# THE EFFECTS OF VIDEOTAPED MODELING AND IMAGINED ANALGESIA ON ACUTE PAIN: A SIGNAL DETECTION ANALYSIS

by

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Dissertation submitted to the Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of
DOCTOR OF PHILOSOPHY
in
Psychology

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July, 1982

Blacksburg, Virginia

Acknowledgements

I would like to express my appreciation to my chairman, George Clum, for his support, advice, encouragement, and respect. Our friendship will be one of the better memories of my graduate career. I would also like to thank:

My other committee members, Dick Winett, Marshall Tessnear, Connie Schmidt, and Chris Peterson for the advice and assistance they provided.

, for the assistance his previous work gave me in completing this project.

for helping me maintain my sanity while completing my dissertation.

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## Introduction

The reduction of pain by psychological techniques has been investigated by many authors, and these studies indicated that a variety of interventions can be effective in attenuating pain. One group of studies has shown that modeling can be used to manipulate effectively pain responding in the laboratory. The work of Craig and his associates (Craig & Coren, 1975; Craig & Neidermayer, 1974; Craig & Weiss, 1971; Craig & Weiss, 1972; Craig, Best, & Ward, 1975) has demonstrated that exposure to a tolerant model will significantly increase a person's tolerance of painful stimulation, while exposure to an intolerant model will significantly decrease a person's tolerance. There is also evidence (Craig & Coren, 1975; Craig, Best, & Ward, 1975) that exposure to an intolerant model will result in an increased physical sensitivity to pain.

Another promising type of psychological intervention for reducing pain is the suggestion of analgesia. Several studies have found that short verbal suggestions for analgesia presented with or without hypnosis are effective in reducing reports of pain (Barber & Hahn, 1962; Chaves & Barber, 1974; Evans & Paul, 1970; Hilgard, Ruch, Lange, Lenox, Morgan, & Sachs, 1974; Hilgard, Cooper, Lenox, & Voevodsky, 1967; Johnson, 1974; McGlashan, Evans, & Orne, 1969; Spanos, Barber, & Lange, 1974; Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979; Hall, 1977). The study proposed here will examine the effect of pain responding of a treatment combining modeling and the suggestion of analgesia and of treatments involving exposure to tolerant and intolerant models. In the literature review that follows, the reader

will first be introduced to the various theories of pain and to the problematic task of measuring pain responding. Then the research concerning the use of modeling and suggestion in manipulating pain responding will be reviewed in detail.

## Review of Literature

### Theories of Pain

Aristotle believed that pain was an increase in the sensitivity of touch which was conveyed by the blood to the heart, where pain was experienced. He believed that pain was a negative emotion and was the eptiome of unpleasantness (Bonica, 1980). This conception remained the prevalent theory of pain until the Renaissance. The conception of pain as an increase in the responsivity of a sense, however, endured much longer.

The modern scientific study of pain really began with the emergence of physiology as an experimental science in the first half of the 1800s (Bonica, 1980). There are four theories of pain that have or are at present enjoying noteriety in the field of pain research. One of the theories is specificity theory. This theory hypothesizes that pain is a specific sensation with its own specific sensory receptors. This theory was espoused by Descartes, but was not specifically formulated until 1858 when Schiff published studies which proved that touch and pain were independent sensations. In the late 1890s von Frey extended these studies by beginning to map out pain and touch receptors through histological examination of the skin. On the basis of his work, he deduced that there were four major cutaneous sense modalities: touch, warmth, cold, and pain. Subsequent researchers espoused the position that if pain is a separate modality then it must have its own receptors and nervous pathways that service these receptors (Bonica, 1980). Present variants of specificity theory state that A-delta and C neural

fibers are specifically associated with two types of pain. The A-delta fibers respond to short latency pricking pain, while the C fibers respond to long latency burning pain. The afferent impulses of either of these fibers are necessary and sufficient to evoke painful sensations. It is felt that the pricking pain (A-delta) impulses enter the dorsal spinal cord and ascend to the somatic sensory areas of the cerebral cortex via the anterolateral system and the thalamic centers. The burning pain (C) impulses are thought to follow the same route, but to different centers in the thalamus and cerebral cortex. The exact nature and location of these thalamic and cortical pain centers have not been specified (Weisenberg, 1977; Mountcastle, 1974).

An implicit assumption of specificity theory is that when a pain-specific receptor is stimulated it will result in the perception of pain. There are research and clinical findings, however, that run counter to this assumption. Beecher (1956) demonstrated that people can experience little or no pain despite extensive tissue damage. Of 215 men seriously wounded on the battlefield only twenty-five percent wanted a narcotic for analgesia while over eighty percent of a comparable civilian group with similar surgical wounds wanted narcotic analgesics. Beecher attributed the difference in the two groups to the significance of the wound to the person. In wartime the wound meant a ticket to safety, while to the civilian the surgery meant disaster. If specificity theory were correct then the wounded soldiers should be experiencing as much pain as the civilians since their pain-specific receptors were being stimulated similarly. Clinical pain phenomena such as phantom limb pain also can not be accounted for by specificity theory. If

pain arises through the stimulation of pain-specific receptors how can a person experience severe pain in a limb that has been amputated?

A second theory of pain is what is referred to as pattern or intensity theory. This theory proposes that every sensory stimulus is capable of producing pain if it reaches sufficient intensity. This theory was espoused in the 1840s by Weber and was later supported by Wundt, Külpe, Titchner, and Goldscheider. In 1894 Goldscheider published the first fully developed variant of this theory, which identified stimulus intensity and build up of stimulation through central summation as the critical determinates of pain (Bonica, 1980). Later theorists included spatial summation in the theory, so in more recent variants the theory proposes that pain is a result of the summation of spatial and temporal patterns of sensory input (Weisenberg, 1977; Crue & Carregal, 1975). Pattern theory runs counter to specificity theory in that it suggests that there are no specific receptors or neural fibers for pain. Despite this difference it also can not account for the findings of Beecher (1956). Presumably Beecher's subjects received stimulation of similar intensity and patterning and as a result pattern theory would predict no difference between civilians and soldiers.

The third notable modern theory of pain is a biochemical theory. Lindahl (1974 a & 1974 b) suggests that pain is caused by an elevation of pH or hydrogen ion concentration in a nerve or in the vicinity of a nerve or nerve ending. It has been found that pus from septic abcesses is acidic, whereas pus from painless tuberculoes has a neutral pH. The tissues in and around gastric ulcers, painful hematomas, fractures, and

painful malignant tumors have been found to have an acidic pH. In addition the tissue in an area experiencing ischemic pain has been found to have an acidic pH. This biochemical theory appears promising but has not yet been tested. Like pattern and specificity theories it can not account for Beecher's (1956) findings. If Beecher's subjects' wounds were similar then one would expect that the pH levels in the nerves and surrounding tissues should not have drastically differed. Thus Lindhal's theory would predict no difference in pain between civilians and soldiers.

The fourth major modern pain theory is the gate control theory of Melzack and Wall (1965, 1970). Gate control theory seeks to account for both physiological and psychological influences on pain phenomena by postulating that there is a neural gate in the spinal cord through which all pain sensations must pass. This gating mechanism involves three components (Wall, 1980). The first is a transfer mechanism. Specifically there must be a system to transfer painful stimulation from the peripheral afferents to the spinal cells that project to the brain. This mechanism is in the substantia gelatinosa and is referred to as the spinal gate. The gate modulates the afferent impulses of the A-delta, A-beta, and C nerve fibers. The second component is a system by which the peripheral afferents can influence the transfer mechanism. Along this line Melzack and Wall postulate that the afferent activity of the A-beta fibers close the spinal gate preventing transmission of painful stimulation up the spinal cord. The afferent activity of the A-delta and C fibers are thought to open the gate and the impulses are transmitted up the spinal cord. The third component is a system by

which cognitive processes can influence the gating mechanism. This is accounted for by the postulation of decending neural pathways from cortical centers to the gating mechanism.

Gate control theory goes beyond previous pain theories and attempts to delineate the factors that influence an individual's response to painful stimulation. Melzack & Casey (1968) hypothesize three dimensions which influence responding to painful stimuli. The first dimension is sensory-discriminative. Neural systems which project to the neospinothalamic area are thought to determine the sensory-discriminative aspects of painful phenomena. The second dimension is motivational-affective. Neural systems which activate the reticular and limbic systems of the brain are thought to control the motivational drive and unpleasant affect which accompanies painful stimulation. The third dimension is cognitive-evaluative. The cortical areas are thought to control evaluation of painful phenomena and exert influence over both the motivational-affective and sensory-discriminative dimensions.

Figure 1 depicts how both the gating mechanism and the three dimensions of pain responding fit together in the gate control conceptualization of pain. Unlike the specificity, pattern, and biochemical theories of pain, the gate control theory tries to incorporate psychological variables in its explanation of pain. As can been seen in Figure 1, "the gate control theory proposes that cognitive activities such as anxiety, attention, and suggestion can influence pain by acting at the earliest levels of sensory transmission (Melzack, 1973, p. 199)." In addition, these cognitive activities can affect both the

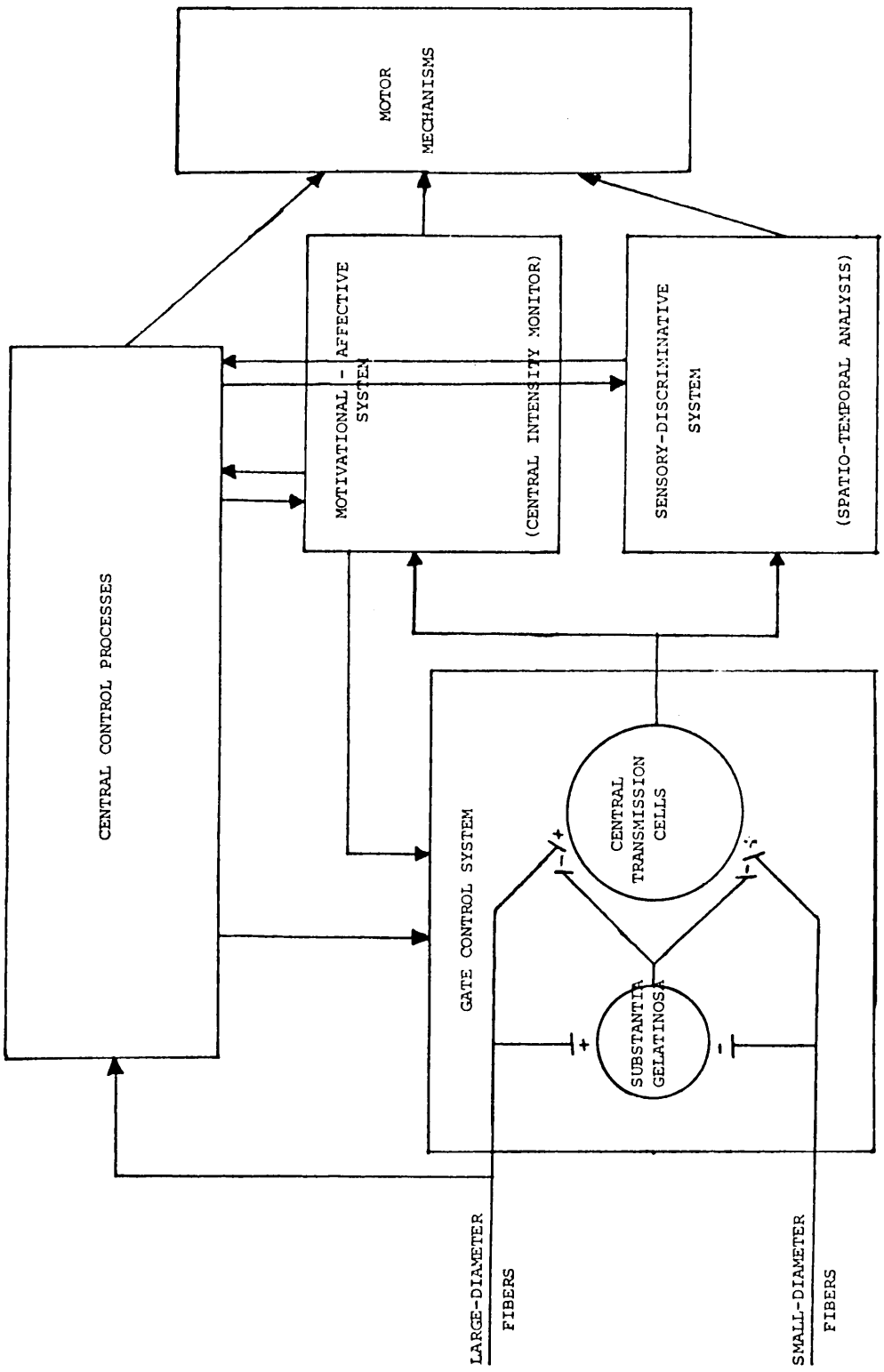


Figure 1. Gate-Control Model of Pain (Adapted from Melzack and Casey (1968) and Melzack and Wall (1965)).

motivational-affective and sensory-discriminative dimensions. As a result it can account for both the findings of Beecher (1956) and clinical states such as phantom limb pain.

The gate control theory is not without its detractors (e.g., Iggo, 1972). Most of them argue about the accuracy of the neural pathways specified by Melzack and Wall. Regardless of the accuracy of their specifics, Melzack and Wall's theory has been the most influential current theory of pain. Its attempt to tie together psychological and physiological variables in pain perception have greatly influenced pain research and clinical treatment of pain.

## Measurement of Pain

The measurement of pain is a difficult but important task (Weisenberg, 1977). Whether one is a researcher or a clinician, it is vital to be able to describe, quantify, and localize the pain that a person is feeling. This task is made difficult by the complex nature of pain phenomena. There are three broad types of pain measurement: physiological, behavioral observation, and self-report. While there is undoubtedly overlap in the information each approach provides, they each deal with a different category of responses made by an individual who is experiencing pain.

