and pain measures (Haslam, 1966, Malow, 1981).

In the present study, stress intensity ratings, HR, and EMG data were obtained during a waiting period to validate that the instructions evoked a stressful experience. The effects of the instructions upon these measures were similar to the effects of anxiety and stress. Each set of instructions appeared to evoke a different cognitive orientation toward the nociceptive

event.

Multimodal assessment of the pain experience. Many research projects assessing the relationship between anxiety and pain have not employed a variety of pain measures. Most research has employed either pain threshold or tolerance as the single pain pain measure (eg., Haslam, 1966; Malow, 1981; Nisbett & Schachter, 1966). However, neither of these measures reflects the pain experience in its entirety (Chapman et al., 1985). The present study employed a number of direct pain measures and these measures were influenced in different ways by the experimental manipulation. This underscores previous reports of the importance of employing multimodal assessment.

Utility of postexperimental questionnaires. The poatexperimental questionnaire data indicated that the experience of pressure nociception was painful and moderately stressful. However, these questionnaire data did not produce the significant differences among groups that were obtained by the direct and indirect measures during the nociceptive period. In addition, the postexperimental data did not produce differences between the two groups that did and did not report tolerance with the exception of two subscales (Gracely painfulness scale and the effectiveness of coping strategies). Few studies have been conducted to evaluate to reliability of questionnaires completed during a pain-free period. Klepac et al. (1981) reported that, since subjects endorsed higher ranked words on the MPQ after tolerance than after threshold, the MPQ could be used in laboratory research. The results from this study do not support this conclusion. It is possible that large changes in perceived magnitude of nociceptive stimuli (e.g., from threshold to

tolerance) are measured by postexperimental questionnaires yet other changes in the pain experience are not. The validity of questionnaire data during a pain-free period must continue to be explored. Future research should assess the pain experience during and after nociception and then determine whether or not the memory for the experience is different from the report of this experience during nociception.

Theoretical and clinical implication of the research findings

The importance of psychological mechanisms. The recognition that pain is a complex perceptual experience has created the need for clarification of the mechanisms through which psychological and physiological processes codetermine pain (Schneider & Karoly, 1983). Melzack and Wall (1982) have proposed a neurophysiological model describing the mechanisms responsible for the influence of anxiety on pain processes. However, this model explains how these effects can occur at a physiological level and does not predict when anxiety will create these effects. Knowledge of anxiety level per se is not adequate to predict the reaction to nociceptive input (Weisenberg et al., 1984). In order to make predictions concerning the influence of anxiety on pain, reference must be made to psychological and social constructs (Degenaar, 1979). Two psychological theories that have been proposed to explain this relationship were compared in this study.

The research findings from the present study indicate that individuals who experience increased levels of anxiety immediately preceding nociception also report higher pain and stress intensity ratings during nociception. When the anxiety stems from a relevant source, (a threat to physical or emotional

well-being that is related to the injury), the arousal and facial indicants of distress associated with pain also increase. If the source of the anxiety is not relevant to the nociceptive stimulus, these measures do not increase. Pain threshold was not influenced by experimentally induced anxiety. It is not clear how pain tolerance was affected by experimentally induced anxiety. These findings support a modified perceptual disruption theory in which anxiety in general influences the evaluation of nociceptive input, and the type of anxiety influences the motivational-affective processes associated with the pain experience.

The results are also consistent with Melzack and Wall's (1982) proposal that altering one component of the pain process (sensory-discriminative, motivational-affective, or cognitive evaluative) affects the other components of the triad. In this study, creating an emotional experience in addition to applying a nociceptive stimulus altered the evaluation of the intensity of the stimulus, and the type of anxiety created influenced the motivational-affective component associated with this experience. Further research is necessary to determine if the influence of anxiety upon pain intensity affected sensory discriminative processes as well as cognitive-evaluative ones. Empirical investigations that incorporate SDT analyses into the design may be able to resolve this issue.

Clinical implications. These data confirm Volicer's (1978a) hospital setting findings that both general life stress (irrelevant anxiety) and stress related to medical and surgical (relevant anxiety) influenced the pain experience.

Because relevant anxiety increases the affective dimensions

associated with pain, these findings suggest that relevant anxiety may influence the decision by individual's to seek aid from professionals following physical trauma. Crook et al.'s (1984) epidemiological study found that 66% of the subjects reporting pain had not sought aid for its relief. Many of these respondents who did not seek aid explained that the problem was not serious enough to require professional help. Although these authors did not assess the possibility that relevant anxiety determines the 'seriousness' of the pain, it is consistent with Cassel's (1982) report that the experience of a threat to physical integrity (relevant anxiety) increases the drive to obtain relief and to search for help.

Melzack, Wall, and Ty (1982) reported that 37% of patients presenting themselves for aid at an emergency clinic with various types of tissue damage had a pain-free period following the injury. Although they did not ask why patients came to the clinic if they did not feel pain, the reason may relate again to anxiety over physical integrity. Individuals may go to a clinic following injury not necessarily due to the severity of the pain they experience, but because of their belief that the injury warrants medical attention and treatment in order to restore physical integrity.

Finally, the results of the present study suggest that psychological treatments designed to reduce general as well as relevant anxiety will be more effective in producing pain relief than treatments aimed to reduce relevant anxiety alone. Weisenberg (1984) reviewed the literature comparing various psychological treatments for pain. In one laboratory study, a full stress inoculation procedure (education and coping skills)

was more effective at producing increases in threshold and tolerance to cold pressor nociception than either component alone. In a second laboratory study, coping skills were more effective than cognitive skills (imagery and distraction) in increasing tolerance.

Two clinical studies also support the efficacy of psychological techniques that combine both types of anxiety reduction procedures. Melzack and Perry (1975) reported that hypnosis (including progressive relaxation) plus alpha training (a type of distraction) was more effective than hypnosis alone in relieving chronic pain. Mitchell and White (1977) reported that progressive relaxation training followed by behavioral self-management skills acquisition was more effective than progressive relaxation alone in reducing headache frequency. Progressive relaxation alone was more effective than the self management skills alone.

The reason why coping skills packages that include both a general relaxation procedure plus specific pain reduction techniques are more effective than either component alone may relate to the effect of these packages upon general as well as specific types of anxiety in the pain experience.

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APPENDIX A

Study Materials

EXPERIMENT INFORMATION FORM

EXPERIMENT Bodily and Cognitive Responses to Stress

EXPERIMENTER'S NAME Anne Cornwall

OFFICE NUMBER N7/17 PHONE # 392-5894

RESEARCH SPONSOR Dr. Don C. Donderi

BRIEF DESCRIPTION OF RESEARCH

Physiological monitoring of your responses to a briefly presented stressor that involves either a pressure pain at the index finger or participation in a psychologically stressful interview situation or both. We will also ask you to fill out some questionnaires related to this experience.

COURSES RELATED TO: Perspectives on Human Nature; Physiological Psychology; Behavioral Medicine

SPECIAL SUBJECT REQUIREMENTS Males only. Individuals who did not participate in the experiment 'Thinking and Pressure Pain'. English as a first language.

SCHEDULING INFORMATION:

NUMBER AND LENGTH OF SESSIONS 1 session; one hour in length

TIMES AVAILABLE Morning and afternoon

LOCATION Clinical Psychophysiology Laboratory, Stewart Biological Sciences Building, N7/26

CONTACT Anne Cornwall in N7/17 or at 392-5894 or leave name and numberbelow.

OTHER INFORMATION Each subject is paid $10 for participation.

PARTICIPANT'S MANUAL

This manual has been designed to provide you with a better understanding of the nature and methodology of my research. The purpose of this study will explained, as will the various testing and measurement procedures. It is important that you understand the study and feel comfortable with the procedure, so do not hesitate to ask questions or to have something clarified.

Anne Cornwall
392-5894

INTRODUCTION

The standard of past research on stress has been to assess subjective and physiological responses to stress with little regard to how the subject's thoughts are affecting these responses. In addition, little regard has been given to the type of stressor employed by the experimenter. In attempting a more realistic assessment of human behavior and emotion, this study is concerned with the effect that thinking about certain issues has upon the experience of two different types of stressors. Thus, it is important for you to realize that this experiment involves either the application of a painful stimulus that will terminate when you can no longer tolerate it or your involvement in a psychologically anxiety producing situation designed to produce stress or your participation in both these conditions. Both of these stimuli have been shown to produce anxiety and associated effects on the circulatory and muscle systems. In addition, your bodily responses to this stimulus will be recorded and you will be asked to monitor your emotional state and to complete a number of questionnaires. This is necessary to determine how what you are thinking is affecting the discomfort you experience.

In order to entice you to participate, you will be paid $10 at the end of the experiment. Although you are free to terminate your participation at any time, you will be paid only if you complete the entire experiment (which should last about one hour). Other gains to be expected from participating are 1) obtaining information concerning your bodily reactions to stress, and 2) contributing information that will advance the scientific understanding of stress and its control.

An outline of the project is discussed in greater detail below.

PROJECT OUTLINE

1. All participants will be asked to abstain from consuming any coffee, drugs, alcohol, exercise, heavy food and cigarettes for a 2 hour period prior to the experimental session. This is necessary to ensure that all participants are in a similar physiological state prior to the experiment.

2. When you arrive at the lab, you will be asked to complete a series of questionnaires which will take about 15 minutes to finish. You will be asked to answer questions pertaining to your background and your attitudes and beliefs about various issues. These questionnaires address themselves to individual differences that may be responsible for the relationship between your thoughts and your experience of stress. All your responses are strictly confidential. You can refuse to answer a particular question. However, your candidness is greatly appreciated since this information is very important.

3. You will be asked to sit in a comfortable chair while one set of electrodes is attached to your chest (to monitor your heart rate) and a second set is attached to your forehead muscles (to monitor muscle contractions). There is no discomfort to this procedure. You will be asked to sit quietly throughout this part of the experiment since any movement will disturb the recordings being made. A video recorder will be used to monitor your movements so that if you should move, we can correct for it on the polygraph record.

4. After the electrodes have been attached, a board will be placed over your lap which houses a rating box and stress producing apparatus. You will be asked to use this rating box to record your level of stress and of pain throughtout the experiment. You will be randomly assigned to one of 3 stress inducing conditions: 1) Pressure pain; this involves the application of a pressure wedge to the index finger. This will produce an uncomfortable sensation at first that will increase over time. Once you report that you can no longer tolerate this pressure, the wedge will be released immediately, 2) Stress interview; this task involves your participation in a variety of cognitive tasks that previous research has shown to produce discomfort and anxiety in individuals undergoing the tasks, or 3) both stressors. You will not be assigned to any of these groups until you reach the lab. After the stress condition, you will be asked to remain seated until your bodily and psychological reactions return to normal.

5. The electrodes will be removed and you will be asked to complete another set of questionnaires. Following this, any question you have will be answered and you will be paid $10 for your participation. I will again state at this time that your response are kept confidential. The laboratory part of the experiment should take about 45 minutes and the entire questionnaire periods will take about 30 minutes.

A FINAL NOTE

I would like to encourage you to participate in the experiment. As you can actually see your recordings after the session and discuss them with the experimenter this is a rather unique opportunity. Not only is this a chance for you to pick up $10 but in addition, you will also contribute scientific information that will advance the understanding of stress. Your participation in this research project will be greatly appreciated.

CONSENT FORM - A

Date_____

I,_____,freely and voluntarily and without undue inducement or any element of force, fraud, deceit, duress or other form of constraint ~~or coercion~~ consent to be a research participant in the research project entitled "Bodily and Cognitive Responses to Stress" to be conducted at McGill University, department of psychology, during the period of April, 1983 to December, 1984 with Anne Cornwall as principal investigator. The procedures to be followed and their purpose have been explained to me. As I understand it, the study is concerned with measuring a person's reactions to laboratory pain and stress inducing interview and evaluating the effects that pressure pain on the experience of this stress interview. I realize that this study will last one hour and I will be: 1) Required to complete several questionnaires before and after this experiment, 2) asked to experience pressure pain in my non dominant index finger produced by a pressure pain stimulator that will disappear within several seconds after I request that the pressure cease, 3) asked to monitor pain and stress ratings throughout the lab experiment, and 4) asked to complete a stress interview following the pressure pain.

The benefits for participating in this experiment have been explained to me and are as follows: 1) $10 for participating in the experiment, 2) obtaining information on my bodily and psychological reactions to pain and to stress, and 3) contributing knowledge that will advance the scientific understanding of bodily reactions to pain and stress.

I understand that this consent and data collected on me may be withdrawn at any time. I have asked and received answers on any questions concerning this consent form. Questions, if any, have been answered to my satisfaction. I have read and understand this consent form.

