THE EFFECTS OF EXPERIMENTALLY-INDUCED
ANXIETY ON THE REPORT OF PAIN: A SIGNAL DETECTION ANALYSIS

by

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter I

INTRODUCTION

It has long been believed that experimentally-induced anxiety tends to exacerbate subjects' reports of pain. For example, one early study (Hall & Stride, 1954) found that the presence of the word "pain" in a set of instructions resulted in anxious subjects reporting as painful a level of electric shock that was not regarded as painful when the word was missing from the instructions. It was suggested that the anticipation of pain presumably heightened subjects' levels of anxiety and resulted in pain being reported. However, it seems equally plausible that the subjects given the instructions containing the word "pain" learned to label their responses as painful but did not actually experience more pain.

Similarly, it has long been thought that abating anxiety decreases the amount of pain reported. Another early study (Hill, Kornetsky, Flanary, & Wikler, 1952) found that the same level of electric shock or burning heat was reported to be significantly less painful when anxiety was presumably reduced by reassuring subjects that they had control over the aversive stimulus compared to conditions of no control. It was reasoned that control over an aversive stimulus reduces the anxiety associated with it. Unfortunately, no attempt was made to determine whether the effects were due to increased control or reduced anxiety or both. In fact, in neither of these studies was there any empirical demonstration that anxiety levels were actually altered.

These particular studies vividly illustrate two of the major shortcomings associated with research on pain and anxiety. First, little has been done to assess the effectiveness of manipulations intended to increase or decrease anxiety in the laboratory. Second, it has often been inferred that any change in the amount of pain reported <u>must</u> be due to a difference in the amount of pain experienced. The usual intepretation is that anxiety-invoking situations increase the perception of pain. An alternative explanation, one which has largely been overlooked until relatively recently, is that anxiety-producing situations alter the attitude of subjects such that they <u>report</u> more pain whether or not they actually <u>perceive</u> more pain. That is to say, subjects may exhibit a bias toward reporting more pain under certain conditions whether or not they actually feel more pain. Consequently, a subject who consistently rates one stimulus as more painful than another in an experiment does not necessarily experience a greater amount of pain even though he reports it.

Up until about a decade ago, pain researchers were without an effective solution to this dilemma. An experimenter could either use subject self-reports of pain, which may be confounded by elements of response bias, or he could turn to other more circumscribed methods such as physiological measurement. Physiological measures, however, have been shown to be unreliable and possibly even inappropriate in light of current pain theory (Hall, 1977).

In 1969, Clark introduced to pain research a technique called sensory-decision theory analysis, more commonly referred to as signal detection theory (SDT). Signal detection studies have been popular in the psychophysiological literature for many years, especially in the fields of visual and auditory perception (Swets, 1973). But it was Clark who first applied this statistical approach to the study of pain. SDT offers a potential solution to the problem encountered by previous researchers. It permits an independent assessment of both the sensory experience of pain and response bias as each contributes to the report of pain.

The purpose of the present study was to examine the effects of somatic and cognitive state anxiety on the report of pain using an SDT analysis. In order to facilitate an understanding of the current investigation, first it will be necessary to provide a theoretical framework for conceptualizing the vast amount of clinical evidence and laboratory data that exist on pain. The results of pain research involving experimentally-induced anxiety will then be presented. Typically, pain researchers have studied threshold and tolerance levels and drawn their conclusions on the basis of significant changes in these measures. Some of the problems associated with traditional methods of pain measurement have already been mentioned, but they deserve and will receive closer review. An explanation of SDT, how it overcomes the difficulties inherent in traditional pain research, and findings from experiments using SDT in the study of pain are provided as well. In addition, the need to distinguish between a unidimensional and multidimensional concept of anxiety is discussed as it pertains to the measurement of state anxiety.

