THE EFFECTS OF SUGGESTED ANALGESIA ON RADIANT HEAT PAIN
AS A FUNCTION OF HYPNOTIC SUSCEPTIBILITY:
A SIGNAL DETECTION ANALYSIS
by
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INTRODUCTION

Pain is a complex, multidimensional phenomenon which has proved difficult to define empirically. Because of this very complexity, there is no one generally accepted definition of pain which adequately accounts for all the parameters of the pain experience.

This introduction will first review definitions and theories of pain in addition to techniques used to measure pain. Secondly, the rationale and procedures of Signal Detection Theory (SDT), which is a specialized pain measurement paradigm, will be reviewed. Thirdly, the research evidence for the effectiveness of "cognitive" strategies in the treatment of pain will also be reviewed. Finally, the effectiveness of a particular kind of cognitive strategy, suggested analgesia (versus an expectancy control group) will be investigated in the present study.

Definitions of Pain

In a review of the pain literature, Weisenberg (1977) observed that on the basis of previous research (Casey, 1977, Melzack, 1973), pain may be viewed as a sensation on the one hand and/or an emotional-motivational phenomenon which leads to escape and avoidance behavior on the other hand. Weisenberg (1975) noted that physiologists and sensory psychologists have traditionally conceptualized pain as a separate sensation, along with temperature and other cutaneous senses. In spite of the fact that emotional and motivational factors have been mentioned as being very important in determining the individual's reaction to pain, the traditional approach has focused almost exclusively on the sensory component of pain (Weisenberg, 1977). The
traditional view has defined pain largely as a reaction to actual or impending tissue damage, i.e., the greater the tissue damage, the greater the pain reaction. This emphasis on the sensory aspects of pain has serious limitations in that it fails to consider the psychological (or emotional-motivational) component of pain. That non-sensory determinants of pain are very important was shown in a study by Beecher (1956); he concluded that the setting (environmental) can affect the pain reaction more than the actual tissue destruction does. He found that of 215 soldiers with serious battle wounds, only 25% requested narcotics for pain relief. This was in contrast to requests made in civilian life with a similar surgical wound made under anesthesia; in this case, over 80% of the group wanted pain relief. Beecher speculated that the differences in pain reaction between the two situations (battlefield vs. civilian life) were due to the significance assigned to the wound rather than to the degree of actual tissue damage. On the battlefield the wound meant an honorable exit from a life-threatening situation; in civilian life surgery was threatening and potentially disastrous.

Weisenberg (1977) rejected stimulus-response (e.g., that tissue damage is directly related to pain reaction) definitions of pain as clinically inadequate. He observed that these definitions implied that in order to stop pain all that is required is to interrupt the pain pathway. In fact, surgical results generally report a disappointing success rate (Weisenberg, 1975). The phenomenon of central pain (Loeser, 1977) seems to occur in the absence of antecedent stimuli. The fact that psychiatric illness, especially depression, has
been associated with complaints of pain (Sternbach, 1974) is taken as evidence that psychological/emotional factors can cause pain.

In summary, attempts to define pain have proved largely inadequate because they cannot account for its complex multidimensional properties. The problems in defining pain are well summarized by Liebeskind and Paul (1977):

Pain means many different things; and the variables which correlate with, inhibit or enhance one kind of pain, and the neural mechanisms which underlie it, may not be associated with or influence other kinds. Thus one must distinguish between the normal perception of noxious stimuli and pain of pathological origin, and between acute pathological pain and chronic, intractable pain conditions.... While it is often useful to distinguish between various aspects of pain experience (e.g., "sensory-discriminative" versus "motivational-affective" components), other dichotomous terms used in an attempt to specify the origin of pain ("physiological" versus "psychological", "organic" versus "functional") connote a Cartesian dualism and should have been discarded long ago. (1977, pp. 41-42)

**Theories of Pain**

As in the case with definitions of pain, there does not appear to be a single pain theory which is adequate to account for the wide range of pain phenomena. Gate Control Theory (Melzack, 1973), proposed originally in 1965 and later updated (Melzack & Wall, 1965, 1970), has been proven at least partially inaccurate in terms of physiology (e.g., a proposed differential response of small and large fibers at the gating mechanism; see Vyklicky, Rudomin, Zajal and Burke, 1969) but is still the most important and influential current theory of pain perception. The importance of the gate control theory of pain lies in its integration of motivational-affective components
with sensory-discriminative components of pain, regulated by a gate control system which is mediated by central (cognitive) mechanisms (see Figure 1 for diagram of updated model).

Gate Control Theory combined aspects of two other current (but individually inaccurate) theories:

1. **Specificity theory** - refers to a pain system based on a specific set of peripheral nerve fibers that are nonciceptive in function, i.e., two sets of peripheral nerve fibers associated with two qualities of pain: (a) A-delta fibers with short-latency pricking pain; and (b) C-fibers with long-latency burning pain (Mountcastle, 1974). Melzack (1973) rejected specificity theory as not adequately accounting for pain phenomena and as making unwarranted assumptions about the perception of pain.

2. **Pattern theory** - opposes the idea that there is a set of specialized pain receptors; instead, pain perception is based on stimulus intensity and central summation. In this view, pain results from the summation of spatial and temporal patterns of input. Melzack (1973) rejected pattern theory as inconsistent with physiological evidence.

Weisenberg (1977) noted that Gate Control Theory rejected specificity but accepted specialization, i.e., specialization can be found at receptor sites, such as A-delta and C-fibers, that respond to particular types and ranges of physical energy. The point is made that specialization is not specificity; specificity responds to one and only one given kind of stimulus.

In summary, Gate Control Theory appears to account fairly well for a wide range of pain phenomena, but is not adequate to account for all. It is undoubtedly the most useful and practical model to date, but has little predictive utility for individual pain behaviors. Perhaps its primary utility lies in its conceptualization of pain as a
Figure 1. Gate-Control Model of Pain (Adapted from Melzack and Casey (1968) and Melzack and Wall (1965)).
tri-partite phenomenon composed of motivational-affective and sensory-discriminative dimensions which are regulated by and interrelated with central (cognitive-evaluative) mechanisms.

The schema of the gate control theory has been concisely summarized by Weisenberg (1977):

The gate control theory of pain contains elements of both the specificity and pattern theories. It attempts to account for psychological influences on pain perception, as well as such clinical findings as spread of pain and persistence of pain after tissue healing. Conceptually, gate control theory proposes a dorsal spinal gating mechanism in the substantia gelatinosa that modulates sensory input by the balance of activity of small-diameter (A-delta and C) and large-diameter (A-beta) fibers. Activity of large fibers closes the gate and prevents synaptic transmission to centrally projecting T (transmission) cells, whereas small-diameter fibers open the gate and facilitate T-cell activity once a critical level is reached. Small fiber activity is believed to be responsible for prolongation of pain and its spread to other parts of the body. A central control trigger can also influence the gate. Thus, cognitive processes can either open or close the gate.

(1977, p. 1011)

A more recent conceptualization of pain has focused on pain as behavior and is largely grounded in the work of Fordyce (1976) and reiterated by Gentry and Bernal (1977). Briefly, pain behavior is the focus of analysis and can be viewed in an operant-respondent scheme. This work is well summarized in a review by Sanders (1979) and will not be elaborated upon here. Sanders (1979) has extended upon this work and devised a conceptualization system of trimodal pain and well behavior (in the overt, covert and physiological response modes). The value of this model is that although it is not a theory per se, it is an outgrowth of a theoretical system which emphasizes pain as behavior.
Measurement of Pain

Techniques to measure pain are many and varied. This state of affairs is probably a function of the fact that pain is multidimensional, subjective and hard to define. Wolff (1978) has maintained that the use of operational definitions (with cautions regarding generalization and requirements for validation) is an acceptable and necessary strategy, given the uncertainties in defining and measuring pain.

Techniques to measure human pain are grounded in the methods of psychophysics pioneered by German researchers in the late 19th century. This research was based on the Classical Threshold model of Fechner (1860).

Threshold has been defined simply as the point at which the subject first perceives the stimulation as painful; this is in contrast to the measure of tolerance, which refers to the point at which the individual is not willing to accept stimulation of a higher magnitude or to continue to endure stimulation at a given level of intensity, i.e., terminal threshold (Weisenberg, 1977). Threshold has typically been associated with physiological variables, whereas tolerance has been related to psychological factors, which are attitudinal or motivational in nature (Gelfand, Gelfand & Rardin, 1965). This hypothesis has by no means been proven, however, and there is conflicting evidence on the issues. On the basis of a series of studies, Wolff and his associates (e.g., Wolff & Horland, 1967) concluded that "while both threshold and tolerance had sensory and
psychological components, the experimental pain threshold was more highly loaded with sensory than with psychological variables, whereas the pain tolerance had proportionally higher loadings of psychological than of physiological (sensory) components" (Wolff, 1978, p. 145).

These conclusions have been disputed by Clark and Goodman (1974), who used an SDT analysis. This latter study will be discussed in further detail in the section below on SDT.

In addition to the pain measurement techniques mentioned above, several others are noteworthy. With the exception of the McGill Pain Questionnaire (Melzack, 1975), the scales which will be discussed below have been used primarily for the measurement of laboratory pain. As the name implies, verbal rating scales rely on the subjective responses of the individual on verbal (semantic) pain dimensions. Studies which have examined verbal rating scales (e.g., Dubisson & Melzack, 1976; Melzack, 1975; and Tursky, 1976) have generally concluded that they can reliably differentiate quantitative and qualitative aspects of pain. Another technique which has been used is the visual analogue scale, which consists of a straight line with fixed end points representing extremes of the sensation measured (e.g., no pain versus excruciating pain). This type of scale is technically a kind of cross-modality matching. When using the technique of cross-modality matching, the subject matches several standard stimuli along one sensory modality (e.g., brightness) by adjusting the value on a second sensory modality (e.g., loudness). These scales have the advantage of not introducing verbal rating bias into the response, but a disadvantage is that the matching procedure has not been well standardized.
Physiological indices have also been used to measure human pain. In general, results have been equivocal across a range of specific response types. The search for an idiosyncratic, pain-specific physiological response has largely resulted in failure (Sanders, 1979; Sternbach, 1968; Wolff, 1978). Physiological measures have been largely abandoned in favor of the subject's verbal pain report (Hilgard, 1969, p. 107).

To summarize the literature on the measurement of pain, a multitude of assessment instruments have been used to assess what is admittedly a multidimensional phenomenon. Measurement techniques are often a function of the researcher's theoretical orientation. By way of review, Melzack and Wall's (1965) gate-control theory hypothesizes that there are three interrelated pain dimensions: (1) sensory-discriminative; (2) motivational-affective; and (3) a central, cognitive-evaluative dimension. The McGill Pain Questionnaire (Melzack, 1975) was an outgrowth of the gate-control theory and is designed to distinguish between the sensory versus affective component of pain. The MPQ has not been proven to distinguish between the affective and sensory components of pain, nor have any of the measures discussed previously. Evidence will now be presented to demonstrate that the signal detection approach presents the best available method for differentiating between these two components. First the general SDT model will be considered and then examined as it applies to pain research.

Signal Detection Theory (SDT)

A relatively recent approach (given the long history of pain research) has involved the use of signal detection theory (SDT). This
technique purports to distinguish between changes in pain behavior as a function of either sensory detection (the actual physical discrimination of the stimulus) or response bias (i.e., the likelihood that the subject will change his pain behavior as a function of expectancy or other judgmental criteria which may be introduced by the experimenter).

The simplest SDT paradigm consists of ratings in a "yes-no" task. The subject answers "yes" if he believes a signal plus noise stimulus was presented and "no" if he believes that a noise stimulus was presented. According to this paradigm there are four possible response categories: (1) a "hit" response, which occurs when a subject correctly perceives a stimulus; (2) a "false alarm" response, which occurs when a subject incorrectly reports that a signal was present when it was not; (3) a "correct rejection" response in which the subject correctly reports that no signal was present; and (4) a "miss" response, in which a signal was present during a given trial but the subject responded negatively. The correct rejection and miss responses are redundant in that they can be computed if one knows the hit and false alarm rates and the total number of both noise and signal plus noise presentations.

The probabilities of both the hit and false alarm rates can be calculated by dividing each rate by the number of signal presentations (for the hit rate) and by the number of noise presentations (for the false alarm rate). Two conditional probabilities result: \( P(S/s) \) and \( P(S/n) \), respectively. If these conditional probability estimates are represented graphically, a set of two curves can be plotted which represent noise and signal plus noise. A criterion point can also be
estimated, with the areas under the two curves representing noise and signal plus noise to the right of the criterion.

The statistic $d'$ is defined as the difference between the means of the two distributions divided by the standard deviation of the noise distribution. Given normal distributions with equal variance, $d'$ can be defined as the difference between the standardized normal (i.e., $z$ scores) of the hit and false alarm probabilities. The formula is $d' = z(P(S/s)) - z(P(S/n))$. A low $d'$ value means that the subject showed a tendency to confuse stimuli of different intensities. According to Clark (1974), this occurs when either the physical intensities of the stimuli are close together or when the subject's sensory system is insensitive. The criterion represents an overall bias to favor a particular response. The likelihood-ratio criterion ($L_x$) consists of the ratio of the ordinate of the two distributions as defined by the conditional probabilities, $P(S/s)$ and $P(S/n)$, i.e., the conditional probabilities of hits and false alarms (Clark, 1974). Although the criterion is generally termed $L_x$, an analogous but alternative criterion measure, called $C_x$ is also used. It reflects the fact that criteria at different locations along the sensory continuum will have identical likelihood ratios when more than two stimulus intensities are being judged (Luce, 1963, pp. 103-109). Clark emphasizes that SDT maintains that the likelihood ratio criterion is independent of changes in values of either stimulus intensity or the subject's physical sensitivity.

If several sets of hit and false alarm rates are plotted they yield what is called a receiver operating characteristic (ROC) curve, which is
Figure 2. Sample signal and noise distributions with a criterion designated \( P(S|s) \) and \( P(S|n) \).
a plot of the different $P(S/s)$ by $P(S/n)$ pairs (Green & Swets, 1966). This is represented in Figure 3. The ROC function contains information about both the subject's sensory discrimination and response bias.

Chapman (1980) noted an advantage to using a rating scale (vs. a simple yes-no paradigm) in SDT research: an ROC curve can be generated from hit rate and false alarm rate values at each rating scale category. This eliminates the need for varied experimental conditions and is more economical in the laboratory. To obtain multiple points on an ROC curve using a yes-no task, experimental conditions must be varied (e.g., instructions to "Be conservative when guessing," vs. "Be moderate," vs. "Be liberal and do not worry about mistakes" - Chapman, 1980). Hall (1977) emphasized that the yes-no task only provides a single point on the ROC curve and advocated the use of a rating scale task instead. The rating scale task is used in order to estimate several (usually three or more) points on the ROC curve. As noted by Hall (1977) the data obtained from the rating-scale task are analyzed similarly to data obtained from the yes-no task except that the response frequencies in each category are cumulated across categories before being converted to conditional probabilities. In addition, the cumulated response frequencies are then converted to provide a set of inferred hit and false alarm rates -- the hit and false alarm rates that would have resulted if the observer had used each category as a single criterion in a yes-no task (Lee, 1969, p. 105). The result is a plot of points on the ROC curve equal to one less than the number of stimulus intensities. Hall concluded that the advantages of accuracy and
Figure 3. Example of an ROC curve plotting HIT by FALSE ALARMS.
economy afforded by the rating scale task outweighed shortcomings regarding possible failures of SDT assumptions. He noted that researchers in visual and auditory perception have been satisfied that this is not a serious problem because d' values obtained from yes-no and rating-scale tasks agree (Green & Swets, 1966, p. 110ff).

An ROC curve can also be generated to compute nonparametric statistics for the SDT model. The nonparametric estimate of d' is called P(A) and represents the proportion of the area under the ROC curve (McNichol, 1972). The P(A) statistic is useful for those occasions when the assumptions of normal distribution and equal variance are not satisfied. Rollman (1977) has also recommended the use of nonparametric statistics when a relatively small number of trials per stimulus intensity is given.

According to Chapman (1980) classical SDT procedures are based on the assumption that the ROC curve would have a slope of 1.0 when the hit and false alarm rates were scaled in "normal deviate units." Another assumption is that all data points would fall on the single line representing the ROC plot. Chapman advises the use of P(A) and a nonparametric index of response bias when the aforementioned assumptions cannot be met, as nonparametric indices do not require these assumptions. The appropriateness of nonparametric statistics will be discussed further in the Results section of this study.

For detailed reviews of the appropriateness of various statistical models of SDT and the assumptions underlying them (especially for a
discussion of a special form of SDT called the Thurstonian Scaling Model) the reader is referred to Hall (1977).

**Signal Detection Studies in Pain Research**

The use of SDT in pain research is still relatively recent (within the past 10 to 15 years) and the absolute number of studies is modest and primarily emphasizes drug treatments. Accordingly, only a handful of studies has evaluated treatments having "psychological" components (e.g., placebos/suggestions for pain relief, distractions, hypnosis, etc.). Following is a review of SDT studies in pain research to date.

A summary of Clark's (1969) original study will serve as an introduction to other SDT pain research. He used 22 paid volunteers to serve in a control and placebo group to assess the effects of radiant heat stimulation in both an SDT and threshold analysis. Subjects were administered a placebo (a virtually inert substance consisting of 392 mg. lactose and 8 mg. quinine), preceded by a check on health and previous drug reactions and followed by a check list on the side effects of medication. These manipulations were designed to reinforce the subjects' impression that a potent drug had been given. Results were calculated under each condition and cumulated across categories to yield estimates of $d'$ and $C_X$. Statistical analysis indicated that the placebo did not have an effect on $d'$ but did have an effect on $C_X$. Values of $C_X$ were more conservative for reporting pain after treatment, and Clark attributed this to "a placebo-induced set (which) increased the embarrassment or social cost of a 'pain' response" (1969, p. 369).
When the data were subjected to a traditional threshold analysis (using the method of constant stimuli), however, the conclusion was that the placebo had raised the pain threshold. This comparison of a traditional threshold analysis with an SDT analysis for the same data represented a milestone in laboratory pain research. The results were clear: "According to decision theory analysis (i.e., SDT)... the change in the constant stimuli threshold was due to the effect of suggestion on the attitude or response bias of Ss (subjects)" (Clark, 1969, p. 369).

In a follow-up to this study, Clark and Goodman (1974) tested Wolff and Horland's (1967) hypothesis that the pain tolerance threshold ("tolerance") is more susceptible to instruction and suggestion than the pain detection threshold ("threshold"). In this study, Clark and Goodman posited a contrary hypothesis — that the influence of instructional set would have equal susceptibility for the pain detection and pain tolerance threshold. Clark and Goodman based this hypothesis on the belief that the pain detection and pain tolerance thresholds are merely "different criterion locations along the same pain decision-axis" (p. 365). The 40 subjects (both male and female) were divided into four experimental conditions (equally balanced for sex) after judging heat stimuli in a control (pretest) situation. A 12 point rating scale (including withdrawal) was used. Two groups of subjects were given instructions which were intended to affect their detection thresholds. One of these groups was instructed that they would have more difficulty detecting painful stimuli as a result of their pretest exposure. The other group was given similar instructions, but, in this case, was told
that they would find it easier to detect pain. The other two groups in
the experiment were given instructions intended to result in analogous
changes in the pain tolerance threshold. As with the two pain detection
groups, the two pain tolerance groups were given instructions that it
would be (for the first group) easier to tolerate pain versus (for the
second group) more difficult to tolerate pain.

For the criterion measure, $L_x$ was used. The category "very faint
pain" was used as a measure of the detection threshold, and the
condition of withdrawal was used as a measure of pain tolerance. In
addition, $d'$ values were computed for each subject. Results indicated
that none of the instructions had any effect on $d'$. Effects on
criterion measures for detection and tolerance thresholds were as
follows: as expected, subjects who were given instruction to raise
versus lower the detection threshold in fact did so by reporting a
higher versus lower criterion for detection; results for subjects
given instructions regarding tolerance threshold were highly similar
to those for detection threshold. There were, however, somewhat
different effects for instructions on the threshold measure which was
not mentioned in the experimental instructions (i.e., the effect of
detection suggestions on tolerance suggestions and vice versa).
Instructions intended to raise or lower the tolerance threshold
resulted in parallel changes in the detection threshold. On the other
hand, instructions designed to cause changes in the detection
threshold did not affect the tolerance threshold.

On the basis of these data, Clark and Goodman drew two main
conclusions: (1) that instructions had no effect on sensitivity to pain
(d'); (2) the effect of instructions on detection and tolerance thresholds was due to the fact that "instructions increased the psychosocial 'cost' of a pain response and the subjects complied by raising their criteria" (1974, p. 370). Clark and Goodman also speculated that many pain threshold changes produced by "cognitive control," hypnosis, and other techniques such as counter-irritant stimulation are not due to decreased pain sensitivity but rather to changes in the criterion for reporting pain. It is the contention of the present author that this speculation has not been borne out in all subsequent research in this area. Particulars will be discussed in later sections of this paper.

To summarize, the importance of the Clark (1969) and Clark and Goodman (1974) studies lies in the fact that, when compared with threshold and tolerance measures of pain response, SDT analyses yielded a more fine-grained measure which provided indices of sensory/discriminative versus attitudinal/response bias components of pain.

Clark's (1969) study of the effects of a placebo on pain was replicated in a somewhat abbreviated version by Feather, Chapman and Fisher (1972), who found that the placebo did not affect the ability of the subjects to discriminate between stimuli (i.e., d') but it did affect response bias (C_X), in that subjects were more conservative about reporting pain after receiving the placebo.

A subsequent attempt to replicate these findings was made by Hall (1977). Results for d' were that there was no difference between placebo and control conditions, no difference between heat and pain judgments and no interaction effects. For the measure C_X, only the test
for the main effect for category labels achieved significance. Tests for other measures of $C_x$ indicated that there were no differences between control and placebo conditions in mean $C_x$ and no interaction between treatment condition and rating-scale label.