_____

Research participant

CONSENT FORM - B

Date_____

I,_____, freely and voluntarily and without undue inducement or any element of force, fraud, deceit, duress or other form of constraint or coercion consent to be a participant in the research project entitled "Bodily and Cognitive Responses to Stress" to be conducted at McGill University, department of psychology, during the period of April, 1983 to December 1984, with Anne Cornwall as principal investigator The procedures to be followed and their purpose have been explained to me. As I understand it, the study is concerned with measuring a person's reactions to laboratory pain. I realize that this study will last one hour and I will be: 1) Required to complete several questionnaires before and after this experiment, 2) asked to experience pressure pain in my nondominant index finger produced by a pressure pain stimulator that will that will disappear within seconds after I request that the pressure cease, 3) asked to monitor pain and stress ratings throughout the lab experiment, and 4) given information that may affect the experience of the pain.

The benefits for participating in this experiment have been explained to me and are as follows. 1) $10 for participating in the experiment, 2) obtaining information on my bodily and psychological reactions to pain and to stress, and 3) contributing knowledge that will advance the scientific understanding of bodily reactions during pain and stress.

I understanding that this consent and data collected on me be withdrawn at any time. I have asked and received answers on any questions concerning this consent form. Questions, if any, have been answered to my satisfaction. I have read and understand this consent form.

_____

Research participant

CONSENT FORM - C

Date_____

I,_____freely, and voluntary and without undue inducement or any element of force, fraud, deceit, duress or other form of constraint or coercion consent to be a participant in the research project entitled "Bodily and Cognitive Responses to Stress" to be conducted at McGill University, department of psychology, during the period of April, 1983 to December, 1983, with Anne Cornwall as principal investigator. The procedures to be followed; and their purpose have been explained to me. As I understand it, the study is concerned with measuring a person's reactions while answering questions in a stress-inducing interview I realize that the study will last one hour and I will be: 1) Required to fill out several questionnaires before and after the experiment, 2) asked to participate in a stressful interview during the experiement, and 3) asked to monitor pain and stress ratings throughout the lab phase of the experiment. The purpose of the experiment is to determine my subjective and bodily ractions to this interview.

The benefits for participating in this experiment have been explained to me and are as follows: 1) $10 for participating in the experiment, 2) obtaining information my bodily and psychological reactions to the stress interview, and 3) contributing knowledge that will advance scientific understanding of bodily reactions during a stressful situation.

I understand that this consent and data collected on me may be withdrawn at any time. I have asked and received answers on any questions concerning this consent form. Questions, if any, have been answered to my satisfaction. I have read and understand this consent form.

_____

Research participant

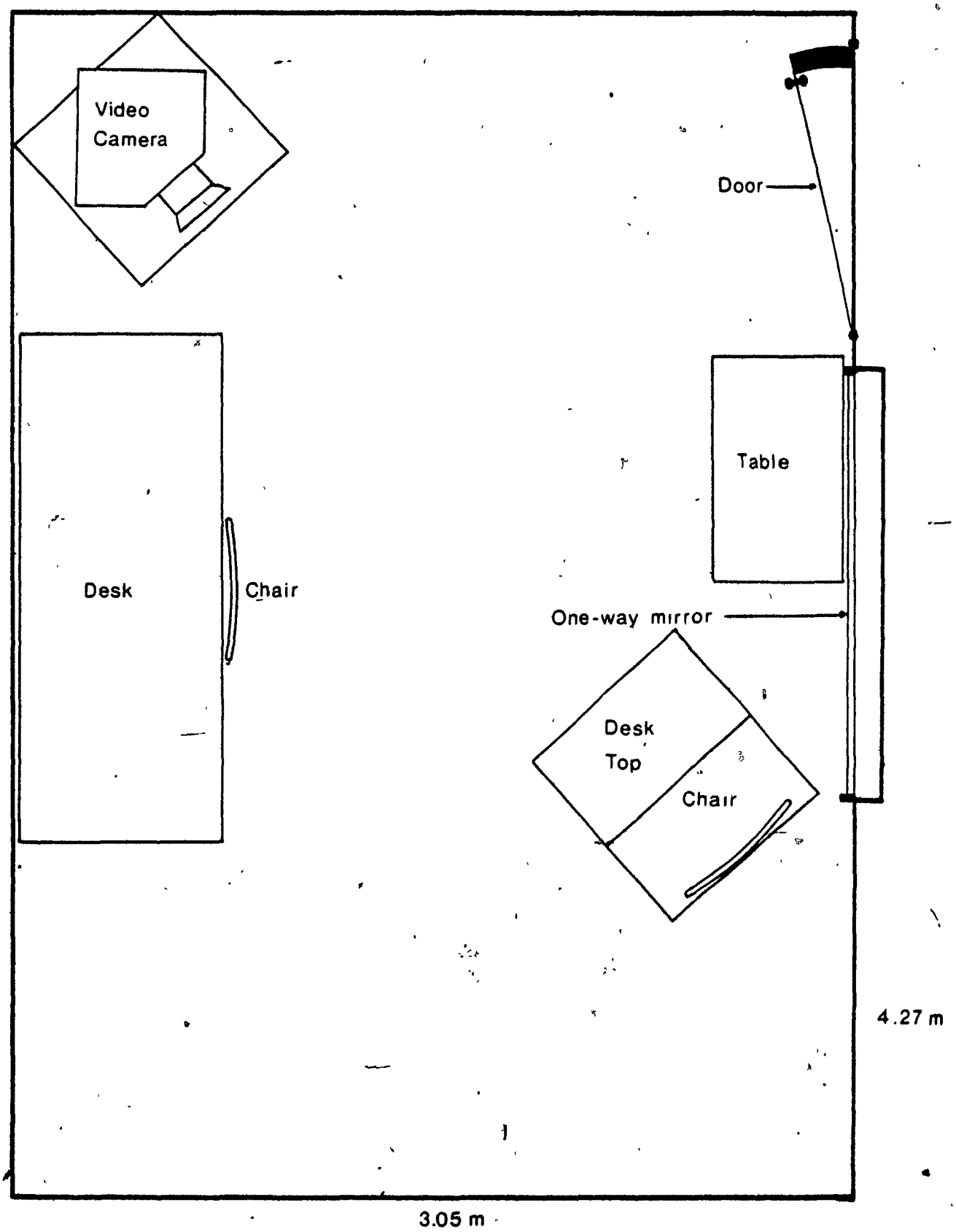
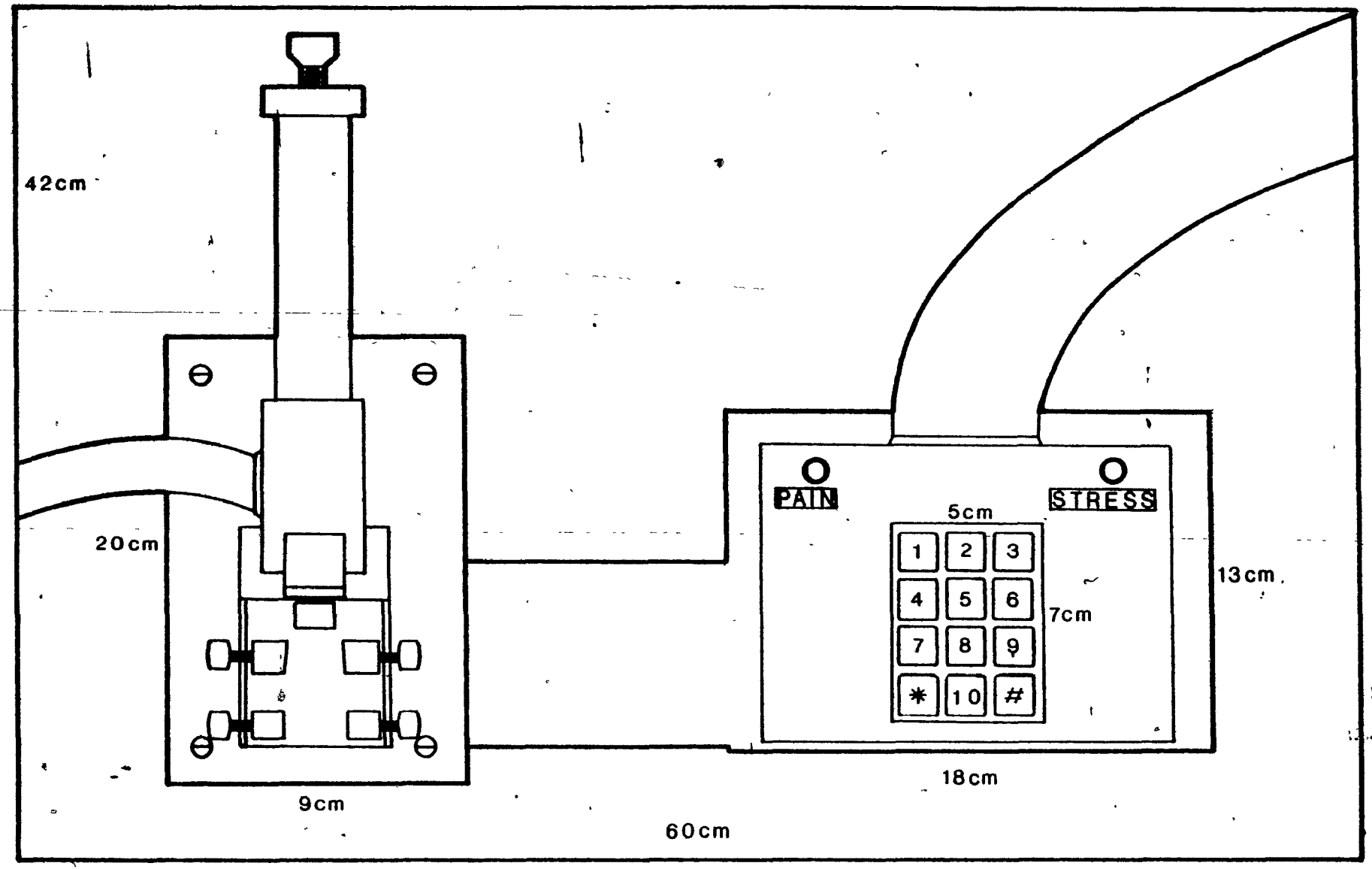


Diagram of the Subject room



Subject's desk top housing experimental apparatus

APPENDIX B

Original pre and post experimental questionnaires

Demographic Data

Subject #:_____ Date:_____

Age:_____ Sex:_____

Weight._____

First Language:_____

Religious Affiliation:_____

Do you attend a church/synagogue?_____

If yes, how often?_____

Occupation:_____

Mother's Occupation_____

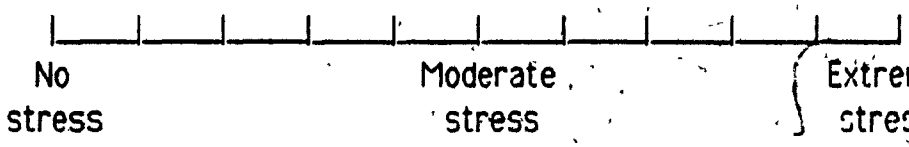
Father's Occupation:_____

EVALUATION OF STRESS LEVEL

Name:_____ Date:_____

Time._____ Form._____

Instructions: On the line below, put a slash to indicate your present level of psychological stress. Stress refers to a state of mental tension, feelings or thoughts of constraint, worry, anxiety or aprehension. A slash on the extreme left of the line would indicate that you are experiencing no stress at all and a slash on the extreme right would mean that you are experiencing an extreme degree of stress. A slash on the middle of the line would indicate that you are moderately stressed.



Post Laboratory Phase Interview - I

Rate the degree to which your response to the painful experience you just encountered resembles your general response to other painful situations you may have encountered.

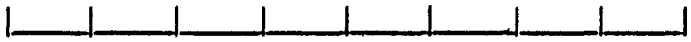
Not all representative | Moderately representative | Identical

What is the worst pain you have ever experienced, e.g. migraine headache, absess tooth, broken bone, etc?

_____

Post Laboratory Phase Interview - II

Rate the degree to which your response to the painful experience you just encountered resembles your general response to other painful situations you may have encountered.



Not at all representative — Moderately representative — Identical

What is the worst pain you have ever experienced, e.g. migraine headache, absess tooth, broken bone, etc.?

_____

_____

Rate the degree to which you feel your pain experience was influenced by the stressfulness of the impending interview or whether the impending interview acted as a distractor from your pain.

Affected by stressfulness of the impending interview

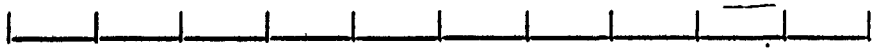
Not at all — Very much

Affected by distraction

Not at all — Very much

Post-Laboratory Phase Interview - III

Rate the degree to which your response to the painful experience you just encountered resembles your general response to other painful situations you may have encountered.



Not all representative | Moderately representative | Identical

What is the worst pain you have ever experienced, e.g. migraine headache, absess tooth, broken bone, etc.?

_____

Rate the degree to which you feel your pain experience was influenced by the stressfulness of the warning of impending pain given prior to application of the pressure stimulus.

Affected by stressfulness of the warning

Not at all | Very much

Subjective Impressions Questionnaire:

Now that the experiment is over, we would appreprecíate your feedback about your participation in this experiment. Please be as candid as possible since the results of this study depend on it.