Chapter II

PAIN THEORIES

Presently, there exist three major categories of pain theories: (1) specificity theories, (2) pattern theories, and (3) the more recently devised gate-control theory of Melzack and Wall (1965). Up until about a decade ago specificity theory was by far the most widely accepted explanation of the pain perception process (Sternbach, 1968). Melzack (1973) has made the claim that specificity theory was so deeply rooted in medical school teaching, until recently, that it was more often taught as fact rather than theory. Since that time, however, a revised version of Melzack and Wall's original gate-control theory has gained in prominence (Melzack & Casey, 1968). Each of the three primary theories of pain will be briefly presented, and the relative merits and defects of each will be noted. Before proceeding, it should be re-emphasized that the purpose of the following summary presentation is to provide a theoretical framework for conceptualizing the large body of literature on pain. To this end, no attempt has been made to produce a comprehensive, or by any means, fully evaluative review. The descriptions and criticisms offered are extracted largely from the work of three investigators--Hall (1977), Melzack

(1973), and Sternbach (1968). A more complete treatment of the topic is available from these sources.

There are a variety of specificity theories, but all share some of the same basic properties. For the sake of brevity and clarity, only those features that are most widely accepted by specificity theorists will be entertained. In general, specificity theory proposes the existence of a direct pain pathway that leads from receptors in the skin to one or more pain centers in the pain. Because of the one-to-one relationship between stimulus intensity and pain perception implied by specificity theory, there has been considerable research into the degree of specialization present in the nervous system. At the receptor level, free nerve endings that branch out into the upper layers of the skin appear to be particularly responsive to noxious stimuli. It has been suggested that each of the two peripheral fiber systems that lead from the free nerve endings indirectly subserve one of two distinctly different pain experiences.

The A- fiber system, as described by Hall, consists of large, myelinated fibers that enter the spinal cord and synapse in the posterior horn with the spinothalamic tract.[1]

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[1]Not all A- fibers are large in diameter (e.g., A- delta fibers), but Hall (1977) does not make this distinction. "A-" indicates that the fibers are myelinated.

The spinothalamic tract ascends to specific relay nuclei in the thalamus which project to the parietal cortex and other areas of the brain. It has been postulated that these nuclei control the neural areas which subserve the experience of sharp, well-localized pain.

C- fibers are smaller by contrast and are unmyelinated.[2] They enter the spinal cord and synapse on the substantia gelatinosa of the dorsal horn with the paleospinothalamic tract. This tract passes through the reticular system of the lower brain stem and terminates on those thalamic nuclei having poor cortical representation. Activity in the paleospinothalamic system may subserve the experience of dull, diffuse pain.

The preceding schematic account presents a grossly oversimplified view of the structural complexities involved in the pain system. This depiction is provided for illustrative purposes only and should not be taken as wholly accurate or complete.

The strength of specificity theory lies in the vast amount of evidence supporting physiological specialization of skin receptor cells. This proposition is so widely accepted, in fact, that it "has achieved the proportions of a genuine biological law" (Melzack, 1973, p. 132). Skin

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[2]"C-" indicates that the fibers are unmyelinated.

receptors do respond to particular stimulus dimensions. The drawbacks of specificity theory are that it predicts tissue damage will automatically and invariably result in pain and that noxious stimulation is a prerequisite for pain. Because specificity theory assumes a fixed, direct-line nervous system, there must be a one-to-one relationship between pain perception and stimulus intensity. This contention does not, however, appear to be supported by the evidence. As an argument against the validity of specificity theory, Melzack cites Beecher (1959) who reported that American soldiers injured in battle at Anzio beach-head denied pain from their extensive wounds or felt so little they refused medication, unlike a control group of civilians who had received similar physical insult. Presumably, psychological factors had modified the pain experience for the soldiers. Specificity theory makes no provisions for modulations in pain sensitivity, however.