Thus, in contrast to the findings of Clark (1969) and Feather, et al., (1972), who found changes in response bias after subjects were given a placebo, Hall (1977) did not find that a placebo affected the subjects' response bias. Hall (1977) speculated that his failure to replicate the findings of earlier studies was due to at least three differences in details of experimental design, all of which affected the perceived potency of the placebo, e.g., (1) that the two earlier studies had been conducted in hospital settings, whereas Hall's study was conducted in a university department; (2) that the experimenter was probably less credible to his subjects in that he was only slightly older (versus the more senior status of Clark and Feather, et al., to their subjects); and (3) that expectancy effects in Hall's study were not as powerful as, e.g., Clark's "elaborate ruses" and that the placebo was not presented as a narcotic analgesic. Regarding the finding of no difference on the sensory/discriminative ($d'$) index as a function of rating-scale labels, Hall concluded that "subjects do not use heat categories any more reliably than they use pain categories when making judgments about thermal stimuli" (1977, p. 94).

Given that the aforementioned early studies had demonstrated the superiority of SDT (for evaluating sensory vs. response bias
comparisons) over traditional threshold and tolerance measures and the usefulness of SDT in evaluating equivalent components of the placebo-induced response, the next trend in the literature was to evaluate the effect of drugs (e.g., analgesics and tranquilizers) on pain response by means of SDT analysis. Other procedures which have been evaluated via SDT for assessing their effect on the pain response include such "physical" manipulations as acupuncture, transcutaneous electrical nerve stimulation (TNS), brain implants and other electrical (e.g., dental) stimulation. The use of "psychological" or cognitively oriented treatments in an SDT analysis has been generally rare and often not well controlled experimentally. Following is a review of SDT studies on drug treatments and a cursory review of other "physical" treatments. This will be followed by a review of the few studies using "psychological" treatments. Finally, consideration will be given to some of the criticisms of the use of SDT in pain research and some of the issues that should be considered in using it for this purpose.

One of the first studies to use SDT to evaluate the effectiveness of drugs on pain response was done by Chapman, Murphy and Butler (1973). The drug used was an analgesic, 33% nitrous oxide. Results indicated that the nitrous oxide solution reduced sensory discriminability (d') between the zero stimulus and each of the non-zero stimuli but not for adjacent non-zero stimuli. Regarding the response bias measure, results indicated the subjects showed more conservative criteria for reporting pain after receiving nitrous oxide. The authors concluded that their findings were in accordance with Melzack and Casey's (1968) gate control theory of pain.
A second study, or rather a series of three experiments, was done by Chapman and Feather (1973) to evaluate the effect of diazepam (a tranquilizer) on human pain response. In the first experiment, the dependent measure was tolerance of ischemic pain (induced by the sub-maximum effort tourniquet technique) and the independent variables were diazepam versus a placebo. Briefly, the results were that diazepam increased the subjects' tolerance of pain significantly over the placebo condition. For the second experiment ischemic pain tolerance was again used as the dependent measure, but, in this case, the independent variables were diazepam versus aspirin. Results for this experiment were that diazepam was significantly more effective than aspirin in increasing pain tolerance time. In the third experiment the authors used an SDT approach to assess whether the extended pain tolerance as a result of diazepam was due to a reduction in sensory discriminability or changes in response bias. A placebo condition was also used.

Results were that there were no significant differences between the diazepam and placebo conditions on either the sensory-discriminative measure (d') or the response bias measure (C_x). Chapman and Feather compared these results to those for the two previous (tolerance) experiments and concluded that the evidence suggested that:

The sensory-discriminative aspect of pain was not affected by diazepam nor was the central control process. This strongly suggests that the extended pain tolerance observed in the first two experiments reflects the effects of the drug on the motivational-emotional aspects of pain alone. (1973, p. 339)

Another study which used an SDT model to investigate the effectiveness of various drugs on pain perception was done by Lineberry, Kulics,
Tung, and Tenicella (1975). This study used "SDT reaction time methods" to analyze the analgesic effectiveness of a placebo, two concentrations of codeine (30 mg. and 60 mg.) and two concentrations of diazepam (5 mg. and 10 mg.). Results were that the placebo condition had no effect on discrimination and that "codeine reduced pain sensitivity in a dose-related manner in contrast to 10 mg. of valium (diazepam) which had no effects upon pain perception" (Lineberry, et. al., 1975, p. 99).

One final study which used SDT methodology to evaluate the effectiveness of a drug (33% nitrous oxide) for its analgesic properties on human pain was done by Chapman, Gehrig and Wilson (1975). In addition, a control group and a group receiving acupuncture (for 20 minutes at the Hoku point on the hand) were also used. A total of 42 males served as subjects, 14 in each group. Stimuli consisted of four intensities of electrical stimulation (including zero), with 75 presentations of each stimulus intensity. The stimulated area was on a standardized section of the subjects' tooth pulp. A seven-point rating scale, ranging from "nothing" to "strong pain," was used. Briefly, results were that, for the measure d', a small (but statistically significant) decrease occurred for both the acupuncture and nitrous oxide conditions, in comparison to the control condition. The two treatment conditions were approximately equal in effectiveness. In addition, both treatment conditions were associated with a change in response bias such that subjects were much more conservative about reporting pain.

In general, results of studies using an SDT analysis of acupuncture are somewhat equivocal. Although changes in response bias are
occasionally found in these studies, changes in d' have been found to be nonexistent (e.g., Clark & Yang, 1974) or relatively small (e.g., Chapman, Chen & Bonica, 1977). Hall (1977) noted that the apparently discrepant findings regarding changes in d' after acupuncture (e.g., the studies of Chapman and his colleagues on the one hand, versus Clark & Yang on the other) are probably explainable because of a number of procedural differences among the studies. Hall cited several of these procedural differences: site of acupuncture stimulation (e.g., cheek, hand, volar surface of forearm); type of noxious stimulation (e.g., electrical stimulation or radiant heat); and measures of discrimination (d') and response bias (e.g., nonparametric versus traditional use of adjacent stimulus intensity distributions.

A relatively new procedure which has been subjected to SDT analysis in order to assess its effectiveness in attenuating pain involves external placement of electrical stimulators on the subject's skin and stimulation with low electric currents in localized areas. This procedure has been termed transcutaneous electrical stimulation (TES) (Chapman, Wilson & Gehrig, 1976), or transcutaneous electrical stimulation (TENS) (McCreery & Bloedel, 1978). For the purpose of convention, it will be referred to as "TENS" in the present review. A brief review of the few studies using this procedure in an SDT analysis will follow and, insofar as possible, a summary statement on the overall effectiveness of TENS will be made.

The study by Chapman, et al., (1976) mentioned above compared the treatment effectiveness of acupuncture versus the TENS procedure. In
general, both acupuncture and TENS reduced the sensory (d') component to a small but significantly different degree over a placebo group which also did not show any changes in response bias. The two treatment groups, however, did show a change in response bias, toward a more conservative criterion for reporting pain. The TENS procedure had a broader range of effectiveness across all levels of the dental stimuli. The authors concluded that:

The effects of acupuncture were most pronounced at the lowest level of stimulation, while TES (i.e., TENS) affected the perception of all levels of dental stimuli. The observed effects appeared to be small, reliable, and dependent on the stimulation of a particular anatomical locus. (1976, p. 256).

Somewhat different results were reported by McCreery and Bloedel (1978), who used thermal stimuli as the pain agent and who also assigned subjects to "real" vs. "sham" TENS groups. The study reported 2 experiments, the second of which involved presentation of the TENS to different anatomical locations. The design and results were somewhat complex and will not be reported in detail but are interesting for at least two major points: (1) discriminability (d') changed in one of the sham groups, "suggesting that discriminability can be affected by changes in mental state which may occur during the evaluation of an analgesic" (p. 38); (2) response bias changes without accompanying changes in discriminability, which was interpreted by the authors as "reflecting actual analgesic, rather than only changes in response bias, produced by TENS" (p. 38). The authors acknowledged, however, that the results could have been confounded at least in small part by the
alternation of "on" (real) and "off" (sham) trials, so that the "maximal analgesic effect of electrical stimulation may not have been achieved" (p. 56). In spite of this possible confound, the authors do raise the criticism of SDT (amplified by Rollman, 1979) that response bias changes without changes in discriminability do not preclude actual analgesia. These issues will be addressed at the end of this section on SDT, when various criticisms of the theory as it applies to pain research are considered.

Another study which evaluated the effectiveness of the TENS procedure was reported by Malow and Dougher (1979). The pain stimulus was produced by the Forgione-Barber Focal Pressure Stimulator (Forgione & Barber, 1971). The TENS procedure was given in an ipsilateral and contralateral location and compared to a control group. Results of a traditional pain threshold analysis were that "the ipsilateral TENS condition significantly reduced ratings and increased pain thresholds relative to the contralateral and control conditions which did not significantly differ from each other" (p. 101). Using an SDT analysis of the data, findings were that the ipsilateral TENS condition reduced pain sensitivity (d') and also changed response bias in the direction of adopting a more conservative criterion for reporting pain. The effects of the ipsilateral TENS were found to be strongest at the higher levels of stimulation. The authors concluded that the results were consistent with gate control theory, which postulates that ipsilateral TENS reduces pain sensation.
The only other study to report an evaluation of TENS procedures also evaluated the use of implanted electrical stimulators (dorsal column, anterior cord, and sciatic nerve stimulators). This study was done by Bloedel, McCree and Erickson (1975), who noted that "implantable devices produced changes in both discrimination and response bias" (1977, p. 78), whereas transcutaneous stimulators produced in what Bloedel, et. al., expressed as "changes primarily in bias" (p. 185).

In summary, findings regarding the effectiveness of TENS for pain via SDT analysis are equivocal, with Bloedel and colleagues generally finding no changes in discriminability (d') and others finding clear changes in the same measures. Again, differences may be attributable to different pain stimuli, different procedural approaches and the site of stimulation. Overall, however, the TENS procedure does appear to hold promise as a viable technique for clinical pain relief and more controlled SDT research is needed.

The SDT approaches discussed so far have all involved "physical" manipulations (e.g., drugs, acupuncture, TENS, electrical implants) and "attitudinal" variables have been only indirectly investigated. Only a very few studies using SDT analyses have been done which have assessed human pain response as a function of "non-physical" (i.e., "attitudinal/psychological") procedures and, as will be seen, even these have been plagued by methodological problems.

Before proceeding to a review of the relatively few studies which have directly investigated psychological/cognitive variables, it should
be noted that earlier cited studies have looked at "psychological" components of pain via placebo groups. Some of the aforementioned studies which investigated the effect of a placebo on pain response using SDT analysis (e.g., Clark, 1969; Feather, Chapman & Fisher, 1972; Hall, 1977) compared a placebo to no placebo. Other studies cited above have compared a placebo to a drug treatment condition using an SDT analysis (e.g., Chapman & Feather, 1973; Lineberry, et al., 1975). To date, these are apparently, however, studies which have investigated purely attitudinal/psychological variables using an SDT analysis of the pain response.

The earliest apparent attempt to evaluate attitudinal/psychological variables using an SDT approach evaluated the effects of experimenter modeling on subjects' pain perception (Craig & Coren, 1975). It was an outgrowth of earlier work by Craig and his colleagues which demonstrated that a subject's report of pain can be influenced by a confederate/model who is tolerant or intolerant of electric shock. Briefly, subjects were assigned to one of three groups: (1) control; (2) exposure to a tolerant model (who rated shocks at lower intensity than the subject); and (3) exposure to an intolerant model (who rated shocks at higher intensity than the subjects). These results cannot be considered valid as there was a serious methodological confound: the shock intensities given the subjects varied as a function of treatment group (Hall, 1977, p. 79).

A second study which investigated psychological variables as they affected pain responses via SDT analysis was reported by Hall (1977).
This was the second in a series of three SOT studies reported by Hall in the same paper. (The first was an attempted replication of Clark's (1969) original placebo effect.) In experiment two, Hall sought to investigate the effects of suggestion and distraction on pain perception using an SOT analysis. He used a 2 x 2 factorial design. Two conditions varied the way the experimental instructions (i.e., suggestion) were presented: (1) as a pain experiment vs. (2) a memory experiment; two other conditions varied whether the distraction task (memory for digits presented over headphones) was presented: (1) during vs (2) between presentation of the noxious stimulus.

For d', findings were that there were no significant main effects and no interaction effect for all treatment conditions. Hall concluded that "instructions and task had no effect upon discrimination, and there was no interaction between instructions and task" (1977, p. 105). Similarly, non-significant findings were reported for Cx. A separate analysis of a memory score indicated that those subjects who did the memory task between noxious stimuli presentation scored higher (i.e., did better) than those who performed the task during the presentation of the noxious stimuli. Hall speculated that the negative findings may have been due primarily to: (1) "clinical" impressions that attempts to manipulate suggestion were not successful (e.g., the subjects may have been unclear about instructions); and (2) the distraction task may not have been difficult enough to adequately divert the subjects' attention (e.g., an average of 75% of all subjects made correct responses).
Another study which sought to investigate the effects of psychological variables on pain perception via an SDT analysis was also reported by Hall (1977) as the third in a series of three experiments. In this experiment, he investigated the effects of what he termed "hypnotic analgesia" versus "imagined analgesia". For the hypnotic analgesia condition, he used a conventional 10-minute hypnotic induction procedure followed by suggestions of analgesia. For the imagined analgesia condition, he gave subjects instructions to imagine insensitivity in the arm where they received the noxious stimuli. A no-treatment control condition was also used. In addition, within each group subjects were equally divided for high versus low hypnotic susceptibility, on the basis of their scores on form A of the Harvard Group Scale of Hypnotic Susceptibility (Shor and Orne, 1962). Results were that, with regard to $d'$, the hypnotic and imagined analgesia groups differed significantly from the no treatment control group. There was also an interaction between level of susceptibility and treatment level. To summarize the findings for $d'$, Hall concluded that the hypnotic and imagined analgesia procedures reduced discrimination relative to the no-treatment groups, but that the amount of reduction in $d'$ varied with the susceptibility of the subjects, being largest for the low susceptible subjects. The findings for $C_X$ all failed to achieve statistical significance, i.e., there were no main effects for treatment or susceptibility nor any interaction effects. On a separate measure of hypnotic depth subjects who were high in susceptibility rated themselves as in a deeper state of hypnosis than did subjects low in susceptibility; this effect held
regardless of subject's treatment condition. Hall's conclusions regarding these findings are noteworthy:

The fact that the hypnotic and imagined analgesia groups differed from the control group but not from each other on d' suggests that the hypnotic induction is not necessary for the production of analgesia. It also provides support for the conjecture of Barber, Spanos and Chaves (1974) that the hypnotic analgesia procedure provides an implicit cognitive strategy for controlling pain. The absence of any differences between high and low susceptible subjects in the effects of hypnotic and imagined analgesia suggests that a high level of susceptibility is not necessary for the production of analgesia. Taken together these results seem to 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. (emphasis added) The presence of an interaction between level of susceptibility and treatment group complicates this conclusion but does not fundamentally change it....(added below). The qualification is: highly susceptible subjects are more likely to become involved in the type of imaginative activity required to produce analgesia. (1977, pp. 122-123)

Hall's findings may be considered surprising in that they represent the first conclusive evidence that a psychological ("cognitive") variable can affect sensory discrimination (d'). It should be noted that the Craig and Coren (1975) study mentioned above did not present conclusive evidence for a change in sensory discrimination because of serious methodological flaws. For this reason alone, replication attempts should be made. But Hall's findings that the hypnotic analgesia and imagined analgesia groups showed a significant reduction in d' over the control group and that this implies that the hypnotic induction procedure is unnecessary, is not completely warranted because of the design of his treatment group instructions. In fact, it appears that the actual "suggestion of analgesia" part of the hypnotic analgesia conditions differ from the suggestions given in the imagined analgesia
condition. The crucial point is this: in the hypnotic analgesia condition, the suggestions appear to focus entirely on feelings of numbness in the arm (see Appendix D.2, p. 187). This discrepancy in instructions does not per se invalidate the findings of a change in $d'$ for both treated conditions. No empirically sound conclusion can be made regarding the role of the hypnotic induction in Hall's experiment given the non-equivalence of the analgesia suggestions. It can be concluded, however, that analgesia suggestions (imagining the arm as numb or imagining the arm as numb plus reinterpreting the noxious heat stimulus as pleasantly warm) have demonstrated effectiveness in reducing discrimination ($d'$) for radiant heat stimuli. This conclusion will serve as a basis for the present study.

A final study which investigated the effect of psychological variables on pain perception using an SDT analysis was reported by Dougher (1977). In this study, the author investigated the effects of trait anxiety and experimental instructions on pain response produced by the Forgione-Barber focal pressure pain stimulator. Threshold values were used and the data were also subjected to an SDT analysis. The experimental instructions were designed to affect the subject's response bias. Subjects were divided into two groups, high versus low anxious, on the basis of their scores on a forced choice version of the Taylor Manifest Anxiety Scale (TMAS). Within each of these two groups, half were given facilitative instructions (i.e., were told that a reluctance to report pain is associated with emotional problems); the other half within each groups were given inhibitory instructions (i.e., were told
that a tendency to report pain too quickly is often associated with emotional problems). The design was thus 2 x 2 factorial design, with two levels of anxiety and two of instructions. Subjects were the 49 lowest and 49 highest scorers on the TMAS (who met inclusion criteria) of an original pool of approximately 500 undergraduate volunteers.

Threshold analysis findings were that high anxious subjects reported pain significantly sooner than low anxious subjects. The effect of instructions was, as expected, also significant, i.e., the subjects who received facilitative instructions reported pain sooner than those who received inhibitory instructions. This effect of instructions occurred only on the last four trials (relative to the first two). There were no significant interactions between anxiety and instructions.

For the SDT analysis, nonparametric measures of sensitivity and response bias were employed (see Green, 1964) "because the data did not strongly confirm the assumptions of normality and homogeneity of variance necessary for a traditional signal detection analysis" (Dougher, 1977, p. 140). Individual data were combined to yield group indices which were computed as if each group were a single observer receiving 144 trials. Results of the SDT analysis were that there were no differences in sensitivity among the four groups but that there were significant differences for response bias. Anxious subjects used lower criteria for reporting sensations as painful than did non-anxious subjects. Dougher noted that although his findings indicated that the
anxious subjects were no more sensitive than non-anxious subjects to pain stimuli, the precise relationship between trait anxiety and the tendency to report pain is still not clear. He further noted that the effect of state anxiety on sensitivity and response bias had not yet been ascertained and is a fruitful area for future research.

Two recent studies which have used an SDT model to investigate the influence of "psychological" treatments on acute pain were done in the same laboratory as the present study (Hatcher, 1982; Luscomb, 1980).

The first of the two (Luscomb, 1980) compared three treatment groups: (1) relaxation training; (2) cognitive attention-redirection; and (3) distraction (listening to a story) with a no treatment control and an expectancy control group.

The main findings were: (1) none of the treatments affected a change in the sensitivity component; (2) only for the cognitive attention-redirection group was there a change in response bias (it became more conservative). The cognitive attention redirection group was the only one which had significantly lower mean pain ratings. All groups except the cognitive attention-reduction group showed significant reductions in state anxiety on the posttest.

The second study from this lab (Hatcher, 1982) investigated the effects of videotaped modeling and imagined analgesia instructions via a comparison of four treatment groups: (1) tolerant modeling (2) intolerant modeling; (3) modeled imagined analgesia (composed of a combination of tolerant modeling plus imagined analgesia instructions);
and (4) imagined analgesia instructions with no treatment and expectancy control groups.

Nonparametric measures of sensitivity and bias were used. The main findings were: (1) three of the treatment groups (tolerant modeling, modeled imagined analgesia and imagined analgesia instructions) showed a significant reduction in response bias; (2) there was a very low level reduction (for comparisons of stimulus levels 0 and 1) in sensitivity measures for the tolerant modeling and imagined analgesia instructions. In addition results revealed a significant reduction in state anxiety from baseline to posttest.

In summary, there has been a dearth of studies using SDT to analyze the effectiveness of psychological/attitudinal/cognitive treatments (hereafter referred to as "psychological" treatments) on subject's responses to noxious stimuli. A particular lack of attention has been given to cognitive strategies (i.e., those strategies which use imagery to somehow alter or reinterpret the stimulus). These strategies have been evaluated fairly extensively, however, using traditional tolerance (and threshold) measures. This body of literature will be reviewed in the following section, but first brief consideration will be given to some of the criticisms which have been leveled against the use of SDT for pain research.

A Critique of SDT

A full review of all of the theoretical and statistical considerations and the various models of SDT are beyond the scope of this review, but are covered amply elsewhere (e.g., Green & Swets, 1966;
Hall, 1977; McNicol, 1972). To date, the weight of evidence supports the viability of SDT as it applies to pain research (Chapman, 1977; Grossberg & Grant, 1978; Hodos, 1970; Jones, 1979; Velden, 1979). Criticisms have been several and varied, but, in this reviewer's opinion, have not been substantive enough to challenge the fundamental applicability of SDT to pain research. Clark and Mehl (1973), for example, assessed the measurement of d' as determined by different types of rating tasks: (1) a one-interval binary rating task; (2) a one-interval 12 point confidence rating task; (3) a one interval 12 point magnitude rating task; and (4) a two-interval forced choice task. Seventeen radiant heat intensities were judged. A d' value was determined for each of the four tasks. Results were that d' differed between tasks and also varied with stimulus intensity. In addition, the d' values for binary tasks (both one and two-intervals) were larger than the d' obtained by rating scale tasks (i.e., confidence and magnitude ratings). As noted by Hall, Clark and Mehl (1973) concluded that "to eliminate this problem investigators should employ a binary high-low decision or forced choice decision to estimate d' and a rating-scale task to determine the location of the pain criterion" (Hall, 1977, p. 70). A follow-up to Clark and Mehl's (1973) study was done by Clark and Dillon (1973), who made comparisons of d' values for binary and magnitude-rating tasks when these two judgments were made singly or concurrently. As noted by Hall, the findings of Clark and Dillon agreed in part with the earlier results of Clark and Mehl (1973): d' values for the binary task were larger than those for the rating scale task.
Clark and Dillon (1973) did not, however, find any significant differences between the d' or $L_\infty$ values determined from single versus concurrent judges. Clark and Dillon repeated the earlier recommendation of Clark and Mehl that, for d' a binary task should be used, whereas for locating the subject's pain criterion, a rating scale task should be used. After a cursory review of these studies, Hall concluded that "neither Clark nor any other reseacher has followed this recommendation for the very good reason that the increase in labour which it entails procures only a small increment in experimental precision" (1977, p. 70).

The effects of age on sensitivity and response bias have also been investigated (Clark & Mehl, 1971; Harkins & Chapman, 1976) and results indicate that changes in both sensitivity and willingness to report pain (response bias) occur in the elderly. In addition, Clark and Mehl (1971) found that the sex of the subject (especially for older females) may influence the SDT values obtained. Again, however, these findings suggest precautions to be taken in interpreting results involving the variables of ages and sex. They are not a fundamental challenge of the theory.