1. Have you taken the course "Perspectives on Human Nature"?

   Yes_____ No_____

2. After hearing of the upcoming pressure pain, did you think of any strategies you could use to cope with the pain?

   Yes_____ No_____

   If yes, what were they?

   _____

   _____

   _____

3. To what degree did you believe that the pressure would be painful?

   1 2 3 4 5

   not painful ... very painful

4. What hypotheses do you think this experiment was trying to test, i.e., what do you think this experiment was about?

   _____

   _____

   _____

5. Have any friends or acquaintances participated in this experiment?

   Yes_____ No_____

   If yes, did you discuss this experiment with them?

   Yes_____ No_____

   If yes, what information did they give you that was not contained in the manual?

   _____

   _____

   _____

Thank-you for your candidness. Please recheck your answers to ensure that they are corect (as it applies to you personally).

Subjective Impressions Questionnaire:

Now that the experiment is over, we would appreciate your feedback about your participation in this experiment. Please be as candid as possible since the results of this study depend on it.

1. Have you taken the course "Perspectives on Human Nature"?

   Yes_____ No_____

2. After hearing of the upcoming stress interview, did you think of any strategies you could use to cope with the stress?

   Yes_____ No_____

   If yes, what were they?

   _____

   _____

   _____

3. To what degree did you believe that the stress interview would be stressful?

   1 2 3 4 5

   not stressful ——— very stressful

4. What hypotheses do you think this experiment was trying to test, i.e., what do you think this experiment was about?

   _____

   _____

   _____

5. Have any friends or acquaintances participated in this experiment?

   Yes_____ No_____

   If yes, did you discuss this experiment with them?

   Yes_____ No_____

   If yes, what information did they give you that was not contained in the manual?

   _____

   _____

   _____

Thank-you for your candidness. Please recheck your answers to ensure that they are correct (as it applies to you personally).

APPENDIX C

Behavioral response categories and operational definitions

List of behaviours commonly seen during baseline, pain and recovery periods.

BASELINE PERIOD	PAIN PERIOD	RECOVERY PERIOD
- moving head right, left, up, down	- moving head right, left, up, down	- moving head right, left, up, down
- moving eyes right, left, up, down	- moving eyes right, left, up, down	- moving eyes right, left, up, down
- frown	- frown	- frown
- pout	- grimace	- grimace
- opening/closing eyes	- opening/closing eyes	- opening/closing eyes
- opening/closing mouth	- opening/closing mouth	- opening/closing mouth
- smile, laugh	- smile, laugh	- smile, laugh
- motionless	- motionless	- motionless
- talking	- talking	- talking
- sigh, deep breath	- sigh	- sigh, deep breath
- leaning forward, backward, right, left	- rocking, swaying	- leaning forward, backward
- staring	- staring	- looking at hand
- hand to face	- hand to face	- hand to face
- moving hand	- shaking/nodding head	- hand touching other hand
- cough, yawn	- looking to the left	
- blinking eyes	- blinking eyes	
- head straight		

OPERATIONAL DEFINITIONS FOR NONVERBAL BEHAVIORS

I. HEAD MOVEMENTS

a. UP/DOWN/RIGHT/LEFT/TILT: All head movements across the center line to the right or left and all head movements above or below the lower horizontal line and all head movements in which the head tilts to the left or right or forward or backward. Chin position is usually the best indicator of this.

b. LEFT DOWN: All head movements to the left and down, i.e., head is positioned to the left of the middle line and directed down toward the lower quadrant of the screen. Eye direction can be used as a determinant if unclear if head is only to the left.

c. STRAIGHT AHEAD: Head positioned straight ahead, eyes can be down right left or straight ahead. Look at chin position if unclear.

d. NODDING/SHAKING: Head movements up and down at least once in rapid succession and head movements right and left at least once in rapid succession. Includes head bobbing.

II. MOUTH MOVEMENTS

a. TALKING : Rapid mouth movements.

b. SMILING/LAUGHING: Stretching of the corners of the mouth in an upward fashion with or without the appearance of the teeth or any sound of laughter, head jerks, accompanying smiles.

c. COUGH /YAWN: Sound of cough, body or head jerking; mouth opening of wide upening of mouth and/or sound of yawn.

d. OPEN/CLOSE: Mouth opening wider or closing if and only if not accounted for by above categories.

III. BODY MOVEMENTS

a. ARMS: Any arm movements up, down, right left without the appearance of the hand.

b. HAND TO SHOULDER/FACE/NECK: Any movement of either hand to the upper part of the body. Sometimes an arm movement may precede hand movements, then count both. However, if arm and hand movements go together, only count hand movement.

c. SHOULDERS/DOWN/FORWARD/BACK: Any movement of the shoulders to the left, right, up, down, forward, or backward from a previous position. Eg. hunching shoulders, raising them or pushing back.

d. ROCKING/SWAYING Any body movements in which the upper part of the body moves to the right, left, forward or back several times in rapid succesion.

IV. GRIMACE

a. PARTIAL: Lip pursing (apposition of both the upper and lower lip) or lip biting (application of the teeth to the upper or lower lip) without other facial involvement. Do not include if lip pursing is part of smiling.

b. FULL: Lip pursing or biting with any of the following additional facial features; raising or lowering of the eyebrows, closing the eyes tightly or opening them wider, widening of the nostrils.

GENERAL NOTES

1. Record start of period head position first at the beginning of each period.

2. Categories need not be mutually exclusive.

3. If you are not sure that a particular movement took place, <u>do not</u> indicate that it occured.

4. If an apparent body movement may be due to a tape tremor, <u>do not</u> check it as occuring.

5. While eye position may serve as an indicator of head position, <u>do not</u> register changes in eye movements as changes in head position.

6. Be careful to include multiple co-occuring behaviours. eg. often shoulders move up and down when mouth opens and closes and often open/close mouth when bringing hand to face, etc.

APPENDIX D

Statistical analyses conducted for validity checks on the main measures.

Repeated measures ANOVA obtained from the analysis of the bridge transducer data and Scheffé's tests comparing the increases across successive minutes.

Source	Mean Square	df	F	p	Huynh Feldt p
Mean	1853887143.60	1	26922.97	0.0000	
Group	1313.13	2	0.02	0.9811	
Error	68858.93	45			
R	13054.60	9	4.42	0.0000	0.0138
RG	2768.30	18	0.94	0.5331	0.4581
Error	2953.26	405			

Scheffé's Tests

Comparisons	F	p
P1-P2	10.81	<0.01
P2-P3	0.32	ns
P3-P4	1.67	ns
P4-P5	1.08	ns
P5-P6	0.31	ns
P6-P7	0.02	ns
P7-P8	0.18	ns
P8-P9	1.92	ns
P9-P10	2.94	ns

Mean force (in grams) exerted during each minute of the pain period.

Group	PO	P+PW	P+S1	All Group
Minutes	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean
1	1945 (61)	1933 (93)	1894 (85)	1924
2	1967 (58)	1962 (88)	1952 (86)	1960
3	1956 (87)	1972 (97)	1982 (63)	1967
4	1990 (58)	1972 (97)	1982 (67)	1982
5	1975 (65)	1967 (106)	1968 (94)	1970
6	1978 (85)	1963 (91)	1988 (88)	1976
7	1984 (64)	1969 (97)	1972 (76)	1975
8	1971 (48)	1964 (94)	1975 (81)	1970
9	1934 (62)	1970 (98)	1960 (79)	1954
10	1982 (70)	1967 (97)	1972 (79)	1974
Mean	1969	1964	1964	1965

Repeated measures ANOVA obtained from analyses of the baseline and anticipatory stress intensity ratings of the PO and P+PW groups.

Source	Mean Square	df	E	p	Huynh Feldt p
Mean	1043.28	1	219.04	0.00	
Group	10.92	1	2.29	0.14	
Error	4.76	30			
R	0.60	10	1.98	0.04	0.07
RG	0.23	10	0.77	0.66	0.60
Error	0.30	30			

Repeated measures ANCOVA and Scheffé's tests obtained from analyses of the baseline and anticipatory HR minutes from the PO and P+PW groups.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	13.57	1	1.14	0.30	
Covariate[1]	12443.06	1	1041.59	0.00	
Error	11.95	29			
R	31.96	4	4.83	0.00	0.00
RG	26.98	4	4.08	0.00	0.01
Error	6.62	120			

Scheffé's tests:

Comparisons	F	p
B6	1.20	ns
B7	0.19	ns
B8	1.46	ns
B9	0.43	ns
AP	15.61	<0.01

Note:

[1]Covariate=First five minutes of baseline HR.

Repeated measures ANCOVA and Scheffé's tests obtained from the analyses of the baseline and anticipatory EMG minutes data from the PO and P + PW groups.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	773.48	1	5.46	0.03	
Covariate[1]	102468.58	1	723.38	0 00	
Error	141.65	29			
R	225.16	4	2.03	0.09	0.14
RG	460.94	4	4.16	0.00	0.02

Scheffé's tests:

Comparisons	F	p
B6	0.01	ns
B7	0.00	ns
B8	0.21	ns
B9	0.32	ns
AP	23.15	<0.01

Note:

[1] Covariate = First five minutes of baseline EMG.

Repeated measures ANOVA obtained from the the stress intensity ratings, and the HR and EMG data from the SI group during baseline , AP, and recovery minutes.

Stress Intensity Rating Data:

Source	Mean Square	df	E	p	Huynh Feldt p
Mean	1412.00	1	90.50	0 00	
Error	15.60	15			
R	0.96	21	2.95	0.00	0.01
Error	0 93	315			

HR data:

Source	Mean Square	df	E	p	Huynh Feldt p
Mean	1489930.80	1	800.51	0.00	
Error	1861.23	15			
R	26.56	19	2.79	0.00	0.00
Error	9.54	285			

EMG data:

Source	Mean Square	df	E	p	Huynh Feldt p
Mean	90731341.71	1	189.81	0.00	
Error	644685.15	15			
R	5934.26	19	1.08	0.37	0.38
Error	5507.34	285			

Scheffé's tests conducted using the stress intensity ratings obtained from the SI group across Baseline, Anticipatory, and Recovery periods.

Comparisons	F	p
B1-B2	4.33	0.05
B2-B3	0.49	ns
B3-B4	3.12	ns
B4-B5	0.00	ns
B5-B6	0.20	ns
B6-B7	0.00	ns
B7-B8	0.78	ns
B8-B9	1.76	ns
B9-B10	0 00	ns
B10-AP1	33.00	0.01
AP1-AP2	0.20	ns
AP2-R1	15.82	0.01
R1-R2	1.75	ns
R2-R3	0.20	ns
R3-R4	0.00	ns
R4-R5	0.00	ns
R5-R6	1.76	ns
R6-R7	3.12	ns
R7-R8	0.78	ns
R8-R9	0.72	ns
R9-R10	0.72	ns

Scheffé's tests conducted using the HR data obtained from the SI groups across the Baseline, Anticipatory and Recovery periods.

Comparisons	F	p
B1-B2	0.20	ns
B2-B3	2.26	ns
B3-B4	1.69	ns
B4-B5	0.10	ns
B5-B6	0.27	ns
B6-B7	0.04	ns
B7-B8	0.04	ns
B8-B9	0.21	ns
B9-AP1	10.29	0.01
AP1-AP2	2.67	ns
AP2-R1	12.46	0.01
R1-R2	0.16	ns
R2-R3	0.51	ns
R3-R4	3.86	ns
R4-R5	3.86	ns
R5-R6	0.27	ns
R6-R7	0.17	ns
R7-R8	0.08	ns
R8-R9	0.16	ns

ANOVAs from the analyses SDS scores obtained from the 4 experimental groups and TMAS /SDS composite scores obtained from the 3 groups receiving pressure pain.

SDS[1]	Mean Square	df	E	p
Between	16.64	3	0.59	0.62
Within	28.18	60		

TMA S^2/SDS

Source	Mean Square	df	E	p
Between	0.56	2	1 32	0.27
Within	0.43	45		

Notes:

[1]SDS-Social desirability Scale

[2]TMAS =Taylor Manifest Anxiety Scale

APPENDIX E

Statistical analyses and tables pertaining to the pre-experimental questionnaire data.

Univariate ANOVAs obtained from the analyses of differences between the four groups in age, height to weight ratio, and SES.