Melzack also points to the wide body of clinical evidence pertaining to phantom limb pain, causalgia, and the neuralgias. Again, the data appear to refute the notion of specificity. For instance, surgical lesions of the peripheral and central nervous system have been unsuccessful in permanently abolishing these kinds of pain. Non-noxious stimuli, such as gentle touch or vibration, can trigger excruciating pain in these patients, and sometimes pain occurs for long periods of time without any identifiable stimulus. New pains or trigger zones for eliciting pain may spread to parts of the body that are devoid of pathology. Finally, the onset of pain from hyperalgesic skin areas may be delayed and continue long after the stimulus has been removed.

None of these occurrences can be adequately explained in terms of strict specificity theory, which posits a direct-line pathway between receptor and brain. Even though specificity theory assumes that internal sensitivity to pain is constant, it is arguable within the theory that the criterion for reporting pain is altered. The wounded soldiers in Beecher's study could have denied the existence of pain in spite of their predicament because, presumably, they were terrifically relieved to be out of battle and still alive. In addition, it is possible they might have felt that taking medication signified a lack of "manliness" and an inability to cope with the realities of war. But in the absence of strong environmental demand characteristics, it does seem unlikely that attitudinal differences could produce a change in response bias dramatic enough to account for the findings from studies involving causalgia, neuralgia, and phantom limb pain.

Pattern theories of pain reject the notion of a fixed, straight-through conceptual nervous system like the specificity theories. What is proposed instead depends, however, on the particular pattern theory under discussion. There are three general categories of pattern theories as described by Melzack: (1) peripheral pattern theory, (2) central summation theory, and (3) sensory interaction theory.

Peripheral pattern theory in its simplest form maintains that pain is a function of excessive peripheral stimulation that produces a pattern of nerve impulses. This pattern is interpreted centrally as pain. Central summation theory assumes that at some location in the pain system there exists a mechanism that produces patterning of neural activity. As noted by Melzack (1973) and Hall (1977), Livingston (1943) located the site of patterning in neuron pools of the spinal cord, while Hebb (1949) believed the process to take place in the thalamocortical neural circuits.

Sensory interaction theory, related to central summation theory, proposes that an input monitoring system normally impedes the occurrence of summation. If this system is destroyed, the consequence is pathological pain. One version of this theory (Noordenbos, 1959) as described by Melzack designates specialized functions for large, myelinated fibers and small, unmyelinated fibers. The small fibers are believed to carry impulse patterns that produce pain, while the large fibers are thought to inhibit transmission. Selective destruction of large fibers relative to small fibers results in increased neural transmission and therefore excessive pain.

Pattern theory is superior to specificity theory to the extent that it allows for the coding of nerve impulses. Although there remains considerable debate as to where this coding occurs, Melzack has proclaimed, "there can no longer be any doubt that temporal and spatial patterns of nerve impulses provide the basis of our sensory perceptions" (p. 150). Furthermore, the concepts of central summation and input control can account for many of the clinical conditions of pain left unexplained by specificity theory. For instance, Melzack noted that Livingston proposed the existence of self-sustaining reverbatory loops in the spinal cords of phantom limb patients. Minor irritation of the skin might then produce pain by feeding into these active loops and summating with them. As a result, volleys of impulses could be sent to the brain. In support of sensory interaction theory, Melzack has noted there is ample evidence that pathological pain is often associated with the loss of large, myelinated fibers.

The primary criticism of pattern theories is that they are too vague. The mechanism responsible for coding has yet to be identified. There is no specification of the kinds of patterns that might be related to pain. Nor is there any explanation as to how the code might be interpreted by central cells. Little mention, if any, is made of how psychological variables might modify the pain experience. It is not the case with pattern theory, as it is with specificity theory, that the effect of psychological factors is directly at odds with the underlying theoretical principles. It is just that pattern theorists, for the most part, have failed to incorporate psychological influences into their theories. It is concluded, then, that pattern theory has limited utility, and thus will be excluded from further discussion.