The primary critic of SOT as it is used in pain research has been Rollman (1977, 1979). His earlier criticisms attacked the technical feasibility of SOT (e.g., necessity for numerous trials, repeatability of discrete stimuli, etc.). In addition, he has implied (1977, 1979) that SDT is not an appropriate methodology for pain research. Replies to this implication by Rollman have focused in large part on lack of
acknowledgment that some (of the several) SOT models seem indeed to be relevant to pain research (Chapman, 1977; Jones, 1979; Velden, 1979). Jones (1979) in particular has made a cogent argument that:

Rollman misleadingly gives the impression that he is questioning the general validity of the application of TSD (i.e., SOT) to answering questions in pain research. This is not the case. Procedures derived from TSD may play an important part in answering questions of interest to pain researchers especially as Rollman has exaggerated the practical difficulties associated with TSD experiments. It must be emphasized that there is no one TSD model as Rollman often seems to imply but a variety of models (Green & Swets, 1966; McNichol, 1972). The usefulness of any model in any situation is a matter for experiment. Rollman has certainly criticized effectively one dubious analogy of TSD procedures which pain researchers have used (i.e., their failure to realize, particularly with studies using analgesics, that the discriminability of two ordinarily painful stimuli may not be related to their reported painfulness). This is not the same as questioning the general usefulness of TSD models. (1979, p. 305).

As can be seen by the above review, SOT appears to offer a valid and reliable means of assessing sensitivity (discriminability) and response bias estimates of pain. It has been little applied to psychological treatments, per se, especially to "cognitive" strategies, which involve primarily imaginal alterations or reinterpretations of the pain stimulus. Research on these strategies will be reviewed below.

"Cognitive" Strategies in the Treatment of Pain

This section will present an overview of the research (primarily laboratory) on "psychological" treatments for pain which have not used SDT models. "Psychological" techniques for pain treatment are here defined as those which generally involve suggestion, distraction, reinterpretation, or other strategies which affect the individual's cognitions and, hence, affect attitudinal and/or motivational
variables. There certainly does not appear to be a consensus on what are defined as "psychological" variables, nor is there apparently any consensus even on a system for determining where a given specific technique is classified. Two specific classification systems will be mentioned briefly below (cf. Hall, 1977; Meichenbaum & Turk, 1976).

Hall (1977, pp. 1-11) reviewed psychological methods for reducing pain and classified them generally into two categories: "suggestion" and "distraction" procedures. His definitions of these categories were as follows:

The suggestion category includes methods in which suggestions of pain reduction are given verbally, alone or accompanying a placebo, or are communicated to the person by means of a model. The distraction category includes methods in which an attempt is made to "distract" the person by having him attend to stimuli presented by the experimenter or engage in fantasy and imagining during the presentation of noxious stimuli. (1977, p. 2).

Hall also includes a third category which he terms "hypnotic analgesia". He maintained that this category had attributes which could place it in either the suggestion or distraction category, but he favored assigning it to a separate category.

Hall concluded that evidence indicated that both distraction and hypnotic analgesia techniques were successful in reducing pain. He did not offer a summary statement regarding suggestion procedures, but apparently did not evaluate them as favorably, especially for specific techniques (e.g., modeling).

Meichenbaum and Turk's (1976) conceptual schema regarding psychological intervention strategies for pain reduction is somewhat different from that of Hall. They conceptualized a number of the techniques as
cognitive strategies. They also described coping skills strategies and self-reinforcement strategies within a stress-inoculation framework. They maintained that the cognitive strategies affect the "motivational-affective" component of pain. They conceptualized the cognitive strategies under three major categories: (1) attention-diversion; (2) somatization; and (3) imagery manipulations. Under this third category was included "imaginative transformation of pain," which involved suggestions for analgesia.

One technique which can be found in these conceptual schemata and which has proven effective in the reduction of laboratory-induced pain involves the use of suggestions for "analgesia" in the part of the body subjected to pain. This treatment typically involves the use of cognitive strategies to imagine the affected body part as numb and/or insensitive. This procedure may be given with or without an hypnotic induction. In general, results have shown that the procedure is just as effective without an hypnotic induction although there is still some controversy about this issue (Hilgard, 1977; Spanos, Radtke-Bodorick, Ferguson & Jones, 1979). Evidence also points to the fact that subjects who are rated as highly susceptible (on standardized hypnotizability scales) are more responsive to suggested analgesia treatments, at least when tolerance measures are used. As noted above, virtually all of the studies investigating the effects of suggested analgesia have been done in the laboratory and have virtually all involved traditional measures of tolerance and/or threshold. Overall, the results have indicated that suggested analgesia significantly reduces subjects' verbal reports of
pain (and in some instances physiological responses) when compared with control group subjects. In addition, suggested analgesia has been generally shown to be at least equivalent, if not superior to, other cognitive strategies for pain reduction. Following is a review of these studies. A rationale will then be offered for investigating this variable in the present study. Use of a signal detection methodology will then be proposed to partial out the sensory vs. the response bias components of the pain response in the present study.

The first study to investigate the effects of suggested analgesia was done by Barber and Hahn (1962). Subjects were 48 female undergraduates who scored high (5.5 or higher on 8 point scale) on a Suggestibility scale. Subjects were equally divided into four experimental groups: (1) hypnotically suggested analgesia, i.e., imagining hand to be numb and insensitive (including an hypnotic induction); (2) waking-imagined analgesia (no hypnotic induction); (3) uninstructed (no analgesia); and (4) controls. All subjects except those in the control group immersed one hand in 2°C ice water. Main findings were that hypnotically-suggested analgesia appeared to be no more effective than waking-imagined analgesia in reducing pain reactivity. Both conditions produced reports of reduced pain, showed a reduction in muscle tension and respiratory irregularities, but did not alter cardiac acceleration or produce a drop in GSR. On the average, both the hypnotically-suggested analgesia and the waking-imagined analgesia groups reported significantly less pain than the uninstructed
group. The control group subjects also, on the average, differed significantly on mean pain ratings from the three experimental groups.

A second study to examine the effects of suggested analgesia, with and without hypnotic induction was done by Evans and Paul (1970). Sixty-four female undergraduate students served as subjects, and were equally divided among 4 treatment groups: (1) hypnotic induction plus analgesia suggestion; (2) hypnotic induction alone; (3) waking self-relaxation plus analgesia suggestion; and (4) waking self-relaxation alone. Subjects were given the Harvard Group Scale of Hypnotic Susceptibility (HGSHS) and equally distributed among the four groups four levels of hypnotic susceptibility. Main findings were, for self-report data: (1) subjects given analgesia suggestions reported a greater reduction in subjective distress in response to the ice-water stressor than those subjects not given such suggestions; (2) hypnotic induction alone did no better than self-relaxation instructions; and (3) hypnotic induction did not significantly facilitate the effects of suggested analgesia. Also, for self-report data, the effect of suggestion varied with hypnotic susceptibility, whether or not the suggestion was accompanied by hypnotic induction. For the physiological response measures, there was no evidence for the effectiveness of hypnotically suggested analgesia, hypnotic induction alone or suggested analgesia alone in suppressing the physiological responses to the ice-water stressor, whether subjects were high or low on hypnotic susceptibility.
A study by Greene and Reyher (1972) investigated the effects of electric shock on highly suggestible subjects who were given suggestions for analgesia, pleasant imagery, or the two conditions combined. Subjects were 36 female undergraduates who scored 8 or higher on the HGSHS. Half were placed in a simulator group (i.e., asked to act as if they were hypnotized) and half in a group given a hypnotic induction prior to each of the three experimental conditions. Groups were matched on the basis of subjects' hypnotic susceptibility. Main findings were that, for hypnosis subjects, increases in tolerance for the analgesia and analgesia plus pleasant imagery conditions were significant (at the .01 level) but not for the pleasant imagery alone conditions. The tolerance increases for simulators were significantly lower than that for hypnotic subjects. It was also found that, contrary to the experimenters' hypothesis, individuals with the highest levels of state anxiety prior to the tolerance tests did not exhibit the largest changes in tolerance under experimental conditions. An analysis of the extent to which subjects in the analgesia vs. pleasant imagery conditions used involved (non-body oriented) imagining also revealed no significant differences. Overall "natural" (non-body oriented) imagery resulted in increased tolerance across groups.

A study done by Johnson (1974) compared the effects of suggestions for brief relaxation alone, relaxation with suggestions to imagine the hand as warm, and relaxation with suggestions to imagine the hand as numb. An uninstructed control group was also used. The pain stimulus
was 40° F ice water, into which subjects immersed their hand in a
tolerance test (maximum of 3 minutes). Subjects consisted of 40 male
volunteers who were enlisted men in the army. Main findings were that,
for subjective pain scores, both the relaxation only group and the
imagined numbness group reported levels of pain significantly below that
of the uninstructed control group. Although the relaxation and the
imagined numbness groups did not differ significantly from each other,
the imagined numbness group reported slightly lower pain levels. The
imagined warmth group did not report a level of pain significantly lower
than the control group, but it also did not differ significantly from
pain levels reported by the relaxation group. None of the physiological
response measures for the treatment groups differed significantly from
the control group.

A study by Chaves and Barber (1974) compared the effects of
suggestions to imagine pleasant events with suggestions to imagine a
finger as insensitive; in addition, a regular control group and an
expectancy control group (i.e., subjects given the expectancy of a
reduction in pain, but not provided with cognitive strategies) were also
employed. An experimenter modeling condition (i.e., with the
experimenter modeling increased tolerance of pain) was also given to
half of the subjects in each experimental group. Main findings were that
subjects who used the cognitive strategies of imagining pleasant events or
imagining the finger as insensitive showed a reduction in self-reported
pain as compared with uninstructed controls. Subjects given the expectancy
of a reduction in pain, but not provided with cognitive strategies also
reported decreased pain in the posttest when compared with control subjects, but the reduction was smaller than for subjects using the cognitive strategies (i.e., imagine pleasant events or imagine the finger as insensitive). Overall, the experimenter modeling procedure was not effective. It was effective in reducing verbal reports of pain only for those subjects with high pretest pain levels who were asked to imagine pleasant events. Experimental subjects who reported greater use of cognitive strategies also reported greater reductions in pain.

Barber and his group (Spanos, Barber, & Lang, 1974) reported yet another study regarding suggestions for anesthesia the same year as the Chaves and Barber study mentioned above. Spanos, et. al., (1974) used a 2 x 2 x 2 factorial design, varying hypnotic induction vs. no induction; anesthesia instructions vs. no instructions; and demands for honesty vs. no demands for honesty. Eighty subjects, both male and female, participated in the experiment. The pain stimulus was administered via the Forgione-Barber pain stimulator, with a 2,000 gram weight applied to the finger for a maximum tolerance time of no longer than one minute. Main findings were as follows: (1) subjects who received the anesthesia instructions reported significantly lowered pain ratings than those who did not receive anesthesia instructions; (2) combining the anesthesia instructions with the hypnotic induction procedure did not produce a greater reduction in pain than giving the anesthesia instructions alone; (3) subjects who were not exposed to demands for honesty did not report significantly different degrees of pain than those who were exposed to demands for honesty; and (4) there were no significant interactions
between anesthesia instructions, hypnotic induction procedure and demands for honesty. It should also be noted that subjects who rated themselves as more deeply hypnotized did not show a significantly greater reduction in pain than those who rated themselves as less deeply hypnotized. Spanos et. al., hypothesized that the active ingredients in the anesthesia instructions may consist of: (1) the subjects' being informed of, and believing in, the fact that he/she could control their sensory experiences and that such control had been successfully exercised by previous subjects; and (2) the suggestions provided the subjects with a strategy for exercising control over pain in which the stimulated body part is thought of in a manner which is inconsistent with the perception of pain (emphasis added). Spanos, et. al., also cited evidence (e.g., Barber & Hahn, 1962, Hilgard, 1969) that physiological indices of pain, which are not under direct voluntary control, are reduced (together with verbal reports of pain) when suggestions for anesthesia are given. This kind of evidence recommends itself to testing the effects of anesthesia suggestions using a signal detection format, with which sensory discrimination and response bias can supposedly be partialled out.

Stacher, Schuster, Bauer, Lahoda and Schulze (1975) used a within-subjects' design to investigate the effects of suggestions of relaxation vs. suggestions of analgesia in both the "hypnotic" and "waking" states. Subjects were four female volunteers who were selected because of high susceptibility (rigid left arm catalepsy and posthypnotic amnesia) scores based on a prescreening. The pain stimulus was provided via an electrical stimulus attached to the ear lobe. Subjects signalled both
threshold and tolerance measures by use of a manually operated key. The
four treatments were given to each subject in a counterbalanced order.
Main findings were: (1) suggestions of relaxation given in the waking or
in the hypnotic state were found to be less effective in elevating pain
threshold and tolerance than suggestions of analgesia under the same
conditions; (2) suggestions of both relaxation and analgesia resulted in
higher increases of threshold and tolerance under hypnotic than under
waking conditions. The authors hypothesized that "the more accentuated
changes in pain threshold and tolerance under hypnosis may result from
the fact that the subject's attention is focused more intensely on the
experimenter than in the non-hypnotic condition" (p. 264). This
statement appears to be more in line with the view of Hilgard (cf.
1969), i.e., that hypnosis involves an altered state of consciousness,
and is in opposition to the views of Barber (as stated above), i.e., that
"hypnotic" suggestions do not in fact differ from "waking" suggestions.
Evidence from a recent article (Stam and Spanos, 1980) indicates,
however, that the alleged superiority of "hypnotic" vs. "waking"
suggestions may be an artifact of within-subject experimental designs and
expectancies attached to "hypnosis". These issues will be dealt with in
greater detail at the end of the present section reviewing studies which
investigated the effects of suggestions for analgesia.

A study which specifically investigated several parameters of
analgesia suggestion was done by Spanos, Radtke-Bodorik, Ferguson and
Jones (1979). There were a total of 96 subjects, 50% of whom were males
and 50% females. Subjects were pretested using the HGSHS and were
classified as low (score of 0-3), medium (score of 5-7), or high (score of 9-12) susceptible. There were 32 subjects (16 males and 16 females) at each susceptibility level. The four groups were: (1) hypnotic induction procedure alone; (2) hypnotic induction procedure plus analgesia suggestion; (3) analgesia suggestion alone; and (4) control (no induction - no suggestion). Subjects were given cold-pressor pain via arm immersion in ice water (0° - 2° C). There was a post-experimental interview with ratings for: (1) distraction; (2) imagining inconsistent with pain; (3) relaxation; and (4) catastrophizing. Main findings were: (1) subjects given analgesia suggestions reported significantly less pain on the second immersion trial than on the first; (2) there were no significant differences for subjects not given suggestions; (3) there were no significant effects for the hypnosis/non-hypnosis variable. It was also noted that high and medium susceptibles reported significant drops in pain from first to second immersion; low susceptibles, however, showed no significant change. High susceptibles also used significantly more cognitive strategies than low susceptibles. There was a trend for subjects who were given a suggestion for analgesia to use more strategies than those not given a suggestion (and also to report significantly less pain). The single best predictor of reporting or not reporting a pain decrement was catastrophizing: 88% of catastrophizers reported no decrement or increment in pain (whereas only 39% of non-catastrophizers failed to report a decrement). In summary, this study replicated previous studies by finding that pain reduction was enhanced by both high susceptibility and by administration of suggestions for analgesia.
A study by Knox, Gekoski, Shum and McLaughlin (1981) was designed primarily to investigate the effect of hypnotic susceptibility of acupuncture analgesia, but also was designed to compare acupuncture and hypnotic analgesias. Experimental subjects were given a series of trials consisting of acupuncture followed by "hypnotic analgesia" (i.e., an hypnotic induction followed by suggestions for insensitivity in the hand and arm). The rating scale varied from 0 (no pain) to 10 (intense pain). Although the primary hypothesis was disconfirmed (i.e., responsivity to acupuncture did not increase over sessions), the effectiveness of hypnotic analgesia was demonstrated. High-susceptible subjects (in both experimental and control groups), but not low susceptibles, showed dramatically lower mean pain reports after receiving suggestions for hypnotic analgesia.

A study mentioned above (Stam & Spanos, 1980) focused on several variables which mediate response to "hypnotic analgesia". Two significant findings were concerned with biases that could be introduced by within-subjects' experimental designs. First of all, it was found that significantly different effects could be obtained by varying the order of suggested "waking" vs. "hypnotic" analgesia treatments. Secondly, and perhaps as an extension of the first point, it was found that different expectancies could be introduced, e.g., if a subject was given a "hypnotic" condition after a "waking" condition. A brief review of this study will highlight some of these issues and serve as a springboard to summarize findings on the effects of suggestions of analgesia on experimentally induced pain.
Subjects for the Stam and Spanos study were 20 male and 20 female undergraduates who scored high (9-12) on the HGSHS. A cold pressor test, consisting of submersion of the subject's hand and forearm in ice water (0° to 2°) was given. Subjects were assigned to one of four groups, which each had a series of three trials, beginning with a baseline trial. Trials two and three differed for the four groups as follows: (1) for Group One, suggested "waking" analgesia (called "suggested analgesia" henceforth) was followed by hypnosis plus suggested analgesia; (2) for Group Two, suggested analgesia was followed by another trial of suggested analgesia; (3) for Group Three, hypnosis plus suggested analgesia was followed by suggested analgesia; and (4) for Group four, no treatment was given for both trials. The primary dependent variables consisted of a magnitude estimation measure (giving a numerical value to a line of a certain length) and category ratings (ranging from 0 for "no pain" to 10 for "excruciating pain"). Other dependent measures consisted of demands for honesty and estimates of cognitive activity during the pain rating task (e.g., "coping" vs. "catastrophizing" vs. coping plus catastrophizing). Main findings for the magnitude estimation and category scale ratings were that hypnotic analgesia was more effective than, less effective than, or equally as effective as waking analgesia, depending on the order of trials presented. The authors made the general observation that subjects who knew that they were going to be hypnotized refrained from performing maximally during the waking condition in order to enhance performance during hypnosis. It was also observed that the findings strongly
supported the hypothesis that the superiority of hypnotic to waking analgesia found in within-subjects experiments (cf. Hilgard, 1969) is due to a carryover of expectations that results when the same subjects are tested under both treatments. The authors also noted that the findings contradicted the hypothesis that hypnotic analgesia is intrinsically more effective (in terms of significantly decreased reports of pain) than waking analgesia. The authors also maintained that this argues strongly against the hypothesis that hypnotic analgesia involves special cognitive activity, it was found that for the third (and final) trial the higher the percentage of time that subjects engaged in coping imagery, the less pain they reported. In addition, treatments that were associated with the greatest reductions in reported pain on the third trial were also associated with the highest levels of coping imagery. Stam and Spanos emphasized that suggested analgesia is mediated by the subjects' use of coping strategies. Stam and Spanos' conclusions are worth quoting in detail:

Our view emphasizes the strategic nature of hypnotic responding. This perspective conceptualizes good hypnotic subjects as strongly invested in validating their hypnotic enactments by moderating their behavior to conform with treatment-generated expectations (Barber, Spanos & Chaves, 1974; Coe & Sarbin, 1977; Orne, 1959; Spanos, in press; Spanos, et. al., 1980). As mentioned above, this perspective views reports of nonvolition as reflecting subjects' attributions concerning the causes of their own behavior. These self-attributions are an aspect of the role subjects enact and are influenced by such psychological variables as preconceptions concerning "appropriate" responding and the wording of suggestions (Spanos & Barber, 1982; Spanos, et al., 1980). None of this implies that subjects fake analgesia in the absence of corresponding changes in experienced pain or report their activities as feeling effortless when they in fact feel effortful. Instead, it
suggests that subjects modify their subjective experiences as well as their overt behavior in conformance with treatment-induced expectations. (Stam & Spanos, 1980, p. 760)

Stam and Spanos further hypothesized that, to say that subjects have reduced pain following suggestion does not mean that they were necessarily less able to discriminate between different intensities of painful stimulation or were less sensitive to pain (cf. Rollman, 1977). The authors hypothesized that "reduced pain" under these circumstance may indicate that subjects have relabeled or redefined (rather than lessened the intensity of) their sensory experiences. Following this line of reasoning further, Stam and Spanos concluded:

Therefore, sensory events previously labeled as "high pain" may now be categorized in some alternative way (e.g., intense but not painful, very cold, numb, strong prickly sensation). Although difficult to discriminate at an operational level, it is important to maintain the theoretical distinction between the notion of redefining intense sensory experiences as "not painful" and the notion of privately defining sensory experiences as painful while publicly reporting that they are not. (1980, p. 760)

A signal detection theory methodology would enable this distinction between "public" verbal report (i.e., response bias) and "private" sensory experience (i.e., sensory discrimination) to be partialled out. Apparently only two studies have effectively used this methodology to investigate the effects of suggested analgesia (Hall, 1977; Hatcher, 1982). They involved very brief (under 10 minutes) analgesia instructions. The rationale for the present study was based on a longer, more intensive set of suggested analgesia instructions and exercises.
Rationale and Hypotheses

Based on the above review, evidence indicates that suggestions for analgesia are effective in reducing verbal reports of experimentally induced pain. To date, all but a very few of the studies have used traditional tolerance or threshold measures. The two reported studies (Hall, 1977; Hatcher, 1982) which have investigated suggested analgesia using an SDT paradigm have employed short (no longer than 5 to 10 minutes) one-time instructions and have provided little or no in vivo laboratory practice with the analgesia technique. There is evidence to indicate that "live" (i.e., in vivo) training is more effective than tape recorded training, e.g., with relaxation training (Israel & Beiman, 1977). It could be that the failure to find significant and prevailing changes in d' with "psychological" treatments (e.g., Hatcher, 1982) may have been due at least in part to use of taped and/or brief "live" training.