Age:

Source	Mean Square	df	F	p
Between	0.56	3	0.05	0.98
Within	10.89	60		

Height to weight ratio:

Source	Mean Square	df	F	p
Between	0.00	3	1.50	0.22
Within	0.00	60		

SES:

Source	Mean Square	df	F	p
Between	590.50	3	2.59	0.06
Within	227.99	60		

Demographic characteristics of the groups and of the entire sample

Measure	Age	Season (1 - 4)	Language (1 or 2)	Citizenship (1 or 2)	Religion (1 - 5)	Height/ Weight	SES
Group	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
PO	21.4 (2.8)	1.8 (1.2)	1.0 (0.0)	1.0 (0.0)	3.3 (1.4)	0.45 (0.04)	50.2 (18)
P+PW	20.4 (3.8)	1.6 (1.0)	1.0 (0.0)	1.0 (0.0)	2.7 (1.4)	0.45 (0.04)	54.9 (15)
P+SI	21.1 (3.6)	1.9 (1.1)	1.0 (0.0)	1.1 (0.4)	3.5 (1.6)	0.46 (0.06)	64.8 (10)
SI	21.2 (2.9)	2.2 (0.5)	1.1 (0.3)	1.0 (0.0)	2.8 (1.3)	0.42 (0.07	60.2 (16)
Grand Mean	21.0 (3.4)	1.6 (0.9)	1.0 (0.1)	1.0 (0.1)	3.1 (1.4)	0.44 (0.05)	57.5 (14)

ANOVAs obtained from the analyses of group differences on the TMAS[1], SDS[2], GTAR-EA[3] and GTAR-PD[4].

TMAS:

Source	Mean Square	df	F	p
Between	42.60	3	2.22	0.0949
Within	19.18	60		

SDS:

Source	Mean Square	df	F	p
Between	16.64	3	0.59	0.6236
Within	28.18	60		

GTAR-EA:

Source	Mean Square	df	F	p
Between	89.35	3	0.75	0.5247
Within	118.59	60		

GTAR-PD:

Source	Mean Square	df	F	p
Between	21.85	3	0.14	0.9372
Within	158.93	60		

[1] TMAS= Taylor Manifest Anxiety Scale
[2] SDS = Social Desirable Scale
[3] GTAR-EA= Inventory of General Trait Anxiety Revised-Physical Danger
[4] GTAR-PD= Revised - Evaluation Anxiety

Personality characteristics of the groups and the total sample.

Measure:	TMAS[1]	SDS[2]	GTAI-EA[1]	GTAI-PD[1]
Sample	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
PO	8.8 (4.3)	13.9 (3.6)	40.1 (10.3)	50.4 (11.1)
P+PW	5.5 (4.5)	16.3 (5.6)	39.1 (11.5)	51.5 (15.3)
P+SI	9.1 (4.8)	14.6 (4.7)	34.8 (10.3)	52.8 (10.7)
SI	7.6 (3.9)	14.9 (6.8)	37.1 (11.4)	51.9 (12.8)
Mean	7.7 (4.5)	15.0 (5.3)	37.8 (10.8)	52.6 (12.3)
Population norms	14.9 (n.a)	15.1 (5.6)[3]	32.4 (9.8)[4]	52.6 (10.8)

Notes:

[1]See notes on previous page

[2]Taylor (1953) norms obtained from the general population

[3]Crowne and Marlowe (1964) norms obtained from male college students.

[4]Endler and Magnusson (1976) norms obtained from male college students.

ANOVA obtained from the 4 (groups)x 2 (GTAI-EA and GTAI-PD)[1] analysis.

Source	Mean Square	df	F	p
Mean	25 7313.45	1	1399.66	0.0000
Group	17.22	3	0.09	0.9633
Error	183.84	60		
Anxiety Type	6398.63	1	68.30	0.0000
AG	93.99	3	1.00	0.3977
Error	93.68	60		

Note:

[1] GTAR-EA= General Trait Anxiety Revised Scale-evaluation anxiety
GTAR-PD= General Trait Anxiety Revised Scale-Physical Danger

ANOVA obtained from the analyses of group differences on the STAI-S and present "stress" rating.

STAI-S

Source:	Mean Square	df	E	p
Between	86.23	3	1.49	0.22
Within	57.76	60		

Present stress:

Source:	Mean Square	df	E	p
Between	10.93	3	2.80	0.05
Within	3.90	60		

Note.

STAI-S=State-Trait Anxiety Inventory-State measures.

Group values obtained on the STAI - S and present stress rating.
Population norms are presented from the STAI - S[1].
(A high STAI - S score indicates a low level of anxiety.)

Measure:	STAI - S	Present Stress
Sample	Mean SD	Mean SD
PO	65.0 (6.6)	3.3 (1.8)
P+PW	64.1 (9.6)	4.4 (1.7)
P+SI	61.0 (7.4)	4.2 (1.4)
SI	66.0 (6.4)	2.6* (1.8)
Mean	64.2 (7.7)	3.6 (2.1)
Population norms	33.8 (7.4)[1]	

*p<0.05

Notes:
[1] STAI - S = State - Trait Anxiety Inventory - State measures

[1] Pancheri, Bernadet, Bellaterra, and Tartaglione (1976) norms obtained from college students.

APPENDIX F

Analyses conducted using the data obtained from the direct pain measures

ANCOVA conducted with the pain intensity ratings obtained from all subjects using group means to fill missing data cells.

Source	Mean Square	df	F	p	Huynn Feldt p
Group	57.85	2	8.26	0.00	
Covariate	31.90	1	4.56	0.04	
Error	7.00	44			
R	209.71	30	221.69	0.0	0.0
RG	3.00	60	3.17	0.0	0.0
Error	0.95	1350			

Note:
[1] Covariate = SES

Baseline orthogonal analysis conducted with the pain intensity ratings obtained from all subjects.

SOURCE	Mean Square	df	F	p	Huynh Feldt p
Group	0.164	2	0.52	0.60	
Covariate [1]	0.034	1	0.11	0.74	
Error	0.316	44			
R (1)	0.016	1	1.97	0.17	
R (1) G	0.004	2	0.52	0.61	
Error	5.064	45			
R (2)	0.031	1	0.75	0.39	
R (2) G	0.046	2	1.12	0.34	
Error	0.41	45			
R (3)	0.002	1	1.97	0.17	
R (3) G	0.001	2	0.52	0.61	
Error	0.001	45			
R (5)	0.039	1	1.87	0.18	
R (5) G	0.012	2	0.56	0.58	
Error	0.021	45			
R6	0.003	1	0.82	0.38	
R (6) G	0.004	2	1.09	0.34	
Error	0.004	45			
R 7	0.001	1	0.13	0.72	
R (7) G	0.009	2	1.43	0.25	
Error	0.006	45			
R (8)	0.018	1	1.32	0.26	
R (8) G	0.012	2	0.84	0.44	
Error	0.14	45			
R (9)	0.004	1	0.84	0.37	
R (9) G	0.006	2	1.9	0.35	
Error	0.005	45			
R	0.013	9	1.12	0.35	0.32
RG	0.011	18	0.94	0.52	0.43
Error	0.011	405			

Note:

[1] Covariate = SES

Pain period orthogonals conducted with the pain intensity ratings using group means to fill missing data cells.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	126.39	2	7.21	0.01	
Covariate	69.49	1	3.97	0.03	
Error	17.52	44			
R(1)	507.04	1	100.13	0.00	
R(1)G	18.79	2	3.71	0.04	
Error	5.06	45			
R(2)	139.17	1	46.76	0.00	
R(2)G	4.24	2	1.42	0.23	
Error	2.98	45			
R(3)	13.60	1	11.99	0.00	
R(3)G	4.45	2	3.93	0.03	
Error	1.14	45			
R(4)	1.47	1	1.35	0.25	
R(4)G	0.70	2	0.64	0.53	
Error	1.09	45			
R(5)	0.09	1	0.17	0.67	
R(5)G	0.86	2	1.65	0.20	
Error	0.52	45			
R(6)	0.71	1	2.09	0.16	
R(6)G	0.32	2	0.97	0.39	
Error	0.34	45			
R(7)	0.61	1	1.50	0.23	
R(7)G	0.56	2	1.37	0.26	
Error	0.41	45			
R(8)	0.01	1	0.01	0.91	
R(8)G	2.23	2	4.17	0.02	
Error	0.53	45			
R(9)	1.01	1	3.66	0.06	
R(9)G	0.03	2	0.12	0.89	
Error	0.27	45			
R	73.74	9	53.75	0.00	0.00
RG	3.76	18	2.61	0.00	0.01
Error	1.37	405			

Note: Covariate=SES

Recovery period orthogonal analysis conducted with the pain intensity ratings.

Source	Mean Square	df	F	p	Huynn Feldt p
Group	4.511	2	2.15	0.13	
Covariate[1]	3.357	1	1.60	0.21	
Error	2.101	44			
R(1)	26.264	1	21.15	0.00	
R(1) G	1.946	2	1.57	0.22	
Error	1.242	45			
R(2)	14.299	1	31.28	0.00	
R(2) G	0.704	2	1.54	0.23	
Error	0.457	45			
R(3)	4.931	1	17.49	0.00	
R(3) G	0.011	2	0.04	0.96	
Error	0.282	45			
R(4)	0.535	1	2.97	0.09	
R(4) G	0.061	2	0.34	0.71	
Error	0.180	45			
R(5)	0.106	1	1.23	0.27	
R(5) G	0.126	2	1.45	0.24	
Error	0.086	45			
R(6)	0.011	1	0.44	0.51	
R(6) G	0.029	2	1.12	0.33	
Error	0.026	45			
R(7)	0.018	1	1.03	0.32	
R(7) G	0.010	2	0.57	0.57	
Error	0.018	45			
R(8)	0.000	1	0.00	0.95	
R(8) G	0.002	2	0.10	0.90	
Error	0.016	45			
R(9)	0.004	1	0.25	0.62	
R(9) G	0.012	2	0.74	0.48	
Error	0.016	45			
R	5.130	9	19.88	0.00	0.00
RG	0.322	18	1.25	0.22	0.80
Error	0.258	405			

Note:

[1]Covariate = SES

Scheffé's tests comparing the PO and P+PW, PO and P+SI, and P+PW and P+SI during the pain minutes with the pain intensity data using group means to fill missing data cells.

Comparisons	PO and P+PW		PO and P+SI		P+PW and P+SI	
Minutes	F	p	F	p	F	p
P1	0.20	ns	7.07	<0.01	4.31	<0.05
P2	0.11	ns	3.12	ns	2.06	ns
P3	1.17	ns	5.85	<0.01	1.84	ns
P4	0.57	ns	6.89	<0.01	3.49	<0.05
P5	3.37	<0.05	15.97	<0.01	4.67	<0.05
P6	3.76	<0.05	16.10	<0.01	4.31	<0.05
P7	3.37	<0.05	13.72	<0.01	3.49	<0.05
P8	19.20	<0.01	25.77	<0.01	0.47	ns
P9	12.60	<0.01	18.83	<0.01	0.62	ns
P10	10.40	<0.01	8.85	<0.01	0.65	ns

ANCOVA conducted with the pain intensity rating data obtained from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	34.98	2	3.22	0.06	
Covariate [1]	19.65	1	1.81	0.19	
Error	10.85	19			
R	82.28	30	75.96	0.0	0.0
RG	2.11	60	1.95	0.00	0.10
Error	1.08	600			

Note:
[1] Covariate = SES

Baseline orthogonal conducted with the pain intensity ratings obtained from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huyhh Feldt p
Group	0.230	2	0.33	0.72	
Covariate[1]	0.033	1	0.05	0.83	
Error	0.692	19			
R(1)	0.001	1	0.89	0.36	
R(1)G	0.010	2	0.93	0.41	
Error	0.011	20			
R(2)	0.075	1	0.89	0.36	
R(2)G	0.079	2	0.93	0.41	
Error	0.085	20			
R(3)	0.001	1	0.89	0.36	
R(3)G	0.002	2	0.93		
Error	0.002	20			
R(4)	0.000	1	0.89	0.36	
R(4)G	0.001	2	0.93	0.41	
Error	0.001	20			
R(5)	0.029	1	0.89	0.36	
R(5)G	0.030	2	0.93	0.41	
Error	0.032	20			
R(6)	0.000	1	0.89	0.36	
R(6)G	0.000	2	0.93	0.41	
Error	0.000	20			
R(7)	0.009	1	0.89	0.36	
R(7)G	0.009	2	0.93	0.41	
Error	0.010	20			
R(8)	0.025	1	0.89	0.36	
R(8)G	0.026	2	0.93	0.41	
Error	0.028	20			
R(9)	0.010	1	0.89	0.36	
R(9)G	0.010	2	0.93	0.41	
Error	0.011	20			
R	0.018	9	0.89	0.54	0.37
RG	0.019	18	0.93	0.54	0.42
Error	0.020	180			

Note:

[1] Covariate=SES

Pain period orthogonals conducted with the pain intensity ratings obtained from subjects who did not report tolerance