The physiological measurements of pain responses used by researchers have varied. They include blood pressure (Hilgard, 1969), GSR (Riley & Richter, 1975), heart rate (Lenox, 1970), skin temperature (Shapiro & Surwit, 1976), and EMG (Epstein & Abel, 1977). Barber and Hahn (1962) examined forehead EMG, respiratory irregularity, heart rate,

and GSR as physiological pain indicies in a cold pressor task. These authors found that subjects with hypnotically-suggested analgesia and subjects with wakening-imagined analgesia showed reduced muscle tension and respiratory irregularity when compared to a control group. The treatment groups showed no difference in heart rate or GSR. In addition, the hypnotically-suggested analgesia and the wakening-imagined analgesia groups reported experiencing less pain than the control group. Victor, Mainardi, and Shapiro (1978) examined the relationship between heart rate and subjective report of pain in a cold pressor task. They found that the higher the subject's heart rate the greater the pain he/she reported. Hilgard, Morgan, Lange, Lenox, McDonald, Marshall, and Sachs (1974) also found a positive correlation between heart rate and subjective verbal report of pain.

There are, however, problems with physiological assessment of pain responding. Lacy and Lacy (1970) have shown that physiological responses can show "response stereotypy." Specifically each individual responds to a given stimulus event in an idiosyncratic manner. So a painful stimulus may produce large changes in GSR for one subject whereas the same stimulus may produce large changes in heart rate for another subject. In addition, Hilgard et al (1974) suggest that these individual differences are not necessarily related to the amount of subjectively reported pain.

The second approach to measurement of pain is behavioral observation. Several authors (e.g., Fordyce, Fowler, & Delateur, 1968; Sternbach, 1968) have suggested that a major component of a person's response to painful stimulus is overt publicly observable pain behavior

such as moaning, crying, grimacing, verbal complaints, distorted gait, and taking analgesics. In behavioral observation techniques, these behaviors are usually measured via observational ratings made by family or medical personnel. These behavioral observation techniques are "first generation" methods and are not without problems. At present the biggest problem is lack of demonstrated inter- and intra-observer reliability. There has been a tendency among researchers to neglect to demonstrate accuracy and reliability. Those authors who do examine reliability show positive results, but unfortunately the majority of authors do not examine this (Saunders, 1979).

The third approach to the measurement of pain is to use the subject's own self-report as the basis of quantifying and localizing the pain. This is by far the approach most often used in both the laboratory and the clinic. The methods typically used include threshold and tolerance measurements, ratings on Likert-type scales, indicating on a ten-centimeter line the length of line equal to the intensity of pain felt, cross-modality matching tasks (e.g., squeezing a hand dynamometer to indicate the intensity of pain), and adjective checklists.

A promising instrument utilizing the self-report approach is the McGill Pain Questionnaire (Melzack & Torgerson, 1971). Consistent with the gate control theory of pain, they developed a self-report scale which appears to differentiate between a sensory component and an affective component on the basis of verbal descriptions of pain qualities. Statistical analyses of McGill Pain Questionnaire responses from patients suffering from one of eight pain syndromes

indicated that each type of pain syndrome appears to be characterized by a distinctive constellation of verbal descriptors. Data from studies of the McGill Pain Questionnaire indicate that there are "appreciable and quantifiable differences in the way various types of pain are described (Melzack, 1980, p. 144)."

The work of Melzack and his associates both with the McGill Pain Questionnaire and on the gate control theory of pain indicate that it is vital to understand what part or parts of the pain phenomenon have been tapped by your measurement instrument. All three approaches to pain measurement can tap more than one component of the subject's pain experience. This fact has particular significance for researchers involved in the experimental analysis of pain in the laboratory. The most commonly used measurement method for such studies has involved determination of threshold and tolerance levels for pain. Pain threshold is defined as, "that point at which pain is perceived 50% of the time (Wolff, 1980, p. 177)." Pain tolerance is defined as, "that point at which the individual will withdraw from or terminate noxious stimulation (Wolff, 1980, p. 178)." The pain threshold level is considered to be a measurement of the sensory component of pain, whereas the pain tolerance level is thought to reflect psychological (i.e., affective, cognitive, and motivational) aspects of pain.

In a pain treatment study, changes in pain threshold level from preintervention to postintervention are thought to indicate changes in the sensory component of the subject's pain experience. If the pre-post threshold change is an increase then it is thought that the treatment worked by making the subject less sensitive to the pain

stimulus. Conversely, if the pre-post change is a decrease in pain threshold level it is thought that the treatment had caused the subject to become more sensitive to the pain stimulus. Changes in pain tolerance level are viewed similarly. A decrease in tolerance from pre- to post-intervention could be thought of as reflecting a change in attitude or anxiety which resulted in the subject lowering his/her criterion for what constitutes a very painful stimulus. An increase in tolerance from pre- to post-intervention could be interpreted as reflecting a change in attitude or anxiety which resulted in the subject raising his/her criterion for what constitutes a very painful stimulus. There is research which suggests, however, that the conceptualization of pain tolerance and pain threshold levels is not as clearcut as outlined above.

Blitz and Dinnerstein (1968) evaluated the effects of different instruction on pain threshold and "quit point." One group of subjects received instructions which were intended to raise their pain threshold. A second group received instructions which were intended to increase their pain tolerance level. A third group received no specific instructions. The pain stimulus was electric shock. The authors found that the threshold instructions increased both the pain threshold level and the quit point, while the instructions to increase tolerance raised the quit point. The authors concluded that it was not clear whether the increase in threshold level was due to a shift in the subject's criterion for what constituted pain. They stated, "one cannot conclude from the elevated pain threshold that Ss were less sensitive to painful noxious stimulation; in this sense the

elevation in pain threshold may be interpreted as response bias (Blitz & Dinnerstein, 1968, p. 278)." Blitz and Dinnerstein's findings cast doubt on the conceptualization of pain threshold as a "pure" sensory measure and pain tolerance level as a "pure" psychological measure. These findings point to the unreliability of the traditional fifty percent threshold measure. Using this method, a low number of reports of pain indicates a high threshold by definition and a high number of pain reports indicates a low threshold. Traditional threshold analysis assumes that both high and low thresholds reflect sensory factors, but it is equally likely that the subject's response criterion has been biased by nonphysiological factors (Lloyd and Appel, 1976).

In recent years, an alternative pain measurement procedure has been developed. This procedure involves the application of signal detection theory to the analysis of pain responding. This method allows one to separate changes in response bias from changes in sensory discrimination. More specifically, two kinds of performance measures are obtained, perceptual capability and criterion location. The first reflects the ability of the subject to perceive the stimulation or to differentiate one level of stimulation from another. The second measure reflects the willingness of the subject to classify his/her verbal reports in various ways (Chapman, 1980).

"The theory assumes that a given stimulus leads to a sensory experience x, which can be scaled, rated, or ranked. Given an infinite number of presentations of a "noise" stimulus, there results a (normal) distribution of sensory experience events on the continuum. Infinite presentations of a second stimulus consisting of noise plus

signal leads to a (normal) distribution on the same continuum but further along, since the stimulus is of higher intensity (Lloyd and Appel, 1976, p. 80)." Signal, in this case, refers to the "target" stimulus in pure form while noise refers to any background or additional stimulation other than the target. A subject is typically presented with numerous trials consisting of noise alone or noise plus signal and asked to decide whether or not a signal is present on each trial. On any one trial a subject can make one of our categories of response. If the subject correctly judges that the signal is present the response is a "hit." If the subject correctly judges that no signal is present then the response is a "correct rejection." If the subject incorrectly judges that a signal is present when it is not, then the response is a "false alarm." If the subject incorrectly judges that a signal is not present when in fact it is present, the response is a "miss."

Figure 2 shows distributions of signal and noise from a hypothetical signal detection analysis. d' is a measure of the individual's ability to discriminate between two levels of a stimulus (in this case signal and noise). d' is considered to be a relatively pure measure of discriminability which remains unaltered when variables such as attitude, expectation, and motivation are manipulated (Clark, 1974). The definition of d' is the difference of the means of the two distributions divided by the standard deviation of the noise distribution. If one assumes normal distributions with equal variance, d' can be defined as the difference between the z scores of the hit and false alarm probabilities. The probabilities of both hits and false alarms

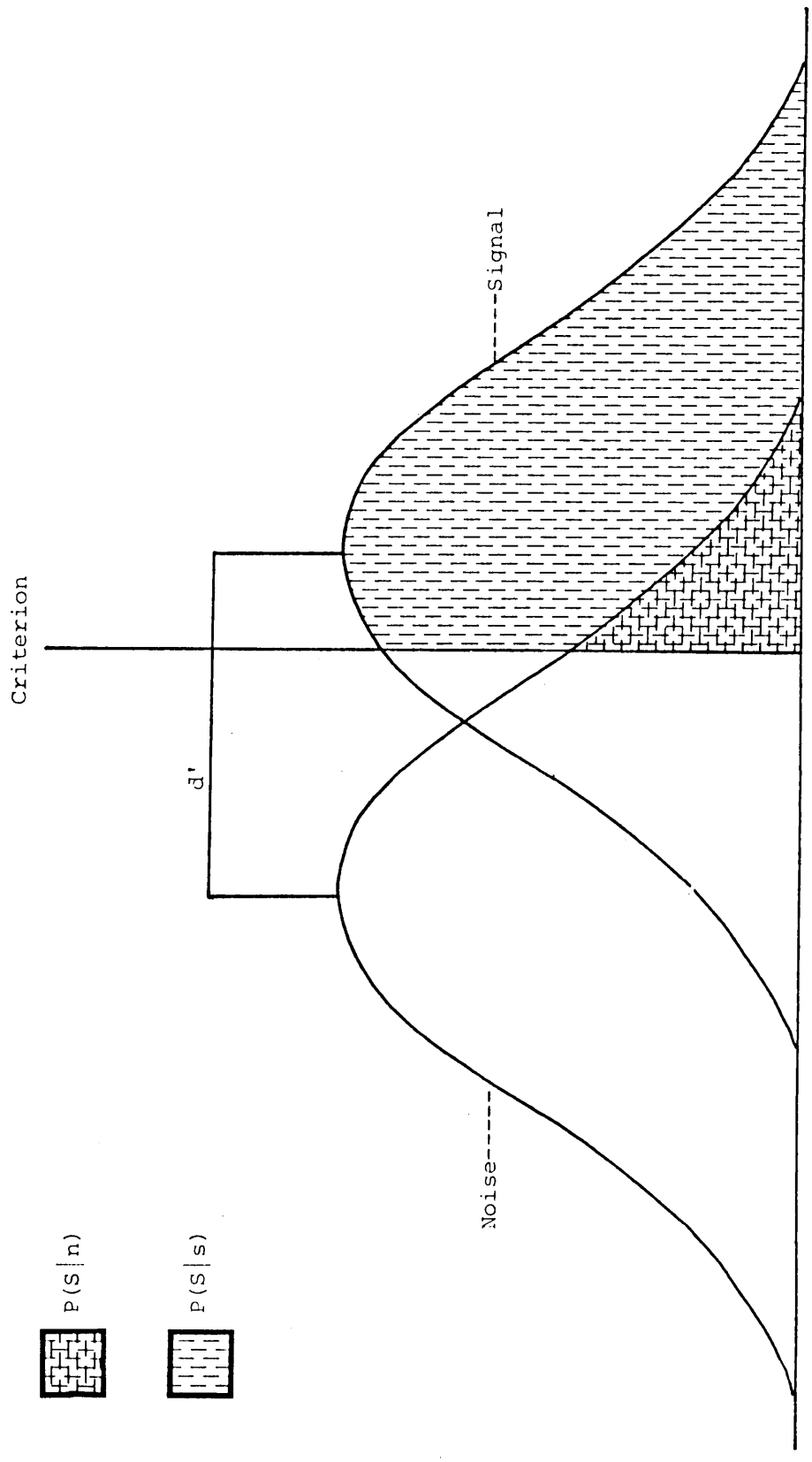


Figure 2. Sample signal and noise distributions with a criterion designated P(S|s) and P(S|n).

can be computed by dividing the rate of each by the number of signal presentations or the number of noise presentations, respectively. A low d' means that the subject tended to confuse stimuli of different intensities. This typically happens when either the physical intensities of the stimuli are close together or when the subject's sensory system is insensitive (Clark, 1974).

The subject's response bias is represented by his/her criterion for responding that a signal is present or absent. This criterion can fall anywhere on the distributions. It is defined as a likelihood ratio (Lx). Lx is the ratio of the ordinate of the signal plus noise distribution to the ordinate of the noise distribution as defined by the conditional probabilities of hits and false alarms (Clark, 1974). Since signal detection theory assumes normal distributions with equal variance, then p/n and p/fa can be found in a normal curve probability table and the ordinate of the signal plus noise distribution can be divided by the ordinate of the noise distribution (Lloyd and Appel, 1976). A basic tenet of signal detection theory is that the likelihood ratio criterion is independent of changes in values of stimulus intensity or the subject's physical sensitivity (Clark, 1974). If the stimulus on a given trial falls to the right of the criterion point, the subject will report that a signal has occurred. If the stimulus falls to the left of the criterion point, the subject will report that a signal has not occurred. If the subject shifts his/her criterion to the right, he/she is more biased toward reporting that a signal did not occure. Conversely, if the subject shifts his/her criterion to the left, he/she is biased toward reporting that a signal

did occur. Since Lx is independent of d', these shifts in Lx could occur while d' remained constant, and d' could change while Lx remained constant (Lloyd and Appel, 1976).

In a typical signal detection study more than one set of hit and false alarm rates are usually obtained for a given experimental manipulation. With data from many sets of hit and false alarm rates a graph can be drawn in which the conditional probabilities of hits are plotted against the conditional probabilities of false alarms. This is referred to as a receiver operating characteristic (ROC) curve (see Figure 3). The points on the graph give an index of response bias because they represent a range of criteria from very strict to very lenient, the criteria becoming more lenient as the points move upward and to the right (Lloyd and Appel, 1976).

The ROC curve can provide an alternative measure of sensory sensitivity, P(A). This is a nonparametric estimate of d' which is the proportion of the area under the ROC curve (McNicol, 1972). This measure is of interest because there are times when the assumptions of normal distribution and equal variances will not be met. The classical signal detection theory procedures are predicated on the assumption that the ROC curve would have a slope of one when the hits and false alarms were scaled in "normal deviate units." It is also assumed that all data points would fall on the single line of the ROC curve. When these assumptions can not be met it is advisable to use P(A) and a nonparametric index of response bias since they do not require such assumptions (Chapman, 1980).