There is evidence from laboratory studies using threshold and tolerance measures and SDT measures that a hypnotic induction procedure is not necessary for an increase in analgesia (i.e., a decrease in reported pain) to occur. There is also no conclusive evidence that suggestions of analgesia preceded by a hypnotic induction are superior to suggested analgesia without the hypnotic induction. Accordingly, the present study sought to investigate the effects of suggestions of analgesia (without a hypnotic induction) using more extended (three 30 minute training sessions) practice, using "live" (in vivo) training.
There is also a body of experimental evidence, largely with the use of traditional (threshold and tolerance) measures, that suggests that level of hypnotic susceptibility affects pain response. Specifically, in the threshold and tolerance studies, subjects who are high in hypnotic susceptibility typically give significantly lower post-training pain ratings than subjects low in susceptibility. Hall's study (1977) is the only reported study to investigate the effects of high vs. low susceptibility on suggested analgesia using an SDT methodology. Hall found a decrease in mean $d'$ for both the hypnotic analgesia and the imagined analgesia groups when compared to the control group, but the difference was greater for low susceptibility subjects, i.e., the low susceptible subjects in the control group had the highest mean $d'$ scores. Hall attributed this finding at least in part to the fact that

The discrimination of high susceptibility subjects in the control group has been underestimated because, unlike the low susceptible subjects, they spontaneously engaged in the type of fantasy and imagining that subjects in the other treatment groups had been instructed to engage in. (Hall, 1977; p. 123)

Giving support to this assertion was the fact that for the treatment groups the highly susceptible subjects reported themselves to be more deeply hypnotized regardless of treatment condition. Hall also noted that:

"the absence of any differences between high and low susceptible subjects in the effects of hypnotic and imagined analgesia suggests that a high level of susceptibility is not necessary for the production of analgesia. (1977, p. 123)

It should also be noted that when manipulation checks have been performed (e.g., self-report of depth of hypnosis or use of cognitive
strategies) greater hypnotic depth or use of imaging have been correlated with a decreased pain report.

The present study compared two groups: (1) an experimental group given suggested analgesia and (2) an expectancy control group given information and expectancy of reduced pain.

Based on the above review the following hypotheses were made for the present study:

1. The suggested analgesia groups would show a significant decrease in sensitivity to pain relative to the expectancy control group, and these differences would occur for comparisons of all adjacent levels of stimulus pain (e.g., stimulus level zero compared with level one, level one compared with level two, etc.). This is based on Hall's (1977) findings of decreased sensitivity across all levels and Hatcher's (1982) findings of changes in sensitivity for level zero and level one comparisons for his modeled imagined analgesia treatment. This change would be reflected in non-significant differences on pretreatment dependent measures and significant differences on post-treatment measures. There would also be a significant treatment by sessions interaction. This would also apply to the tests on the dependent measures in hypotheses 3 and 6 below.

2. Both the suggested analgesia and expectancy control groups would show significant and nondifferential decreases in response bias. This would be reflected in nonsignificant differences in pretreatment dependent measures and nonsignificant differences on post-treatment measures, There would be a nonsignificant treatment by sessions
interaction. These criteria would also apply to hypotheses 4 and 5 below. Hatcher (1982) found significant reductions in response bias for both of his imagined analgesia groups, although Hall (1977) did not. Previous studies which used powerful expectancy suggestions (e.g., placebo or strong expectations to decrease pain report) have found decreased response bias, although findings are somewhat equivocal.

3. The hypotheses in (1) and (2) above would be further expanded in that the effects would be significantly greater for high vs. low susceptibility subjects. This hypothesis was based largely on more traditional (threshold and tolerance) studies but at least in part on Hall's (1977) findings.

4. Mean pain ratings would decrease for both treatment groups. This hypothesis parallels findings by Hatcher (1982) and Luscomb (1980) and is in agreement with much of the research using traditional (tolerance and threshold) measures.

5. There would be no significant decrease in trait anxiety as a function of treatment group. This has been verified by two studies in the present laboratory (Hatcher, 1982; Luscomb, 1980).

6. State anxiety scores would significantly decrease across both the suggested analgesia and the expectancy control group. This has been verified by Hatcher (1982) and Luscomb (1980) and at least suggested by Dougher (1979).
METHOD

Subjects

A total of 32 subjects completed this study. All were undergraduate volunteers enrolled in Introductory Psychology at Virginia Tech. Twenty-two were male and 10 were female. Twenty-eight subjects were Caucasian, two were Black, one was from India and one was Oriental. Subjects were given course credit for their participation in the experiment. Of the 33 subjects who began this study one dropped out because he stated that he no longer wanted to endure the radiant heat stimuli.

Subjects were assigned to the treatment and expectancy control group after being screened for level of hypnotic susceptibility. The Harvard Group Scale of Hypnotic Susceptibility (HGSHS), Form A (Shor & Orne, 1962) was used to screen an original pool of 224 subjects. For purposes of this study subjects were assigned to the high susceptibility group if they scored 10 or higher (on a 12 point scale) and to the low susceptibility groups if they scored 4 or lower. Thus there were eight high and eight low susceptible subjects in both the suggested analgesia treatment group and the expectancy control group. Of the original pool of 224 subjects 32 (or 14.3%) had HGSHS scores of 4 and below; 147 (65.6%) had scores between 5 and 9; and 45 (20.1%) scores of 10 or higher.

Dependent Measures

A total of four different dependent measures was used: (1) the trait anxiety portion of the State-Trait Anxiety Inventory (Spielberger,
Gorsuch & Lushene, 1970); (2) the Present Affect Response Questionnaire (PARQ) from Endler (1976); (3) a scale for rating the radiant heat stimuli; (4) a scale for rating depth of analgesia (for the suggested analgesia group) and effectiveness of cognitive strategies (for the expectancy control group).

**State-Trait Anxiety Inventory**

The trait portion of the State-Trait Anxiety Inventory is a self-report inventory which purports to measure individual differences in anxiety proneness which are relatively stable over time (Spielberger, Gorsuch & Luschene, 1970). It is not designed to measure change which occurs as a function of situational stress. There are 20 items on the scale and the subject is asked to respond to how he/she generally feels. Each time is based on a 4-point scale: 1 = almost never; 2 = sometimes; 3 = often; 4 = almost always. A total score is obtained by summing each item (Items 1, 6, 7, 10, 13 and 19 are reverse scored). A higher score indicates a higher level of trait anxiety. See Appendix A for the complete scale.

**Present Affect Response Questionnaire**

This scale was designed by Endler (1976) as a self-report measure of state anxiety. It supposedly measures transitory emotional responses which occur in individuals when they interpret specific situations as personally treatening (Lamb, 1968). The scale consists of 24 items wherein the subject is asked to respond as to how he/she feels “at this particular moment.” Items are summed to yield a total score. Each item is based on a 5-point scale where 1 = not at all and 5 = very much so. See Appendix A for the complete scale.
Pain Rating Scale

Radiant heat stimuli were rated using a 7 point scale, where 0 = something; 2 = warm; 3 = hot; 4 = faint pain; 5 = painful; and 6 = very painful. An additional response category was included if the subject withdrew his/her arm and this response was assigned a value of 7 for purposes of data analysis. See Appendix B for the criteria to be used in scoring a withdrawal response.

Depth of Analgesia and Effectiveness of Coping Strategies Scales

Both of these scales were designed as a manipulation check for the suggested analgesia and the expectancy control groups respectively, and were given during the posttreatment session. The Depth of Analgesia scale is a 7 point scale anchored at one end where value 0 = not at all numb and at the other with 6 = completely numb. The Effectiveness of Coping Strategies scale is also a 7 point scale anchored at both ends. The expectancy control subjects were asked if they had used any cognitive strategies during the posttest portion of the experiment. If they responded negatively, they were assigned a zero response. If they responded affirmatively they were asked to rate the effectiveness of the strategy where 0 = not at all effective and 6 = very effective.

Apparatus

The apparatus used in this experiment was a projector-type heat source which was similar in design to the Hardy-Wolff-Goodell dolorimeter (Hardy, Wolff & Goodell, 1952). There were two main components to the apparatus. The first was an oblong metal projector-type box consisting of a variac input, a moveable spotlight (GE 150 volt), a convex lens
through which the light was focused and an electromagnetically operated shutter. When the aperture was open a concentrated beam of light (focused in a circle one centimeter in diameter) was concentrated on the subject's forearm. Five different heat intensities were used during the experiment: 0 mcal/sec/cm²; 61 mcal/sec/cm²; 106 mcal/sec/cm²; 152 mcal/sec/cm²; and 198 mcal/sec/cm². These values were calibrated for each testing session using a Jodon Model 450B power meter. It should be noted that although the values reported for the present study were below those reported in two previous studies used in the same lab (Luscomb, 1980; Hatcher, 1982), the stimuli were judged to be functionally equivalent based on pilot study data. A primary reason is that the light beam in this study was apparently focused more intensely than that in the aforementioned two studies. Each of the five stimuli were presented a total of 30 times and were in random order. Thus, each session consisted of 150 separate presentations of a radiant heat stimulus. Each stimulus was presented for three seconds and was followed by a 15 second inter-stimulus interval. Stimulus presentations were presented and automatically controlled by the second main component of the apparatus, which consisted of a relay-rack type switching board.

Treatments

Two treatment groups were used in this experiment: a suggested analgesia group and an expectancy control group. Within each group half of the subjects were assigned as having low scores on hypnotic susceptibility and half having high scores. Thus the paradigm consisted basically of a 2 x 2 design.
Subjects in the suggested analgesia group were seen on three separate days and given 30 minute training sessions (within a five day period) designed to give them practice with exercises and cognitive strategies which instructed them to imagine their arms as numb and insensitive. See Appendix C for a transcript of the training.

Subjects in the expectancy control group were also seen for 3 separate 30 minute training sessions (within a five day time period). Their training consisted primarily of informational material regarding pain, including theories of pain, a review of Gate Control Theory, demographic variables affecting pain response, and applied questions involving coping strategies for pain. See Appendix D for a transcript of this training.

Procedure

There were three phases in the experimental procedure: (a) pretreatment (baseline); (b) treatment; and (c) post-treatment. Before the pretreatment phase the experimenter reviewed the informed consent form (see Appendix A) with the subject and answered any questions which the subject may have had. Upon receiving consent, the experimenter had the subject complete the trait and state anxiety forms.

Pretreatment (baseline)

After filling out the consent form and the two anxiety measures subjects were seated in the laboratory beside the radiant heat apparatus and were told that they would be receiving a set of instructions via headphones. This introduction included a brief explanation of the experimental equipment, a review of the rating scale, and instructions regarding the format of the experiment (see Appendix E).
At the conclusion of the audiotaped instructions India ink was applied to the volar surface of the subject's right forearm by means of a plastic template that consisted of six circles approximately 2.5 cm in diameter.

The subject was shielded from the dolorimeter by a dark curtain (which went over the subject's arm) so as to obscure visual cueing from the brightness of the light source. The experimenter then went over the rating scale with the subject once again and reminded him/her that the task consisted not of a test of pain endurance but rather of the ability to discriminate heat intensities. At this point the subject was reminded that he/she could withdraw their arm from the apparatus at any time if they felt the heat source was too intense. The subject was reminded that immediately following a stimulus presentation a tone sounded through the earphones; this was a signal for the subject to rate the response. A copy of the rating scale was posted at the subject's eyelevel on the wall approximately three feet away. The experimenter explained that he would reposition the subject's arm after each trial (during the 15 second interstimulus interval). This resulted in each of the six ink spots being stimulated sequentially such that any one spot only received a stimulus once every 108 seconds. Twenty practice trials were then given and all levels of stimuli which were presented during the 150 experimental trials were included in the practice trials.

After the completion of the practice trials the rating scale was reviewed again with the subject. The 150 experimental trials then were given. After trial 40, 80, and 120 a two minute break was given and the
subject was given three 30 minute appointment times for training sessions on separate days within the ensuing five day period. The subject was also reminded that the post-treatment session would be scheduled for seven days following the pretreatment session.

Treatment

As stated above, treatments consisted of three 30 minute training sessions on three separate days. For the suggested analgesia group the training consisted of exercises and guided cognitive strategies. Subjects in this groups were also asked to give ratings for depth of analgesia and were given behavioral checks (e.g., a needle prick) to test for analgesia. The expectancy control group was also given three separate 30 minute training sessions which were informational in nature and covered theories of pain, variables effecting pain and some applied questions involving the rationale behind different coping strategies. The subjects were not instructed in the use of different coping strategies. They were, however, given the strong expectancy that they would experience a decreased sensitivity to the radiant heat stimuli (in the post-treatment phase) as a result of training.

Posttreatment

The post-treatment phase was very similarly formatted as the pretreatment phase. The subject was again asked to complete the trait and state anxiety measures. As before, the subject listened to taped instructions which were only slightly modified from the pretreatment phase (see Appendix F). If the subject was assigned to the experimental group a short series of in vivo instructions and exercises was given by
the experimenter (see Appendix G). If the subject was in the control group he/she also listened to taped instructions and was given a brief *in vivo* series of instructions along with the expectation that they would experience a decreased sensitivity to the radiant heat stimuli (see Appendix H).

Prior to receiving the radiant heat stimuli the subject was reminded that this was not a test of pain tolerance but rather of the subject's ability to discriminate between different heat intensities. The subject was also reminded that he/she was free to withdraw their arm from the apparatus if a given stimulus was too intense. A series of 20 practice trials was then begun. The experimental subjects were asked to rate their level of analgesia. The stimulus intensities, number and order of stimuli, stimulus durations and interstimulus intervals were identical to those used in the pretreatment phase. The 150 experimental trials were given and, as in the pretreatment phase, the subject was given two-minute rest periods at the end of trials 40, 80 and 120. At the end of the 150 trials, the experiment was completed and the subject debriefed as appropriate.
RESULTS

The data analyses for this study can be divided into three main categories. First are the signal detection analyses, which are broken down into analyses of the sensory discrimination and response bias measures for the treatment effect of the suggested analgesia vs. the expectancy control group. Second are the analyses of the pain rating scale data. Third are the analyses of the data on trait and state anxiety measures.

Signal Detection Theory Data Analysis

When small numbers of trials per stimulus intensity are used, McNicol (1972) and Grossberg and Grant (1978) have recommended the use of nonparametric indicators of sensory discrimination and response bias. Although there are no definitive rules on what qualifies as a sufficient number of trials, a minimum of fifty trials per stimulus level has been recommended by Rollman (1977). Because of the relatively small number of trials per stimulus level used in the present study (30) nonparametric measures of sensory discriminations and response bias were computed. Cumulated conditional probabilities were calculated for all rating scale categories and for each stimulus level.

In the data analysis four (adjacent) stimulus pairs were used: Level 0 and Level 1, Level 1 and Level 2, Level 2 and Level 3, and Level 3 and Level 4. As discussed before (in the Methods section), when comparing a pair of stimulus intensities the lower stimulus level was considered the "noise" and the higher stimulus level considered the "signal plus noise". Within a given rating, then, the hit rate $P(S/s)$
is defined as the cumulated 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 measures of sensory discrimination and response bias were calculated from these inferred hit and false alarm rates.

**Sensory Discrimination Measures**

$P(A)$ was used as the nonparametric measure of sensory discrimination. The procedure used to compute $P(A)$ was to calculate the areas of the triangle and subsequent trapezium formed by joining the hit rate and false alarm rates on an ROC curve with straight lines (see Figure 4). The total of these areas is the value of $P(A)$. Thus, in Figure 4, $P(A)$ is the sum of the areas 1, 2, 3 and 4.

A $P(A)$ value was computed for each subject for every stimulus level pair at both pre- and posttreatment. If these scores are transformed using $2 \arcsin \sqrt{P(A)}$, the skewness of the distribution can be reduced (McNicol, 1972). Thus, in the present analysis of $P(A)$ the transformed values have been used.

The first hypothesis predicted there would be a significant decrease in the sensory discrimination measure for the suggested analgesia group relative to the expectancy control group. In addition, the pretreatment difference between mean values of $P(A)$ were predicted to be nonsignificant and the posttreatment differences significant comparing the treatment and control groups. The mean $P(A)$ values and standard deviation for both the experimental and control groups for each pair of stimulus level comparisons for pre- and posttreatment can be seen in Table 1.
Figure 4. Example of describing the area under the ROC curve for obtaining the non-parametric statistic $P(A)$. 
Table 1
Mean Transformed P(A) Values and Standard Deviations

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Level 0 - Level 1</th>
<th>Level 1 - Level 2</th>
<th>Level 2 - Level 3</th>
<th>Level 3 - Level 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Suggested Analgesia</td>
<td>2.3427</td>
<td>1.8839</td>
<td>2.2324</td>
<td>1.9825</td>
</tr>
<tr>
<td>(0.2540)</td>
<td>(0.2872)</td>
<td>(0.1631)</td>
<td>(0.2612)</td>
<td>(0.2179)</td>
</tr>
<tr>
<td>Expectancy Control</td>
<td>2.3893</td>
<td>2.1522</td>
<td>2.2356</td>
<td>2.1886</td>
</tr>
<tr>
<td>(0.1811)</td>
<td>(0.2741)</td>
<td>(0.1787)</td>
<td>(0.2064)</td>
<td>(0.1460)</td>
</tr>
</tbody>
</table>
A t-test comparison revealed that there were no group significant differences for any of the pretreatment values of P(A). On the other hand, t-test comparisons on posttreatment differences were significant for levels 0 and 1 (t(30) = -2.70, \( p < .01 \)), levels 1 and 2 (t(30) = -2.48, \( p < .05 \)) and levels 2 and 3 (t(30) = -2.15, \( p < .05 \)) but not for levels 3 and 4.

For these and all subsequent dependent variable analyses of variance a three-way ANOVA for Treatments x Sessions x level of Hypnotic Susceptibility with repeated measures over Sessions and Subjects nested within Treatment and Hypnotic Susceptibility was conducted. The analysis of variance on the P(A) values for levels 0 and 1 is presented in Table 2. There was a significant main effect for both treatment and session \( (F(1, 28) = 5.80, \ p < .05 \) and \( F(1, 28) = 29.54, \ p < .0001 \) respectively). From an analysis of group means (Table 1) it can be seen that both the suggested analgesia and expectancy control had a significant pre- to posttreatment decrease in P(A) for levels 0 and 1. The pre- to posttreatment differences for the control group was significant for levels 0 and 1 (t(30) = 2.89, \( p < .01 \)). Next, a comparison was made for P(A) values for stimulus levels 1 and 2. An analysis of variance table is presented in Table 3. The only significant main effect was for session \( (F, (1, 28) = 12.02, \ p < .001) \). It can be seen from Table 3 that there was a significant treatment by sessions interaction. The sensitivity values for the suggested analgesia group showed a significant decrease relative to the expectancy control group for levels 1 and 2 (F (1, 28) =
Table 2
Summary of Analysis of Variance for P(A) Values for Levels Zero and One

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>.3965</td>
<td>5.80*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>1.9368</td>
<td>29.54**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0078</td>
<td>.11</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.1966</td>
<td>3.00</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0167</td>
<td>.24</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0331</td>
<td>.50</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0163</td>
<td>.25</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0683</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0656</td>
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</tbody>
</table>

*p < .05

**p < .001
Table 3
Summary of Analysis of Variance for P(A) Values for Levels One and Two

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>.1752</td>
<td>3.15</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>.3526</td>
<td>12.02**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0366</td>
<td>.66</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.1646</td>
<td>5.61*</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0000</td>
<td>.00</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1250</td>
<td>4.26*</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0004</td>
<td>.01</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0556</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0293</td>
<td></td>
</tr>
</tbody>
</table>

*p < .05

**p < .001
It should also be noted that there was a significant session by level of hypnotic susceptibility interaction ($F(1, 28) = 4.26, p < .05$).

For the P(A) comparison of levels 2 and 3 there were no significant main effects (see Table 4). There was, however, a significant treatment by session interaction ($F(1, 28) = 7.29, p < .05$).

In comparing P(A) values for levels 3 and 4 there were no significant main effects (see Table 5) or significant interactions.

To summarize, Hypothesis 1 was partially confirmed, i.e., there were significant pre-to posttreatment decreases in P(A) values for the suggested analgesia group relative to the control group for comparisons between levels 1 and 2 and between 2 and 3. Both groups, however, showed significant decreases in P(A) for the level 0 -- level 1 comparison. Further comparisons made between expectancy and control groups at posttreatment resulted in less sensitivity for the analgesia group given levels 0 -- 1, 1 -- 2, 2 -- 3.

Taken together, these data indicate that the suggested analgesia treatment decreased the physical sensitivity of subjects for all but the highest (levels 3 and 4) stimulus intensities. The expectancy control group had a significant decrease in sensitivity for the lowest (levels 0 and 1) stimulus pair.

**Response Bias Measures**

Hodos (1970) was the first to describe the nonparametric measure of response bias used in this study. The statistic is called $B'$. According to Grier (1971), this statistic can be computed using the formula:
Table 4

Summary of Analysis of Variance for P(A) Values for Levels Two and Three

<table>
<thead>
<tr>
<th>Source</th>
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</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>.0056</td>
<td>.15</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>.0366</td>
<td>.96</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0408</td>
<td>1.07</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.2783</td>
<td>7.29*</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0162</td>
<td>.43</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0014</td>
<td>.04</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0135</td>
<td>.35</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0381</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0382</td>
<td></td>
</tr>
</tbody>
</table>

*p < .05
Table 5

Summary of Analysis of Variance for P(A) Values for Levels Three and Four

<table>
<thead>
<tr>
<th>Source</th>
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<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
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<td>Treatment</td>
<td>1</td>
<td>.0181</td>
<td>.35</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>.0205</td>
<td>.94</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0017</td>
<td>.03</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.0264</td>
<td>1.21</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0010</td>
<td>.02</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0291</td>
<td>1.33</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0055</td>
<td>.25</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0523</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0219</td>
<td></td>
</tr>
</tbody>
</table>
In this formula $X = \text{false alarm rate } (P(S/n))$ and $Y = \text{hit rate } (P(S/s))$ at a given rating category and a given pair of adjacent stimuli. The formula yields values of $B''$ ranging from -1.00 to +1.00. The absolute value of $B''$ indicates the degree of response bias whereas the direction of bias is determined by the valence, i.e., a negative valence signifies a bias to respond. For the present analysis a $B''$ value was computed for each stimulus level pair for each subject. These values were computed using the cumulative probability of a rating of four (faint pain) on the intensity scale for each stimulus level. The $B''$ values obtained represent each subject's bias toward responding with a rating of four or higher. Thus a $B''$ value which is negative indicates a tendency to make a response of four or higher, and a positive $B''$ value indicates a hesitancy to respond with a four or higher. Mean $B''$ values and standard deviations for each treatment group for each pair of stimulus comparisons can be found in Table 6.