Source	Mean Square	df	F	p	Huynh Feldt p
Group	95.448	2	3.53	0.05	
Covariate[1]	67.688	1	2.51	0.13	
Error	27.011	19			
R(1)	378.530	1	68.59	0.00	
R(1)G	11.598	2	2.10	0.15	
Error	5.519	20			
R(2)	37.689	1	17.99	0.00	
R(2)G	5.316	2	2.54	0.00	
Error	2.095	20			
R(3)	1.692	1	2.12	0.16	
R(3)G	0.372	2	0.47	0.63	
Error	0.79	20			
R(4)	0.000	1	0.00	0.97	
R(4)G	0.452	2	1.21	0.32	
Error	0.374	20			
R(5)	0.004	1	0.01	0.92	
R(5)G	0.950	2	2.44	0.11	
Error	0.390	20			
R(6)	0.262	1	1.48	0.23	
R(6)G	0.017	2	0.10	0.90	
Error	0.17	20			
R(7)	0.181	1	0.62	0.44	
R(7)G	0.14	2	0.51	0.61	
Error	0.292	20			
R(8)	0.037	1	0.19	0.66	
R(8)G	0.100	2	0.52	0.60	
Error	0.194	20			
R(9)	0.000	1	0.00	0.98	
R(9)G	0.033	2	0.25	0.78	
Error	0.133	20			
R	46.488	9	41.96	0.00	0.0
RG	2.110	18	1.90	0.02	0.09
Error	1.107	180			

Note:

[1] Covariate=SES

Recovery period orthogonals conducted with the pain intensity ratings obtained from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	1.999	2	1.20	0.32	
Covariate[1]	0.042	1	0.03	0.88	
Error	1.660	19			
R(1)	8.478	1	5.90	0.02	
R(1)G	3.618	2	2.52	0.11	
Error	1.438	20			
R(2)	2.978	1	11.32	0.00	
R(2)G	0.604	2	2.30	0.13	
Error	0.263	20			
R(3)	0.162	1	0.82	0.38	
R(3)G	0.114	2	0.57	0.57	
Error	0.199	20			
R(4)	0.129	1	0.62	0.44	
R(4)G	0.228	2	1.09	0.35	
Error	0.208	20			
R(5)	0.009	1	0.10	0.75	
R(5)G	0.135	2	2.19	0.14	
Error	0.065	20			
R (6)	0.010	1	0.51	0.49	
R(6)G	0.15	2	0.78	0.47	
Error	0.020	20			
R(7)	0.022	1	1.19	0.29	
R(7)G	0.005	2	0.26	0.77	
Error	0.019	20			
R(8)	0.001	1	0.03	0.86	
R(8)G	0.008	2	0.34	0.72	
Error	0.025	20			
R(9)G	0.000	1	0.01	0.92	
R(9)G	0.024	2	2.32	0.12	
Error	0.010	20			
R	1.310	9	5.20	0.0	0.01
RG	0.534	18	2.12	0.01	0.11
Error	0.252	180			

Note:

[1]Covariate=SES

Scheffé's tests comparing overall pain intensity rating means during the Pain period.

Comparisons	F	p
PO and P+PW	3.74	<0.05
PO and P+SI	10.35	<0.01
P+PW and P+SI	0.94	ns

ANCOVAs and Scheffé's tests obtained from the analyses of the overall mean pain intensity rating during the pain period.

SOURCES	Mean Square	df	F	p
Group	9.52	2	3.73	0.03
Covariate[1]	4.05	1	1.59	0.21
Error	2.05	44		
Equality of Slopes	1.85	2	0.72	0.49
Error	2.59	42		

Scheffé's test

Comparisons	F	p
PO and P+PW	0.62	ns
PO and P+S1	4.36	<0.05
P+PW and P+S1	1.69	ns

Note:
[1]Covariate=SES

ANCOVAs obtained from analyses of between groups differences on pain threshold, tolerance, and endurance. Kolnogorov-Smirnov tests on the cumulative proportion of dropouts.

PAIN THRESHOLD

SOURCE	Mean Square	df	F	P
Group	6029.63	2	0.31	0.74
SES	7419.82	1	0.38	0.54
Error	19457.48	44		

PAIN TOLERANCE

SOURCE	Mean Square	df	F	P
Group	16967.58	2	0.51	0.61
SES	74163.67	1	2.21	0.14
Error	33568.14	44		

PAIN ENDURANCE

SOURCE	Mean Square	df	F	P
Group	13152.60	2	0.41	0.66
SES	34667.36	1	1.09	0.30
Error	31877.80	44		

KOLMOGOROV-SMIRNOV TEST

COMPARISON	D	P
P0 and P+PW	.3125	ns
P0 and P+S1	.0000	ns
P+PW and P+S1	.4175	<0.05

ANCOVAs obtained from the analyses of the post experimental pain questionnaires.

GRACELY - INTENSITY SCALE

SOURCE	Mean Square	df	F	p
Group	6.45	2	1.44	0.25
SES	0.33	1	0.07	0.79
Error	4.47	44		

GRACELY-UNPLEASANTNESS SCALE

SOURCE	Mean Square	df	F	p
Group	0.56	2	0.08	0.92
SES	1.68	1	0.25	0.62
Error	6.66	44		

GRACELY-PAIN SCALE

SOURCE	Mean Square	df	F	p
Group	3.85	2	0.44	0.65
SES	6.19	1	0.70	0.41
Error	8.78	44		

MCGILL PAIN QUESTIONNAIRE (MPQ)-PAIN RATING INDEX (PRI) SENSORY SCALE

SOURCE	Mean Square	df	F	p
Group	49.33	2	1.31	0.28
SES	55.59	1	1.47	0.23
Error	37.76	44		

MPQ-PRI-AFFECTIVE SCALE

SOURCE	Mean Square	df	F	p
Group	7.72	2	2.16	0.13
SES	14.27	1	4.00	0.05
Error	3.57	44		

ANCOVA tables from postexperimental pain questionnaires (continued)

MPQ-PRI-EVALUATIVE SCALE

SOURCE	MEAN SQUARE	df	F	p
Group	0.33	2	0.14	0.87
SES	2.84	1	1.22	0.31
Error	2.33	44		

MPQ-PRI-MISCELLANEOUS SCALE

SOURCE	MEAN SQUARE	df	F	p
Group	1.38	2	0.17	0.84
SES	15.83	1	1.99	0.16
Error	7.94	44		

MPQ-PRI-TOTAL OF ALL SCALES

SOURCE	MEAN SQUARE	df	F	p
Group	74.68	2	0.74	0.48
SES	224.94	1	2.22	0.14
Error	101.44	44		

APPENDIX G

Statistical analyses conducted with the data obtained from the stress intensity rating data.

ANCOVA performed using the stress intensity rating data obtained from the PO, P+PW, and P+SI groups using group means to fill missing data cells.

Source	Mean Square	df	F	p	Huyhn Feldt p
Group	53.93	2	2.31	0.11	
Covariate	9.03	1	0.34	0.56	
Error	23.30	44			
R	39.58	30	40.00	0.00	0.00
RG	2.13	60	2.15	0.00	0.00
Error	0.99	1350			

Note: Covariate=SES

Baseline orthogonals conducted with stress intensity ratings from all subjects

SOURCE	Mean Square	df	F	p	Huynh Feldt p
Group	4.040	2	0.76	0 47	
Covariate[1]	0.141	1	0.03	0.87	
Error	5.312	44			
R(1)	0.056	1	0.09	0.77	
R(1)G	0.550	2	0.83	0.44	
Error	0.667	45			
R(2)	0.171	1	0.41	0.52	
R(2)G	0.047	2	0.11	0.89	
Error	0.418	45			
R(3)	0.547	1	1.87	0.18	
R(3)G	0.159	2	0.55	0.58	
Error	0.292	45			
R(4)	0.198	1	0.70	0.46	
R(4)G	0.384	2	1.35	0.27	
Error	0.285	45			
R(5)	0.009	1	0.05	0.83	
R(5)G	0.401	2	1.98	0.15	
Error	0.202	45			
R(6)	0.0	1	0.0	1.00	
R(6)G	0.676	2	4.87	0.01	
Error	0.138	45			
R(7)	0.401	1	3.99	0.05	
R(7)G	0.037	2	0.037	0.37	
Error	0.100	45			
R(8)	0.088	1	1.04	0.31	
R(8)G	0.000	2	0.01	0.99	
Error	0.084	45			
R(9)	0.44	1	2.73	0.11	
R(9)G	0.059	2	0.37	0.70	
Error	0.161	45			
R	0.212	9	0.8	0.60	0.55
RG	0.257	18	0.98	0.48	0.48
Error	0.261	405			

Note:

[1]Coveriete=SES

Pain Orthogonals conducted using the stress intensity ratings from all subjects using group means to fill missing data cells.

SOURCE	Mean Square	df	F	p	Huynh Feldt p
Source	79 669	2	5 64	0.01	
Covariate[1]	34.376	1	2.43	0 13	
Error	14.120	44			
R(1)	54.710	1	11.87	0.00	
R(1)G	3.108	2	0.67	0 51	
Error	4.611	45			
R(2)	10 102	1	7 74	0.01	
R(2)G	9 699	2	7 43	0 00	
Error	1 306	45			
R(3)	0.912	1	0.71	0.46	
R(3)G	1.341	2	1.04	0.36	
Error	1.292	45			
R(4)	4.627	1	3.89	0 05	
R(4)G	1 781	2	1 50	0 23	
Error	1.190	45			
R(5)	1.401	1	2.92	0 09	
R(5)G	3.399	2	7.07	0 00	
Error	0.480	45			
R(6)	0.558	1	0 59	0 45	
R(6)G	0.403	2	0.42	0 66	
Error	0.949	45			
R(7)	0.140	1	0.12	0.73	
R(7)G	3.964	2	3 52	0.04	
Error	1.127	45			
R(8)	2.116	1	1 49	0 23	
R(8)G	2.514	2	1.77	0.18	
Error	1.417	45			
R(9)	0.275	1	0.32	0.53	
R(9)G	1.60	2	1.84	0.17	
Error	0.872	45			
R	8.317	9	5.65	0.0	0.00
RG	3.091	18	2.10	0 01	0 03
Error	1.472	405			

Note:

[1]Covariate =SES

Recovery orthogonals conducted using the stress intensity ratings from all subjects.

SOURCE	Mean Square	df	F	p	Huynh Feldt p
Group	4.378	2	0.35	0.71	
Covariate[1]	0.309	1	0.02	0.88	
Error	12.581	44			
R(1)	10.202	1	6.26	0.02	
R(1)G	5.311	2	3.57	0.04	
Error	1.630	45			
R(2)	9.167	1	15.29	0.00	
R(2)G	0.297	2	0.49	0.61	
Error	0.599	45			
R(3)	6.539	1	17.25	0.00	
R(3)G	0.698	2	1.84	0.17	
Error	0.379	45			
R(4)	0.027	1	0.06	0.81	
R(4)G	0.159	2	0.34	0.71	
Error	0.464	45			
R(5)	0.444	1	1.44	0.24	
R(5)G	0.166	2	0.54	0.59	
Error	0.309	45			
R(6)	0.250	1	2.81	0.10	
R(6)G	0.362	2	4.07	0.02	
Error	0.089	45			
R(7)	0.002	1	0.02	0.90	
R(7)G	0.030	2	0.23	0.80	
Error	0.131	45			
R(8)	0.194	1	1.08	0.30	
R(8)G	0.094	2	0.53	0.59	
Error	0.179	45			
R(9)	0.041	1	0.37	0.55	
R(9)G	0.023	2	0.20	0.82	
Error	0.113	45			
R	2.985	9	6.90	0.0	0.00
RG	0.849	18	1.96	0.01	0.06
Error	0.43272	405			

Note:

[1]Covariate =SES

Scheffé's tests comparing groups during the pain minutes on the stress intensity rating data from all subjects.

Comparisons	PO and P+PW		PO and P+SI		P+PW and P+SI	
MINUTES	E	p	E	p	E	p
P1	2.87	ns	2.24	ns	0.04	ns
P2	2.24	ns	3.64	<0.05	0.17	ns
P3	4.85	<0.05	5.84	<0.01	0.05	ns
P4	4.93	<0.05	6.92	<0.01	0.24	ns
P5	0.33	ns	6.92	<0.01	4.22	<0.05
P6	2.34	ns	6.33	<0.01	1.52	ns
P7	0.21	ns	1.40	ns	0.53	ns
P8	3.21	<0.05	13.32	<0.01	3.45	<0.05
P9	17.30	<0.01	5.84	<0.01	3.04	ns
P10	10.38	<0.01	4.43	<0.05	1.25	ns

Scheffé's tests comparing groups during the recovery period on the stress intensity rating data from all subjects.