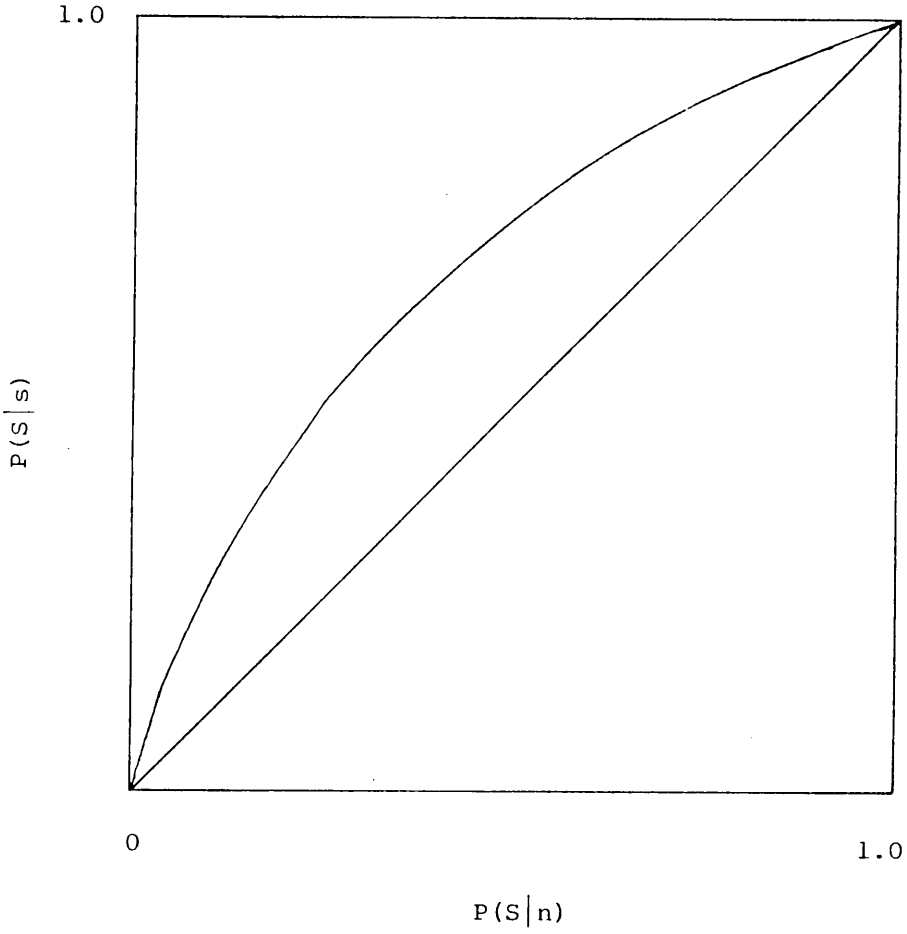


Figure 3. Example of an ROC curve plotting HIT by FALSE ALARMS.

Modeling and the Manipulation of Pain Responses

There is considerable clinical and experimental evidence to indicate that an individual's pain response can be manipulated through various psychological interventions (see Weisenberg, 1977; Saunders, 1979; Turk, 1978). One psychological approach to manipulating pain responses is through social-learning. Studies of modeling and vicarious reinforcement processes indicate that report of pain can be manipulated through exposure to variably tolerant models. A model is any stimulus array organized such that an observer can extract and act on the important information conveyed by environmental events without needing to first perform overtly (Rosenthal and Bandura, 1978). It encompasses a wide range of modes and content formats, and is considered by some to be the form of most learning by humans (Bandura, 1969; Rosenthal, 1976).

Craig and Weiss (1971) had subjects rate the intensity of electric shocks on a five point scale ranging from undetectable to painful after observing the rating of a confederate model. The shocks were given in .5 milliampere increments beginning with 0 milliamperes. There were three treatment groups. In one treatment the model consistently rated the shocks as more intense and painful than the subject (intolerant model). In the second treatment the model consistently rated the shocks as less intense and painful than the subject (tolerant model). In the third treatment the model "responded in a manner that would represent a typical, uninfluenced, naive subject (p.55)" (control group). The subjects exposed to the tolerant model rated as painful shocks with a mean intensity of 8.65 milliamperes,

the control subjects rated as painful shocks with a mean intensity of 6.35 milliamperes, and the subjects exposed to the intolerant model rated as painful shocks with a mean intensity of 2.50 milliamperes. The pain criterion of the subjects exposed to the intolerant model was seventy percent less than the subjects exposed to the tolerant model and thirty-five percent less than the control subjects. In addition, the control subjects' pain criterion was significantly less than that of the subjects exposed to the tolerant model.

Craig and Weiss (1972) further examined the influence of a model over subjects' pain ratings. In this study, subjects were repeatedly exposed to a nonaversive shock. One group of subjects was exposed to a confederate model who rated the shocks as increasingly painful, while a second group of subjects was given the shocks with no model present. The subjects exposed to the model consistently rated the shocks as painful while the control subjects did not.

Craig and Neidermayer (1974) examined autonomic correlates of pain perceptions of subjects whose pain responses were manipulated through exposure to a confederate model. The authors took skin conductance and heart rate measures of subjects who rated the painfulness of shocks either a) in the presence of a tolerant model; b) in the presence of an intolerant model; c) in the presence of a noncontingently responding confederate; or d) with no confederate present. The subjects' pain criterion levels replicated the findings of the 1971 Craig and Weiss study. The four treatment groups did not differ on the autonomic measures. Considering the autonomic measures as indicies of subjective discomfort, the authors concluded that the

subjects exposed to the tolerant model were induced into tolerating high levels of shock without experiencing any more discomfort than the subjects exposed to the intolerant model or even to no model. There is, however, the possibility that the autonomic measures taken were not discriminative enough or were sufficiently variable to mask group differences.

Craig, Best, and Ward (1975) examined the influence of modeling on the psychophysiological power functions describing the relationship between the subject's subjective judgment of pain and the intensity of the shock experienced. Subjects were asked to rate the painfulness of electric shocks in the presence of a tolerant model, an intolerant model, or no model. As in the studies above, subjects exposed to the tolerant model accepted significantly stronger shocks without expressing pain than subjects exposed to an intolerant model or no model, and the control group accepted significantly greater shocks than the intolerant model group. Hilgard (1969) found that magnitude estimations of experimentally induced pain grew as a power function of a stimulus intensity:

$$\psi = \alpha \varnothing^{n}$$

Where:

$\varnothing$ = the physical stimulus value

$\psi$ = the quantitative estimate of intensity

$n$ = exponent

$\alpha$ = the unit of the scale the subject uses

Analysis of the power functions in this study indicated that exposure to modeling influenced the unit of the scale. More specifically, the

subjects exposed to the tolerant model used smaller units than both the control and intolerant model groups, while the intolerant model group used units larger than those used by the other two groups. Because the unit serves as a multiplicative constant for any given increment in stimulus intensity, the corresponding increment in response magnitude is determined by the size of the unit of scale. A smaller unit would mean a proportionately small response magnitude all along the power function. The smaller unit used by the group exposed to the tolerant model meant that a stronger stimulus would be needed to generate a particular response than would be needed to generate the same response in the group exposed to the intolerant model. The authors interpreted these results as providing evidence that modeling affects the perception of pain via "central processing systems."

Craig and Coren (1974) applied a signal detection analysis to the effects of modeling on pain. Subjects were asked to rate the painfulness of electric shocks in the presence of a tolerant model, an intolerant model, or a model responding noncontingently. The results indicated a significant difference between the treatment groups as to the shock level which elicited a "painful" response. The subjects exposed to the tolerant model had the highest, the control group was intermediate, and the subjects exposed to the intolerant model had the lowest shock level. The signal detection analysis indicated that the subjects exposed to the intolerant model manifested greater sensitivity to pain than the control or tolerant model groups. The authors concluded that the sensory component of pain can be influenced by modeling. They state:

> If you tell a patient that the ongoing stimulation is painful, via the presence of an intolerant model, you may actually be increasing his vulnerability to the sensory experience of pain. If you tell him that the ongoing stimulation is not painful you do not affect the actual internal sensory experience, but rather reduce his willingness to report his distress (p. 111).

Unfortunately their conclusion of a response bias shift in the subjects exposed to a tolerant model can not be supported. Response bias was not analyzed in this study because subjects received different shock levels sccording to their initial response patterns in the presence of the model.

Neufeld and Davidson (1971) compared the effects of modeling and detailed preparatory information on pain tolerance of radiant heat. One group of subjects observed a model undergo sixty seconds of radiant heat stimulation "in a serene manner with no overt signs of stress." A second group of subjects observed a model "serenely" undergo sixty seconds of pressure algometer stimulation. A third group of subjects was given a detailed verbal description of the radiant heat procedure and of the sensations associated with the radiant heat stimulus. A fourth group of subjects was given a detailed verbal description of a pressure algometer procedure and of the sensations associated with pressure algometer stimulation. A fifth group was simply asked to wait for fifteen minutes before beginning the experiment. After receiving their treatment, each subject's pain tolerance to radiant heat stimulation was measured twice. The results indicated that there was no difference between relevant modeling and relevant preparatory information in increasing tolerance. There was, however,

a difference in relevance versus irrelevance. Subjects receiving irrelevant modeling or preparatory information had significantly lower tolerance on the second heat stimulus exposure than subjects receiving relevant modeling or information. There was not any difference among these groups on the first exposure to the heat stimulus. The authors suggest that the initial exposure to the pain stimulus made the subjects receiving the irrelevent modeling or information aware that their information was irrelevant and that this awareness increased their anxiety resulting in a decreased tolerance upon a second exposure to the pain stimulus.

Not all modeled studies have had positive results. Chaves and Barber (1974) found that modeling was effective in reducing pain only for those subjects with high pretest levels of stress reaction to pressure stimulation. Subjects experienced two minutes of pressure stimulation via the Forgione-Barber device, then for one-half of the subjects the experimenter modeled one of four treatments with his own finger in the device for two minutes and the other half of the subjects received verbal explanations of one of four treatments. After receiving their treatment the subjects experienced two more minutes of stimulation using the treatment given to them. The four treatments were: imagining pleasant scenes, imagining insensitivity to pain in the finger, expectancy control, and no treatment control. The authors postulated that the modeling may not have been as effective as in other studies because the model was an older male and the subjects were all female undergraduates. This is very plausible since research has shown that models who are similar to the observer and/or appear to

be realistic reference figures to compare with oneself are most effective (Kazdin, 1974; Kornhaber & Schroeder, 1975; Thellan, Dollinger, & Roberts, 1975; Brown, Brown, & Danielson, 1975; Festinger, 1954; Kanfer, Karoly, & Newman, 1974).

There is some clinical evidence for the effectiveness of modeling to manipulate pain responses. Vernon (1974) exposed hospitalized children to one of two modeling films. One film depicted children receiving injections without experiencing pain or expressing negative emotion. The other film depicted children receiving injections and expressing moderate negative emotions and experiencing slight pain. The subjects' reactions to later injections were rated by hospital personnel. Those children exposed to models who experienced no pain experienced more pain than the children exposed to models experiencing moderate pain. On the surface the results of this study seem to contradict the modeling literature discussed above. It is likely, however, that relevance of modeling is a key issue in this study. One group was indeed exposed to a tolerant model, but the model was unrealistically tolerant and therefore not providing relevant information or modeling to the child.

Overall, the literature indicates that exposure to a modeling treatment can have profound influence on a person's pain responding. It appears that one can manipulate a subject's pain response in both a positive and negative fashion with modeling. The precise mechanism is not clear but the evidence suggests that it influences both sensory and cognitive-motivational components.

Suggestion of Analgesia and the Manipulation of Pain Responding

Suggestion can be an effective way to manipulate pain responding. As discussed above, Blitz & Dinnerstein (1968) found that instructions designed to increase both pain threshold and tolerance indeed can increase both measures. One promising type of suggestion is that of analgesia. More specifically, giving the subject a set of instructions which direct him/her to imagine that his/her arm is insensitive to pain appears to be an effective way to manipulate pain responding.

Johnson (1974) compared three different suggestions to no treatment in the effects on cold pressor pain. The suggestions were relaxation instructions, instructions to imagine warmth in the hand with relaxation, and instructions to imagine numbness in the hand with relaxation. The relaxation instructions and the relaxation plus suggested numbness instructions significantly decreased reported pain relative to the control group, while instructions for relaxation and imagined warmth did not have a significant effect.

Barber and Hahn (1962) compared hypnotically-suggested analgesia to a suggestion of analgesia given to awake subjects and to no treatment. The noxious stimulus was a cold pressor task. The hypnotically-suggested subjects were given a standardized twenty-minute hypnotic induction procedure followed by the suggestion of numbness and insensitivity in their hands. The waking-suggested analgesia group was asked to imagine that their hand was relaxed, comfortable, and warm. The subjects were given their treatment and then asked to immerse their hand in near freezing water for three minutes. The authors found that the waking-suggested analgesia was as effective as hypnotically-suggested

analgesia in reducing subjective reports of pain, and both groups reported less than the control group.

Stacher, Schuster, Bauer, Lahoda, and Schultze (1975) also compared hypnotically-suggested analgesia to a suggestion of analgesia given to awake subjects. The noxious stimulus in this study was electric shock delivered to the ear lobe. Whether hypnotized or awake, subjects received the same suggestion of analgesia. The subjects were told that they would feel the shocks but they would not feel pain from them. Stacher et al found that both suggestions significantly increased tolerance for electric shock but hypnotically-suggested analgesia was more effective than waking-suggested analgesia.

In another comparison of hypnotically-suggested versus waking-suggested analgesia, Spanos, Barber, and Long (1974) gave one group of subjects a suggestion of anesthesia while awake, while another group was given the same suggestion after hypnotic induction. A third group was given the hypnotic induction with no suggestion of analgesia and a fourth group was given neither the hypnotic induction nor the analgesia suggestion. The analgesia suggestion was to imagine "that your hand is numb, insensitive, and like a piece of rubber (p. 148)." The painful stimulus in this study was induced via the Forgione-Barber pain stimulator. The suggestion of analgesia was found to produce a significant reduction in subjective pain intensity reports. There was no difference in reported pain between the group receiving the suggestion while awake and the hypnotically-suggested group.

Hall (1977) also compared hypnotically-suggested analgesia to waking-suggested analgesia, and to no treatment. The noxious stimulus

was radiant heat. Hall used a signal detection paradigm. The analysis of d' (sensory discriminability) found that there was a significant difference between the mean d's of both the hypnotically-suggested analgesia group and the waking-suggested analgesia group and that of the no treatment controls. This indicates that the analgesia suggestion, given either awake or under hypnosis, altered the physical sensitivity of the subjects, causing them to feel less pain. No group differences in response bias were found. Hall concluded that "these results...imply that the only condition required for the production of analgesia is a set of instructions which direct the subject to imagine that his arm is numb or insensitive (p. 123)."