The gate-control theory of pain posits the existence of a neural mechanism that acts like a gate in the dorsal horn of the spinal cord. It is believed to affect the transmission of nerve impulses from peripheral fibers to the central nervous system (Figure 1). When the gate is open, summation of neural activity is facilitated; when the gate is closed, summation is inhibited. How much the gate is open or closed depends on the relative activity of the large myelinated (A-beta) fibers and the small, unmyelinated (A- delta and C-) fibers in addition to descending impulses from the brain.

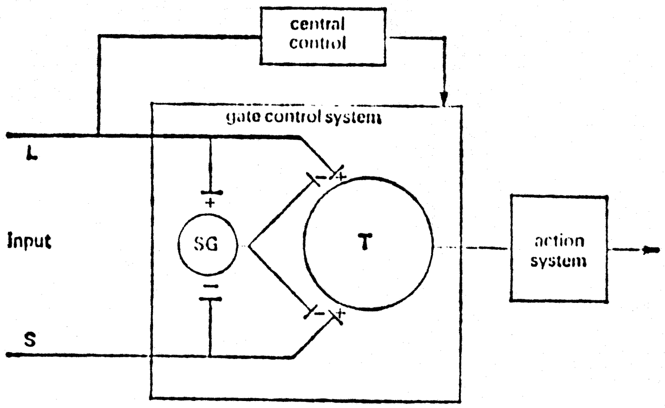

Figure 1. Schematic diagram of the gate-control theory of pain mechanisms: L, the large-diameter fibers; S, the small-diameter fibers. The fibers project to the substantia gelatinosa (SG) and first central transmission (T) cells. The inhibitory effect exerted by SG on the afferent fiber terminals is increased by activity in L fibers and decreased by activity in S fibers. The central control trigger is represented by a line running from the large fiber system to the central control mechanisms; these mechanisms, in turn, project back to the gate-control system. The T cells project to the entry cells of the action system. +, excitation; -, inhibition. (Reprinted from Melzack and Wall, 1965.)

Some of the large and small fibers project to the substantia gelatinosa that is thought to be the site of the spinal gating mechanism. It is believed that activity in these large-diamter fibers tends to inhibit transmission by closing the gate and blocking summation, while activity in the small-diameter fibers tends to facilitate summation by opening the gate. In addition, both kinds of fibers act directly on the spinal cord transmission (T) cells that project to the entry cells of those neural areas directly responsible for the experience of pain. When the level of output from the T cells exceeds a critical value, those neural areas are activiated. Therefore, any event or bodily condition that continuously and selectively exhibits an excitatory effect on small fibers, but not on large fibers, will tend to promote pain.

Because of the known influence variables such as past experience can have on the pain response (Beecher, 1959), Melzack and Wall (1965) have proposed the existence of a central control trigger. The function of the central control trigger is to activate certain brain processes (e.g., memories of prior experience) that can then exert immediate influence over the more slowly arriving sensory input. Via this mechanism, the brain can modify spinal cord transmissions. This, in part, accounts for the spontaneous effect that cognitive factors such as anxiety, anticipation, and attention have on pain processes. Melzack and Wall (1965) have suggested that the dorsal column--medial lemniscal and dorsolateral systems could perform the functions of the central control trigger. Other descending influences on the gate-control system include projections from the midbrain reticular areas and the cortex. Because of the growing accumulation of evidence suggesting that an area in the reticular formation brainstem is capable of having a powerful inhibitory influence on transmission at all levels of the somatosensory system, Melzack has postulated the existence of a central biasing mechanism there. A portion of the brainstem reticular formation is thought to exert a tonic inhibitory influence on transmissions at all synaptic levels of the somatic projection system.