Comparisons	PO and P+PW		PO and P+SI		P+PW and P+SI	
Minutes	E	p	E	p	E	p
R1	10.21	<0.01	8.75	<0.01	0.06	ns
R2	2.27	ns	3.27	<0.05	0.09	ns
R3	0.52	ns	4.71	<0.05	2.09	ns
R4	0.82	ns	3.96	<0.05	1.18	ns
R5	0.01	ns	0.66	ns	0.82	ns
R6	0.02	ns	0.23	ns	0.11	ns
R7	0.29	ns	0.48	ns	0.02	ns
R8	0.01	ns	0.09	ns	0.11	ns
R9	1.38	ns	0.07	ns	0.82	ns
R10	0.61	ns	0.01	ns	0.48	ns

ANCOVA conducted using the stress intensity data obtained from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynn Feldt p
Group	6.25	2	0.14	0.87	
Covariate[1]	5.49	1	0.13	0.73	
Error	43.11	19			
R	17.99	30	15.78	0.0	0.0
RG	1.62	60	1.42	0.02	0.13
Error	1.14	600			

Notes:

[1] Covariate=SES

Baseline Orthogonals using stress intensity data from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynn Feldt p
Group	0.808	2	0.10	0.90	
Covariate[1]	2.874	1	0.36	0.56	
Error	8.015	19			
R (1)	0.194	1	0.19	0.67	
R (1) G	0.225	2	0.22	0.80	
Error	1.013	20			
R (2)	0.424	1	1.24	0.28	
R (2)G	0.314	2	0.91	0.42	
Error	0.343	20			
R (3)	0.740	1	2.66	0.12	
R (3) G	0.834	2	2.99	0.07	
Error	0.278	20			
R (4)	0.877	1	2.47	0.13	
R (4) G	0.437	2	1.23	0.31	
Error	0.355	20			
R (5)	0.221	1	0.97	0.37	
R (5) G	0.327	2	1.43	0.26	
Error	0.228	20			
R (6)	0.155	1	0.96	0.34	
R (6) G	0.302	2	1.88	0.18	
Error	0.161	20			
R (7)	0.226	1	1.32	0.26	
R (7) G	0.090	2	0.52	0.60	
Error	0.171	20			
R (8)	0.029	1	0.33	0.57	
R (8) G	0.052	2	0.60	0.56	
Error	0.087	20			
R (9)	0.259	1	1.02	0.32	
R (9) G	0.306	2	1.20	0.32	
Error	0.255	20			
R	0.347	9	1.08	0.38	0.37
RG	0.321	18	1.00	0.46	0.45
Error	0.321	180			

Note:

[1]Covariate= SES

Pain period orthogonals using stress intensity ratings from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynn Feldt p
Group	25.738	2	1.00	0.39	
Covariate	8.149	1	0.32	0.58	
Error	25.790	19			
R(1)	74.401	1	17.45	0.00	
R(1) G	13.814	2	3.24	0.06	
Error	4.264	20			
R(2)	11.690	1	9.08	0.01	
R(2) G	6.874	2	5.34	0.01	
Error	1.28	20			
R(3)	0.385	1	0.47	0.50	
R(3)	0.634	2	0.78	0.47	
Error	0.811	20			
R(4)	0.021	1	0.02	0.88	
R(4) G	1.145	2	1.29	0.30	
Error	0.887	20			
R(5)	0.000	1	0.00	0.99	
R(5) G	0.953	2	3.34	0.06	
Error	0.285	20			
R(6)	0.115	1	0.11	0.74	
R(6) G	1.33	2	1.27	0.30	
Error	1.047	20			
R(7)	0.080	1	0.05	0.82	
R(7) G	2.617	2	1.71	0.21	
Error	1.528	20			
R(8)	1.395	1	0.99	0.33	
R(8) G	0.631	2	0.45	0.64	
Error	1.408	20			
R(9)	0.691	1	0.51	0.49	
R(9) G	0.207	2	0.15	0.86	
Error	1.365	20			
R	9.864	9	6.89	0.0	0.00
R	3.134	18	2.19	0.0	0.01
Error	1.431	180			

Note:
[1]Covariate= SES

Recovery orthogonals using stress intensity ratings from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	2.796	2	0.13	0.88	
Covariate[1]	24.167	1	1.11	0.31	
Error	21.758	19			
R(1)	0.94	1	0.06	0.80	
R(1) G	3.905	2	2.65	0.09	
Error	1.475	20			
R(2)	1.095	1	4.07	0.06	
R(2) G	0.265	2	0.99	0.39	
Error	0.269	20			
R(3)	3.044	1	10.50	0.00	
R(3) G	0.556	2	1.92	0.17	
Error	0.290	20			
R(4)	0.85	1	0.21	0.65	
R(4) G	0.143	2	0.35	0.71	
Error	0.405	20			
R(5)	0.088	1	0.31	0.58	
R(5) G	0.335	2	1.19	0.33	
Error	0.282	20			
R(6)	0.063	1	0.87	0.36	
R(6) G	0.189	2	2.62	0.10	
Error	0.072	20			
R(7)	0.318	1	2.77	0.11	
R(7) G	0.040	2	0.34	0.71	
Error	0.115	20			
R(8)	0.202	1	0.70	0.41	
R(8) G	0.104	2	0.36	0.70	
Error	0.287	20			
R(9)	0.054	1	0.36	0.56	
R(9) G	0.036	2	0.24	0.79	
Error	0.152	20			
R	0.560	9	1.51	0.15	0.20
RG	0.619	18	1.67	0.05	0.11
Error	0.372	180			

Note:

[1] Covariate= SES

Scheffé's tests conducted comparing the three groups' stress intensity ratings during the pain period from subjects who did not report tolerance.

Comparison	PO and P+PW		PO and P+SI		P+PW and P+SI	
Minutes	F	P	F	P	F	P
P1	0.06	ns	0.05	ns	0.00	ns
P2	0.00	ns	0.52	ns	0.43	ns
P3	0.04	ns	1.29	ns	0.71	ns
P4	0.08	ns	4.21	<0.01	2.55	ns
P5	0.01	ns	2.48	ns	1.79	ns
P6	0.36	ns	2.11	ns	0.52	ns
P7	2.43	ns	2.92	ns	0.00	ns
P8	2.07	ns	6.03	<0.01	0.60	ns
P9	14.67	<0.01	2.80	ns	6.07	<0.01
P10	5.40	<0.05	1.81	ns	1.32	ns

ANCOVA and Scheffé's tests obtained from the analyses of the overall stress intensity rating obtained during the pain period.

Source	Mean Square	df	F	p
Group	13.82	2	5.61	0.01
Covariate	13.87	1	5.63	0.02
Error	2.46	44		
Equality of slopes	1.89	2	0.70	0.47
Error	2.49	42		

Scheffé's tests.

Comparisons:	F	p
PO and P+PW	4.19	<0.05
PO and P+SI	5.21	<0.01
P+PW and P+SI	0.06	ns

Note:
[1]Covariate = SES.

APPENDIX H

Statistical analyses conducted with the psychophysiological data

ANCOVA conducted with the HR data obtained from all subjects using group means to fill missing data cells.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	1785.10	2	12.12	0.00	
Covariate	65688.41	1	446.02	0.00	
Error	147.28	44			
R	161.14	23	9.00	0.0	0.0
RG	70.64	46	3.95	0.0	0.00
Error	17.90	1035			

Note:

[1]Covariate= First five minutes of baseline HR.

Baseline polynomials conducted with the HR data obtained from all subjects.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	1.720	2	0.16	0.85	
Covariate[1]	15725.132	1	1436.91	0.00	
Error	10.944	44			
R (1)	4.538	1	0.82	0.37	
R (1) G	8.028	2	1.45	0.241	
Error	5.522	45			
R (2)	0.188	1	0.05	0.83	
R (2) G	4.044	2	0.98	0.38	
Error	4.116	45			
R (3)	13.067	1	1.90	0.18	
R (3) G	3.795	2	0.55	0.58	
Error	6.894	45			
R	5.931	3	1.08	0.36	0.36
RG	5.290	6	0.96	0.45	0.45
Error	5.511	135			

Note:

[1]Covariate= First five minutes of Baseline HR.

Pain period polynomials conducted with the HR data obtained from all subjects using groups means to fill missing data cells.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	3663.124	2	20.22	0.00	
Covariate[1]	19241.442	1	106.21	0.00	
Error	181.159	44			
R (1)	282.356	1	2.91	0.09	
R (1) G	270.307	2	2.79	0.07	
Error	97.023	45			
R (2)	108.821	1	6.75	0.01	
R (2) G	39.711	2	2.46	0.10	
Error	16.117	45			
R (3)	32.551	1	2.05	0.16	
R (3) G	42.282	2	2.67	0.08	
Error	15.860	45			
R (4)	0.161	1	0.01	0.90	
R (4) G	15.613	2	1.44	0.25	
Error	10.866	45			
R (5)	58.703	1	4.74	0.03	
R (5) G	2.183	2	0.18	0.84	
Error	12.377	45			
R (6)	15.092	1	2.76	0.10	
R (6)	1.634	2	0.30	0.74	
Error	5.460	45			
R (7)	27.254	1	5.10	0.03	
R (7) G	0.655	2	0.12	0.88	
Error	5.347	45			
R (8)	45.439	1	9.31	0.00	
R (8) G	7.293	2	1.49	0.24	
Error	4.882	45			
R (9)	11.542	1	3.02	0.09	
R (9) G	7.416	2	1.94	0.16	
Error	3.824	45			
R (10)	10.490	1	1.95	0.17	
R (10) G	8.746	2	1.62	0.21	
Error	5.385	45			
R	59.24	1	3.34	0.00	0.02
RG	39.584	2	2.23	0.00	0.04
Error	17.714	450			

Note:

[1] Covariate= First five minutes of baseline HR

Recovery polynomials conducted with the HR data obtained from all subjects.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	0.186	2	0.02	0.981	
Covariate[1]	35771.008	1	3560.70	0.00	
Error	10.046	44			
R(1)	18.050	1	1.19	0.28	
R(1)G	0.279	2	0.02	0.98	
Error	15.106	45			
R(2)	0.344	1	0.04	0.84	
R(2)G	17.406	2	2.06	0.14	
Error	8.44	45			
R(3)	2.128	1	0.58	0.45	
R(3)G	15.455	2	4.21	0.02	
Error	3.670	45			
R(4)	0.104	1	0.03	0.87	
R(4)G	2.705	2	0.66	0.521	
Error	4.091	45			
R(5)	0.040	1	0.01	0.93	
R(5)G	1.529	2	0.30	0.74	
Error	5.023	45			
R(6)	0.230	1	0.06	0.81	
R(6)G	4.167	2	1.03	0.37	
Error	4.057	45			
R(7)	0.063	1	0.02	0.90	
R(7)G	0.205	2	0.06	0.95	
Error	3.670	45			
R(8)	2.920	1	1.11	0.30	
R(8)G	3.899	2	1.48	0.24	
Error	2.633	45			
R	2.985	8	0.51	0.85	0.77
RG	5.705	16	0.98	0.48	0.46
Error	5.857	360			

Note:

[1]Covariate= First five minutes of Baseline HR.

Scheffé's tests conducted with the AP and pain minutes HR data obtained from all subjects.

Comparisons	PO and P+PW		PO and P+SI		P+ PW and P+S	
Minutes	F	p	F	p	F	p
AP	7.59	<0.01	3.79	<0.05	<0.65	ns
P1	12.03	<0.01	2.77	ns	3.26	<0.05
P2	12.70	<0.01	1.19	ns	6.10	<0.01
P3	14.81	<0.01	9.25	<0.01	0.65	ns
P4	15.93	<0.01	7.86	<0.01	1.41	ns
P5	21.24	<0.01	16.30	<0.01	0.33	ns
P6	18.69	<0.01	15.18	<0.01	0.18	ns
P7	25.36	<0.01	17.88	<0.01	0.65	ns
P8	35.30	<0.01	20.37	<0.01	2.03	ns
P9	38.15	<0.01	18.28	<0.01	3.61	<0.05
P10	43.61	<0.01	13.74	<0.01	8.40	<0.01

ANCOVA conducted with the HR obtained from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	268.35	2	1.61	0.23	
Covariate[1]	33121.69	1	198.27	0.00	
Error	167.05	19			
R	28.77	23	2.68	0.00	0.02
RG	9.75	46	0.91	0.65	0.54
Error	10.74	460			

Note:

[1] Covariate=First five minutes of Baseline HR.

Baseline polynomials obtained with the HR data from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	2.027	2	0.19	0.82	
Covariate	6395.789	1	600.27	0.00	
Error	10.654	19			
R (1)	2.316	1	0.27	0.60	
R (1) G	4.405	2	0.52	0.60	
Error	8.493	20			
R (2)	8.793	1	1.61	0.21	
R (2) G	1.825	2	0.33	0.71	
Error	5.453	20			
R (3)	0.386	1	0.06	0.81	
R (3) G	10.974	2	1.60	0.22	
Error	6.872	20			
R	3.831	3	0.55	0.64	0.64
RG	5.735	6	0.83	0.55	0.55
Error	6.939	60			

Note:

[1] Covariate= First five minutes of Baseline HR.