Spanos, Radtke-Bodorik, Ferguson, and Jones (1979) examined the effects of four different treatments on subjective reports of pain during a cold pressor task. The four treatments were: hypnotic induction, hypnotic induction plus the suggestion of analgesia, the suggestion of analgesia while awake, and no treatment. The suggestion of analgesia was that the subject's hand would become numb and insensitive. The results indicated that the hypnotic induction plus analgesia suggestion and the waking-suggested analgesia groups reported significantly less pain than the hypnotic induction alone and control groups. There was no difference between the waking-suggested analgesia and hypnotically-suggested analgesia groups in reported pain.

As discussed earlier, Chaves and Barber (1974) also examined waking-suggested analgesia. They compared instructions to imagine pleasant scenes, experimenter modeled imagining pleasant scenes, instructions to imagine that their finger had become numb and insensitive,

experimenter modeled imagining analgesia, expectancy control, experimenter modeled expectancy control, no treatment control, and experimenter modeled no treatment control. Subjects were pretested with two minutes of painful stimulation via the Forgione-Barber device, given their treatment, and then post-tested with two more minutes of painful stimulation. Imagining pleasant scenes and imagining analgesia, whether experimenter modeled or not, significantly reduced subjective reported pain relative to the expectancy control and no treatment control groups. In addition, the expectancy control groups reported significantly less pain than the no treatment groups.

The studies examining suggested analgesia indicate that suggesting to a subject that he/she should imagine a part of his/her body becoming numb and insensitive to pain, whether in a hypnotic state or not, is an effective way to attenuate the amount of pain that is reported. Hall's (1977) study suggests that this pain reduction is due to a reduction in physical sensitivity to pain and not the result of a shift in response bias.

Rationale and Hypotheses

As reviewed above, research evidence indicates that exposure to modeling and the suggestion of imagined analgesia can manipulate the subsequent pain responding of subjects. Exposure to variably tolerant models can effect both the sensory and the cognitive-motivational components of pain responding, while imagined analgesia appears to effect primarily the sensory component of pain responding. This has implications for the treatment of pain in the clinical setting. There are, however, several unanswered questions concerning these two intervention

modes. One such question is, "What is the effect of exposure to variably tolerant models on response bias?" Craig and Coren (1975) did not analyze response bias data and therefore their conclusion of a response bias shift in subjects exposed to a tolerant model is unsubstantiated. Another unanswered question is, "Will instructing a subject in an effective psychological intervention via modeling increase the effectiveness of that intervention?" It seems reasonable that since modeling is a potent manipulator of pain responding it could increase the effectiveness of other interventions. Chaves and Barber (1974) did examine modeling a treatment relative to instructing the subject in a treatment and found that the addition of modeling did not result in a treatment that was more effective than simply instructing the subject verbally. As discussed above, however, the modeling used by Chaves and Barber was not appropriate because the model was a middle-aged male and the subjects were female undergraduates. A test of the combination using more appropriate modeling appears to be in order.

This study examined the effects of exposure to various videotaped modeling treatments using a signal detection analysis. Specifically, six different treatment conditions were examined: a) Exposure to a videotape of a tolerant model; b) exposure to a videotape of an intolerant model; c) exposure to a videotape of a model who is imagining analgesia in her arm coupled with instructions to the subject to imagine that her arm is insensitive to pain; d) instructions to the subject to imagine that her arm is insensitive to pain; 3) an expectancy control in which the subject is told to expect a decrease in the amount of experienced pain; and f) a no treatment control.

As noted earlier, the work of Craig and Coren (1975) and Hall (1977) indicates that exposure to modeling or imagined analgesia results in a change in physical sensitivity to pain with no accompanying change in response bias. Therefore a signal detection paradigm was used to allow examination of the effects of the treatments on both response bias and physical sensitivity to pain.

Several hypotheses were investigated. They are:

1. Subjects exposed to an intolerant model would show an increase in sensitivity to pain (d'), when compared to the control groups.
2. Subjects exposed to a model using imagined analgesia will show a decrease in sensitivity to pain (d'), when compared to the control groups.
3. Subjects instructed to imagine analgesia will show a decrease in sensitivity to pain (d'), when compared to the control groups.
4. Subjects exposed to a tolerant model will show an increase in response bias to a higher criterion for judging a stimulus as painful.
5. Subjects exposed to an intolerant model, modeled imagined analgesia, imagined analgesia instructions, expectancy control, or no treatment will show no change in response bias.

# METHOD

## Subjects

A total of 68 females participated in this study. They were all volunteers from the students enrolled in an introductory psychology course at Virginia Tech. Each subject received course credit for their participation in the experiment. Of the 68 subjects who participated, 60 completed all facets of the experiment. The remaining 8 subjects did not complete the study for various reasons. Two subjects were dropped from the study due to equipment malfunctions. Five subjects failed to return for the second testing session for unknown reasons. One subject withdrew from the study due to illness. The subjects were randomly assigned to one of six treatment conditions such that each treatment group was comprised of 10 subjects.

## Measures

Three different measures were used in this study: a) The trait portion of the State-Trait Anxiety Inventory (Spielberger, Gersuch, & Luschene, 1970), b) the Present Affect Response Questionnaire (Endler, 1976), and c) a scale for rating the intensity of the radiant heat stimuli presentations.

### State-Trait Anxiety Inventory

The trait portion of the State-Trait Anxiety Inventory is a self-report measure of relatively stable individual differences in anxiety proneness. It is, therefore, considered to measure that anxiety which is stable over time and not apt to change as a function of situational stresses. The scale consists of twenty items which the subject is

asked to respond to in such a way as to indicate how she generally feels. The subject responds to each item using a four-point scale: 1 = almost never, 2 = sometimes, 3 = often, 4 = almost always (see Appendix A for the complete scale).

Present Affect Response Questionnaire

The Present Affect Response Questionnaire is a self-report measure of state anxiety. State anxiety refers to the complex emotional reaction that occurs in individuals once they interpret specific situations as personally threatening (Lamb, 1968). It is composed of twenty-four items which the subject is asked to respond to indicating how she feels "at this particular moment." The subject responds to each item using a five-point scale with 1 = not at all and 5 = very much so (see Appendix A for the complete scale).

Rating Scale

Each presentation of a radiant heat stimulus was rated by the subject using a seven-point scale: 0 = nothing, 1 = something, 2 = warm, 3 = hot, 4 = faint pain, 5 = painful, and 6 = very painful. If a subject pulled, rotated, or pivoted her arm away from the radiant heat source during a stimulus presentation it was scored as a withdrawal. (See Appendix B for the specific criteria for scoring a withdrawal response.) In tabulating and analyzing the data, a withdrawal was given a score of 7.

Apparatus

The painful stimulus in this study was radiant heat. The apparatus used to present radiant heat stimuli was very similar to the Hardy-Wolff-Godell dolormeter (Clark, 1969). It consisted of two major parts,

a heat source and a switching board. The heat source was an oblong metal box with a small aperture at one end. Inside the box was a movable spotlight and an electromagnetically operated shutter. The spotlight was aimed so as to send a beam of light through the aperture when the shutter was open. Five different radiant heat intensities were used in the experiment: 0 mcal/sec$^{-1}$/cm$^{-2}$, 98 mcal/sec$^{-1}$/cm$^{-2}$, 155 mcal/sec$^{-1}$/cm$^{-2}$, 215 mcal/sec$^{-1}$/cm$^{-2}$, 256 mcal/sec$^{-1}$/cm$^{-2}$. These stimulus intensities were calibrated before and after each testing session to assure consistency. The switching board was designed so that the presentation of the stimuli was automatic once the experiment began a series of trials. Each heat stimulus was automatically presented for three seconds and followed by an interstimulus interval of fifteen seconds. Each of the five stimulus intensities was presented for a total of thirty times, with the order of presentation randomized. Therefore, each session consisted of one hundred fifty separate presentations of a radiant heat stimulus.

Treatments

Six different treatment conditions were used in this study. They were: tolerant modeling, intolerant modeling, modeled imagined analgesia, verbal instructions for imagined analgesia, expectancy control, and no treatment. The individuals in the tolerant modeling group saw a five-minute videotape which showed a model performing twenty trials of the signal detection task. The model was female and looked, dressed, and acted like a typical undergraduate student. The model gave consistently low ratings of stimulus intensity (no higher than 4) and showed no overt

signs of distress or anxiety. In addition, the model made several positive self-statements aloud (e.g., "This isn't so bad.").

The subjects in the intolerant modeling group saw a five-minute videotape which showed a model performing twenty trials of the signal detection task. The model was female and looked, dressed, and acted like a typical undergraduate student. In the intolerant modeling tape, the model gave consistently high ratings of stimulus intensity (no lower than 4) and showed overt signs of mild distress and uneasiness.

The subjects in the imagined analgesia modeling group saw an eight-minute videotape which showed a model instructed how to imagine numbness and insensitivity in her arm and then performed twenty trials of the stimulus rating rask giving predominantly lower ratings of stimulus intensity (nothing higher than 4). Then the subjects were instructed, in the same manner as the model, how to imagine numbness in their own arms (see Appendix E).

The subjects in the imagined analgesia instructions group listened to a three-minute audiotape which instructed them to imagine their arm to be numb and insensitive (see Appendix F). The subjects in the expectancy control group listened to a short audiotape which instructed them that they would feel fess pain because prior exposure to radiant heat stimuli had desensitized them to further pain (see Appendix G). The subjects in the no treatment control group received no treatment or specialized instructions.

Procedure

The experimental procedure consisted of three phases: a) baseline, b) treatment, and c) post-treatment testing. Prior to the baseline phase, the experimenter went over the informed consent form with the subject and answered any questions she might have. Upon receiving consent, India ink was applied to the volar surface of the subject's right arm in six circular patches approximately 2.5 cm in diameter. Then the subject was asked to listen to an audiotape, via headphones, which explained the experiment and the task to her (see Appendix C). After the tape was finished the experimenter answered any additional questions. Then the baseline phase began.

Baseline Phase

The subject was asked to answer the STAI-trait and the PARQ questionnaires. The experimenter read each item to the subject and recorded her answers on a separate form. After completing the anxiety questionnaires, the experimenter went over the rating scale with the subject once again and reminded her that this was not a test of pain endurance but rather of her ability to discriminate between heat intensities. The subject was also reminded that she was free to withdraw her arm from the apparatus if she found a stimulus too painful.

An opaque cloth curtain was placed between the subject and the apparatus and she began a series of twenty practice trials. The stimuli were presented for three-second durations with an interstimulus interval of fifteen seconds. Immediately following a stimulus presentation a tone sounded signifying that the subject should rate the stimulus intensity. Each time a stimulus was presented the subject held her arm

against the heat source box so that an inked spot was over the aperture. The inked spot was alternated so that any one spot was stimulated only once every 108 seconds. The twenty practice trials included all levels of stimuli which were used during the experiment.

After the practice trials were completed the rating scale was again reviewed with the subject. Then the one hundred fifty experimental trials began. After forty, eighty, and one hundred twenty trials, two-minute breaks were taken. Upon completion of trial one hundred fifty, the baseline phase was completed and an appointment was made for two days later to complete the treatment and post-treatment testing phases.

Treatment Phase

The treatment phase took place in the first portion of the second experimental session. Before beginning the treatment, India ink was applied to each subject's right arm in the manner described above. After the ink was applied each subject received the treatment for the group to which she was assigned.

Tolerant Modeling

Each subject in this group first listened to an audiotape which gave a rationale for the modeling treatment (see Appendix D) and then watched the tolerant modeling videotape.

Intolerant Modeling

Each subject in this group listened to an audiotape which gave a rationale for the modeling treatment (Appendix D) and then watched the intolerant modeling videotape.

Modeled Imagined Analgesia

Each subject in this group listened to an audiotape which gave a rationale for the imagined analgesia treatment (see Appendix E) and then watched the modeled imagined analgesia videotape. After the videotape, each subject listened to an audiotape which instructed them how to imagine that their arm was numb.

Imagined Analgesia

Each subject in this condition listened to an audiotape which gave a rationale for the imagined analgesia treatment and then instructed them how to imagine that their arm was numb (see Appendix F).

Expectancy Control

Each subject in this condition listened to an audiotape which gave a rationale for why the subjects should feel less pain during the post-treatment stimulus rating task (see Appendix G).

No Treatment Control

No treatment or specialized instructions were given.

Post-Treatment Phase

After completion of the treatment phase each subject listened to an audiotape which reviewed the instructions for the stimulus rating task. The subject was then asked to complete the STAI-trait and PARQ. The experimenter read each item to the subject and recorded her answers on a separate form. After completion of the anxiety questionnaires the experimenter reviewed the rating scale with the subject and reminded her that this was not a test of pain endurance but rather of her ability to discriminate between heat intensities. The subject was also reminded that she was free to withdraw her arm from the apparatus if a stimulus

became too painful. Before beginning the practice trials, the experimenter gave the subject a verbal reminder about the effects and/or utilization of her treatment (see Appendix I). Then the subject began a series of twenty practice trials. The same stimulus intensities, durations, interstimulus intervals, and numbers of stimuli were used in this phase as in the baseline phase. After completion of the twenty practice trials the rating scale was again reviewed with the subject and the verbal reminder about her treatment was repeated. Then the one hundred fifty experimental trials were begun. These trials were identical to the one hundred fifty baseline phase experimental trials. After forty, eighty, and one hundred twenty trials, two-minute breaks were taken and the verbal reminder about the subject's treatment was repeated. Upon completion of trial one hundred fifty, the post-treatment phase was completed and the subject was appropriately debriefed.

# RESULTS

There are three major areas of data analysis for the present study. The first is the analysis of treatment effect on sensory discrimination and response bias as determined through signal detection theory analysis. The second is the analysis of treatment effect on the stimulus intensity rating scale data. The third is the analysis of treatment effect on the anxiety questionnaire data.