Melzack and Casey (1968) have suggested that there are three major psychological dimensions of pain, each subserved by specialized physiological systems in the brain. The neospinothalamic fibers provide, in part at least, the neurological basis of the sensory-discriminative dimension. This component of pain is what might be termed the raw sensory experience, devoid of any meaning or affective quality. The paramedial ascending system projects to the processes influencing the motivational-affective dimension. This could be thought of as that urgent, unpleasant feeling that often accompanies pain. Both of these brain systems are supposedly activated when the output of the T cells exceeds a critical level (Figure 2). The third element of pain is the cognitive-evaluative dimension, which uses information like past experience to modify the contributions made by the other two dimensions. Neocortical and higher central nervous system processes are thought to underlie the cognitive-evaluative component of pain.

It is assumed that all three systems interact with one another to provide: (1) information about the perceptual characteristics of the noxious stimulus, (2) a strategy for coping with the pain in view of relevant cognitive variables, and (3) an incentive to respond based on the aversive quality of the stimulus. Furthermore, each of these systems is believed to influence the complex motor mechanisms that make up the pain response.

It can safely be said that in terms of the evidence supporting it, gate-control theory is by far the most comprehensive theory of pain yet developed. As noted by Sternbach and the authors of the theory, it takes into consideration spatial and temporal summation phenomena, sensory facilitation and central inhibition, and the data on physiological specialization. Hall has commended the theory for its abil-

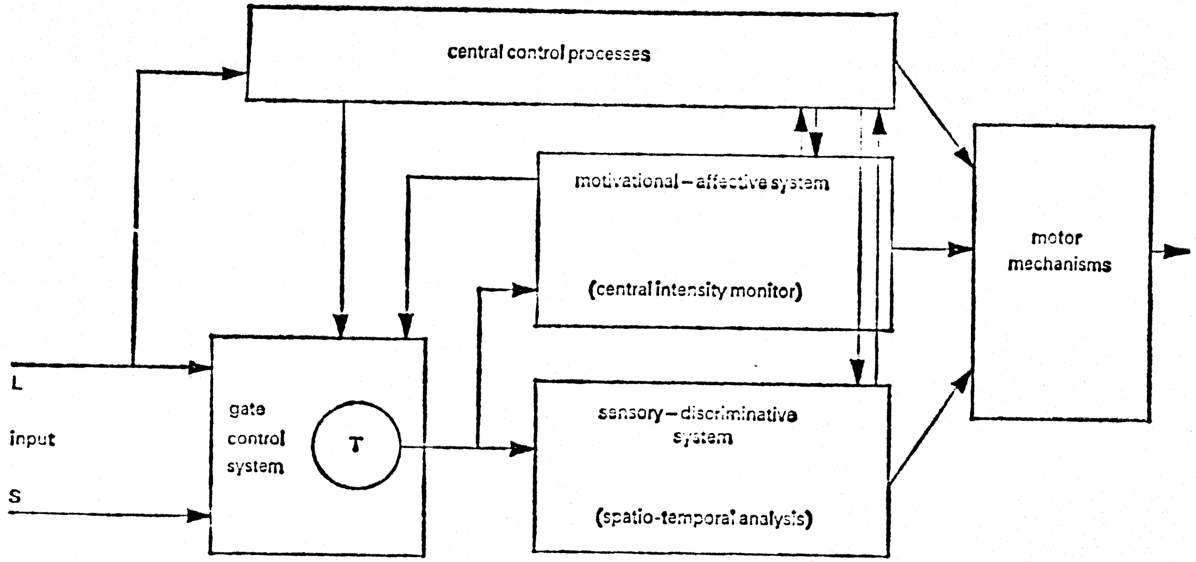

Figure 2. Conceptual model of the sensory, motivational and central control determinants of pain. The output of the T cells of the gate-control system projects to the sensory-discriminative system (via neospinothalamic fibers) and the motivational-affective system (via the paramedial ascending system). The central control trigger is represented by a line running from the large fiber system to central control processes; these, in turn, project back to the gate-control system, and to the sensory-discriminative system and motivational-affective system. All three systems interact with one another, and project to the motor system. (Reprinted from Melzack and Casey, 1968.)