To examine Hypothesis 2 comparisons were performed on both pretreatment and posttreatment $B''$ values for each of the adjacent stimulus level pairs. A comparison of pretreatment $B''$ values between treatment groups for each stimulus pair revealed no significant differences. The equivalent comparison of posttreatment $B''$ values found significant differences between groups only for the highest stimulus level pair ($t(30) = 2.14, p < .05$), i.e., there was a differential decrease in response bias (a greater tendency to respond with a rating of
<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Level 0 - Level 1</th>
<th>Level 1 - Level 2</th>
<th>Level 2 - Level 3</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td></td>
<td>.6250 (.5000)</td>
<td>.0625 (.2500)</td>
<td>.5188 (.3463)</td>
<td>.2291 (.4166)</td>
</tr>
<tr>
<td>Suggested Analgesia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.0465 (.6515)</td>
<td>.2648 (.5588)</td>
<td>-.5825 (.5628)</td>
<td>.3682 (.5216)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expectancy Control</td>
<td>.5711 (.4794)</td>
<td>.0625 (.4425)</td>
<td>.5084 (.5170)</td>
<td>.3396 (.4610)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>-.1266 (.3849)</td>
<td>.4117 (.5650)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.6880 (.3104)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.0810 (.6572)</td>
</tr>
</tbody>
</table>
Table 7

Summary of Analysis of Variance for B" Values for Levels Zero and One

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
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<td>0.0116</td>
<td>0.05</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>4.5892</td>
<td>25.62*</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>0.0751</td>
<td>0.35</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>0.0116</td>
<td>0.06</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>0.0006</td>
<td>0.00</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>0.0511</td>
<td>0.29</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
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<td>0.0006</td>
<td>0.00</td>
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<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
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<td>0.2118</td>
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</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
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<td>0.1791</td>
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*p < .0001
<table>
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<tbody>
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<td>Treatment</td>
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<td>.0401</td>
<td>.40</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>.8409</td>
<td>2.94</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.3335</td>
<td>3.31</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.0584</td>
<td>.20</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1244</td>
<td>1.23</td>
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<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.2699</td>
<td>.94</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
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<td>.0592</td>
<td>.21</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
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<td>.1009</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.2857</td>
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Table 9
Summary of Analysis of Variance for B" Values for Levels Two and Three

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<td>.01</td>
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<td>Session</td>
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<td>2.2895</td>
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</tr>
<tr>
<td>Hypnotic Susceptibility</td>
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<td>.0003</td>
<td>.00</td>
</tr>
<tr>
<td>Treatment x Session</td>
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<td>.4098</td>
<td>1.27</td>
</tr>
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<td>.01</td>
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<tr>
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<td>.3650</td>
<td>1.13</td>
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<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
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<td>.2337</td>
<td>.72</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.2999</td>
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<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
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</tbody>
</table>

*p < .01
Table 10

Summary of Analysis of Variance for B' Values for Levels Three and Four

<table>
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<tr>
<td>Treatment</td>
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<td>1.2310</td>
<td>5.20*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>9.7063</td>
<td>30.20**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.8105</td>
<td>3.42</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.4727</td>
<td>1.47</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1367</td>
<td>.58</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0544</td>
<td>.17</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1280</td>
<td>.40</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.2367</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.3214</td>
<td></td>
</tr>
</tbody>
</table>

* _p < .05

** _p < .0001
4 or higher) for the expectancy control group vs. the suggested analgesia group.

The analysis of variance on $B^*$ for each stimulus level comparison pair are presented in Tables 7 through 10. For the comparison of stimulus levels zero and one there was a significant main effect for session ($F(1, 28) = 25.62, p < .0001$). The comparison between stimulus levels one and two revealed no significant main effects. For the level two and three comparison there was a significant main effect for sessions ($F(1, 28) = 7.08, p < .01$). For the comparison of levels three and four there was a significant main effect for session ($F(1, 28) = 30.20, p < .0001$) and treatment ($F(1, 28) = 5.20, p < .05$). There were no significant interaction effects at any level of pair comparisons.

To summarize, hypothesis two was partially confirmed by the present findings, i.e., there was a relative decrease in response bias (i.e., a tendency to report less pain) for both treatment groups for the two highest stimulus level pairs. In addition, however, there was a relative increase in response bias (i.e., a tendency to report slightly more pain) for the level zero and level one pair comparison. Although there was a trend towards an increase in response bias (i.e., tendency to report greater pain) for the level one and two comparison, the effect of session was nonsignificant. Overall, then, there was no differential response bias change between treatment groups. The only exception to this statement was for the level three and four comparisons, for which the suggested analgesia group was significantly less likely to report pain.
Level of Hypnotic Susceptibility Data Analysis

Concerning hypothesis 3, an inspection of Tables 2 through 5 and 7 through 10 will reveal that, overall there were no significant main effect or interaction differences found between the suggested analgesia and expectancy control groups for the hypnotic susceptibility (high versus low). There was one exception to this statement: for levels 1 and 2 there was a significant interaction effect for session by hypnotic susceptibility for \(P(A) (F(1, 28) = 4.26, p < .05)\). Thus hypothesis 3 was not confirmed.

Mean Rating Scale Data Analysis

A mean score was computed for each stimulus level for each session by subject. This score consisted of the average of the 30 responses at each stimulus level during each session. (Note that withdrawals were assigned a value of 7). Thus, each subject had five pretreatment and five posttreatment scores, or one for each stimulus level. These groups means are given in Table 11.

Hypothesis 4 was examined first by comparing the pre- and post-treatment mean rating scale values for each stimulus level. With the exception of the zero level stimulus \(t(30) = -2.35, p < .05\) no significant differences were found for pretreatment values. For equivalent posttreatment comparisons, however, there were significant differences for both the suggested analgesia and expectancy control groups at each stimulus level: zero = \(t(30) = -4.70, p < .0001\), one \(t(30) = -3.55, p < .001\), two \(t(30) = -3.40, p < .01\), three \(t(30) = -3.22, p < .01\), and four \(t(30) = -2.65, p < .01\).
Table 11
Mean Rating Scale Scores and Standard Deviations

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Level 0 Pre</th>
<th>Level 0 Post</th>
<th>Level 1 Pre</th>
<th>Level 1 Post</th>
<th>Level 2 Pre</th>
<th>Level 2 Post</th>
<th>Level 3 Pre</th>
<th>Level 3 Post</th>
<th>Level 4 Pre</th>
<th>Level 4 Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suggested Analgesia</td>
<td>.3333 (.2449)</td>
<td>.0229 (.0483)</td>
<td>1.5771 (.5524)</td>
<td>.4083 (.4164)</td>
<td>2.8104 (.8368)</td>
<td>1.0062 (.8219)</td>
<td>3.9223 (.8431)</td>
<td>1.7667 (.8368)</td>
<td>5.1312 (.8496)</td>
<td>2.8833 (.9817)</td>
</tr>
<tr>
<td>Expectancy Control</td>
<td>.6479 (.4761)</td>
<td>.3083 (.2380)</td>
<td>2.0375 (.7526)</td>
<td>1.0667 (.6146)</td>
<td>3.3104 (.8135)</td>
<td>2.0021 (.8366)</td>
<td>4.3770 (.8438)</td>
<td>2.9979 (.9817)</td>
<td>5.6282 (.9015)</td>
<td>4.1083 (1.1455)</td>
</tr>
</tbody>
</table>
Table 12
Summary of Analysis of Variance of Mean Rating
Scale Values for Level Zero

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>1.44</td>
<td>10.66*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>1.69</td>
<td>37.44**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.01</td>
<td>.06</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.0034</td>
<td>.08</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0711</td>
<td>.53</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0100</td>
<td>.22</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0506</td>
<td>1.12</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.1350</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.0451</td>
<td></td>
</tr>
</tbody>
</table>

*p < .01

**p < .0001
Table 13

Summary of Analysis of Variance of Mean Rating
Scale Values for Level One

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>5.0064</td>
<td>8.21*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>18.3113</td>
<td>131.99**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>0.0077</td>
<td>.01</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.1567</td>
<td>1.13</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1567</td>
<td>.26</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0689</td>
<td>.50</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.1438</td>
<td>1.04</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.6100</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.1387</td>
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</tr>
</tbody>
</table>

*p < .01

**p < .0001
Table 14
Summary of Analysis of Variance of Mean Rating
Scale Values for Level Two

<table>
<thead>
<tr>
<th>Source</th>
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<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>8.9501</td>
<td>7.75*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>38.7506</td>
<td>144.55**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.1534</td>
<td>.13</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>.9834</td>
<td>3.67</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0367</td>
<td>.03</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.9506</td>
<td>3.55</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0667</td>
<td>.25</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>1.1553</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.2681</td>
<td></td>
</tr>
</tbody>
</table>

*p < .01

**p < 0.0001
Table 15
Summary of Analysis of Variance of Mean Rating
Scale Values for Level Three

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
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</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>11.3703</td>
<td>7.59*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>49.9804</td>
<td>106.43***</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0049</td>
<td>.00</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>2.4119</td>
<td>5.14**</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0216</td>
<td>.01</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>1.3877</td>
<td>2.96</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0020</td>
<td>.00</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>1.4976</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment X Hypnotic Susceptibility)</td>
<td>28</td>
<td>.4696</td>
<td></td>
</tr>
</tbody>
</table>

*p < .01

**p < .05

***p < .0001
Table 16
Summary of Analysis of Variance of Mean Rating
Scale Values for Level Four

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>11.8599</td>
<td>6.04*</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>56.7836</td>
<td>78.72**</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>.0002</td>
<td>.00</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>2.1205</td>
<td>2.94</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0964</td>
<td>.05</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>4.0086</td>
<td>5.56*</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>.0937</td>
<td>.13</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>1.9631</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>.7213</td>
<td></td>
</tr>
</tbody>
</table>

*p < .05

**p < .0001
An inspection of the analysis of variance scores for each stimulus level indicates that there were significant main effects for both treatment ($F(1, 28) = 10.66, p < .01; F(1, 28) = 8.21, p < .01$; $F(1, 28) = 7.75, p < .01; F(1, 28) = 7.59, p < .01; F(1, 28) = 6.04, p < .05$) and session ($F(1, 28) = 37.44, p < .0001; F(1, 28) = 131.99, p < .0001; F(1, 28) = 144.55, p < .0001; F(1, 28) = 106.43, p < .0001; F(1, 28) = 78.72, p < .0001$). There was also a significant treatment by session interaction at stimulus level 3 ($F(1, 28) = 5.14, p < .05$) and a significant session by hypnotic susceptibility interaction ($F(1, 28) = 5.56, p < .05$) for stimulus level four.

In summary, Hypothesis 4 was confirmed, i.e., both groups showed a significant decrease in reported pain ratings from the pre- to post-treatment. In addition, the two groups did not differ significantly in the amount of decrease. The one exception to this statement was for level 3. At only one level (zero) were there significant pretreatment mean differences between groups.

**State and Trait Anxiety Analysis**

Hypothesis 5 was concerned with the trait anxiety scores which are presented in Table 17. T-test comparisons revealed that there were no significant differences between groups on trait anxiety scores for both pre- and posttreatment measurements.

A summary of the analysis of variance scores for trait anxiety is presented in Table 18. As can be seen, there were no significant main effects and no significant interactions. Thus, Hypothesis 5 was confirmed.
<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suggested Analgesia</td>
<td>35.5625</td>
<td>35.0625</td>
</tr>
<tr>
<td></td>
<td>(8.4061)</td>
<td>(9.6917)</td>
</tr>
<tr>
<td>Expectancy Control</td>
<td>34.5625</td>
<td>32.7500</td>
</tr>
<tr>
<td></td>
<td>(6.2925)</td>
<td>(7.3439)</td>
</tr>
</tbody>
</table>
Table 18
Summary of Analysis of Variance of Trait Anxiety Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>43.8906</td>
<td>.34</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>21.3906</td>
<td>2.40</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>34.5156</td>
<td>.27</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>6.8906</td>
<td>.77</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>8.2656</td>
<td>.06</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>2.6406</td>
<td>.30</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>8.2656</td>
<td>.06</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>127.4576</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>8.9040</td>
<td></td>
</tr>
</tbody>
</table>
Table 19

Means and Standard Deviations of State Anxiety Scores

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Pretreatment</th>
<th>Posttreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suggested Analgesia</td>
<td>46.5625</td>
<td>37.3750</td>
</tr>
<tr>
<td></td>
<td>(11.5641)</td>
<td>(9.3086)</td>
</tr>
<tr>
<td>Expectancy Control</td>
<td>50.1875</td>
<td>38.6875</td>
</tr>
<tr>
<td></td>
<td>(16.7102)</td>
<td>(10.9953)</td>
</tr>
</tbody>
</table>
Hypothesis 6 pertained to the analysis of the state anxiety data. Means and standard deviations are presented in Table 19. A comparison of both pretreatment and posttreatment mean state anxiety scores revealed no significant differences between the suggested analgesia and expectancy control groups.

As can be seen in Table 20, the analysis of variance scores show a significant main effect for session ($F(1, 28) = 24.83, p < .0001$). There were no other main or interaction effects.

Thus, Hypothesis 6 was also confirmed, i.e., both the suggested analgesia and the expectancy control group showed a significant decrease in state anxiety scores. In addition, the rate of decrease did not significantly differ between the two groups.
Table 20

Summary of Analysis of Variance of State Anxiety Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>1</td>
<td>97.5156</td>
<td>.40</td>
</tr>
<tr>
<td>Session</td>
<td>1</td>
<td>1711.8906</td>
<td>24.83*</td>
</tr>
<tr>
<td>Hypnotic Susceptibility</td>
<td>1</td>
<td>17.0156</td>
<td>.07</td>
</tr>
<tr>
<td>Treatment x Session</td>
<td>1</td>
<td>21.3906</td>
<td>.31</td>
</tr>
<tr>
<td>Treatment x Hypnotic Susceptibility</td>
<td>1</td>
<td>284.7656</td>
<td>1.16</td>
</tr>
<tr>
<td>Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>165.7656</td>
<td>2.40</td>
</tr>
<tr>
<td>Treatment x Session x Hypnotic Susceptibility</td>
<td>1</td>
<td>58.1406</td>
<td>.84</td>
</tr>
<tr>
<td>Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>244.6987</td>
<td></td>
</tr>
<tr>
<td>Session x Subject (Treatment x Hypnotic Susceptibility)</td>
<td>28</td>
<td>68.9397</td>
<td></td>
</tr>
</tbody>
</table>

*p < .0001
DISCUSSION

There were several noteworthy findings in the present study, both from the signal detection data analysis compared with the mean pain rating scale analyses and for the anxiety scores.

Perhaps the most salient finding, was the fact that there were significant pre- to posttreatment differences in sensitivity (P(A)) for the suggested analgesia group for three of the four (i.e., all but the highest) pair stimulus level comparisons. A finding of significant changes in sensitivity using "psychological" treatments has rarely been demonstrated. The only other study to conclusively demonstrate pervasive decreases in sensitivity was reported by Hall (1977) and also involved the use of analgesia instructions, similar to those in the present study. For reasons of confounding in the experimental design (discussed previously) Craig and Coren's (1975) findings of increases in sensitivity using a modeling procedure cannot be considered conclusive.

Several points regarding differences in experimental design and statistical analyses are also in order when comparing the present study to Hall's (1977) study on the effects of analgesia suggestion. First of all, Hall's design was not a true pre-post experiment, i.e., he did not obtain baseline measurements. Rather, he used a treatment - post-treatment measurement design. He also used the same subjects on two occasions each in order to have a larger N per cell (10 as opposed to five). Hall justified this type of analysis in that the scores for occasion one and occasion two were found to be nonsignificantly
different. Hall also summed and averaged his sensitivity (d') values across all stimulus levels, rather than making comparisons for adjacent stimulus pairs, as in the present study. The present study represented a more valid pre-post experimental design and thus, it can be argued, the results of the present study are more generalizable because the assumption of equality between experimental and control groups was demonstrated empirically.

The major findings of the present study — that, for all but the highest stimulus level pair, there was a significant pre-post decrease in the sensitivity measure of pain for the suggested analgesia group — argue persuasively for the effectiveness of the suggested analgesia techniques.

The lack of a significant decrease in sensitivity in the analgesia group for the highest stimulus pairs comparison may be explainable in terms of Gate Control Theory. Thus, it is possible that the cognitive mechanism which overrides the threshold level (i.e., raises it) at the gate control site is not as effective for higher stimulus intensities.

Some conjectures of Chapman (1980) may also be appropriate in explaining these findings. Chapman hypothesized that "pain normally generates arousal because of its disruptive effects on perception" (1980, p. 119). He further cited findings from experiments by Hilgard and Hilgard who took both overt and covert (or "hidden") pain measures for subjects under hypnotic analgesia:

the hidden pain was sensory pain of high intensity, but unaccompanied by suffering. After amnesia for the covert report was lifted, subjects commonly remembered what their
covert reports were, but could not remember actually feeling the pain reported in that way. (Hilgard & Hilgard, 1975, p. 44)

Chapman referred to the theory that "hypnotic suggestion is a form of social control of the perceiver's figure-ground functioning so that the process becomes rigidly exclusive of perceptual organizations in which pain is the figure" (1980, p. 118). Chapman invoked this theory to explain the phenomenon of hidden pain during hypnotic analgesia as a kind of background pain that has been prevented by the hypnotist from emerging into the foreground. In addition, Chapman observed that "when the pain cannot emerge as the figure, it apparently does not provoke the emotional and aversive responses that one normally observes with patients in pain" (1980, p. 119). The weight of experimental evidence using both SDT (e.g., Hall, 1977) and tolerance paradigms (e.g., Barber & Hahn, 1962) has determined that there is virtually no difference between the effectiveness of "hypnotic" (i.e., with an induction) and "suggested" ("waking") analgesia. Following this line of reasoning, it seems likely that the subjects in the suggested analgesia group of the present experiment did not experience pain as a salient part of the "figure" in the figure-ground analogy, and for all but the highest comparison pair experienced a decrease in sensitivity. In other words, the subjects couldn't discriminate between the "figure" vs. "ground" as a function of treatment. It should be noted that although there were no significant pre-post differences for the highest stimulus pair comparison (levels 3 and 4) for the suggested analgesia group there was a slight downward trend. It may be that future efforts to decrease
sensitivity measures using suggested analgesia could profitably focus on different treatment parameters at the higher stimulus level comparisons (i.e., the highest comparison pair in the present study). For example, different treatment parameters could be investigated: using shorter vs. longer relaxation instructions, longer suggestions, and more extended in vivo practice.

An unexpected finding in the present study was a significant pre-post decrease for sensitivity measures for the expectancy control group at the lowest stimulus pain comparison (levels 0 and 1). This effect occurred in conjunction with a greater change from pre to post for the suggested analgesia group (vs. the expectancy control) which approached significance ($F(1, 28) = 3.00, p < .09$). Thus the effect of decreased sensitivity for the comparisons of levels 0 and 1 was slightly greater for the suggested analgesia than the expectancy control group.

In retrospect, there is evidence that the expectancy control group may not have been a true control group, in the sense of an inactive treatment. A manipulation check after the posttreatment session revealed that all but two expectancy control subjects did use some type of cognitive strategy. Preferred strategies were numerous and varied from distraction to imagined analgesia, according to the subjects' anecdotal reports. Other anecdotal evidence indicated that at least a few subjects in the expectancy control group asked the experimenter after the third training session whether they should employ the cognitive strategies previously described in the upcoming posttreatment session. Although the subjects were only given the repeat expectancy
that increased knowledge regarding pain research would result in a decreased sensitivity to radiant heat stimuli in the posttreatment session, evidence was that some subjects used cognitive strategies (and in their opinion quite effectively) in the posttest. It seems, then, that the change in sensitivity at the level 0 -- 1 comparison for the expectancy control group may have been due to a weak treatment effect, although it is difficult because of the lack of a no treatment control group to ascribe the effect to the use of a particular strategy. One previous study (Hatcher, 1982) has found slight changes (for the level 0 -- 1 comparison) for treatment groups (tolerant modeling and modeled analgesia) when brief videotaped instructions were given. Although the expectancy control in the present study was not given active training *per se*, the apparently strong expectancy induced and the use of *in vivo* training seemed to result, at least anecdotally, in many subjects using active imagery. Thus, the decrease in sensitivity at lowest levels for the expectancy control seems also to be explainable in terms of Gate Control theory and/or a figure/ground confusion.

Results for responses bias are somewhat more complicated to explain. It is somewhat perplexing that the predicted decrease in response bias (i.e., a posttreatment tendency to report less pain relative to pretreatment) occurred only for the two highest stimulus level comparison pairs, while there was a significant increase in response bias (i.e., a relative tendency to report slightly more pain) for the comparison of levels zero and one. It should be emphasized, however, that both pre- and posttreatment values of $B^*$ for the lowest
stimulus pair were positive, i.e., both suggested a general hesitancy to respond with a rating of pain. The pre-post tendency, for the lowest pair was for a relatively greater tendency to respond with a rating of pain.

Taken together, the results for response bias suggest the lack of a differential treatment effect, although the directionality of the effects varied according to level of stimulus intensity. The only exception to this occurred in the comparison of the highest stimulus pair, when the analgesia group had a greater hesitancy to respond with a pain rating that did the control group. It may be argued that this finding could have been random. It may also be that subjects in the suggested analgesia group experienced a significant decrease in response bias at the highest level because the suggestions were more powerful at the highest intensities. It appears that, at the highest stimulus levels, subjects in the experimental group may have compensated for their lack of sensitivity change with a tendency to report less pain (response bias). The overall results for response bias are not suggestive of a differential effect of treatment, however.

When the findings for sensitivity are combined with those for response bias, there is compelling evidence that there was a significant treatment effect towards reduced sensitivity for the suggested analgesia group which occurred concomitantly with an overall non-differential treatment group change for response bias.

Perhaps the most puzzling finding of the present study was the virtually complete lack of significance of the level of susceptibility
variable. There was only one significant interaction for the hypnotic susceptibility variable: a treatment by susceptibility interaction for sensitivity measures for comparison of levels 1 and 2; the overall lack of significance of the level of susceptibility variable lends credence to the fact that this interactions probably occurred by chance.