## Signal Detection Theory Analysis

McNicol (1972) and Grossberg and Grant (1978) have recommended the use of nonparametric indicators of sensory discrimination and response bias when small numbers of trials per stimulus intensity are used. There is no clearcut rule as to what constitutes a sufficient number of trials in order to use parametric methods, but Rollman (1977) suggests that the minimum number of trials per stimulus should be fifty. Since the present study used only thirty trials per stimulus level, nonparametric indicators of sensory discrimination and response bias were calculated.

Before calculating the nonparametric indicators it was first necessary to convert the responses within each catagory (rating) and radiant level into conditional probabilities. To obtain the conditional probability of a given response at a given level of radiant heat, the number of a given response at a given level of radiant heat, the number of responses at that rating were divided by the number of stimulus presentations at that particular stimulus level. The conditional probability is the probability of a response in a category for a given stimulus level. The conditional probabilities were then

cumulated from highest rating category to lowest category. The cumulative conditional probability for a particular rating category represents the probability of a response in that category or higher for a given stimulus level. Cumulative conditional probabilities were calculated in this manner for each individual for the pretreatment and post-treatment sessions.

Four different pairs of radiant heat stimuli were used in the analysis: Level zero and Level one, Level one and Level two, Level two and Level three, Level three and Level four. When examining a pair of stimulus intensities the lower stimulus level was considered the "noise" and the higher was considered the "signal plus noise." Therefore, within a given rating, the hit rate P(S/s) is defined as the cumulative conditional probability of the higher level stimulus and the false alarm rate P(S/n) is defined as the cumulative conditional probability of the lower level stimulus. The nonparametric measures of sensory discrimination and response bias were calculated from those two rates.

Sensory Discrimination Measures

The nonparametric measure of sensory discrimination used was P(A). The method used to calculate P(A) was to calculate the areas of the triangle and subsequent trapeziums formed by joining the hit rate and false alarm rates on a ROC curve with straight lines (see Figure 4). These areas were then totaled to obtain P(A). Thus, in Figure 4, P(A) is the total of areas 1, 2, 3, and 4. This total area, P(A), reflects the discriminative ability of the individual. The greater the area, the greater the discriminative ability. A P(A) value of 0.5 represents no ability to discriminate and a value 1.0 represents perfect

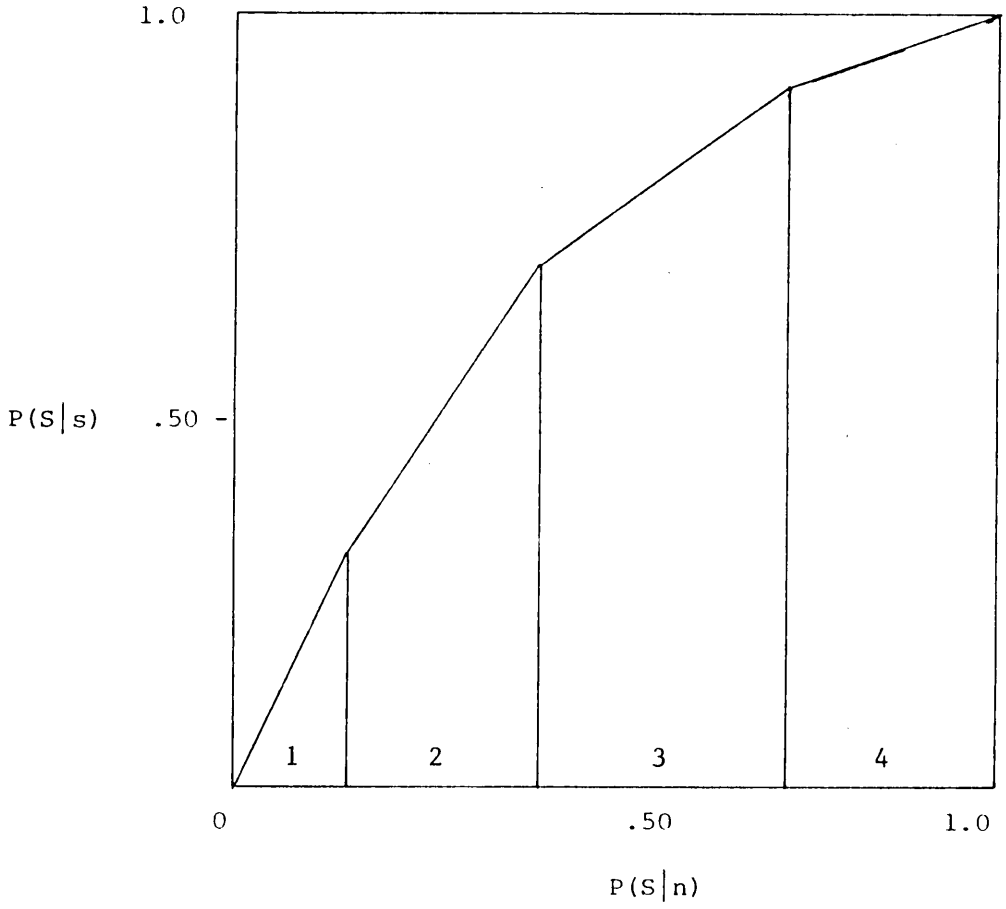


Figure 4. Example of describing the area under the ROC curve for obtaining the non-parametric statistic P(A).

discrimination. P(A) can result in a skewed distribution. The skewedness of the distribution can be reduced by transforming the scores using the question: 2 arcsin $\sqrt{P(A)}$ (McNicol, 1972).

A value of P(A) was calculated for each subject for each stimulus level pair for both the pretreatment and post-treatment testing sessions. These scores were transformed using 2 arcsin $\sqrt{P(A)}$. The mean P(A) for each treatment group for each pair of stimulus levels compared can be seen in Table 1. It was upon these values that the following analyses were performed.

To evaluate hypotheses one, two, and three, planned comparisons were conducted to compare the treatments in question to the expectancy and no treatment control groups. These comparisons were conducted on both the baseline and post-treatment phase P(A) values for each of the stimulus level comparison pairs. To evaluate hypothesis one the intolerant modeling group was compared to the control groups. No significant differences were found in either the baseline or post-treatment phases for any of the stimulus level comparison pairs. To evaluate hypothesis two the modeled imagined analgesia treatment group was compared to the control groups. Significant differences were found only for the comparison of levels zero and one in the post-treatment phase. In that instance the modeled imagined analgesia groups was significantly different from the expectancy control ($\underline{t}$ (54) = -2.738, $\underline{p} < 0.008$) and the no treatment control ($\underline{t}$ (54) = -3.323, $\underline{p} < 0.002$) groups. No other significant differences were found for the other stimulus level comparison pairs. To evaluate hypothesis three the imagined analgesia

TABLE 1

Mean P(A) Values

| Treatment Group | Level 0 - Level 1 | | Level 1 - Level 2 | | Level 2 - Level 3 | | Level 3 - Level 4 | |
|---|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| Tolerant Model | 2.527 | 2.365 | 2.128 | 2.189 | 2.039 | 2.057 | 2.545 | 2.572 |
| Intolerant Model | 2.578 | 2.563 | 2.065 | 2.096 | 2.031 | 2.083 | 2.576 | 2.603 |
| Modeled Analgesia | 2.454 | 2.316 | 2.212 | 2.040 | 2.012 | 2.056 | 2.565 | 2.598 |
| Imagined Analgesia | 2.486 | 2.440 | 2.107 | 2.116 | 2.053 | 2.108 | 2.582 | 2.539 |
| Expectancy Control | 2.515 | 2.646 | 2.118 | 2.141 | 2.008 | 2.121 | 2.561 | 2.577 |
| No Treatment Control | 2.646 | 2.577 | 2.040 | 2.131 | 2.066 | 2.010 | 2.564 | 2.582 |

group was compared to the control groups. No significant differences were found in either the baseline or post-treatment phases for any of the stimulus level comparison pairs.

For each stimulus pair a two-way analysis of variance was performed for Treatment x Sessions with repeated measures over Sessions and subjects nested within Treatment. Summary tables of these analyses are presented in Tables 2-5.

For the comparison of stimulus levels zero and one the main effects of Treatment and Sessions were significant ($F$ (5,54) = 2.54, $p < 0.038$; and $F$ (1.54) = 4.02, $p < 0.0050$ respectively). The Treatment x Sessions interaction was not significant. Post-hoc analyses using the Duncan's Multiple Range test indicated that for the baseline sessions there was no significant difference between any treatment groups. In the post-treatment session, however, significant differences were found. The tolerant modeling group was found to have significantly lower P(A) values than the intolerant modeling group, expectancy control group, and the no treatment control group ($p < 0.05$). In addition, the modeled imagined analgesia group was found to have significantly lower P(A) values than the intolerant modeling group, the expectancy control group and the no treatment control group ($p < 0.05$). Additional Duncan's Multiple Range tests indicated that the post-treatment P(A) values were lower than the baseline values for all groups.

For the comparisons of stimulus levels one and two, two and three, and three and four no significant main or interaction effects were found (see Tables 3-5). An interesting finding was noted in computing the P(A) values for the comparison of stimulus levels three and four.

TABLE 2

Summary of Analysis of Variance

for

P(A) Values for Levels Zero and One

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.142 | 2.54* |
| Session | 1 | 0.145 | 4.02* |
| Treatment x Session | 5 | 0.023 | 0.64 |
| Subjects (Treatment) | 54 | 0.056 | |
| Session x Subjects (Tr.) | 54 | 0.036 | |

* = $p < .05$

TABLE 3

Summary of Analysis of Variance

for

P(A) Values for Levels One and Two

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.017 | 0.27 |
| Session | 1 | 0.002 | 0.02 |
| Treatment x Session | 5 | 0.043 | 0.67 |
| Subjects (Treatment) | 54 | 0.063 | |
| Session x Subjects (Tr.) | 54 | 0.031 | |

TABLE 4

Summary of Analysis of Variance

for

P(A) Values for Levels Two and Three

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.006 | 0.11 |
| Session | 1 | 0.043 | 1.71 |
| Treatment x Session | 5 | 0.015 | 0.60 |
| Subjects (Treatment) | 54 | 0.542 | |
| Session x Subjects (Tr.) | 54 | 0.025 | |

TABLE 5

Summary of Analysis of Variance

for

P(A) Values for Levels Three and Four

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.003 | 0.09 |
| Session | 1 | 0.004 | 0.15 |
| Treatment x Session | 5 | 0.004 | 0.14 |
| Subjects (Treatment) | 54 | 0.030 | |
| Session x Subjects (Tr.) | 54 | 0.030 | |

Many subjects received P(A) values of less than 0.50. Such values are rare, but not impossible. They indicate that the subjects were unable to discriminate between levels three and four and at times would rate a level three stimulus as stronger or more painful than a level four stimulus.

The analyses of the P(A) data indicates that hypotheses one and three were not supported and that hypothesis two was only partially supported. More specifically, the modeled imagined analgesia group had significant post-treatment decrease, relative to the controls, only for the comparison of stimulus levels zero and one. Post-hoc analyses indicated that the modeled imagined analgesia group had lower post-treatment P(A) values than the intolerant modeling group at the comparison of levels zero and one. Taken together these data suggest that the modeled imagined analgesia treatment decreased the physical sensitivity of the subjects, but only for the lowest level of heat stimulation. In addition, the post-hoc analyses indicated that the tolerant modeling group had a similar effect at the lowest level of heat stimulation.

Response Bias Measures

The nonparametric measure of response bias used in this study was B" which was first proposed by Hodos (1970). According to Grier (1971) this measure can be computed using the formula:

$$B'' = \frac{(Y(1-Y) - X(1-X))}{(Y(1-Y) + X(1-X))}$$

In this equation X = false alarm rate and Y = hit rate at a given rating and a given pair of adjacent stimuli. This equation will produce values ranging from -1.00 to +1.00. The absolute value of B" indicates the amount of bias while the valence of B" indicates the direction of the

bias. A negative B" value indicates a tendency to respond, while a positive B" value indicates a hestiancy to respond.

A B" value was computed for each stimulus level pair for each subject. This value was computed using the cumulative probability of a four (faint pain) intensity rating for each stimulus level. By using this cumulative probability the B" values obtained represent each subject's bias toward responding with a rating of four (faint pain), five (painful), six (very painful), or seven (withdrawal). Therefore a negative B" value represents a tendency toward responding with a four, five, six, or seven and a positive B" value represents a hestiancy toward responding with a four, five, six or seven. The mean B" values for each treatment group for each pair of stimulus levels can be seen in Table 6.

To evaluate hypotheses four and five, planned comparisons were conducted to compare the treatments in question to the expectancy and no treatment control groups. These comparisons were conducted on both the baseline and post-treatment phase B" values for each of the stimulus level comparison pairs. In evaluating hypothesis four, the tolerant modeling and control groups were compared. No significant differences were found in the baseline phase for any of the stimulus level comparison pairs. For the post-treatment phase, however, significant differences were found. In the comparison of levels zero and one, the tolerant modeling group was significantly different from both the expectancy control ($\underline{t}$ (54) = - 2.527, $\underline{p} < 0.025$) and the no treatment ($\underline{t}$ (54) = -2.665, $\underline{p} < 0.020$) control groups. In the comparison of levels one and two, the tolerant modeling group was significantly different from the expectancy control ($\underline{t}$ (54) = - 2.250, $\underline{p} < 0.038$) and no treatment control ($\underline{t}$ (54) =

TABLE 6

Mean B" Values

| Treatment Group | Level 0 - Level 1 Pre | Level 0 - Level 1 Post | Level 1 - Level 2 Pre | Level 1 - Level 2 Post | Level 2 - Level 3 Pre | Level 2 - Level 3 Post | Level 3 - Level 4 Pre | Level 3 - Level 4 Post |
|---|---|---|---|---|---|---|---|---|
| Tolerant Model | -0.862 | -0.310 | -0.099 | 0.295 | -0.181 | 0.329 | -0.119 | 0.097 |
| Intolerant Model | -0.975 | -0.800 | -0.194 | 0.032 | -0.181 | -0.152 | -0.046 | -0.208 |
| Modeled Analgesia | -0.812 | -0.200 | -0.223 | 0.286 | -0.175 | 0.293 | 0.061 | 0.012 |
| Imagined Analgesia | -0.926 | -0.232 | -0.108 | 0.231 | -0.159 | 0.083 | -0.066 | -0.200 |
| Expectancy Control | -0.587 | -0.614 | -0.138 | -0.124 | -0.086 | -0.152 | 0.063 | -0.029 |
| No Treatment Control | -0.587 | -0.700 | -0.004 | -0.043 | -0.077 | -0.128 | -0.074 | -0.083 |

-2.136, $p < 0.042$). In the comparison of levels two and three, the tolerant modeling roup differed significantly from the expectancy control ($t$ (54) = -2.668, $p < 0.020$) and no treatment control ($t$ (54) = -2.525, $p < 0.027$) groups. In the comparison of levels three and four, no significant differences were found.