ity to explain pain in the absence of adequate stimulation, a condition characteristic of many pathological pain problems. In addition, he has remarked favorably on the ability of the theory to account for the effect of cognitive variables via the spinal-gating mechanism or through the interaction of the various brain systems. Sternbach has praised the theory for the number of specific predictions that can be made and tested from it. For instance, the theory proposes that any activity or bodily condition that selectively recruits large fibers will reduce pain. Sternbach has cited a study by Wall and Sweet (1967) that tested this prediction with eight chronic pain patients, and the results appear to support it. Hall, on the other hand, has cautioned against readily touting the value of the theory. He has acknowledged that the theory has tremendous explanatory power, but he has been more reserved in his comments regarding the testability of predictions that are derived from it. He has argued that the number and complexity of the interactions possible between the various proposed processes make it difficult to test unequivocally many of the predictions that might be made.

Gate-control theory as applied to the present study has several implications. On the one hand, a difference in the report of pain brought on by anxiety could be due to an actual change in body sensitivity. If more pain is reported under conditions of anxiety, then it may be because there is more pain to report. Perhaps, in terms of the theory, anxiety results in an opening of the spinal gate, permitting rapid summation, and subsequently more pain. There are a variety of ways anxiety could conceivably exhibit this kind of effect, but it is beyond the scope of this investigation to try to specify the exact mechanisms involved. On the other hand, a difference in the amount of pain reported could be due to the effect anxiety has on one's attitude, or standard for reporting pain. This is to say, more pain could be reported because the criterion for reporting pain has become more liberal. Finally, there is the possibility that anxiety affects both components of the pain report, sensitivity and response bias.

Until the recent advent of SDT methods in pain research, there was no way to sort out this difference in experiments using subjects' self-reports. The few studies that have been done with induced anxiety have traditionally relied on pain tolerance and threshold measures. It was stated earlier that these approaches are inferior to SDT analysis. Before advancing this argument, it may prove worthwhile to examine some of these studies to see what consistent patterns exist.

Chapter III

PAIN THRESHOLD AND TOLERANCE MEASURES

There have been comparatively few experiments conducted in which the effects of induced anxiety on pain have been studied. The paucity of data in this area may be due to the methodological difficulties often associated with anxiety research. Or it may be because the work that has been done has yielded fairly consistent and, for the most part, unsurprising results.

On the other hand, there is an enormous amount of data on pain available from research investigating anxiety as a personality factor (e.g., Dougher, 1979; Hall & Stride, 1954; Hemphill, Hall, & Crookes, 1952; Hare, 1965; Merskey, 1965a and b; Schalling & Levander, 1964). The general conclusion to be drawn from this body of research is that anxious individuals tend to have a lower threshold and tolerance for pain than normals or psychiatric patients not regarded as pathologically anxious.

Based on the findings of Hill, Kornetsky, Flanary, and Wikler (1952) that control over a stimulus resulted in its being reported as less painful, Lepanto, Moroney, and Zenhausern (1965) examined threshold differences in the same subjects tested under conditions of both stimulus "control" and "no-control." In seven of nine cases, subjects exhibited significantly lower mean threshold levels in the "no-control" experimental treatment. It was concluded that the lack of stimulus control presumably resulted in an increase in anxiety that subsequently lowered the level at which pain was reported. Haslam (1966) has provided further evidence to suggest that anxiety lowers pain threshold levels. She found that threat of electric shock produced significantly lower mean threshold ratings compared to a "no-threat" control group. Lepanto, et al. and Haslam used radiant heat as the pain stimulus.