Pain period polynomials obtained with the HR data from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	406.508	2	2.77	0.09	
Covariate	12918.979	1	87.88	0.00	
Error	147.006	19			
R(1)	7.982	1	0.33	0.57	
R(1) G	26.293	2	1.08	0.35	
Error	24.347	20			
R(2)	72.848	1	15.13	0.00	
R(2) G	4.154	2	0.86	0.43	
Error	4.815	20			
R(3)	2.016	1	0.19	0.67	
R(3) G	13.267	2	1.26	0.30	
Error	10.506	20			
R(4)1	0.094	1	0.01	0.92	
R(4) G	2.190	2	0.23	0.80	
Error	9.718	20			
R(5)	45.885	1	4.00	0.05	
R(5) G	3.259	2	0.28	0.75	
Error	11.46	20			
R(6)	7.793	1	2.65	0.12	
R(6) G	2.818	2	0.96	0.40	
Error	2.945	20			
R(7)	16.167	1	3.45	0.08	
R(7) G	2.089	2	0.45	0.64	
Error	4.687	20			
R(8)1	15.887	1	5.32	0.03	
R(8) G	5.08	2	1.70	0.21	
Error	2.986	20			
R(9)	7.393	1	2.95	0.10	
R(9) G	1.309	2	0.52	0.60	
Error	2.509	20			
R(10)	5.346	1	1.40	0.25	
R(10) G	5.172	2	1.35	0.28	
Error	3.818	20			
R	18.141	10	2.33	0.01	0.04
RG	6.564	20	0.84	0.65	0.59
Error	7.779	200			

Note:

[1]Covariate= First five minutes of Baseline HR.

Recovery polynomials obtained with the HR data from subjects who did not report tolerance.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	1.168	2	0.14	0.86	
Covariate[1]	16144.051	1	1963.34	0.00	
Error	8.223	19			
R(1)	0.000	1	0.00	0.99	
R(1) G	2.330	2	0.20	0.81	
Error	11.585	20			
R(2)	0.011	1	0.00	0.96	
R(2) G	1.844	2	0.34	0.71	
Error	5.413	20			
R(3)	13.349	1	8.45	0.01	
R(3) G	4.255	2	2.70	0.09	
Error	1.579	20			
R(4)	0.710	1	0.22	0.64	
R(4) G	1.356	2	0.43	0.65	
Error	3.158	20			
R(5)	1.907	1	0.63	0.43	
R(5) G	4.233	2	1.39	0.27	
Error	3.036	20			
R(6)	2.408	1	0.62	0.44	
R(6) G	0.127	2	0.03	0.96	
Error	3.878	20			
R(7)	5.99	1	3.60	0.07	
R(7) G	0.568	2	0.34	0.71	
Error	1.66	20			
R(8)	4.527	1	1.85	0.18	
R(8) G	4.495	2	1.84	0.18	
Error	2.450	20			
R	3.613	8	0.88	0.53	0.51
RG	2.401	16	0.59	0.89	0.85
Error	4.095	160			

Note:

[1]Covariate = First five minutes of Baseline HR.

ANCOVA and Scheffé's tests performed using the overall mean HR during the pain period.

Source	Mean Square	df	F	p
Group	168.74	2	6.21	0.00
Covariate[1]	2677.24	1	98.59	0.00
Error	27.16	44		
Equality of slopes	63.10	2	2.46	0.10
Error	25.44	42		

Scheffé's tests

Comparisons	F	p
PO and P+PW	7.21	<0.01
PO and P+SI	0.59	ns
P+PW and P+SI	3.68	<0.05

Note:

[1]Covariate= First five minutes of Baseline HR.

ANCOVA conducted using the EMG data obtained from all subjects using group means to fill missing data cells.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	5156.91	2	3.19	0.05	
Covariate[1]	649671.74	1	401.43	0.00	
Error	1618.40	43			
R	304.67	23	1.64	0.03	0.1
RG	330.11	46	1.78	0.00	0.05
Error	185.79	1012			

Note: [1]Covariate= First five minutes of Baseline EMG.

Baseline polynomials obtained from the EMG data using from all subjects.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	41.858	2	1.19	0.31	
Covariate	110330.337	1	3143.51	0.00	
Error	35.097	43			
R(1)	54.627	1	0.79	0.38	
R (1) G	43.090	2	0.62	0.54	
Error	69.546	44			
R (2)	0.269	1	0.00	0.95	
R (2) G	10.675	2	0.13	0.88	
Error	83.965	44			
R (3)	86.054	1	0.95	0.33	
R (3) G	35.617	2	0.40	0.67	
Error	90.167	44			
R	46.983	3	0.58	0.63	0.59
RG	29.794	6	0.37	0.89	0.86
Error	81.226	132			

Note:

! Covariate=First five minutes of Baseline EMG.

Pain period polynomials obtained from the EMG data using data from all subjects.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	1928.974	2	0.90	0.41	
Covariate[1]	158796.108	1	74.35	0.00	
Error	2135.749	43			
R(1)	168.788	1	0.19	0.66	
R(1)G	2507.299	2	2.87	0.07	
Error	872.565	44			
R (2)	83.805	1	0.39	0.53	
R(2)G	255.206	2	1.19	0.31	
Error	214.554	44			
R(3)	287.093	1	1.31	0.25	
R (3) G	125.133	2	0.57	0.56	
Error	218.851	44			
R(4)	2041.935	1	15.45	0.00	
R(4) G	429.984	2	3.25	0.04	
Error	132.99	44			
R (5)	200.554	1	1.21	0.28	
R (5) G	200.554	2	1.21	0.28	
Error	166.156	44			
R(6)	307.258	1	4.92	0.03	
R(6)G	422.270	2	6.76	0.00	
Error	62.479	44			
R(7)	2.638	1	0.04	0.84	
R(7)G	145.641	2	2.15	0.12	
Error	67.689	44			
R(8)	238.033	1	4.40	0.04	
R (8) G	93.737	2	1.73	0.18	
Error	54.140	44			
R (9)	47.886	1	0.67	0.41	
R (9) G	171.445	2	2.39	0.10	
Error	71.632	44			
R (10)	402.342	1	4.96	0.03	
R (10) G	45.116	2	0.56	0.57	
Error	81.139	44			
R	378.083	10	1.95	0.04	0.12
RG	467.134	20	2.41	0.00	0.03
Error	194.150	440			

Note:

[1]Coveriate= First five minutes of Baseline EMG.

Recovery polynomials obtained from the EMG data using data from all subjects.

Source	Mean Square	df	F	p	Huynh Feldt p
Group	7313.134	2	6.77	0.00	
Covariate[1]	63264.258	1	243.82	0.00	
Error	1079.750	43			
R(1)	3.525	1	0.02	0.90	
R(1)G	29.632	2	0.13	0.87	
Error	221.117	44			
R(2)	29.826	1	0.27	0.60	
R(2)G	32.379	2	0.29	0.75	
Error	111.937	44			
R(3)	5.686	1	0.06	0.81	
R(3)G	4.35	2	0.04	0.95	
Error	99.316	44			
R(4)	8.984	1	0.14	0.71	
R(4)G	36.897	2	0.57	0.57	
Error	65.282	44			
R(5)	159.84	1	3.30	0.08	
R(5)G	3.142	2	0.06	0.93	
Error	48.398	44			
R(6)	73.10	1	1.15	0.29	
R(6)G	47.11	2	0.74	0.48	
Error	63.725	44			
R(7)	0.715	1	0.01	0.90	
R(7)G	5.84	2	0.11	0.89	
Error	52.495	44			
R(8)	15.201	1	0.29	0.59	
R(8)G	35.323	2	0.68	0.51	
Error	52.095	44			
R	37.110	8	0.42	0.91	0.85
RG	24.335	16	0.27	0.99	0.99
Error	89.289	352			

Note:

[1]Covariate= First five minutes of Baseline EMG.

Scheffe's tests comparing groups during the AP and pain minutes, and comparing groups on overall means during the recovery period using the EMG data obtained from all subjects.

Comparisons	PO and P+PW		PO and P+SI		P+PW and P+SI	
Minutes	F	p	F	p	F	p
AP	6.83	<0.01	1.60	ns	1.82	ns
P1	1.75	ns	0.16	ns	2.01	ns
P2	0.32	ns	0.12	ns	1.45	ns
P3	3.52	<0.05	0.84	ns	0.93	ns
P4	8.11	<0.01	0.07	ns	6.67	<0.01
P5	5.09	<0.05	0.01	ns	5.58	<0.01
P6	0.00	ns	0.12	ns	0.11	ns
P7	0.01	ns	0.07	ns	0.13	ns
P8	0.46	ns	0.03	ns	0.21	ns
P9	0.07	ns	0.16	ns	0.02	ns
P10	1.13	ns	0.07	ns	0.66	ns

Recovery period

Comparisons	PO and P+PW		PO and P+SI		P+PW and P+SI	
Overall Mean	F	p	F	p	F	p
	2.07	ns	2.68	ns	9.42	<0.01

ANCOVA obtained from analysis of EMG data using subjects who did not report tolerance.

Source	Mean Square	df	E	p	Huynh Feldt p
Group	446.60	2	2.63	.10	
Covariate[1]	333669.61	1	196.42	00	
Error	1698.78	19			
R	213.12	23	1.81	0.01	0.06
RG	117.61	46	1 00	0.48	0.47
Error	117 76	460			

Note.

[1]Covariate= First five minutes of Baseline EMG

ANCOVA and Scheffé's tests obtained from the analyses of overall mean EMG during the pain period.

Source	Mean Square	df	F	p
Group	792.86	2	3.05	0.06
Covariate[1]	24025.12	1	92.36	0.00
Error	260.12	43		
Equality of slopes	135.84	2	0.5106	0.60
Error	266.04	42		

Scheffé's tests:

Comparisons:	F	P
PO and P+PW	5.2	<0.05
PO and P+SI	0.0	ns
P+PW and P+SI	5.2	<0.05

Note:

[1]Covariate-First five minutes of baseline EMG

APPENDIX I

Analyses conducted with the Behavorial Response data

ANCOVAS from analyses of the overall frequency of behavioral response data.

First pain minute.

Source	Mean Square	df	F	p
Group	8.88	2	0 99	0 38
Covariate[1]	78 8	1	8 79	0 01
Error	8 98	39		

Last pain minute

Source	Mean Square	df	F	p
Group	8 22	2	1 13	0 33
Covariate[1]	9 91	1	1 37	0 25
Error	7 26	39		

Note.

[1]Covariate=baseline behavioral response data.

ANCOVA and Scheffé's test from the analyses of the grimace expression data.

First Pain minute:

Source	Mean Square	df	F	p
Group	1.01	2	3.54	0.0388
Covariate[1]	1.13	1	3.98	0.0531
Error	0.28	39		
Equality of slopes	0.03	2	0.05	0.9497
Error	11.09	37		

Last Pain minute:

Source	Mean Square	df	F	p
Group	0.13	2	0.44	0.6453
Covariate[1]	0.00	1	0.00	0.9395
Error	0.29	39		
Equality of slopes	0.28	2	0.96	0.3924
Error	0.30	37		

Scheffé's tests of first pain minute

Comparisons	F	p
PO and P+PW	3.54	<0.05
PO and P+SI	1.10	ns
P+PW and P+SI	0.71	ns

Note:

[1]Covariate = Baseline grimace data.

APPENDIX J

ANCOVA table obtained from the analyses of the postexperimental indirect pain questionnaires.

Representativeness

Source	Mean Square	df	F	p
Group	2 21	2	0 39	0 68
SES	13 51	1	2.41	0.13
Error	5 60	44		

Stress pre-pain

Source	Mean Square	df	F	p
Group	5 16	2	1.19	0 31
SES	0 97	1	0·22	0 64
Error	4 33	44		

Stress-post

Source	Mean Square	df	F	p
Group	5 6	2	1 41	0 25
SES	3 57	1	0 87	0 36
Error	4 12	44		

Belief

Source	Mean Square	df	F	p
Group	1.13	2	0.87	0 43
SES	0 38	1	0 29	0 59
Error	1 30	44		

State - Trait anxiety Inventory - State measures

Source	Mean Square	df	F	p
Group	170 59	2	2 00	0 15
SES	95 93	1	1 13	0 29
Error	85.19	44		

Effectiveness of coping strategies

Source	Mean Square	df	F	p
Group	0 04	2	0 04	0 96
SES	0.01	1	0.01	0.94
Error	0.96	33		

The mean adjusted (for SES) values obtained from the postexperimental indirect pain questionnaires

Measure	Rep[1]	Belief	Str-Post[2]	Str-pre[3]	STAI-S[4]
Group					
PO					
M	4.6	3.3	2.1	3.0	68.8
SD	2.9	1.0	2.1	1.8	7.4
P+PW					
M	3.9	3.9	2.5	4.1	68.3
SD	2.2	1.1	1.8	2.4	9.2
P+SI					
M	4.1	3.6	3.3	3.2	62.4
SD	2.0	1.2	2.1	1.9	10.8
Mean					
M	4.2	3.6	2.6	3.5	66.5
SD	2.4	1.3	2.0	2.1	9.3

Notes.

[1]Rep = representativeness

[2]Str-Post = Stress during postquestionnaire period

[3]Str-pre = Stress prior to nociception

[4]STAI-S = State-Trait Anxiety Inventory - State measures

APPENDIX K

Comparisons between subjects who did and subjects who did not report tolerance.