To evaluate hypothesis five, several planned comparisons were conducted comparing the intolerant modeling, modeled analgesia, and the imagined analgesia groups to the expectancy and no treatment control groups. No signficiant baseline session differences were found for any treatment group for any stimulus level comparison pair. In addition, no significant post-treatment differences were found for the intolerant modeling treatment group. Significant differences, however, were found for the modeled imagined analgesia and imagined treatments in the post-treatment session. For the comparison of levels zero and one, the modeled imagined analgesia group was significantly different from both the expectancy control ($t$ (54) = -2.047, $p < 0.045$) and no treatment control ($t$ (54) = -2.030, $p < 0.047$). For the same stimulus level pair the imagined analgesia group was significantly different from both the expectancy control ($t$ (54) = -2.148, $p < 0.034$) and no treatment control ($t$ (54) = -2.318, $p < 0.0005$) groups. For the comparison of levels one and two, the modeled imagined analgesia group was significantly different from both the expectancy control ($t$ (54) = -2.390, $p < 0.020$) and no treatment control ($t$ (54) = -2.423, $p < 0.016$) groups. For the same stimulus level pair the imagined analgesia group was significantly different from both the expectancy control ($t$ (54) = -2.801, $p < 0.005$) and the no treatment control ($t$ (54) = -2.682, $p < 0.020$) groups. For the

comparison of levels two and three, the modeled imagined analgesia group was significantly different from both the expectancy control ($t$ (54) = -3.891, $p < 0.001$) and the no treatment control ($t$ (54) = -3.636, $p < 0.002$) groups. For the same stimulus level pair the imagined analgesia group was significantly different from the expectancy control ($t$ (54) = -2.113, $p < 0.038$) and the no treatment control ($t$ (54) = -2.240, $p < 0.032$) groups. For the comparison of levels three and four there were no significant differences between either treatment group and the control groups.

For each stimulus pair, a two-way analysis of variance was performed for Treatment x Sessions with repeated measures over Sessions and Subjects nested within Treatments. Summary tables of these analyses are presented in Tables 7-10.

For the comparison of stimulus levels zero and one, the main effect of Sessions was significant ($F$ (5,54) = 25.43, $p < 0.001$) and the Treatment x Sessions interaction was also significant ($F$ (5,54) = 4.27, $p < 0.001$) (see Table 7). Simple main effect analyses of Session at each Treatment indicates that the modeled imagined analgesia, imagined analgesia, and tolerant modeling groups all changed significantly from baseline to post-treatment sessions ($F$ (5,54) = 15.41, $p < 0.001$; $F$ (5,54) = 12.47, $p < 0.010$; and $F$ (5,54) = 5.98, $p < 0.010$ respectively).

For the comparison of stimulus levels one and two, the main effect of Sessions was significant ($F$ (1,54) = 21.19, $p < 0.001$) and the Treatment x Sessions interaction was also significant ($F$ (5,54) = 2.88, $p < 0.05$) (see Table 8). Simple main effect analyses of Sessions at each Treatment indicates that the tolerant modeling, modeled imagined

TABLE 7

Summary of Analysis of Variance

for

B" Values for Levels Zero and One

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.343 | 1.90 |
| Session | 1 | 3.284 | 25.43** |
| Trestment x Session | 5 | 0.552 | 4.27** |
| Subjects (Treatment) | 54 | 0.180 | |
| Session x Subjects (Tr.) | 54 | 0.129 | |

** = $p < .01$

TABLE 8

Summary of Analysis of Variance

for

B" Values for Levels One and Two

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.153 | 0.86 |
| Session | 1 | 1.734 | 21.19** |
| Treatment x Session | 5 | 0.234 | 2.88* |
| Subjects (Treatment) | 54 | 0.177 | |
| Session x Subjects (Tr.) | 54 | 0.082 | |

* = $p < .05$ ** = $p < .01$

analgesia, and the imagined analgesia groups all changed significantly from baseline to post-treatment sessions ($F$ (1,54) = 15.42, $p < 0.001$; $F$ (1,54) = 14.70, $p < 0.001$; and $F$ (1,54) = 10.48, $p < 0.010$ respectively).

For the comparison of stimulus levels two and three, the main effect of Sessions was significant ($F$ (5,54) = 15.33, $p < 0.001$) and the Treatment x Sessions interaction was also significant ($F$ (5,54 = 4.16, $p < 0.01$) (see Table 9). Simple main effects analyses of Session at each Treatment indicates that the tolerant modeling and modeled imagined analgesia groups both changed significantly from the baseline to the post-treatment sessions ($F$ (1,54) = 17.01, $p < 0.001$; and $F$ (1,54) = 16.58, $p < 0.001$ respectively). For the comparison of stimulus levels three and four, there were no significant main effects or interaction effects found (see Table 10).

The analyses of the B" data indicate that hypothesis four was supported. The subjects receiving the tolerant modeling treatment changed their criterion for labeling a stimulus as painful to a more conservative criterion. That is, they became less willing to give a rating of four, five, six or seven to a heat stimulus. Hypothesis five was only partially supported. As was predicted, the intolerant modeling group did not show any post-treatment change in their response bias. The modeled imagined analgesia and imagined analgesia groups, however, did have a significant post-treatment change in their response bias. Specifically, the subjects in both groups became less willing to give a rating of four or greater after receiving their respective treatments.

TABLE 9

Summary of Analysis of Variance

for

B" Values for Levels Two and Three

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.224 | 1.61 |
| Session | 1 | 1.170 | 15.33** |
| Treatment x Session | 5 | 0.318 | 4.16** |
| Subjects (Treatment) | 54 | 0.138 | |
| Session x Subjects (Tr.) | 54 | 0.075 | |

** = $p < .01$

TABLE 10

Summary of Analysis of Variance

for

B" Values for Levels Three and Four

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 0.104 | 1.52 |
| Session | 1 | 0.038 | 0.42 |
| Treatment x Session | 5 | 0.470 | 1.04 |
| Subjects (Treatment) | 54 | 0.069 | |
| Session x Subjects (Tr.) | 54 | 0.088 | |

Rating Scale Data Analysis

A mean score for each stimulus level for each session was computed for each subject by taking the average of the thirty responses to each stimulus level during each session. This provided each subject with five baseline and five post-treatment scores, one for each stimulus level. The means of these scores for each treatment group are presented in Table 11.

To evaluate these scores a three-way analysis of variance was performed for Treatment x Sessions x Levels with repeated measures over Levels and Sessions and Subjects nested within Treatment. A summary table of this analysis is presented in Table 12. The analysis indicated that there were significant main effects of Sessions ($F$ (1,54) = 52.01, $p < 0.001$) and Levels ($F$ (4,216) = 804.23, $p < 0.001$) and significant interaction effects of Treatment x Sessions ($F$ (5,54) = 9.38, $p < 0.001$), Sessions x Levels ($F$ (4,216) = 7.50, $p < 0.001$), and Treatment x Sessions x Levels ($F$ (20,216) = 3.78, $p < 0.001$).

To explore the significant three-way interaction, simple main effects analyses were done. The analysis of Treatment and Session at Level zero indicated a significant Sessions main effect ($F$ (1,54) = 31.29, $p < 0.001$) and a significant Treatment x Sessions interaction ($F$ (5,54) = 2.87, $p < 0.010$). The Analysis of Treatment and Session at Level one indicated a significant Treatment main effect ($F$ (5,54) = 75.85, $p < 0.001$), a significant Sessions main effect ($F$ (1,54) = 75.85, $p < 0.001$), and a significant Treatment x Sessions interaction ($F$ (5,54) = 6.17, $p < 0.001$). The analysis of Treatment and Session at Level two indicated a significant Sessions main effect ($F$ (1,54) = 36.19, $p < 0.001$)

TABLE 11

Mean Rating Scale Values

| Treatment Group | Level 0 | | Level 1 | | Level 2 | | Level 3 | | Level 4 | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| Tolerant Model | 0.42 | 0.31 | 2.16 | 1.55 | 3.60 | 2.87 | 4.84 | 3.97 | 4.70 | 3.90 |
| Intolerant Model | 0.46 | 0.38 | 2.32 | 2.13 | 3.55 | 3.38 | 4.65 | 4.65 | 4.70 | 4.58 |
| Modeled Analgesia | 0.42 | 0.08 | 2.09 | 1.10 | 3.82 | 1.91 | 5.16 | 3.03 | 5.11 | 3.20 |
| Imagined Analgesia | 0.63 | 0.11 | 2.48 | 1.45 | 3.80 | 2.64 | 5.11 | 3.84 | 5.10 | 3.73 |
| Expectancy Control | 0.74 | 0.51 | 2.41 | 2.07 | 3.58 | 3.32 | 4.70 | 4.66 | 4.66 | 4.54 |
| No Treatment Control | 0.47 | 0.36 | 2.46 | 2.31 | 3.57 | 3.83 | 4.89 | 4.86 | 4.75 | 4.91 |

TABLE 12

Summary of Analysis of Variance

for

Mean Pain Ratings

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 5.47 | 1.25 |
| Session | 1 | 48.51 | 52.01** |
| Level | 4 | 368.30 | 804.23** |
| Treatment x Session | 5 | 8.75 | 9.38** |
| Treatment x Level | 20 | 0.20 | 0.44 |
| Session x Level | 4 | 1.19 | 7.50** |
| Treatment x Session x Level | 20 | 0.60 | 3.78** |
| Subject (Treatment | 54 | 4.384 | |
| Session x Subject (Tr.) | 54 | 0.932 | |
| Level x Subject (Tr.) | 216 | 0.458 | |
| Session x Level x Subject (Tr.) | 216 | 0.159 | |

** = $p < .01$

and a significant Treatment x Sessions interaction ($F$ (5,54) = 8.44, $p < 0.001$). The analysis of Treatment and Sessions at Level three indicated a significant Sessions main effect ($F$ (1,54) - 27.48, $p < 0.001$) and a significant Treatment x Sessions interaction ($F$ (5,54) = 6.61, $p < 0.001$). The analysis of Treatment and Session at Level four indicated a significant Sessions main effect ($F$ (1,54) = 29.69, $p < 0.001$) and a significant Treatment x Sessions interaction ($F$ (5,54) = 7.42, $p < 0.001$).

To explore the significant two-way interactions seen in each simple main effects analysis several sets of simple main effects analyses were done. The analyses of Treatment at each Level and Session indicated that there was a significant main effect of Treatment at each Level on the post-treatment session only. For stimulus Level zero on the post-treatment session the Treatment effect had an $F$ value of 3.47 (df = 5,54; $p < 0.009$). For stimulus Level one on the post-treatment session the Treatment effect had an $F$ value of 5.56 (df = 5,54; $p < 0.0004$). For stimulus Level two on the post-treatment session the Treatment effect had an $F$ value of 4.28 (df = 5,54; $p < 0.003$). For stimulus Level three on the post-treatment session the Treatment effect had an $F$ value of 3.28 (df = 5,54; $p < 0.012$). For stimulus Level four on the post-treatment session the Treatment effect had an $F$ value of 2.73 (df = 5,54; $p < 0.028$). The analyses of Session at each Level and Treatment found significant main effects of Session for the modeled imagined analgesia group at Level one ($F$ (1,18) = 15.18, $p < 0.001$); Level two ($F$ (1,18) = 15.82, $p < 0.0009$); Level three ($F$ (1,18) = 37.61, $p < 0.0001$); and Level four ($F$ (1,18) = 24.99, $p < 0.0001$). A significant

main effect of Session was also found for the imagined analgesia group at Level zero ($F$ (1,18) = 13.25, $p$ 0.0019); Level one ($F$ (1,18) = 8.00, $p < 0.0077$); Level two ($F$ (1,18) = 17.77, $p < 0.0005$); Level three ($F$ (1,18) = 10.07, $p < 0.005$); and Level four ($F$ (1,18) = 6.47, $p < 0.020$). In addition, a significant main effect of sessions was found for the expectancy control group at Level zero ($F$ (1,18) = 10.55, $p < 0.0045$).

The results of these analyses indicate that the subjects rated each increasing stimulus level as more painful, regardless of the treatment or session, except for Level four. Level four was not rated significantly different from Level three. A Duncan's Multiple Range test was performed and it indicated that there was no difference in the ratings of Level three and four ($p < 0.05$). This finding corroborates the trend, mentioned above, in P(A) values of some subjects for the comparison of Levels three and four. The analyses also indicate that the modeled imagined analgesia, the imagined analgesia, and the expectancy control groups were the only ones to make significantly lower post-treatment pain ratings. The modeled analgesia group rated Levels one, two, three, and four significantly lower after treatment, the imagined analgesia group rated all levels significantly lower post-treatment, and the expectancy control group rated the zero level significantly lower post-treatment.

## Anxiety Measures Analyses

### State Anxiety

A two-way analysis of variance for Treatment x Sessions with repeated measures on Sessions and Subjects nested within Treatment, was

performed on the subject's PARQ scores. The results indicated a significant main effect of Sessions ($F$ (1,54) = 6.62, $p < 0.005$) (see Table 14). This indicates that all subjects, regardless of treatment, reported less state anxiety post-treatment than during the baseline session (see Table 13).

Trait Anxiety

A two-way Treatment x Sessions analysis of variance with repeated measures on Sessions and subjects nested within Treatment was performed on the subjects' STAI-Trait scores. No significant main effects or interactions were found (see Table 15). This indicates no group or session differences in trait anxiety reported.