There is at least some precedent for these findings, especially when comparing traditional (i.e. tolerance) measures with SDT measures. The susceptibility variable has generally been found to be a salient one for studies using tolerance measures. The trends reported in the literature often describe greater increases in pain tolerance for high susceptible subjects. The only reported study to examine susceptibility within an SDT format was done by Hall (1977). In comparing his hypnotic vs. imagined analgesia group he found no significant differences for decreases in sensitivity (d') across levels of susceptibility. Hall's conclusion is worth repeating:

The absence of any differences between high and low susceptible subjects in the effects that a high level of susceptibility is not necessary for the production of analgesia. Taken together, these results seem to 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. (1977, p. 123; emphasis added)

Hall's only significant finding for level of susceptibility occurred for sensitivity measures in the control group. Specifically the mean d' values for the control group subjects who were high in susceptibility was significantly lower (i.e., reduced sensitivity) than those for the low susceptible subjects. Hall attributed this to the
fact that the high susceptible control subjects probably engaged in the same type of cognitive strategies as the subjects in the hypnotic and imagined analgesia groups. Hall cited Hilgard's (1974) research as evidence for this conjecture. Indeed, a visual inspection of the mean sensitivity changes for the treatment by session by susceptibility interaction reveals a slight and nonsignificant trend for subjects in the high susceptible expectancy control group to experience a greater decrease (especially for levels 0 - 1 and 1 - 2) in sensitivity than low susceptible subjects. One other possibility, especially for the suggested analgesia group, with regard to nondifferential responding for level of susceptibility is that treatment effects were strong enough to eliminate any differences produced by level of susceptibility. The present findings suggest that level of susceptibility is not a necessary treatment variable to consider when giving treatment (e.g., suggested analgesia) that has demonstrated effectiveness in reducing sensitivity.

Research has suggested that there may be at least one continuum on which to evaluate subjects that is more useful than level of hypnotic susceptibility. Specifically, the use of strategies which allow the subject to generate coping rather than catastrophizing cognitions has been associated with decreased pain, at least for tolerance measures (Spanos, Brown, Jones & Horner, 1981). Given the anecdotal evidence in the present study regarding use of cognitive strategies for the expectancy control group, it would seem that this is a variable worth investigating more systematically within an SDT paradigm.
Data analysis on the mean pain rating scale values essentially confirmed findings from previous SDT studies, i.e., that reported decreases in verbal pain reports frequently occur across groups. The fact that differential group sensitivity changes were found in the present study concomitant with relatively equal changes across groups for mean pain ratings argues for the use of SDT methodology. Specifically, without reported sensitivity changes as a comparison, mean pain ratings in the present study suggest no significant differences between groups. The one reported pretreatment difference for mean pain ratings (for the level 0 - 1 comparison) in the present study does not appear to alter this conclusion and is probably attributable to random sampling error.

The anxiety data for the present study are essentially straightforward: there was no change for trait anxiety between or within groups but there was a significant decrease in state anxiety with groups. The between-group changes were nonsignificant. These findings suggest (as has been suggested before by previous researchers, e.g., Hatcher, 1982) that decreases in sensitivity are often correlated with decreases in reported state anxiety but that decreases in state anxiety need not be accompanied by decreased sensitivity. Within the context of the present experiment there was a significant decrease in reported state anxiety (specific to being in an aversive, potentially painful situation) from pre-to posttreatment. It seems likely that this difference may be attributable to previous exposure to the aversive stimuli. This, however cannot be conclusively determined within the parameters of the present design.
SUMMARY AND CONCLUSIONS

Signal Detection theory has been proposed as a method by which to study the effect of psychological treatments on pain response. An advantage of SDT is that it purports to provide measures of the sensitivity (i.e., physical/sensory) and response bias (i.e., judgmental criterion) components of pain. Signal Detection theory is purportedly superior to traditional (i.e., threshold and tolerance) methods in that it partials out the sensitivity and response bias components from the total pain response (cf. Grossberg & Grant, 1978; Hall, 1977). There have been very few studies which have demonstrated a significant effect on sensitivity as a function of "psychological" treatments. In spite of strong evidence for the effectiveness of suggested analgesia in reducing pain tolerance, studies using an SDT paradigm to investigate this strategy have been rare. Only one previous study has conclusively demonstrated reductions in sensitivity as a function of analgesia suggestions (Hall, 1977) and there are some minor criticisms of the experimental design mentioned previously. One study which found low level (i.e., for stimulus level zero and one comparisons) reductions in sensitivity as a function of analgesia suggestions was done in the laboratory used in the present study (Hatcher, 1982). The two SDT studies mentioned above used relatively short (under 10 minutes) one-time treatments. The present study was designed to use more protracted treatments which were in vivo.

The present study used a signal detection model to test the effectiveness of a suggested analgesia treatment vs. an expectancy
control group. Findings were that sensitivity measures decreased significantly for the suggested analgesia groups for all but the highest stimulus level pair. There was also a significant decrease in sensitivity for the expectancy control group, and this may be attributable to a weak treatment effect generated by spontaneous coping strategies. The effectiveness of suggested analgesia as a method to reduce sensitivity to radiant heat pain was strongly supported by the present findings. The mechanism by which the reductions in sensitivity were attained can be explained in terms of Gate Control theory (Melzack & Wall, 1965) and possibly a figure-ground realignment of the noxious stimulus (Chapman, 1980).

Although the findings for response bias were not quite as straightforward, a pattern emerged: overall the two groups did not differ across levels in the extent to which response bias changed. For only the highest stimulus intensity pair was there a differential change in response bias. For this level the suggested analgesia group showed a significant decrease in the tendency to call a given stimulus painful.

There were, overall, non-differential changes between treatment groups on mean pain ratings, trait and state anxiety. Mean pain ratings decreased significantly from pre- to posttreatment for both groups, as did state anxiety. Trait anxiety scores did not significantly change. The variable level of susceptibility was found to have virtually no differential effect on the dependent measure values.

It appeared that evaluating subjects on other cognitively mediated dimensions (e.g., tendency to use coping vs. catastrophizing strategies)
rather than level of susceptibility might prove fruitful for future studies. Based on the present findings, future research should also focus on the effect of the same treatment (i.e., suggested analgesia) for different stimulus intensities, especially those in the higher range. The use of well-controlled manipulation checks (e.g., depth of analgesia and effectiveness of cognitive strategies), both pre- and posttreatment are also warranted for future research.
Reference Notes

References


Dubuisson, D. & Melzack, R. Classification of clinical pain descriptions by multiple group discrimination analysis. Experimental Neurology, 1976, 51, 480-487.


CONSENT FORM

Participant: ____________________________  Date: ______________

Date of Birth ________  Sex ________  Race ________  Dominant Hand ________

1. I, the undersigned, hereby consent to serve as a participant in a study to be conducted under the direction of Dr. George A. Clum, faculty member in the Department of Psychology, Virginia Polytechnic Institute and State University.

2. The nature of the study has been explained to me by ______ and I understand that its purpose is to examine the effects of different interventions on the response to noxious stimuli.

3. It has also been explained to me that I will be expected to engage in the following activities:

   A. Answer a number of questionnaires.
   B. Be instructed in a technique to deal with pain.
   C. To have radiant heat applied to points on my arm darkened with India ink at differing levels of intensity, some of which will produce a painful sensation. There is a likelihood of some reddening of the stimulated areas and the possibility of slight blisters.

4. I understand that my participation may improve my ability to deal with pain.

5. I understand that my participation is voluntary and that I may terminate my participation at any time. I understand that if I have any questions concerning this project I may call either:

   Project Director - Dr. George A. Clum (961-1997)
   Chairman, Institutional Review Board - Dr. Milton Stombler (961-5283)

6. Although I understand the procedure is safe and has been used by previous researchers, I understand that it is my responsibility to advise Dr. Clum or one of his assistants should any medical problems arise in the course of this experiment. Virginia Polytechnic Institute and State University has a policy which states that no compensation is available if injury should be suffered as a result of any research.

Subject: ____________________________  Co-Investigator: ____________________________

Address: ____________________________  Date: ______________

SS# (for exp. credit): ____________________________

Phone Number: ____________________________
**FORM A-2**

<table>
<thead>
<tr>
<th>Name</th>
<th>Date</th>
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</table>

**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.

<table>
<thead>
<tr>
<th>Statement</th>
<th>1</th>
<th>2</th>
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</thead>
<tbody>
<tr>
<td>1. I feel pleasant</td>
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<tr>
<td>2. I tire quickly</td>
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<td>3. I feel like crying</td>
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<td>4. I wish I could be as happy as others seem to be</td>
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<td>5. I am losing out on things because I can't make up my mind soon enough</td>
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<td>6. I feel rested</td>
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<td>7. I am &quot;calm, cool, and collected.&quot;</td>
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<td>8. I feel that difficulties are piling up so that I cannot overcome them.</td>
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<td>9. I worry too much over something that really doesn't matter</td>
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<td>10. I am happy</td>
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<td>11. I am inclined to take things hard</td>
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<td>12. I lack self-confidence</td>
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<td>13. I feel secure</td>
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<td>14. I try to avoid facing a crisis or difficulty</td>
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<td>15. I feel blue</td>
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<td>16. I am content</td>
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<td>17. Some unimportant thought runs through my mind and bothers me.</td>
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<td>18. I take disappointments so keenly that I can't put them out of my mind</td>
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<tr>
<td>19. I am a steady person</td>
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<tr>
<td>20. I get in a state of tension or turmoil as I think over my recent concerns and interests</td>
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</table>
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"

<table>
<thead>
<tr>
<th>Number</th>
<th>Item Description</th>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<tbody>
<tr>
<td>1</td>
<td>Hands feel moist</td>
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<td>5</td>
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<td>Very moist</td>
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<td>2</td>
<td>Feel relaxed</td>
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<td>Very relaxed</td>
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<td>3</td>
<td>Hands feel unsteady</td>
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<td>Not at all</td>
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<td>Very unsteady</td>
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<td>4</td>
<td>Feel self-confident</td>
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<td>5</td>
<td>Stomach feels tense</td>
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<td>6</td>
<td>Enjoy this situation</td>
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<td>7</td>
<td>Heart beats faster</td>
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<td>Much faster</td>
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<td>8</td>
<td>Feel calm</td>
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<td>Very calm</td>
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<td>9</td>
<td>Perspire</td>
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<td>Feel comfortable</td>
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<td>11</td>
<td>Mouth feels dry</td>
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<td>Very dry</td>
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<td>12</td>
<td>Unable to focus my thoughts</td>
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<td>Able to focus</td>
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<td>Unable to focus</td>
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<td>13</td>
<td>Feel pleasant</td>
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<td>Very pleasant</td>
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<td>Feel nervous</td>
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<td>15</td>
<td>Feel throbbing in my head</td>
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<td>16</td>
<td>Feel secure</td>
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<td>17. Feel upset</td>
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<td>Not at all</td>
<td>Very upset</td>
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<td>18. Hands feel cold</td>
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<td>Not at all</td>
<td>Very cold</td>
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<td>19. Feel good</td>
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<td>Very good</td>
<td>Not at all</td>
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<tr>
<td>20. Feel anxious</td>
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<td>Not at all</td>
<td>Very anxious</td>
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<td>21. Breathing is irregular</td>
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<td>Not at all</td>
<td>Very irregular</td>
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<td>22. Feel uneasy</td>
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<td>Not at all</td>
<td>Very uneasy</td>
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<tr>
<td>23. Want to avoid this situation</td>
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<td>Not at all</td>
<td>Very much</td>
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<td>24. Feel lump in throat</td>
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<td>Not at all</td>
<td>Very much</td>
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</table>

PARQ-75
APPENDIX B
**CRITERIA FOR SCORING A WITHDRAWAL**

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

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 heat stimulation before initiating the withdrawal response.

3. A withdrawal is **not** scored if a gap is present between the subject's arm and the plastic ring on the front of the dolorimeter because: (a) the subject's arm does not fit well against the ring, or (b) the subject relaxes his/her arm, allowing the gap to appear.
SESSION ONE -- SUGGESTED ANALGESIA INSTRUCTIONS*

Over the next 30 minutes I'll be giving you suggestions and cognitive strategies which will help you to relax and to imagine your right arm as numb and insensitive.

Previous research has shown that this training can enable you to experience a decreased sensitivity to radiant heat stimuli. Today's training will be focused on you beginning to learn these techniques, but no actual radiant heat stimuli will be presented today. Later in training you can become proficient in imagining your arm as numb and insensitive. You will also have the opportunity to test your new skills using practice trials involving radiant heat stimuli.

Studies have shown that subjects can achieve the best results when they use active, vivid imagery to think of their arms as numb. Visual and auditory recall are the easiest. It is helpful to keep in mind that we have all experienced analgesia (loss of sensation) in one form or another -- e.g., our legs, feet or arms may "go to sleep", or our limbs may become numb when blood circulation is reduced or cut off.

Another common experience is the sensation of numbness in the cheeks when the dentist gives Novocaine. The experience of numbness in various areas of the body is common. Today we will be concentrating on you producing numbness in your right forearm. This training can allow you to experience lower heat intensities in the posttest portion of this experiment.

In order to concentrate more fully on the experimental instructions I would like you to settle back in the chair and in a moment I will ask
you to close your eyes. You may take off/take out your glasses or contact lenses now if you wish. Please roll up the sleeve on your right arm. Please close your eyes now and let yourself relax. Focus in on the sound of my voice. If you find your thoughts straying at any time, you can let all of the extraneous thoughts float out of your mind. You can become even more alert and at the same time very relaxed by focusing back in on the sound of my voice and the experimental instructions. Keep in mind that if you should feel uncomfortable during the exercises/instructions at anytime you are free to move your body into a more comfortable position.

So that you can become more fully relaxed and eliminate all tension from your body, in a moment we will focus on your patterns of breathing. I'd like you now to take in a deep breath by expanding your abdomen, pushing down with your diaphragm, so that your abdomen sticks out a little. Now, as you've taken in as much air there as you can, hold it...hold it... and feel the tension...feel the tension. Now slowly, slowly let all the air out through your mouth, breathing out at least two or three times slower than you breathed in. Good. Now, let's try that again. Breathe in by pushing out with your abdomen, down with your diaphragm. Fill your (lower) lungs with as much air as your can by this method and hold it, feel the tension as your lungs are full...feel the tension...feel it...and now slowly let it out, twice as slowly as you breathed it in. Good. Let's try that again. Slowly fill your lower lungs with air by pushing out your abdomen and pushing down with your diaphragm. Feel the tension as your hold in the air...feel the tension
feel it...now very slowly and smoothly let out the air and pay attention to the relaxation which follows the tension. By concentrating now on the slow, easy and smooth rhythm you can breathe deeply and steadily. Breathing in...and then breathing out...breathing in...and then breathing out. Some people find that they are better able to keep up this rhythm if they say silently to themselves...breathe in...relax...breathe in...relax. As they establish this rhythm they can concentrate on letting all the tensions in their body flow out as the air is exhaled...Remember...breathe in...relax...breathe in...relax.

Keep in mind that you are in total control of your senses and you can let yourself experience all of the sensations described in these instructions.

I'd like you to begin now by letting your right arm rest limply in your lap, like the arm of a rag doll, completely relaxed. In a moment you will receive a series of instructions and exercises which will allow you to both relax this arm and also feel it as numb and insensitive. As a result of the exercises that I will give you, you may feel that your arm is becoming heavy or you may feel a tingling in your arm. These sensations are normal and have been reported by subjects who have successfully learned these strategies. Do not be concerned if you do not experience these sensations at first because each subject's response is unique. Keep in mind that you are in full control of all of your sensations and you can have these experiences.

I'd like you to begin now by bending your right arm at the elbow and making a big muscle in your upper arm/bicep (like Superman does). Hold
that and feel the tension in your arm. Hold it...feel the tension...and
now just let your arm drop and gently rest on you lap. (Repeat X2)

Now I'd like you to tense the muscles in your lower right arm...your
forearm. Hold it and feel the tension in your forearm...feel the tension
...feel the tension...feel it...and now let your arm completely relax on
the chair arm. Focus in particularly on the feelings of relaxation, the
easing of tension that follows the tightness in your forearm. You may
even begin to feel a sensation of heaviness or tingling in your forearm.
These are all common sensations. Now let's try that again. Tense the
muscles in your lower arm...your forearm. Hold that tension and feel
it...feel it...and now relax (Repeat X2). You're doing well.

Now I'd like you to make a tight fist with your right hand and feel
that tension in your hand and arm. Feel it...feel the tension...and now
relax your arm...let your fingers spread and your hand and arm completely
relax...(Repeat X1).

I would like you to focus intently on your forearm now and also on
any tingling sensations which you have there. You can concentrate on
these sensations and can soon begin to feel your forearm becoming
slightly numb. As you concentrate your forearm can become more and more
numb...more and more numb. Do you feel the numbness? This is a good
sign and is an indication that you are completely in control of your
sensations. I would like you to focus in on your breathing again and
relate it to the numbness in your arm. Your arm is feeling more and more
numb with every breath that you take...breathe in...becoming more and
more numb...breathe out... more and more numb. (Repeat X1) You may still
be able to feel some things but you feel much less than you could before.
Your arm is becoming almost completely without feeling. Though your arm
is numb you can still move it...you can still move it even though it is
almost entirely numb. Move your right arm up and down...up and down...
notice how easy it is and how it stays without feeling. Even as you move
your arm it feels numb...It will stay numb until today's session is over.
You will notice that you can confine the feeling of numbness to your
forearm -- the area between your wrist and the joint at your elbow. It
is like feeling that you have just received an injection of Novocain or
some other numbing drug in your arm.

I would like you to vividly imagine now that you are feeling the
Novocaine being injected just below your wrist up to your forearm. You
can feel the slight needle prick, then the slight burning as the drug is
injected and then you can feel the numbness spreading up your arm. There
is a tingling feeling as it spreads. Your forearm becomes more and more
numb, top and bottom, all up your arm more and more numb...more and more
insensitive. (Repeat X1) Can you feel the numbness? (Please give
rating where 0 = no numbness and 6 = completely numb.) Good. You can
even visualize and then imagine the doctor lightly sticking your skin
with a needle and you can barely feel it because your forearm is numb.
The doctor or dentist injects another syringe of Novocaine, you arm
becomes even more numb. As the image is stronger with practice it will
become more and more numb. [Note: If subject feels no numbness,
instruct him/her that they can do it better with practice. Do you feel
heaviness? Yes? You can feel your arm heavy like a weight is on your
arm and it is difficult to more. Do you feel tingling? It is a form of numbness.

Focus intently now on the sensations of numbness in your right forearm. You can think of this area as being insensitive, just like a piece of rubber. You can eliminate any sensations of heat, pain, or discomfort. Other subjects have achieved success in thinking of their forearms in this way and it's not as hard as it seems. I would like you now to control your thoughts and to continuously think that your right forearm has no feeling. Keep thinking that you are unable to feel any heat, pain or discomfort in your forearm. Continue to think of your forearm as without heat, pain, discomfort or feeling of any kind. You can use your very best ability to think continuously and to imagine vividly that your arm is numb, insensitive, and like a piece of rubber. You can vividly imagine that your right forearm is becoming more and more numb and insensitive...more and more... The clearer a sensation is in your mind's eye, the more vividly you can perceive it. You can continue to experience it more and more intensely, until the end of our training session today, at which time you will have all normal feeling in your arm return.

Many subjects report that they obtain a more vivid image of numbness if they use concrete imagery to gain control of the sensations in their forearms. Many report success in using an imaginary model wherein they think of the nerves in their forearm as controlled by switches in the brain. When the switches turn on the arm feels sensations, when the switches are off the arm is insensitive. Other subjects may find success
in imagining that a local anesthetic (e.g., Novocaine) has been injected into the forearm and the drug is beginning to take effect; your forearm feels like your cheek does when the dentist injects Novocaine.

I would like you to get your own image as vividly in mind as you can. You may use any image that you choose, which is associated with numbness. Each subject has his own unique image for obtaining the best analgesic effect and it is the job of each subject to bring that image into focus as vividly as possible. Now I would like you to choose the image (or images) which are best for you and I would like you to see them vividly and feel the sensations of numbness and insensitivity as vividly as you can. Signal when you have the image vividly in mind by lifting your left index finger. Remember, you are in control as you allow the numbness to spread and intensify throughout your forearm. Feel the numbness, the insensitivity as it spreads in your forearm. You can get the image more and more vividly in your mind...more and more vividly...more and more numb (Repeat X2). Please give a rating now where 0 = no numbness and 6 = completely numb. You have done well. Please take time now to congratulate yourself on making a good beginning today. Do not expect to master the technique completely today, but tell yourself that you have done well and that you can feel relaxed and experience numbness and insensitivity in your arm.

You can suggest to yourself that there is some numbness in your arm, just as if you have slept on it. Imagine your skin feeling thick and insensitive. There might even be a little tingling sensation. Now, pull the sleeve up a little on your right arm and lightly touch the skin of
your right arm with your left hand and feel the slight numbness. Remember, you can suggest to yourself that the feeling will return to your arm after this session is over. We will continue training at your next appointment and you will be able to pick up from where we left off today. In a moment I will slowly count backwards from 5 to 1 and with each succeeding number you will feel more and more alert, and at the number 1, you can open your eyes. With each number the insensitivity and numbness also will fade more and more, and finally disappear with the number 1 (5-4-3-2-1, etc). You are now very alert and all the numbness has gone from your arm.
SESSION TWO -- SUGGESTED ANALGESIA INSTRUCTIONS*

Today we will continue practicing the techniques you learned during the last session -- i.e., how to make your forearm numb and insensitive. You will be able to pick up where you left off. In addition, today we will learn some new techniques and we will obtain some behavioral measures of the extent of your analgesia.

Keep in mind that the more you allow yourself to be relaxed, the more you concentrate on the instructions/suggestions, the more vividly you visualize the images given in the instructions, the greater the insensitivity and numbness you will feel in your forearm.

In order to concentrate more fully on the experimental instructions I would like you to settle back in the chair and in a moment I'll ask you to close your eyes. You may take off/take out your glasses/contact lenses now if you wish. Please also roll up the sleeves on both arms. Please close your eyes now and let yourself relax. Focus in on the sound of my voice. If you find your thoughts straying at any time, you can let all of the extraneous thoughts float out of your mind. You can become even more alert and at the same time very relaxed by focusing back in on the sound of my voice and the experimental instructions. Keep in mind that if you should feel uncomfortable during the exercises/instructions at anytime you are free to move your body into a more comfortable position.