Student's *t* tests and chi-square statistics calculated using the demographic and preexperimental questionnaire data obtained from the RT and NT groups.

Measure	*t* or χ^2	df	p
Age	*t* = -1.62	46	ns
SES	*t* = 2.18	46	0.04
Height/ Weight	*t* = 1.00	46	ns
Season	χ^2 = 0.03	3	ns
Citizenship	χ^2 = 0.03	1	ns
Religion	χ^2 = 12.69	4	0.02
TMAS[1]	*t* =-1.44	46	ns
SDS[2]	*t* = 0.20	46	ns
GTAR-EA[3]	*t* =-2.12	46	0.04
GTAR-PD[4]	*t* = 0.71	46	ns
STAI-S[5]	*t* = 1.25	46	ns
Stress	*t* = 0.07	46	ns

Notes:

[1]TMAS = Taylor Manifest Anxiety Scale
[2]SDS = Social Desirability Scale
[3]GTAR-EA = General Trait Anxiety Revised - Evaluation Anxiety
[4]GTAR-PD = General Trait Anxiety Revised - Physical Danger
[5]STAI-S = State-Trait Anxiety Inventory - State Measures

ANOVA from transducer data comparing RT and NT subjects and group values for each pain minute.

Source	Mean Square	df	F	p	Huynh Feldt p
Mean	1	1852068170.19	28890.29	0.00	
Group	1	152358.09	2.38	0.13	
Error	46	64106.95			
R	9	13077.04	4.48	0.00	0.01
RD	9	4047.53	1.39	0.19	0.25
Error	414	2921.42			

Group values:

Group	RT	NT	Mean
Minute:	Mean (SD)	Mean (SD)	Mean
P1	1913 (82)	1935 (82)	1923
P2	1955 (70)	1966 (85)	1960
P3	1956 (85)	1978 (78)	1966
P4	1974 (70)	1989 (79)	1981
P5	1950 (78)	1991 (94)	1970
P6	1954 (78)	2000 (90)	1975
P7	1958 (72)	1993 (82)	1975
P8	1944 (83)	1997 (85)	1970
P9	1920 (90)	1991 (84)	1954
P10	1954 (71)	1994 (87)	1973
Mean	1948	1983	1965

Student's t Statistics calculated from the threshold and endurance data obtained from RT + NT groups

Measure	t	df	p
Threshold	3.66	46	<0.001
Endurance	3.47	46	<0.002

The mean stress intensity (SI), HR, and EMG values obtained during the AP and Pain (P) periods; and the mean frequency of grimaces during the first and last minute of the Pain period obtained from the RT and NT groups.

Group	RT		NT	
Measure	Mean	(SD)	Mean	(SD)
AP SI	3.0	(0.8)	2.3	(1.3)
P SI	3.8	(1.8)	3.6	(1.6)
AP HR	72	(11.1)	71	(11.1)
P HR	72	(10.5)	71	(10.5)
AP EMG	81	(31.8)	72	(28.5)
P EMG	72	(28.3)	71	(28.4)
Grimaces - First	0.4	(0.6)	0.4	(0.6)
Grimaces - Last	0.3	(0.6)	0.4	(0.5)

Student's t statistics calculated on mean AP stress intensity ratings, mean pain and stress intensity ratings during the pain period, and mean baseline HR and EMG obtained from the RT and NT groups.

Measure	t	df	p
Ap stress intensity	-0.39	46	ns
Pain intensity	-0.45	46	ns
Pain stress intensity	-0.82	46	ns
Baseline HR	0.93	46	ns
Baseline EMG	-0.47	45	ns

ANCOVAS obtained from RT and NT mean HR and EMG data from the AP and pain periods and grimace data the first and last pain period minute.

APHR:

Source:	Mean Square	df	F	p
Group	11.39	1	0.42	0.52
Covariate[1]	4461.80	1	165.45	0.00
Error	26.97	45		
Equality of Slopes	53.66	1	2.04	0.16
Error	26.36	44		

APEMG:

Source	Mean Square	df	F	p
Group	1033.83	1	3.06	0.09
Covariate[2]	26990.63	1	79.82	0.00
Error	338.14	44		
Equality of Slopes	0.28	1	0.97	
Error	345.82	49		

Pain HR:

Source	Mean Square	df	F	p
Group	10.21	1	0.30	0.58
Covariate[1]	3543.09	1	104.75	0.00
Error	33.83	45		
Equality of Slopes	0.86	1	0.02	0.87
Error	34.57	44		

ANCOVAS (continued)

Pain EMG:

Source	Mean Square	df	F	p
Group	60.48	1	0.21	0.65
Covariate[2]	23963.50	1	83.14	0.00
Error	288.23	44		
Equality of Slopes	318.26	1	1.11	0.29
Error	287.55	43		

Grim 1:

Source	Mean Square	df	F	p
Group	0.04	1	0.11	0.73
Covariate[3]	1.06	1	3.25	0.07
Error	0.33	40		
Equality of Slopes	0.56	1	1.74	0.19
Error	0.32	39		

Grim 2:

Group	Mean Square	df	F	p
Source	0.10	1	0.34	0.56
Covariate[3]	0.00	1	0.00	0.94
Error	0.29	40		
Equality of Slopes	0.03	1	0.10	0.75
Error	0.30	39		

Notes

[1]Covariate = first five minutes of Baseline HR

[2]Covariate=First minutes of Baseline EMG

[3]Covariate= Baseline Grimace data

Student's t statistics calculated from the post experimental questionnaire data obtained from RT and NT groups.

Measure	t	df	p
Gracely scales:			
Intensity	-1.62	46	ns
Unpleasantness	-0.83	46	ns
Pain	-2.36	46	<0.03
McGill Pain Questionnaire:			
Pain Rating Index-Sensory	0.01	46	ns
Pain Rating Index-Affective	0.38	46	ns
Pain Rating Index-Miscellaneous	0.15	46	ns
Pain Rating Index-Evaluative	0.83	46	ns
Pain Rating Index-Total	0.19	46	ns
Representativeness	0.39	46	ns
Belief	0.58	46	ns
Stress prepain	-1.87	46	<0.07
Stress postpain	1.09	46	ns
State-Trait Anxiety Inventory-State	0.26	46	ns
Effectiveness	-2.51	35	<0.02

Mean values obtained from the postexperimental questionnaires from the RT and NT groups

Group	RT		NT	
Measure	Mean	(SD)	Mean	(SD)
G-Int	9.3	(1.4)	8.3	(2.6)
G-Unpl	5.1	(2.9)	4.5	(2.1)
G-Pain	7.2	(2.5)	5.3	(3.1)
PRI-S	13.2	(6.4)	13.2	(6.2)
PRI-A	1.1	(2.0)	1.5	(2.0)
PRI-M	4.4	(2.7)	4.5	(2.9)
PRI-E	1.7	(1.4)	2.1	(1.7)
PRI-T	20.5	(9.7)	21.1	(10.8)
Rep	4.1	(2.2)	4.3	(2.6)
Belief	3.7	(1.0)	3.4	(1.2)
Stress-pre	4.0	(2.0)	2.9	(2.1)
Stress	2.3	(1.7)	3.0	(2.4)
STAI-S	66.2	(8.6)	66.9	(10.1)
Effectiveness	3.6	(1.1)	4.4	(0.7)

Key

G-Int = Gracely intensity scale
G-Unpl = Gracely unpleasantness scale
G-Pain = Gracely painfulness scale
PRI-S = Sensory scale of the MPQ
PRI-A = Affective scale of the MPQ
PRI-M = Miscellaneous scale of the MPQ
PRI-E = Evaluative scale of the MPQ
PRI-T = Sum of all MPQ scales
Rep = representativeness
STAI-S = State-Trait Anxiety Inventory - State measures

Correlation coefficients calculated between SES and the pain and stress measures that differed between RT and NT groups

Measure	SES	df	p
GTAI-EA[1]	-.25	46	ns
Threshold	.06	46	ns
Endurance	.18	46	ns
Gracely - Pain scale	-.10	46	ns
Effectiveness	.22	46	ns

Note.

[1]GTAI-EA = Inventory of General Trait Anxiety - Evaluation Anxiety subtest

APPENDIX L

Correlations between dependent measures

Correlations between the direct pain measures

Measures	Thresh	Tol	End	PPI	G-Int	G-Unpl	G-Pain	PRI-S
Thres	1.00	.40*	-.34*	-.43*	-.31*	-.10	-.38*	-.10
Tol		1.00	.73*	.08	.28	-.18	-.16	-.10
End			1.00	.41	-0.60	.32*	.12	.02
PPI				1.00	.49*	.30*	.46*	.00
G-Int					1.00	1.00	.58*	.30*
G-Unpl							.26	.03
G-Pain							1.00	.18
PRI-S								1.00
PRI-A								
PRI-M								
PRI-E								
PRI-T								
RT								

	PRI-A	PRI-M	PRI-E	PRI-T	RT
Thres	.01	-.21	-.21	-.14	.48*
Tol	-.10	-.05	-.10	-.08	.79*
End	-.10	.11	.20	.03	.46*
PPI	.12	.05	.37*	.09	-.07
G-Int	.29*	.29*	.47*	.40*	-.23
G-Unpl	.13	.18	.45*	.16	-.12
G-Pain	.00	.05	.33*	.17	-.33*
PRI-S	.63*	.55*	.29*	.93*	.00
PRI-A	1.00	.44*	.17	.73*	.02
PRI-M		1.00	.43*	.76*	.02
PRI-E			1.00	.48*	.12
PRI-T				1.00	.03
RT					1.00

*p<.05

KEY

Thres= Threshold
Tol= Tolerance
End= Endurance
PPI= Average pain intensity during the pain period
G-Int= Gracely Intensity Scale
G-Unpl= Gracely Unpleasantness Scale
G-Pain= Gracely Pain Scale
PRI=Pain rating index from the McGill Pain Questionnaire
PRI-S PRI -sensory scale
PRI-A=PRI-affective scale
PRI-M=PRI-miscellaneous scale
PRI-E=PRI-evaluative scale
PRI-T=PRI-sum of all scales
RT= Report of tolerance (1=yes; 2=no)

Correlations between anxiety preexperimental questionnaires and the direct pain measures

Pain Measures	Thres	Tol	End	PPI	G-Int
Anxiety Questionnaire					
TMAS	-.15	-.11	.00	.23	.21
SDS	.01	.05	.06	.18	.18
GTAR-EA	.07	-.14	-.19	-.04	.00
GTAR-PD	.10	.12	.04	.03	-.02
STAI-S	.05	.13	.10	-.23	-.21
Present Stress	-.09	.05	.12	.29	.23

	G-Unpl	G-Pain	PRI-S	PRI-A	PRI-M	PRI-E	PRI-T	RT
TMAS	.37*	.30*	.31*	.07	.23	.30*	.31*	.21
SDS	.26	.10	.18	.14	.27	.16	.19	.07
GTAR-EA	.38*	.28	-.05	-.13	.01	.13	-.03	-.29*
GTAR-PD	.39*	.02	-.19	.01	.02	.28	-.08	.11
STAI-S	-.24	-.25	-.09	-.10	-.13	-.27	-.15	.17
Present Stress	.17	.24	.26	.13	.35*	.39*	.34*	.01

KEY

TMAS= Taylor manifest Anxiety Scale *p< .05
SDS= Social Desirability Scale
GTAR-EA= General Trait Anxiety Revised Scale - Evaluation anxiety
GTAR-PD= General trait Anxiety Revised Scale - Physical Danger
STAI- S= State - Trait Anxiety Inventory - State

See key on preceding page for pain measures.

Correlations between the indirect pain measures obtained during the pain period and the direct pain measures.

Pain measures	Thres	Tol	End	PPI	G-Int	G-Unpl	G-Pain
Indirect measures							
PSI	-.23	-.23	-.07	.53*	.51*	.35*	.29*
PHR	-.02	.14	.16	.16	-.04	.15	.03
PEMG	-.18	-.08	-.01	-.05	-.07	-.23	.16
Grim-1	-.06	-.12	-.08	.07	.24	-.12	-.12
Grim-2	.22	.04	-.12	.02	.14	-.13	-.09

	PRI-S	PRI-A	PRI-M	PRI-E	PRI-T	RT
PSI	.15	.30*	.35*	.52*	.33*	-.12
PHR	.00	.20	.27	.07	.12	.06
PEMG	.18	-.10	.10	.07	.11	-.09
Grim-1	.11	.06	.30*	.35*	.22	.11
Prim-2	-.18	.07	-.02	-.06	-.14	.07

*p< 05

KEY

PSI= Average Stress Intensity rating obtained during the pain period
PHR= Average HR (in bpm) obtained during the pain period
PEMG= Average EMG (in uV) obtained during the pain period
Grim-1= Frequency of grimaces during the first minute of nociception
Grim-2= Frequency of grimaces during the second minute of nociception

See Key two pages back for pain measures.