TABLE 13

Mean PARQ Scores

| Treatment Group | Baseline | Post-Treatment |
|---|---|---|
| Tolerant Model | 40.3 | 34.1 |
| Intolerant Model | 38.0 | 36.2 |
| Modeled Analgesia | 49.0 | 42.3 |
| Imagined Analgesia | 50.0 | 40.8 |
| Expectancy Control | 45.9 | 39.6 |
| No Treatment Control | 45.1 | 40.3 |

TABLE 14

Summary of Analysis of Variance

for

Mean PARQ Scores

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 225.07 | 1.63 |
| Session | 1 | 909.13 | 6.62** |
| Treatment x Session | 5 | 72.31 | 1.05 |
| Subjects (Treatment) | 54 | 143.52 | |
| Session x Subjects (Tr.) | 54 | 64.82 | |

** = $p < .01$

TABLE 15

Summary of Analysis of Variance

for

Mean STAI Scores

| Source | df | MS | F |
|---|---|---|---|
| Treatment | 5 | 127.05 | 1.24 |
| Session | 1 | 16.13 | 1.91 |
| Treatment x Session | 5 | 7.52 | 0.89 |
| Subjects (Treatment) | 54 | 85.50 | |
| Session x Subjects (Tr.) | 54 | 8.31 | |

## Discussion

This study, like many others before it, has found that psychological interventions can effect responses to acute painful stimulation. Tolerant modeling, modeled imagined analgesia, and imagined analgesia treatments were all found to alter pain responding. Between groups analyses found that these three treatments decreased the subjects' bias toward calling a stimulus painful, relative to the expectancy and no treatment control groups. In addition, between groups analyses found that the tolerant modeling and modeled imagined analgesia treatments decreased the subjects' physical sensitivity to the lowest levels of heat stimulation relative to the control groups. Within groups analyses indicated that the three treatments significantly reduced response bias from the baseline to post-treatment sessions. The anxiety data analyses indicate that these effects were achieved with no differential effect on state or trait anxiety when compared to other treatments. These results have many implications.

The results of this study indicate that exposure to a tolerant model can have an effect on pain responding. Tolerant modeling was found to reduce physical sensitivity to pain at the lowest level of heat stimulation and to reduce the subjects' bias toward calling a stimulus painful. Rosenthal and Bandura (1978) conclude that modeling can effect pain responding because it alters the subject's own cognitive standards for self-regulation. They felt that the model provided standards for the subject to judge the adequacy and appropriateness of their own performance and that the subjects alter their performance to conform to these standards. According to this explanation, a response bias shift

for tolerant modeling would be due to the subjects altering their willingness to call a stimulus painful to conform to the new standards of performance established by observing the model. It does not, however, readily explain the change in physical sensitivity observed for the tolerant modeling group. The gate control theory, however, does explain how this might work. The gate control theory postulates mechanisms of influence by which motivational-affective components can effect sensory-discriminative components of pain (see Figure 1). Thus, the subjects' altered cognitive performance standards could change physical sensitivity by effecting both the gate control system and the sensory-discriminative system and this could result in a decrease in signal detection physical sensitivity measure.

The results of this study indicate that exposure to intolerant modeling did not significantly alter pain responding. The hypothesis of an increase in physical sensitivity for the intolerant modeling group was based on the findings of Craig and Coren (1975). There are obvious differences in the modeling treatments used in the present study and by Craig and Coren (1975). Craig and Coren (1975) used a live confederate who performed the task with the subject and rated the stimulus before the subject did. The present study used a short videotape which the subject saw before rating the stimuli. Perhaps the treatment used by Craig and Coren (1975) was a more powerful manipulation of cognitive standards since it provided the subjects throughout the task with a set of standards to continually judge his or her performance. If this was the case, however, then it is logical to ask why was the tolerant modeling treatment effective in the present study?

Perhaps the intolerant modeling treatment used by Craig and Coren (1975) was more effective in altering the subjects' cognitive standards because it did not give the subjects as many contradictory messages as the treatment used in the present study. In the present study, the intolerant modeling subjects had three different sources of information which they could draw on to establish their own cognitive performance standards. One source of information was their previous experience with the heat stimuli two days before. Before beginning the post-treatment phase, the subjects were told that they would be rating the same stimuli they had rated in the baseline phase. The second source of information was in the rationale for the modeling treatment. To control for the effects of expectancy all treatment groups were told that they should find the stimuli less painful or uncomfortable (see Appendix F). The third source of information was the modeling videotape itself, which gave the message that someone else found the task to be painful and distressing. Thus, the subjects had three conflicting data bases to use in developing their own cognitive performance standards. In contrast, Craig and Coren's (1975) subjects had no prior exposure to the stimuli, no experimenter expectations of reduced pain, but did have a live model providing them with the message that the stimuli were painful. It seems reasonable to conclude that Craig and Coren's (1975) intolerant modeling treatment was indeed more powerful than the one used in the present study because it did not send the subjects conflicting messages and most likely that is the reason the results of this study did not support hypothesis one.

The present study also indicates that imagined analgesia can be an effective treatment to alter pain responding. It had the effect of reducing the subjects' willingness to label a stimulus as painful. This was not, however, the expected effect. Hypothesis three predicted that subjects using imagined analgesia would show a decrease in physical sensitivity to pain. This was based on Hall's (1977) finding that a suggestion of imagined analgesia resulted in a physical sensitivity decrease with no apparent change in response bias. There are no readily apparent differences between the two studies which could account for the differences in findings. The treatments, while not identical, are comparable and have only subtle differences. A possible reason for the differences in results may be in the data analysis. Hall (1977 choose to combine the P(A) and B" measures for each stimulus pair into an average P(A) and an average B" for each subject. The statistical analyses were then performed on each subject's average P(A) and B" values. That approach is different from the one used in the present study and may account for the different results observed in this study.

Spanos and his associates (Stam and Spanos, 1980; Spanos, Horton, and Chaves, 1975; and Spanos, Radtke-Bodorik, Ferguson, and Jones, 1979) have evidence which may help to explain how an imagined analgesia treatment can effect response bias. They have observed that subjects who successfully use an imagined analgesia treatment to reduce pain are cognitively active during the noxious stimulation. Stam and Spanos (1980) concluded that the successful users of imagined analgesia modified both their overt behavior and subjective experierces in

conformance with treatment induced expectations. They suggest that:

> ...."Reduced pain" in these circumstances may mean that subjects have redefined or relabeled (rather than lessened the intensity of) their sensory experiences. Therefore, sensory events previously labeled as "high pain" may be categorized in some alternative way.

In terms of a signal detection paradigm, this relabeling would translate into a response bias change such that the subject is more hesitant to call a given stimulus painful.

Tolerant modeling plus imagined analgesia appears to have been an effective treatment combination in the present study. It had the effect of reducing response bias and decreasing physical sensitivity at the lowest heat level. Hypothesis two predicted a decrease in physical sensitivity for subjects receiving this treatment and was partially supported. The change in response bias, however, was not predicted. Since the treatment is a combination of both tolerant modeling and imagined analgesia these findings are not surprising and the explanations discussed above are equally applicable to the modeled imagined analgesia treatment.

Modeled imagined analgesia, while an effective treatment combination, was no more effective than tolerant modeling alone. Duncan's Multiple Range tests were performed on both the baseline and post-treatment B" values for the three effective treatment groups for each stimulus pair comparison. No significant differences were found between the modeled imagined analgesia group and either the tolerant modeling group or imagined analgesia groups ($p < 0.05$). In addition, a Duncan's Multiple Range test was performed on both the baseline and post-treatment P(A)

values for these three treatment groups for the comparison of levels zero and one. No significant difference was found between the tolerant modeling and the modeled imagined analgesia groups, but both groups were found to be significantly different from the imagined analgesia group ($p < 0.05$). The results of these tests suggest that there may be a common element or mechanism responsible for the success of tolerant modeling and modeled imagined analgesia treatments.

One possible common mechanism is that the treatments both created and strengthened expectations of personal effectiveness in coping with the noxious stimulation. Bandura (1977) has hypothesized that successful psychological treatment procedures, whatever the format, succeed by increasing the subject's expectation of personal efficacy. Bandura states that expectation of personal efficacy comes from four main sources: a) performance accomplishments, b) the vicarious experiences of observing others cope and succeed, c) verbal persuasion and exhortation, and d) states of physiological arousal from which people judge their level of anxiety and vulnerability to stress. Subjects in the tolerant modeling and imagined analgesia groups were provided information about personal efficacy from all four sources. The subjects in the imagined analgesia group were provided information only from three of the four sources. This could account for the difference in treatment effects. The additional efficacy information provided by the modeling may have created a stronger expectation of personal efficacy for the tolerant modeling and modeled imagined analgesia subjects than for the imagined analgesia subjects. The expectation of personal efficacy may have been strong enough in all three treatment groups to change response bias, but

the increase in expectation due to tolerant modeling could have resulted in the change in physical sensitivity at the lowest heat level for the tolerant modeling and modeled imagined analgesia groups. Gate control theory explains how this could happen. A sufficiently strong motivational-affective component (expectation of self efficacy) could effect the gate control mechanism and the sensory discriminative system which would result in a decrease in physical sensitivity.

The intolerant modeling group received efficacy information from the same four sources as the tolerant modeling and modeled imagined analgesia groups, but it was ineffective as a treatment. The information the intolerant modeling subjects received, however, was contradictory. They were told that their treatment should cause them to feel less pain, but the modeling tape they saw showed a model who was not successful in feeling less pain. The information conveyed by the intolerant model may have outweighed any of the other information efficacy information such that the subjects had little or no expectation of self efficacy, rendering the treatment ineffective. It can not be unequivocally determined whether expectations of self efficacy were the common mechanism in the successful treatments. It does, however, seem to be a likely candidate.

Anxiety and pain often have been reported to correlate. Many clinical and laboratory pain treatment studies have used treatments designed, whole or in part, to reduce anxiety (e.g., relaxation training or hypnosis). The treatments often reduced anxiety along with pain and sometimes reduced anxiety but did not alter pain. The treatments in the

present study were not designed to reduce anxiety and the successful ones did not significantly alter state or trait anxiety. This raises the question of what is the relationship between anxiety and pain. Beecher (1959, 1963, 1972) has argued that laboratory pain studies are missing the anxiety associated with the disease process and the threat of disfigurement or death. He feels that reducing pain reactions in clinical settings involves reducing anxiety while anxiety is not crucial in reducing laboratory pain. The results of this study suggest that Beecher may be correct.

## Summary and Conclusions

In recent years researchers have increasingly turned to signal detection methodology in the analyses of intervention strategies for pain (e.g., Hall, 1977; Craig and Coren, 1975). The use of this paradigm is due to the inadequacy of threshold and tolerance as measures of the complex phenomena of pain. Signal detection methodology has been seen as a way to tap physical sensitivity to pain and response bias relatively cleanly. The present study used signal detection methodology to examine the effects of videotaped modeling, imagined analgesia, and a combination of videotaped modeling and imagined analgesia on pain responding. Tolerant modeling, imagined analgesia, and modeled imagined analgesia were all found to decrease the subject's bias toward labeling a stimulus as painful and tolerant modeling and modeled imagined analgesia were also found to decrease physical sensitivity to low level heat stimulation.

According to signal detection theory, these results suggest that psychological treatments such as modeling or the imagining analgesia have little effect on the experience of pain through the attenuation of the sensory experience. Rather, the major effect of these treatments appears to be in raising the subjects' criterion for calling stimulation painful.

In examining the results of the present study from the perspective of self efficacy theory the treatments used can be rank ordered according to the amount of information they provide the subjects about expectations of their own self efficacy. The tolerant modeling and

modeled imagined analgesia treatments provided the most information, the imagined analgesia treatment provided the next largest amount, the intolerant modeling provided the next largest amount, the expectancy control the next, and the no treatment control treatment provided the least amount of information. In examining the effectiveness of each treatment the tolerant modeling and modeled imagined analgesia treatments were most effective. The imagined analgesia treatment was the only other effective treatment but it was not as effective as the tolerant modeling and imagined analgesia treatments since it did not effect physical sensitivity.

The gate control model of pain (Melzack and Wall, 1965) hypothesizes that cognitive and motivational activities can have an effect on the sensory experience of pain by attenuating neural transmission at the spinal gate (see Figure 1). The results of this study suggest that there is some threshold that the cognitive-motivational activity must cross before it can effect the sensory experience of pain. The tolerant modeling and modeled imagined analgesia treatments were most likely the strongest in inducing expectancies of self efficacy and did have a minimal effect in decreasing physical sensitivity to pain. The other treatments, which were likely less effective in inducing expectancies of self efficacy, did not effect physical sensitivity and either had no effect or changed only the cognitive-motivational component of the pain experience (response bias). If one accepts this hypothesis, then it could be concluded that the tolerant modeling in the modeled imagined analgesia condition increased the effectiveness of the imagined analgesia treatment. Interestingly, it did not make the combined treatment any

more effective than the treatment using tolerant modeling alone. Therefore one could conclude that tolerant modeling is a powerful manipulator of self efficacy and an effective treatment for acute pain.

Only two studies have found that psychological interventions can alter physical sensitivity to pain (Hall, 1977; Craig and Coren, 1975). The results of this study suggests that they may have been able to do so because the treatments were able to significantly alter the subjects' expectations of personal efficacy such that a threshold of cognitive-motivational activity was exceeded and the sensory experience of pain was altered. This suggests that future research should examine the power of interventions to effect expectations of self efficacy and their effect on the sensory experience of pain.

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# APPENDIX A

FORM A-2

Name_____ Date_____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you generally feel.