So that you can become more fully relaxed and eliminate all tension from your body, in a moment we will focus again on your patterns of breathing. I'd like you now to take in a deep breath by expanding your
abdomen, pushing down with your diaphragm, so that your abdomen sticks out a little. Now, as you've taken in as much air there as you can, hold it...hold it...and feel the tension...feel the tension. Now slowly, slowly let all the air out through your mouth, breathing out slower than you breathed in. Good. Now, let's try that again. Breathe in by pushing out with your abdomen, down with your diaphragm. Fill your (lower) lungs with a much air as you can by this method and now hold it; feel the tension as your lungs are full...feel the tension...feel it...and now slowly let it out, twice as slowly as you breathed it in. Good. Let's try that again. Slowly fill your lower lungs with air by pushing out your abdomen and pushing down with your diaphragm. Feel the tension as you hold in the air...feel the tension...feel it...now very slowly and smoothly let out the air and pay attention to the relaxation which follows the tension. By concentrating now on the slow, easy and smooth rhythm you can breathe deeply and steadily. Breathing in...and then breathing out...breathing in...and then breathing out. Some people find that they are better able to keep up this rhythm if they say silently to themselves...breathe in...relax...breathe in...relax. As they establish this rhythm they can concentrate on letting all the tensions in their body flow out as the air is exhaled...remember...breathe in...relax...breathe in...relax. Even though you are relaxed you continue to remain alert.

Keep in mind now that you are in total control of your senses and you can let yourself experience all of the sensations described in these instructions. With practice you can become more and more proficient at
becoming more relaxed and at the same time allowing your right arm to
become numb and insensitive.

I'd like you to begin now by letting your right hand rest limply in
your lap, like the arm of a rag doll, very relaxed. In a moment you will
receive a series of instructions which will allow you to both relax this
arm and also feel it as numb and insensitive. As a result of these
exercises you may again feel that your arm is becoming heavy or you may
feel a tingling in your arm. These sensations are normal and have been
reported by subjects who have successfully learned these strategies. It
is O.K. if you do not experience these sensations at first because each
subject's response is unique. Keep in mind that you are in full control
of all of your sensations and you can have these experiences.

I'd like you to begin now by bending your right arm up at the elbow
and making a big muscle in your upper arm/bicep (like Superman does).
Hold that and feel the tension in your arm. Hold it...feel the
tension...and now just let your arm drop and gently rest on your lap
(repeat X2).

Now I'd like you to tense the muscles in your lower right arm --
your forearm. Hold it and feel the tension in your forearm...feel the
tension...feel it...and now let your arm completely relax on the chair
arm/your lap. Focus in particularly on the feelings of relaxation, the
easing of tension that follows the tightness in your forearm. You may
even begin to feel a sensation of heaviness or tingling in your forearm.
These are all common sensations. Let's try that again. Tense the
muscles in your lower arm...your forearm. Hold that tension and feel it
...feel it...and now...relax. (Repeat X2). You're doing well.
Now, I'd like you to make a tight fist with your right hand and feel that tension in your hand and arm. Feel it...feel the tension...and now relax your arm...let your fingers spread and your hand and arm completely relax (Repeat Xl).

I would like you to focus intently on your forearm now and also any tingling sensations which you have there. You can concentrate on these sensations and soon begin to feel your forearm becoming slightly numb. As you concentrate, your forearm can become more and more numb...more and more numb. Do you feel tingling/numbness? This is a good sign and is an indication that you are completely in control of your sensations. I would like you to focus in on your breathing again and relate it to the numbness in your arm. Your arm is feeling more and more numb with every breath that you take...breathe in...becoming more and more numb...breathe out...more and more numb. (Repeat Xl.) You may still be able to feel something but you feel much less than you could before. Your arm is becoming almost completely without feeling. Though your arm is numb you can still move it...you can still move it even though it is almost entirely numb. Move your right arm up and down. Notice how easy it is and how it stays without feeling. Even as you move your arm it feels numb...It will stay numb until today's session is over. You will notice that you can confine the feeling of numbness to your forearm -- the area between your wrist and the joint at your elbow. It is like feeling that you have just received an injection of Novocaine or some other numbing drug in your arm. I would like you to imagine now that you are feeling the Novocaine being injected just below your wrist, up your forearm. You
can feel the slight needle prick...then the slight burning as the drug is
injected, then you can feel the numbness spreading up your arm, there is
a tingling feeling as it spreads. Your forearm becomes more and more
numb, top and bottom, all up your arm more and more numb...more and more
insensitive. (Repeat X1.) Can you feel the numbness? (Please give a
rating where 0 = no numbness and 6 = completely numb.) Good. You can
even visualize the doctor lightly sticking your skin with a needle and
you cannot feel it because your forearm is numb. You cannot feel it.

In a moment I will take an alcohol swab and lightly run it over the
area of your forearm which you can make more and more numb. More and
more...Ready? Now I will lightly pass the swab over your forearm. The
slightly cooling sensation can help you to focus on the cooling effect of
the numbness you feel in your forearm, especially in the area that has
been outlined with the alcohol swab -- between your elbow joint and your
wrist.

Any heat or warm sensations will disappear as the warmth turns into
a numbing coolness, which is very pleasant. Even though your arm is
resting and still now, later you will be able to move it freely and/or
have it touched by the experimenter and your forearm area will still be
numb, even when your arm moves.

I would like you to focus intently now on the sensations of numbness
in your right forearm. You can eliminate any sensations of heat, pain or
discomfort. Other subjects have achieved success in thinking of their
forearms in this way and it's not as hard as it seems. I would like you
now to control your thoughts and continuously think that your right
forearm has no feeling. Keep thinking that you are unable to feel any heat, pain or discomfort in your forearm. Your forearm is without heat, pain, discomfort or feeling of any kind. Please try to the very best of your ability to think continuously and to imagine vividly that your arm is numb and insensitive. Keep thinking and vividly imagining that your right forearm is becoming more and more numb and insensitive...more and more... (Repeat X1). The clearer a sensation is in your mind's eye, the more vividly you can perceive it. You can continue to experience it more and more intensely, until the end of our training session today, at which time you will have all normal feeling in your arm return.

I would like you to get an image very vividly in mind now (as you have already shown that you are able to do). You still cannot feel anything in your right forearm. Just continue to think of it being quite numb...more and more numb...and all the feeling is going out of it.

And as I go on talking to you...your right forearm is beginning to feel colder and colder...as if it were surrounded with ice. Just picture your forearm being packed round with ice...and as you do so...it is feeling colder and colder...more and more numb and insensitive. As soon as you feel your forearm becoming cold and numb have the image vividly in mind...please lift up the index finger on your left hand.

Your right forearm has now become so cold and numb...that you are losing all feeling in it. Soon you will not be able to have any feeling at all...no discomfort, no warmth, no pain. In a moment or two...I am going to count slowly to three. And when I reach the count of three... your forearm will be insensitive...and you will be able to feel nothing
in your forearm. One...colder and colder...more and more numb and insensitive...losing all sensations. Two...your forearm is now quite numb and dead...there is no feeling in it at all...just as if it had gond to sleep. Three...your forearm is completely numb...cold...and insensitive...you cannot feel anything in your forearm at all! Please give a rating now where 0 = no numbness and 6 = completely numb.

Now I am going to stimulate your other (left) forearm with a sharp probe (pin) and you can feel the stimulation there. [Experimenter stimulates left forearm area of the subject and then right forearm.] Notice the difference between the sensation in your left forearm and the right. Feel the difference. [The right forearm is then pricked lightly. Further firmer pricking will establish the extent of the analgesia.]

Please take time now to compliment yourself on continuing to do well in your exercises. You have allowed yourself today to experience numbness...lack of feeling in your arm...you have compared this to normal feeling in your other arm. Youi can use these skills in following sessions to improve your ability to tolerate radiant heat. We will continue training at your next appointment and you will be able to build on the skills you have already demonstrated.

In a few minutes...your right forearm will become quite normal again. You will be able to feel it becoming warmer and warmer and all of the feeling of numbness will leave it. When I begin to count backwards from 5 to 1 it will gain more and more feeling...it will become more and mores sensitive...it will become quite normal again...it will feel just the same as your other arm. All the sensations will return...and now you will feel everything...just the same as with your other hand and forearm.
Now I will slowly count backwards from 5 to 1 and with each succeeding number you will feel more and more alert and at the number 1, can open your eyes.
SESSION THREE -- SUGGESTED ANALGESIA INSTRUCTIONS*

Today we will continue practicing the techniques you learned during the previous session, i.e., how to make your forearm numb and insensitive. You will, as before, be able to pick up where you left off. Today we will also obtain some behavioral measures of the extent of your analgesia. We will also have you try out your new skills by exposing you to 20 practice trials of radiant heat stimuli at the end of today's training session.

Keep in mind that the more you allow yourself to be relaxed, the more you concentrate on the instructions and suggestions, the more vividly you visualize the images given in the instructions, the greater the insensitivity and numbness you will feel in your forearm. Today you will assume greater and greater input on your own, as you have previously demonstrated skill in imagining your arm to be numb and insensitive.

In order to concentrate more fully on the experimental instructions and use your own images, I would like you to settle back in the chair and in a moment you may wish to close your eyes. You may take off/take out your glasses/contact lenses now if your wish. Please roll up the sleeves on both arms. You may also close your eyes now and let yourself relax. Remember that today the emphasis is on you using your own suggestions, so that you can experience numbness in your forearm and the feeling will return at the end of our training today.

As before, if you find your thoughts straying at any time, you can allow any extraneous thoughts to float out of your mind. By doing so,
you can become even more alert and at the same time very relaxed by focusing back in on the sound of my voice and/or your own images and suggestions. You have done well using guided imagery (suggestions) and today you can successfully use your own suggestions to obtain vivid images. As before, if you should feel at any time during today's session that your body is uncomfortable you may move it into a more comfortable position.

I'd like you to begin now by regulating your breathing...steadily...smoothly...taking a couple of minutes to quietly attain a good breathing rhythm. You can suggest your own steady and smooth rhythm to yourself. Please signal (by lifting up your left index finger) when you have attained a good, steady breathing rhythm and are as relaxed as you can be. [Allow the subject 2-3 minutes]. (Repeat X1)

Keep in mind now that you are in total control of your senses. You have done well with visual images in the past and you can allow yourself to experience all of the sensations which you suggest to yourself. With practice you have become proficient at achieving a deeper state of relaxation and at the same time allowing your right forearm to become more numb and insensitive.

You may now use your own series of exercises (like we used here previously, in training sessions) to relax and tense your arm and forearm. You may do this now, progressing at your own rate. Pay attention to any feelings of tingling or numbness which you may experience. [Here the subject is given 2-3 minutes in which to practice exercises. After this time the experimenter (E) continues.].
Please signal by lifting up the index finger of your left hand as soon as you feel tingling and/or numbness in your right forearm. [S signals] Good. You can now use your own visual image to increase the numbness in your forearm. For example, you may want to allow the image to develop where your forearm becomes more and more numb with every breath that you take. Or you may choose to use the image of the doctor injecting Novocaine. Or you may choose to use the image of submerging your arm in ice-cold water. Or you may choose to use the image that your arm is like rubber. Or you may use another image completely of your own devising.

Whatever your image, allow it to develop more and more vividly. [Allow the image to develop up to approximately two minutes.] Good. You have shown the ability to increase numbness with training. Today you can allow your forearm to feel very numb, perhaps as much if not even greater than ever before, as you assume greater and greater control. Please give a rating of the level of numbness in your right forearm at this time (where 0 = no numbness and 6 = completely numb).

You will also find that you are also able to move your arm and still experience numbness. It is easy; see how you can move your arm up and down but it remains numb. Later in today's training the experimenter will manipulate your arm (during radiant heat trials) and you will continue to feel numbness even though it is easy to move your arm around.

Now we will focus for a moment on your other (left) arm. I would like you to get an image clearly in mind. Imagine you are in a bath-
room and hot water is running from a spigot/faucet. You know its hot because you can see the steam rising from the water as it flows from the faucet. As soon as you can picture steaming water please lift up the index finger on your left hand. [Repeat as needed.]

Fine. You may put your hand down again. Now, you wonder how hot the water is...so you walk over to the running water and put your left forearm (with the underside up) under the water.

Picture this vividly in your mind so that you will feel the heat. Your forearm is beginning to smart...it feels tender and painful. Your arm is tingling...and feels warm and tender. As soon as you feel these sensations...please lift up your left index finger again.

Good. You may put your finger down again. Now I'm going to probe your left arm with a needle. Your left arm is so tender and sensitive...it feels almost like a stab from a knife, so tender and sensitive...it feels terribly painful. [Here the experimenter probes the subject's forearm with a pin.] See...how tender and painful it feels. You'll notice the difference when I touch this (left) arm (with a needle)...and when I touch the other arm with a needle. Now I'm going to touch the other arm (left)...now this one (right) again. Can you feel the difference?

Now I would like you to relax and realize that the sensitivity and painfulness in your left arm is beginning to fade and will completely go away. As I count slowly backwards from three to one,
normal feeling will return to your left arm and any tenderness, sensitivity or painfulness will fade. Ready? [3...2...1, etc.]

In a moment I will take a swab and paint circles of India ink on your forearm, and lightly run it over the areas of your forearm which you can make more and more numb. More and more...Ready? Now I will lightly pass the swab over your forearm. The slightly cooling sensation can help you to focus on the cooling effect of the numbness you feel in your forearm, especially in the area that has been circled with the alcohol swab -- between your elbow joint and your wrist.

Any heat or warm sensations will disappear as the warmth turns into a numbing coolness, which is very pleasant. Even though your arm is resting and still now, later you will be able to move it freely and/or have it touched by the experimenter and your forearm area will still be numb, even when your arm moves.

I would like you to focus intently now on the sensations of numbness in your right forearm. You can eliminate any sensations of heat, pain, or discomfort. Other subjects have achieved success in thinking of their forearms in this way and it's not hard as it seems. I would like you now to control your thoughts and continuously think that your right forearm has no feeling. Keep thinking that you are unable to feel any heat, pain or discomfort in your forearm as without heat, pain, discomfort or feeling or any kind. Please try to the very best of your ability to think continuously and to imagine vividly that your arm is numb and insensitive. Keep thinking and vividly imagining that your right forearm is becoming more and more numb and insensitive.
more and more... (Repeat Xl). The clearer a sensation is in your mind’s eye, the more vividly you can perceive it. You can continue to experience it more and more intensely, until the end of the radiant heat trials today, at which time you will be given the suggestion for all normal feeling in your arm to return again.

I would like you now to get an image very vividly in mind now (as you have already shown that you are able to do). You still cannot feel anything in your right forearm. Just continue to think of it being quite numb...more and more numb...and all the feeling is going out of it.

And as I go on talking to you...your right forearm is beginning to feel colder and colder...as if it were surrounded with ice. Just picture your forearm being packed round with ice...and as you do so...it is feeling colder and colder...more and more numb and insensitive. As soon as you see the image vividly and feel your forearm becoming cold and numb...please lift up the index finger on your left hand. Good.

Your right forearm has now become so cold and numb...that you are losing all feeling in it. Soon you will not be able to have any feeling at all...no discomfort, no warmth, no pain. In a moment or two...I am going to count slowly to three. And when I reach the count of three...your forearm will be insensitive...and you will be able to feel nothing in your forearm. One...colder and colder...more and more numb and insensitive...losing all sensations. Two...your forearm is now quite numb and dead...there is not feeling in it at all...just as
if it had gone to sleep. Three...your forearm is completely numb...
cold...and insensitive...you cannot feel anything in your forearm at all! Your level of numbness may be deeper than ever before. Please give a rating of your level of analgesia now, where 0 = no numbness and 6 = completely numb.

In a moment I will ask you to open your eyes when I count to three. The numbness will remain in your control under the end of today's session. Remember, the experimenter will move your arm but it will remain numb. You have shown the ability to do well in imagining your arm to be numb and insensitive and you have become better with practice. You are in control and can rely on your own best and most vivid images. Focus in on the insensitivity and numbness. You will find that you will experience an overall decreased sensitivity to the radiant heat stimuli...even the higher heat intensities may feel only slightly warm. You can use these skills in the posttreatment phase of this experiment.

After the 20 radiant heat practice trials are completed, all the numbness and insensitivity in your arm will fade when you receive that suggestion. All feeling will return to your arm and it will feel completely normal again.

I will now count to three aloud. At the count of three your right forearm will remain very insensitive and numb and you may open your eyes and slide forward in your chair to the radiant heat apparatus l...arm still numb...2...arm still numb...3. [Subject moves forward in chair.].
You may close your eyes again if you wish after the trials begin (i.e., if you can remember the rating scale) or you may leave them open. Remember, use your images as vividly as you can. Please give me a rating of your level of analgesia now. I will now take your arm and move it after each radiant heat stimulus presentation. It will remain numb even when it is moved. As before, if any stimulus is too intense, you may withdraw your arm. [Twenty practice trials of radiant heat stimuli are then given to the subject. At the end of the 20th trial the experimenter continues.]

That concludes practice trials for today. Please pause and focus in on the level of analgesia in your forearm now and give a rating. Now, I will count backwards from five to one and with each number the numbness will fade more and more from your forearm and with the number 1 it will be completely normal again. 5-4-3-2-1, etc.
FOOTNOTES FOR APPENDIX C

The suggested analgesia instructions are based in part on the following sources:


SESSION ONE -- EXPECTANCY CONTROL INSTRUCTIONS*

First, let me explain that we'll be discussing theories of pain and research on pain, stress, anxiety and coping strategies for dealing with pain. Studies have shown that when subjects receive this information they experience a decreased sensitivity to radiant heat stimuli.

We'll begin by discussing some of the older, traditional theories of pain. One view focused almost exclusively on the sensory (i.e., physical sensation) component of pain. Could you see any problems with this kind of theory?

[Here the subject (S) answers.] If there is no answer then the experimenter (E) asks: Do you think that psychological variables may play a role?

An older view also suggests that the greater the tissue damage, the greater the pain reaction. For example, if a one inch incision were made on the hand (demonstrate) this theory would suggest that a two inch incision would hurt twice as much. Could you see any problems with this theory?

I would like to focus now on some research which evaluated the influence of environmental variables. The study I would first like to describe was done by Beecher (1956). He studied over 200 men, in each of two groups. The first group of men was wounded at Anzio in World War II. They all had "gut" wounds. Only about 25% of these men wanted a narcotic (painkiller) for pain relief. The second group consisted of civilians who received similar surgical wounds under anesthesia. Of
this group over 80% wanted pain relief. Why do you think these
differences were found?

[S responds. The experimenter explains Beecher's hypothesis that
differences were due to the significance assigned to the wound, e.g., in
battle the wound meant a ticket to safety; in civilian life surgery
meant disaster.]

As discussed above, it has been noted that pain reactions are
affected by psychological variables. Now we will discuss several
examples.

If we are dealing with two groups of people, one group which has
high scores on anxiety, depression and stress and the other group which
has low scores, assuming that both groups have similar pains, which do
you think will have the highest pain report? Why? [E explains that
anxiety (depression) stress increases pain report.]

Which of the two do you think would respond most to placebos? [E
explains that placebos are "sugar pills" or inactive treatments given
with the expectation that the treatment is active.] Why? [E explains
that with increased stress, placebos are more effective.]

To use another example, suppose we compare another two groups of
people, one of which has pain of pathological origin (e.g. people in
chronic pain like low back pain) and the other which has pain induced
experimentally (i.e., in the lab). Which group do you think would
report the highest anxiety? [E explains that the group with
pathological pain reports the highest anxiety, possibly because the
injury/pain is permanent/unavoidable.]
Given that there are these differences which group do you think would respond better to narcotic pain medication (e.g. morphine)? Why?

[E explains that the pathological pain group responds best to narcotics because of the greater "physical" component of pain.] We have talked about how different psychological strategies may affect pain response. Can you think of any examples of your own?

[E encourages subject to relate examples. Then the experimenter gives the example of a football player who injures himself during a game and is so "into" the game that he doesn't notice his injury until he goes to the sideline. E asks why the football player may respond this way, i.e., what variables are at work?]

The communications aspect of pain, especially for those in chronic pain, has also been studied. Take for example the instance of a man who is a construction worker and who is injured on the job such that he is severely or even completely disabled, or a woman who is a housewife and who is injured and cannot perform her household duties. We can assume that both are in chronic pain and they they communicate through their pain behavior to significant others (e.g., family and friends). What different messages do you think could be communicated by the person in chronic pain [if S does not understand, ask how the patient's concerns would change behavior as result of chronic pain].

[Here the subject is encouraged to mention different communications/messages, e.g., responses to gain sympathy, bearing up under pain like "a real man" or that it's legitimate to avoid daily responsibilities, or "I'm being punished".]
Next, I would like to discuss one of the theories of pain, probably the most predominant one. According to this theory, pain is not a simple stimulus-response, but is rather a complex, multi-dimensional phenomenon.

[Here the subject is shown a diagram of the Gate Control Theory Model and various components are explained, e.g., the gating mechanism, the sensory-discrimination, the motivational affective, the cognitive-evaluative components of pain and the output (motor response) components.]

One advantage of the Gate Control Theory is that many kinds of clinical ('real-world') responses can be explained. For example have you ever heard of something called a transcantaneous nerve stimulator (TNS)? [If not then explain. A TNS unit is a small, low voltage electrical generator which is applied to the area of the body in pain and is used for persons in chronic pain. For example, for people with low back pain, the TNS unit is placed with electrodes on the low back area. Patients report clinically a reduction in pain.] What features of Gate Control Theory do you think explain this reduction in reported pain?

[Here the subject answers. If he/she does not have an answer then it is explained that the TNS electrical impulses act as overriding or competing stimuli such that the threshold trigger on the gating mechanism is set higher and thus the report is reduced.]

There is also evidence that people's responses to painful stimuli vary as a result of some demographic variables, e.g., cultural/racial; sex; and age.
First of all let me explain that traditionally there have been two ways of measuring pain, threshold and tolerance. For threshold measures the subject signals at the time he/she first feels the stimulus. For tolerance measures the subject signals when he/she can no longer endure the pain.

For cultural/racial variables, on which measure (threshold/tolerance) do you think differences are found? [Subject answers, experimenter explains that threshold is probably more a function of physiological variables and tolerance more a function of psychological variables.]

For example, one study found that, in comparing subjects of Italian vs. Jewish extraction that subjects of Italian extraction who had the highest upper threshold had the highest heart rate and subjects' of Jewish extraction with the highest upper threshold had the lowest heart rate. What do you think could account for this difference?

[Subject answers. The experimenter explains that the hypothesis is that learned cultural/social variables may account for the difference.]

By way of example, let me describe a study that was done using two groups of subjects: (a) one group of children of mothers who showed high dental anxiety and (b) a second group with mothers who showed low dental anxiety. When their children were subjected to experimentally-induced electric shock, which group do you think reported the lowest pain?
Subject answers. Subject indicates that the group with mothers with high dental anxiety reported the greatest pain. The subject is then questioned regarding variables explaining these differences. Subject answers. The experimenter explains, as appropriate, that modeling/learning effects seem to be involved and that this is probably analogous to learning effects regarding cultural differences.

Subject answers. Experimenter explains, as appropriate, that significant differences are found for tolerance (no difference or only slight differences for threshold) and that they are probably due to socially transmitted behavior patterns, i.e., the male must be "macho" with respect to pain responding.

Finally, I would like to talk about age differences with regard to pain responding. Given that we compare the elderly with the young, who do you think shows the highest pain response for a given pain stimulus?

Subject answers. As appropriate, the subject is questioned regarding whether he/she thinks that physiological sensitivity to pain is greater in elderly or young, and it is pointed out that studies show decreased physiological response in the elderly. Then, the subject is asked, as appropriate whether for a given painful stimulus, the role of previous experience with pain has an influence. The experimenter indicates that studies show that for a given stimulus, elderly subjects (who have had more general experience with pain) tend to give a decreased response to pain.
This concludes our session today. Next time we will continue to review research on pain and anxiety and variables which relate to pain and coping strategies which have been found to be effective in dealing with it.
SESSION TWO -- EXPECTANCY CONTROL INSTRUCTIONS*

By way of review, recall that in the last session we discussed the fact that reported pain may be affected by emotional state. Given that we have two groups of people with similar pain and one group reports high depression and anxiety and the other has low reports on these measures, which group would have an average higher pain report? [The experimenter explains that findings are that subjects high in anxiety and depression have a higher pain report.]

Today we will be discussing other psychological variables which affect pain report, including the variable of anxiety, and research on coping styles and coping strategies.

First of all, a study has compared patients who report high scores on depression with patients who have high scores on anxiety and hysteria [The experimenter explains that hysterical symptoms involve over-dramatization and "holding on" to pain symptoms.] Which of the two groups do you think would respond best to (short-term) treatment? [Subject answers. The experimenter explains that the "depression" group responds better to treatment.]

I'd like to spend some time now talking about the concept of anxiety, which has been described in ambiguous terms in older research. Let's talk about your ideas regarding anxiety. Do you think that there are different kinds and/or different intensities of anxiety? [Subject answers.]

Traditionally, theories of anxiety have made the distinction between "trait" vs. "state" anxiety. According to this theory people
with trait anxiety have anxiety virtually all of the time and in all situations. State anxiety refers to anxiety which is specific to a certain situation (e.g., fear of bodily harm). Can you see any problems with the concept of trait anxiety (i.e., that high trait anxious individuals show high anxiety in all situations)? [Subject is asked: "Do you think there are a significant number of high trait anxious individuals?" and E then answers, "It is explained that, clinically the incidence of high trait anxious individuals -- i.e., people highly anxious in all situations, is fairly low."]

The current theory follows the model that people with high trait anxiety probably have a predisposition for high anxiety in specific situations and that they thus exhibit high state anxiety in these situations.

For example, if an individual has high trait anxiety in situations involving the possibility of bodily harm or pain, then they would show high state anxiety in these potentially harmful situations, and this anxiety would then generalize to other, similar situations.

A good deal of research has been devoted to how people cope/deal with pain. How, for example, do you feel that anxiety contributes to pain, (especially using the Gate Control theory model)? [Subject answers.]

One specific stressor that has been studied to examine people's different coping strategies has been the stress of surgery. Can you think of examples, good and bad, of different coping strategies to deal with the stress of impending surgery? [Here the subject is encouraged
to generate examples of coping strategies. Several possible strategies are enumerated by the experimenter, adding to examples as subject mentions them, e.g.

(a) denial - refusal to deal with stress, denial of opportunity for any further information regarding the procedure of surgery, etc.

(b) talking about the stress (sensitizing) - i.e., talking out fears and wanting to know more about the procedure,

(c) rationalizing - e.g., "other people survived",

(d) catastrophizing - imagining the worst,

(e) introjection - blaming self, e.g., "why didn't I take better physical care of myself -- why did I let this happen?"

(f) trusting in the doctor - allowing self to feel confidence in the caretaker.

Perhaps the most research evidence has accumulated on people who deny (also called Reducers or Avoiders) vs. those who try to find out more (also called Sensitizers or Copers) about surgery.

One study which specifically compared these two different coping styles was done in a laboratory setting using radiant heat and (blood) pressure algometer pain. Measures of tolerance were used. What do you think that the different patterns of tolerance were? [Here subject answers]. It was shown that, during the first half of the experiment the repressors yielded higher pain tolerance than sensitizing but showed a significant decrease on tolerance during the second half of the experiment. Sensitizers on the other hand, kept fairly steady tolerance scores, i.e., did not show any significant effects of repeated exposure to painful stimuli. Which strategy do you think may be more effective over time?
A study which was done using the specific stress of surgery looked at the effect of providing vs. not providing surgical patients with information regarding surgical procedures as a function of their coping styles. Three groups of surgical patients were used and were rated as sensitizers, avoiders or nonspecific defenders (who used both some sensitizing and some avoidance strategies).

What do you think that the hypotheses of the experimenters were regarding how the 3 groups would be ranked in terms of quicker recovery times and fewer medications requested (post-surgery)? [Subject answers. It is explained that the hypotheses were that (1) the sensitizers would welcome the information and show both a decreased recovery time and a decrease in amount of medicine requested, relative to other groups, (2) avoidance would show the slowest recovery times, and (3) nonspecific defenders would score in between.]

The actual results, however, were slightly different:

1. Nonspecific defenders who were provided information showed the fastest recovery rates.

2. Avoiders required more medicine but recovery times were not significantly different from other groups.

3. Sensitizers did not appear to be affected by information.

One other area which has been the focus especially of laboratory research has to do with how a particular stessor is appraised or evaluated.

As an example when we compare two different individuals and how they respond to the same stress, do you think there could be different levels of reported stress? What could this be attributed to? [Subject
answers. It is pointed out that differences may be due to previous experiences with the stressor, i.e., how the stressor is evaluated.]

One area of research which has focused on how a stressor may be evaluated has focused on laboratory stress. One study used a film as a stressor and varied how it was presented by use of different narratives or sound tracks. The film was of a circumcision ritual in a primitive tribe; the film showed circumcision in vivid detail. The 4 conditions were as follows:

**Group One** (called the "denial" group) denied the harmful features of the film; this sound track emphasized that the adolescent boys were not mutilated or harmed nor in significant pain. The ritual was depicted as a happy, joyous occasion.

**Group Two** (called the "intellectualization" group) presented an anthropological view (like a documentary) of the ritual. Words to describe the process were detached and unemotional, like in a technical manual.

**Group Three** (called the "trauma" track) emphasized the horror and dread of the boys and the harmful consequences of the procedure.

**Group Four** consisted of a silent version of the film, i.e., no narrative.

What do you think the findings were in terms of measures of stress? [Subject answers. It is explained that the trauma group had highest measures of stress, followed by the silent version and both the denial and intellectualization sound tracks reported a significantly reduced stress reactions (ANS). Subject is then asked what the
implications from this study are for coping with clinical stress? Subject answers. It is pointed out that how a stress is presented/evaluated is important."

Finally, research has shown that several different types of coping strategies are effective in reducing pain and stress.

Generally, there are two major strategies: (1) relaxation and (2) attention-diversion (getting one's mind off the painful stimulus). In addition there are strategies which combine the two, e.g., La Maze (natural childbirth), which involves deep breathing/relaxation and attention-diversion (concentrating on a mental image or physical stimulus like a spot on the wall).

Relaxation involves slow deep breathing and/or tension/relaxation. Attention diversion may involve any of a number of different strategies.

(a) **focusing on physical characteristics of the environment** (e.g., counting ceiling tiles),

(b) **focusing attention on various thoughts** (e.g., doing figures in head/mental arithmetic, singing),

(c) **focusing attention on part of the body feeling painful stimulus** (e.g., cutting finger and focusing in detached manner on, say, biological sensations).

(d) **imaginative inattention** (i.e., getting an image that is incompatible with the experience of pain, e.g., thinking of being at a party, at the beach, or being with a special friend),

(e) **imaginatively transforming the pain**, interpreting sensations as something other than pain (i.e., thinking of part of body receiving painful stimulus as numb, e.g. arm injected with Novocaine; arm like rubber; or arm as like that of "6 Million Dollar Man").

(f) **Imaginatively transforming context** thinking of a scene where the painful/intense stimulus is different from the situation
one is already in (e.g., thinking of being James Bond after being shot in arm and escaping in car going at a high rate of speed).

That concludes today's session. Next time we will discuss other variables affecting perception of pain/stress and summarize research findings and how they may be applied.
SESSION THREE -- EXPECTANCY CONTROL INSTRUCTIONS*

By way of review, keep in mind that in the two previous sessions we have reviewed theories of pain (especially Gate Control Theory) and some variables that influence the pain response (e.g., racial/cultural, sex, age, anxiety) and research evidence for successful coping strategies. Today we will review these findings and I will ask you how some of your knowledge (in terms of research findings) could be applied. Remember, as stated previously, your knowledge regarding research findings about pain will allow you to experience a decreased sensitivity to radiant heat in the follow-up (posttest) session of this experiment.

First of all I'd like to take a few moments for you to think of some examples of coping strategies and how they might be explained by pain theory (especially Gate Control Theory).

[Here the subject give a couple of examples; the experimenter may prompt, e.g., increased anxiety (motivational-affective component of Gate Control Theory), as in anticipating muscle tension before receiving an injection in a doctor's office, can lower the trigger point (at gating mechanisms) and the subject will make pain report more quickly.]

Now, I'd like to review some of the previous studies/findings that we've discussed. Do you recall the study by Beecher (who reported on the different pain responses of soldiers vs. civilians to similar gut wounds)?

[Here subject is asked to summarize findings and experimenter reminds subject of findings if subject cannot remember.]
What do you think the implications of Beecher’s study are for teaching people to cope?

[Here subject answers. Experimenter may prompt if subject cannot give examples, i.e., it may be important to vary the setting in teaching people to cope with pain].

There is also some evidence that inducing positive affect can improve people’s ability to deal with pain. For example, if we have two people with identical pain stimuli and one person shows negative affect (sadness) and one positive affect (in a good mood/happiness), which one do you think would have the lowest pain report? Right, the one with the positive affect. Can you think of ways to apply this knowledge (i.e., in a situation where he/she experiences pain (e.g., falls down)?

[Here subject answers. Experimenter may prompt if subject cannot think of answer, e.g., might give the child positive reinforcement, such as a toy or sucker but not as a reinforcer for e.g., crying per se.]

There is another line of evidence which has to do with inducing positive affect in teaching people to deal with chronic pain. One report was given by the writer Norman Cousins, who had a terminal illness and who decided to start a program involving regular viewing of old comedy films (e.g., Marx brothers) and generally increased exposure to comedy materials. At the same time he was receiving regular medical treatment. Eventually, he found that after implementing his program his disease went into remission. Do you think these findings are explainable in terms of psychological variables impacting on physiological
states? And, how do you think these findings may be explained in terms of Gate Control Theory? [Subject answers. Experimenter prompts if needed, i.e., inducing positive affect may influence motivational affective component of pain.]

By way of review, do you recall the phenomenon of TNS as used to reduce clinical pain? [If not, experimenter reviews.] What do you recall as an explanation in terms of Gate Control Theory? [Subject answers. Experimenter prompts as needed, i.e., notion of competing (physical) stimuli from TNS unit which elevates pain threshold through gating mechanism.]

What do you think the relationship of this principle is to other techniques, i.e., those aimed at distracting?

Do you think that distraction is an ingredient in Cousin's technique? [Subject answers. Experimenter asks, "How do you think it might operate?"]

Do you recall our previous discussion of La Maze (natural childbirth)? How is distraction involved? [Subject answers.]

Based on what we have discussed today, what do you think the implications are for teaching a child to deal with pain? [Subject answers. Experimenter probes regarding positive affect and distraction technique.]

Do you think that the positive affect technique and the distraction technique can be distinguished? [Subject answers. Experimenter may point out that techniques appear to work by different mechanisms but achieve the same ends.]
By way of review, recall our discussion of individuals with chronic pain. We discussed the fact that pain behavior may be influenced by reinforcement from the immediate environment, e.g., family and friends. From the perspective of the individual with chronic pain, what factors do you think would affect his/her pain behavior? How about from the perspective of significant others (family/friends)? [Here subject answers. Experimenter emphasizes effect of reinforcement, e.g., reinforcing pain behavior by being sympathetic.]

Given the example of two groups of people recovering from surgery -- one group high in anxiety and depression and one group low -- which group would you think would report the greatest pain? [Subject answers.] What do you think the implications are for giving medications, i.e., for the anxious patient could narcotics be substituted for with anti-anxiety medications? Why might this work? [Subject answers.]

We have spent considerable time in our training talking about how anxiety contributes to pain and varying coping strategies which could reduce reported pain and anxiety. Do you think that anxiety and pain can be distinguished (or confused)? [Subject answers.] Can you think of ways to train people to distinguish between the two? [Subject answers. If subject cannot elaborate, experimenter asks what bodily sensations are associated for the subject with anxiety? Subject answers and is prompted, as needed regarding anxiety responses such as sweating, tightness in stomach, increased respiration, headaches, etc.
Subject is then asked whether he/she could think of techniques which would decrease physical arousal/anxiety, e.g., relaxation or biofeedback.]

Based on our training, what might be some good ways of instructing a child who is very anxious before a surgical procedure (e.g., tonsillectomy) or before going to the dentist. [Subject responds, and is prompted regarding using strategies such as accurate information imparting, tolerant modeling, teaching slow deep breathing, etc.]

What influence do you think that early experience with pain (e.g., a painful electric shock or trauma; or a burn) might have on future pain responding, especially to later experience with similar stimuli? Do you think that avoidance/phobic responses could result? [Subject answers.]

What do you think might be effective strategies for teaching the (traumatized) individual to deal better with pain? [Subject answers. Probes are made for answers such as: reducing fear by having subject approach feared stimulus gradually, making connection between irrationality of fear toward feared stimulus (e.g., subject not to avoid gas stoves simply because of burn as a child).]

That concludes our training. Evidence has shown that when people have information regarding how pain is increased and regarding how to deal with pain, they often experience less pain. You will find that your new knowledge regarding pain will result in you feeling the radiant heat stimuli as less intense (even at the higher levels) in the follow-up (posttest) portion of this experiment.
Do you have any questions? [Note: if subject asks if he/she should use coping strategies in posttest, answer that new knowledge regarding pain response should result in decreased perception of pain. Do not endorse coping strategies per se.]
FOOTNOTE FOR APPENDIX D

The expectancy control instructions are based in part on the following sources:


INTRODUCTION TO PRETREATMENT PHASE

Before we begin, I 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 this experiment we will use the machine in front of you to produce the radiant heat stimuli. What this is, essentially, is a spot of light, a magnifier and a shutter. We use a light dimmer to control the intensity of the light which will affect 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, you will be shielded from the apparatus by a curtain which will rest on your arm. 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 15 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. '0' indicates that you felt nothing at all, '1' indicates that you felt something but you could not really identify it, '2' means that you felt that the stimulus was warm, '3' indicates that you thought the stimulus was hot, '4' means that it was faintly painful, '5' indicates that the stimulus was painful and '6' 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. 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. (Taken from Clark (1969) with slight modifications.)

'0' indicates that you felt nothing at all, '1' indicates that you felt something but you couldn't really identify it, '2' means that you felt that the stimulus was warm, '3' indicates that you thought the stimulus was hot, '4' means that it was faintly painful, '5' indicates that the stimulus was painful, and '6' 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. Remember, you are to rate the stimulus after the tone. Please speak loudly, clearly and distinctly so that the experimenter can hear you. When the actual experiment begins, you will be handed the printed scale and may refer to it as needed. Are there any questions at this point?
INTRODUCTION TO POSTTREATMENT 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 15 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: '0' indicates that you felt nothing at all, '1' indicates that you felt something but you could not really identify it, '2' means that you felt that the stimulus was warm, '3' indicates that you thought the stimulus was hot, '4' means that it was faintly painful, '5' indicates that the stimulus was painful and '6' indicates that you felt 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. Remember, you are to rate the stimulus after the tone. Please speak loudly, clearly and distinctly so that the experimenter can hear you.
When the actual experiment begins you will be handed the printed scale and may refer to it as needed.
APPENDIX G
POSTTREATMENT ANALGESIA INSTRUCTIONS -- SUGGESTED ANALGESIA

You have shown that, with practice, you have become better and better at imagining your arm as numb and insensitive. Today you will use these skills again during stimulation with radiant heat. You will find that, because of your ability to imagine your arm as numb and insensitive, you will feel the radiant heat less intensely today. You have shown an increased ability to use your own images successfully in imagining your arm as tingling, numb and insensitive. You can continue to use your own self image more and more vividly today. Using self images is called autosuggestion.

In a moment I will allow you some time to practice the exercises and cognitive strategies that you have used successfully in your training sessions. Assume a comfortable position in your chair.

You may close your eyes now if you like. You may begin by focusing on your breathing -- slow, steady and easy. Focus in now on your breathing, at your own pace -- slow, steady and easy. I will allow you a few moments to focus in on your slow...steady and easy breathing [allow 30 seconds to 1 minute or so].

Now you may do any arm exercises which you would like in order to focus in on feelings of tension...and relaxation [allow 1 minute or so]. Be aware of feelings of tingling, heaviness and later, numbness in your right forearm -- the area between your wrist and your elbow joint -- tensing and relaxing...tensing and relaxing.

Please signal by lifting up your left index finger when you feel tingling, heaviness or numbness in your right forearm. [Allow 30 seconds to 1 minute.]
[When subject signals] Good. Use your own vivid image now to allow your forearm to become more tingling, more numb and more insensitive. Get your own image vividly in mind and feel the tingling...the numbness, the insensitivity as vividly as you can.

As soon as you have the image vividly in mind please signal by lifting your left index finger.

[When subject signals] Good. What is your image? [Record this.]

Now, allow the numbness to go deeper, maybe ever deeper than ever before. You are in control and are doing well. More and more numbness...more and more insensitivity.

As soon as you have reached the deepest level of numbness that you can, please give me a signal by lifting your left index finger.

[After subject signals] Very good. Now please give me a rating of the numbness where 0 = no numbness and 6 = complete numbness. [Record this.]

In a moment we will begin the radiant heat trials. You may either keep your eyes closed or open them during the trials. Keep in mind that your right forearm will stay numb and insensitive throughout today's session, even when the experimenter moves it. The insensitivity will fade from your arm and your arm will feel completely normal again at the end of today's session.

Remember to keep a vivid image and allow the numbness and insensitivity to become deeper and deeper as you focus on the image. When you feel the heat intensities they may be only slightly warm if at all. Due to the numbness in your arm, your sensitivity to the heat intensities will be reduced.
You may move forward in your chair now, give me your arm under the curtain and we will begin the radiant heat trials. Ask for a rating (0 - 6). Record.

[NOTE: After the 20 practice trials and after trials 40, 80, and 120 instruct the subject as follows:]

Continue to use the vivid images which work best for you and which allow you to attain the deepest levels of numbness and insensitivity. Your arm will continue to feel numb and insensitive until the end of the experiment.

[NOTE: At the end of the experiment (after trial 150) say:] I would like you to focus intently on your forearm and the feelings of numbness and insensitivity which are there now. Please give a rating (0 - 6). Good. (Record) Now, please concentrate on your forearm and realize that as I count backward from 5 to 1, your arm will regain all feeling - the numbness will fade - and your right arm will be completely normal just like the other arm. 5 - less and less numbness, 4 - feeling returning, 3 - more and more feeling, 2 - almost completely normal, 1 - completely normal...no numbness. [If subject says arm is still numb, say "it will completely go away in 2-3 minutes, before you leave this room."]

Also at the end ask:

(1) What was your deepest level of analgesia during radiant heat trials? [Record this.]

(2) What was your most vivid cognitive image? [Record this and any other comments.]
APPENDIX H
POSTTREATMENT EXPECTANCY CONTROL GROUP INSTRUCTIONS

The information which you have received in your training sessions regarding different theories of pain, variables affecting pain response and coping strategies will enable you to experience decreased sensitivity to the radiant heat stimuli in today's session. Even the higher stimulus intensities may feel only slightly warm.

After the Radiant Heat Trials

Question: Did you use any cognitive/coping strategies during the radiant heat trials?

Question: Which one(s)?

Question: How would you rate the effectiveness of your strategies:

0 1 2 3 4 5 6
not at all effective very effective
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THE EFFECTS OF SUGGESTED ANALGESIA ON RADIANT HEAT PAIN AS A FUNCTION OF HYPNOTIC SUSCEPTIBILITY:

A SIGNAL DETECTION ANALYSIS

by

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(ABSTRACT)

The present study investigated the effects of suggested analgesia and level of hypnotic susceptibility (high vs. low) on acute radiant heat pain using a signal detection theory model. A signal detection paradigm was used in order to differentiate between sensitivity (sensory-discriminative) and response bias (motivational-affective) components of pain. Treatments consisted of: (1) a suggested analgesia group, and (2) an expectancy control group. Both groups had equal numbers of high and low scorers on a scale of hypnotic susceptibility.

Subjects were 32 male and female undergraduate volunteers assigned equally to each of the two treatment groups and counterbalanced for level of hypnotic susceptibility. Five levels of radiant heat (including zero) were presented. Each subject received 30 stimulus presentations per level and rated each stimulus on a scale from zero to six, with seven being a withdrawal. Self-report inventories of trait and state anxiety were also taken. Subjects participated in pre- and posttreatment sessions of radiant heat stimulation and were given three training sessions in the interim.

Results were that sensitivity measures showed a significant decrease for the suggested analgesia group for all but the highest
stimulus level paired comparison. There was also a decrease in sensitivity for the expectancy control group for the lowest stimulus pair.

Measures of response bias for a report of pain or higher changed nondifferentially across groups and levels, except for the highest stimulus pair, for which the experimental group showed a significant hesitancy to respond relative to the control group. There were no significant difference in terms of treatment effects for high vs. low hypnotic susceptibility.

Mean pain ratings decreased for both groups. Trait anxiety did not but state anxiety did decrease significantly after treatment.

Results were taken as supporting Gate Control Theory and a figure-ground realignment model of pain. Suggestions were made for directions in future research.