There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

| | | almost never | sometimes | often | almost always |
|---|---|---|---|---|---|
| 1. | I feel pleasant | 1 | 2 | 3 | 4 |
| 2. | I tire quickly | 1 | 2 | 3 | 4 |
| 3. | I feel like crying | 1 | 2 | 3 | 4 |
| 4. | I wish I could be as happy as others seem to be | 1 | 2 | 3 | 4 |
| 5. | I am losing out on things because I can't make up my mind soon enough | 1 | 2 | 3 | 4 |
| 6. | I feel rested | 1 | 2 | 3 | 4 |
| 7. | I am "calm, cool, and collected." | 1 | 2 | 3 | 4 |
| 8. | I feel that difficulties are piling up so that I cannot overcome them | 1 | 2 | 3 | 4 |
| 9. | I worry too much over something that really doesn't matter | 1 | 2 | 3 | 4 |
| 10. | I am happy | 1 | 2 | 3 | 4 |
| 11. | I am inclined to take things hard | 1 | 2 | 3 | 4 |
| 12. | I lack self-confidence | 1 | 2 | 3 | 4 |
| 13. | I feel secure | 1 | 2 | 3 | 4 |
| 14. | I try to avoid facing a crisis or difficulty | 1 | 2 | 3 | 4 |
| 15. | I feel blue | 1 | 2 | 3 | 4 |
| 16. | I am content | 1 | 2 | 3 | 4 |
| 17. | Some unimportant thought runs through my mind and bothers me. | 1 | 2 | 3 | 4 |
| 18. | I take disappointments so keenly that I can't put them out of my mind | 1 | 2 | 3 | 4 |
| 19. | I am a steady person | 1 | 2 | 3 | 4 |
| 20. | I get in a state of tension or turmoil as I think over my recent concerns and interests | 1 | 2 | 3 | 4 |

Date _____

PRESENT AFFECT REACTIONS QUESTIONNAIRE (PARQ)

Please circle a number from 1 to 5 on this sheet for each of the 24 items to indicate:

"HOW YOU FEEL AT THIS PARTICULAR MOMENT"

| | | | | | | |
|---|---|---|---|---|---|---|
| 1. | Hands feel moist | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very moist |
| 2. | Feel relaxed | 1<br>Very relaxed | 2 | 3 | 4 | 5<br>Not at all |
| 3. | Hands feel unsteady | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very unsteady |
| 4. | Feel self-confident | 1<br>Very much | 2 | 3 | 4 | 5<br>Not at all |
| 5. | Stomach feels tense | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very tense |
| 6. | Enjoy this situation | 1<br>Very much | 2 | 3 | 4 | 5<br>Not at all |
| 7. | Heart beats faster | 1<br>Not at all | 2 | 3 | 4 | 5<br>Much faster |
| 8. | Feel calm | 1<br>Very calm | 2 | 3 | 4 | 5<br>Not at all |
| 9. | Perspire | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very much |
| 10. | Feel comfortable | 1<br>Very much | 2 | 3 | 4 | 5<br>Not at all |
| 11. | Mouth feels dry | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very dry |
| 12. | Unable to focus my thoughts | 1<br>Able to focus | 2 | 3 | 4 | 5<br>Unable to focus |
| 13. | Feel pleasant | 1<br>Very pleasant | 2 | 3 | 4 | 5<br>Not at all |
| 14. | Feel nervous | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very nervous |
| 15. | Feel throbbing in my head | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very much so |
| 16. | Feel secure | 1<br>Very secure | 2 | 3 | 4 | 5<br>Not at all |

| | | | | | | |
|---|---|---|---|---|---|---|
| 17. | Feel upset | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very upset |
| 18. | Hands feel cold | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very cold |
| 19. | Feel good | 1<br>Very good | 2 | 3 | 4 | 5<br>Not at all |
| 20. | Feel anxious | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very anxious |
| 21. | Breathing is irregular | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very irregular |
| 22. | Feel uneasy | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very uneasy |
| 23. | Want to avoid this situation | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very much |
| 24. | Feel lump in throat | 1<br>Not at all | 2 | 3 | 4 | 5<br>Very much |

# APPENDIX B

## Criteria for Scoring Withdrawal

1. A withdrawal occurs when a subject pulls, rotates, or pivots her arm away from the apparatus during the stimulus presentation.

2. A withdrawal is not scored if the subject pulls her arm away from the apparatus as the shutter closes. The rationale is that the subject has already received the full three seconds of stimulation before the withdrawal response was initiated.

3. Do not count as a withdrawal a gap between the arm and the plastic ring if the gap is present because: a) the subject's arm does not fit well against the ring, b) the subject relaxes her arm allowing the gap to appear.

# APPENDIX C

## Introduction to Baseline Phase

Before we begin, we would like to familiarize you with the experiment and the equipment to which you will be exposed. The purpose of this experiment is to examine the effects of different interventions on your response to radiant heat stimulation. During the experiment we will use the machine in front of you to produce the radiant heat stimuli. What this is, essentially, is a spotlight, a magnifier, and a shutter. We use a light dimmer to control the intensity of the light which will effect the amount of heat you feel on your arm. So that all arms, regardless of pigmentation, will absorb uniformly across subjects, we will darken six circles on your arm with India ink which will wash off easily after the experiment. So that you respond to the stimuli and not to the brightness of the light source, a heavy cloth curtain hangs between you and the light source. Additionally, you will wear a set of headphones during the experiment in order to muffle the sound of the shutter opening and closing. You will receive a stimulus every fifteen seconds. Immediately following the presentation of a stimulus a tone will sound in your headphones. At the sound of the tone you should rate the stimulus according to the scale you have in front of you. "Zero" indicates that you felt nothing at all, "one" indicates that you felt something but you could not really identify it, "two" means that you felt that the stimulus was warm, "three" indicates that you thought the stimulus was hot, "four" means that it was faintly painful, "five" indicates that the stimulus was painful, and "six" indicates that you felt that the stimulus was very painful. As noted earlier, in the event that

a given stimulus is too painful, you may remove your arm from the projector.

In this experiment we wish to determine your ability to feel warmth, heat, and pain. A variety of heat intensities, including zero, will be applied to your arm. Some stimuli will be so weak that you will feel nothing at all, others will be hot, while others will produce a pain sensation. Remember, we don't want to see how much pain you can endure; rather, we want to know how good you are at detecting the presence of a just noticeable amount of pain. If the stimulus is too painful, you may remove your arm from the projector. Don't forget, after the presentation of each stimulus we would like you to rate the thermal experience according to the rating scale on the card before you. "Zero" indicates that you felt nothing at all, "one" indicates that you felt something but you couldn't readily identify it, "two" means that you felt that the stimulus was warm, "three" indicates that you thought that the stimulus was hot, "four" means that it was faintly painful, "five" indicates that the stimulus was painful, and "six" indicates that you felt the stimulus was very painful. If at any time you need to stop or you forget the rating scale, please tell the experimenter.

So as to give you practice in rating the stimuli using this scale, we will now go through twenty practice trials which include all levels of stimuli which you will be exposed to during the experiment. Remember, you are to rate the stimulus after the tone. Please speak loudly, clearly, and distinctly so that the experimenter can hear you. Are there any questions at this point?

# APPENDIX D

Introduction to Tolerant and Intolerant Modeling Videotapes

It has been found in several research studies that observing another person's performance on a task involving painful or uncomfortable stimulation will have a positive influence on your own performance on that task. Typically, after observing another person's performance on the task one finds the stimuli less painful or uncomfortable. We wish to examine the effect that observing another person's performance on the heat stimuli rating task will have on your own ability to differentiate the heat stimuli. Therefore, we would like you to watch a short videotape which will show you another person's performance on the stimuli rating task.

# APPENDIX E

Introduction and Instructions for Modeled Imagined Analgesia

It has been found in numerous research studies that an individual feels much less pain and finds painful stimulation much easier to tolerate if they tell themselves that their body is insensitive to pain. Everyone has heard of persons undergoing surgery while hypnotized or yoga masters walking across burning coals. The principle underlying these acts is simply that you can use your mind to produce numbness and insensitivity to pain. We are interested in examining what effects using thought control to produce numbness will have on your ratings of the heat stimuli.

In order to show you how this strategy works we would like you to watch a short videotape which will show you someone who has trained her mind to produce numbness in her arm.

(Subject watches modeled imagined analgesia videotape)

Now we want to teach you how to cognitively produce numbness and insensitivity in your own arm. Make yourself as comfortable as possible in the chair....Now focus on your right arm....Tightly clench your right fist....Hold it....Release it slowly....As you release your fist notice how the tension flows out of your hand and forearm....Let your arm relax ....becoming more and more relaxed each time you breathe out....Let the tension flow out of your arm....with each breath your arm becomes more relaxed....Notice how your arm is beginning to feel heavy....It is as if an iron bar is on your arm weighting it down....Imagine the iron

weighing heavily on your arm....Just let your arm go limp and heavy.... with each breath your arm becomes heavier and more relaxed....As your arm becomes more relaxed you will notice a feeling of warmth spreading throughout your arm....A very pleasant feeling of warmth....increasing with each breath you take....you should begin to notice a tingling sensation beginning in your fingertips and slowly spreading up your hand to your arm ....Focus on this sensation....It is the beginning of numbness and insensitivity in your arm....Your arm is gradually becoming numb and insensitive to pain, as if you have been given a shot of Novocain....with each breath you take the tingling becomes stronger....your arm is becoming more and more numb....you are beginning to lose feeling in your arm.... your arm is becoming more and more insensitive with each breath you take....numb and insensitive as if it were made with rubber....you may still be able to feel some things, but you will feel much less than you could before....your arm will continue to be insensitive and numb throughout the experiment....when a heat stimulus is presented, it will feel only pleasantly warm....remember, the more that you imagine that your arm is numb, the less uncomfortable the heat stimuli will be.

APPENDIX F

Introduction and Instructions for Imagined Analgesia

It has been found in numerous research studies that an individual feels much less pain and finds painful stimulation much easier to tolerate if they tell themselves that their body is insensitive to pain. Everyone has heard of persons undergoing surgery while hypnotized or yoga masters walking across burning coals. The principle underlying these acts is simply that you can use your mind to produce numbness and insensitivity to pain. We are interested in examining what effects using thought control to produce numbness will have on your ratings of heat stimuli.

Now we want to teach you how to cognitively produce numbness and insensitivity in your own arm. Make yourself as comfortable as possible in the chair....Now focus on your right arm....Tightly clench your right fist....Hold it....Release it slowly....As you release your fist notice how the tension flows out of your hand and forearm....Let your arm relax....becoming more and more relaxed each time you breathe out....Let the tension flow out of your arm....with each breath your arm becomes more relaxed....Notice how your arm is beginning to feel heavy ....It is as if an iron bar is on your arm weighting it down....Imagine the iron bar weighing heavily on your arm....Just let your arm go limp and heavy....with each breath your arm becomes heavier and more relaxed....As your arm becomes more relaxed you will notice a feeling of warmth spreading throughout your arm....A very pleasant feeling of warmth....increasing with each breath you take....you should begin to notice a tingling sensation beginning in your fingertips and slowly

spreading up your hand to your arm....Focus on this sensation....It is the beginning of numbness and insensitivity in your arm....Your arm is gradually becoming numb and insensitive to pain, as if you have been given a shot of Novocain....with each breath you take the tingling becomes stronger....your arm is becoming more and more numb....you are beginning to lose feeling in your arm....your arm is becoming more and more insensitive with each breath you take....numb and insensitive as if it were made with rubber....you may still be able to feel some things, but you will feel much less than you could before....your arm will continue to be insensitive and numb throughout the experiment....when a heat stimulus is presented, it will feel only pleasantly warm.... remember, the more that you imagine that your arm is numb, the less uncomfortable the heat stimuli will be.

APPENDIX G

Instructions for Expectancy Controls

Our previous research indicates that repeated thermal stimulation alters pain sensitivity by desensitizing the individual to radiant heat pain. Since the previous experience you have had with thermal stimulation has desensitized your skin receptors to radiant heat, you should now perceive fewer painful stimuli.

APPENDIX H

Instructions for Post-Treatment Phase

As in the first part of the experiment, we again wish to determine your ability to feel warmth, heat, and pain. A variety of heat intensities, including zero, will be applied to your arm. Some stimuli will be so weak that you will feel nothing at all, others will be hot, while others will produce a pain sensation. Remember, we do not want to see how much pain you can endure; rather, we want to know how good you are at detecting the presence of a just noticeable amount of pain. If the stimulus is too painful, you may remove your arm from the projector. After the presentation of each stimulus I would like you to rate the thermal experience according to the rating scale on the card before you.

You will receive a stimulus every fifteen seconds. Immediately following the presentation of a stimulus, a tone will sound in your headphones. At the sound of the tone you should rate the stimulus according to the scale in front of you. "Zero" indicates that you felt nothing at all, "one" indicates that you felt something but you could not really identify it, "two" means that you felt that the stimulus was warm, "three" indicates that you felt that the stimulus was hot, "four" means that it was faintly painful, "five" indicates that the stimulus was painful, and "six" indicates that you felt that the stimulus was very painful. In the event that a given stimulus is too painful, you may remove your arm from the projector. If at any time you need to stop or you forget the rating scale, please tell the experimenter.

So as to give you practice in rating the stimuli using this scale, we will now go through twenty practice trials which will include all

levels of stimuli which you will be exposed to during the experiment. Remember, you are to rate the stimulus after the tone. Please speak loudly, clearly and distinctly so that the experimenter can hear you.

Are there any questions at this point?

# APPENDIX I

## Verbal Prompts Given in Post-Treatment Phase

Tolerant and Intolerant Modeling Treatments:

Don't forget, because you watched the videotape you should find the stimuli less painful or uncomfortable.

Modeled Imagined Analgesia and Imagined Analgesia Treatments:

Don't forget, the more that you imagine that your arm is numb, the less uncomfortable the heat stimuli will be.

Expectancy Control Treatment:

Don't forget, because of your prior experience with thermal stimulation, you should find the stimuli less painful or uncomfortable.

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# THE EFFECTS OF VIDEOTAPED MODELING AND IMAGINED ANALGESIA ON ACUTE PAIN: A SIGNAL DETECTION ANALYSIS

by

Joseph Ward Hatcher, Jr.

(ABSTRACT)

The present study examined the effects of videotaped modeling and imagined analgesia instructions as treatments for acute pain in the context of a signal detection paradigm. A signal detection analysis was used because it allows differentiation between sensory-discriminative and motivational-affective components of pain. The treatment conditions used were: 1) tolerant modeling, 2) intolerant modeling, 3) modeled imagined analgesia which combined tolerant modeling and imagined analgesia instructions, 4) imagined analgesia instructions, 5) an expectancy control treatment in which the subjects were told to expect a decrease in the amount of pain experienced, and 6) no treatment control.

Subjects were sixty female undergraduate volunteers who were randomly assigned to one of five treatment groups. The noxious stimuli were five levels of radiant heat, including zero. Subjects participated in pre- and post-treatment sessions in which they received thirty stimulus presentations per level and were requested to rate each presentation on a scale from zero to six, with seven being a withdrawal. Self-report anxiety measures were also taken.

The results indicated that the tolerant modeling, modeled imagined analgesia, and imagined analgesia treatments reduced the subjects' response bias. In addition, the tolerant modeling and modeled imagined

analgesia treatments reduced physical sensitivity to the lowest level of heat stimulation. Analyses of the anxiety data indicated that all groups experienced a reduction in state anxiety over time.

The implications that these data have with respect to current psychological theories of pain are discussed. In addition, the results of this study are discussed from the perspective of self-efficacy theory to attempt to explain the treatment effects seen.