Bobey and Davidson (1970) attempted to induce anxiety in a group of female subjects by playing a tape recording of women in child labor. Contrary to expectations, they found that "anxious" subjects showed a greater tolerance for pain than a control group of female subjects that listened to a neutral tape on study habits. Furthermore, the tolerance levels for the "anxious" subjects were not any lower than a "cognitive rehearsal" treatment group. The highest tolerance scores were obtained by subjects in the "relaxation" group that received desensitization training. Bobey and Davidson suggested a number of possible explanations to account for their unexpected results. Perhaps the "anxiety" tape served as a cognitive rehearsal instrument signalling the forthcoming painful stimulus. Or maybe the "anxiety" tape functioned as a distraction, disguising the actual pain stimulus.

Indirect evidence on this issue comes from the work of Chapman and Feather (1973) who investigated the effects of reducing anxiety on the report of pain. In a series of three experiments (one of which will be discussed later), they found that subjects who were administered the tranquilizer diazepam showed a significantly greater tolerance for tourniquet pain and lower state anxiety ratings than subjects given a placebo. Diazepam was also shown to be more effective in extending tolerance time than aspirin, but no differences in state anxiety scores were observed between the two drug groups.

It appears, then, that experimentally-induced anxiety lowers pain threshold values. The effect of induced anxiety on tolerance levels is somewhat less certain given mixed results and the small amount of data relevant to this question. In light of the large body of evidence from personality research, it seems that anxiety lowers pain tolerance as well, but definitive conclusions await further testing.

Some of the problems encountered with the use of traditional threshold and tolerance measures will now be explained, and an argument for employing SDT will be forwarded.

Chapter IV

PROBLEMS WITH TRADITIONAL PAIN MEASURES

For many years, the technique of measuring pain consisted of little more than an evaluation of differences in threshold and tolerance levels. The pain threshold, by convention, is the stimulus intensity at which pain is reported 50 percent of the time. Pain tolerance is the length of time a noxious stimulus is endured. A number of criticisms have been directed at threshold and tolerance indices of pain.

Beecher (1959) has questioned the reliability of the pain threshold by pointing to a number of studies in which a large degree of threshold variability was obtained using radiant heat. Lloyd and Appel (1976) in their review of SDT and the psychophysics of pain "judged [the 50 percent threshold measure] to be unreliable, because it cannot account for at least one crucial determinant of behavior, response likelihood" (p. 79).

The most widely used argument by supporters of SDT is the fact that the threshold measurement of pain requires subjects to make decisions that are immensely susceptible to response bias. Simple threshold analysis is totally incapable of distinguishing between the actual pain sensation and the tendency to report (or not to report) pain. Barber

(1959) has noted that pain thresholds are easily affected by non-sensory variables (e.g., instructions), which may influence the subject's judgment or interpretation. Hall (1977) has cited evidence (Clark & Goodman, 1974; Wolff & Horland, 1967) to suggest that pain tolerance is just as vulnerable to the effect of judgmental variables as pain threshold.

For these reasons, many pain reseachers in the past decade have turned to SDT, which offers an independent assessment of pain sensitivity and response bias. To demonstrate this point, the rationale and principle properties of SDT will now be presented.

Chapter V

AN EXPLANATION OF SIGNAL DETECTION THEORY

In order to facilitate an understanding of SDT, a basic, non-mathematical description of the theory will be provided first. But because this view of SDT has numerous shortcomings in the context of pain research, a presentation of the methods intended for use in the current study will follow.

SDT is typically applied to research problems where subjects decide whether or not a particular stimulus has occurred within a specified time period. Ideally, the stimulus should be ambiguous enough so that the subject cannot always tell with complete certainty if the stimulus was actually presented. Experiments of this type usually require a large number of trials and stimulus presentations before stable response patterns begin to emerge (Green & Swets, 1966).

SDT assumes that in any stimulus detection task there exist two sensory distributions lying along the same continuum (Figure 3).

One is the noise distribution, which represents any ongoing activity that may interfere with detecting the stimulus. The source of this interference may be internal (e.g., random neural discharge) or external (e.g., low level sensory input). The other distribution represents the effects of

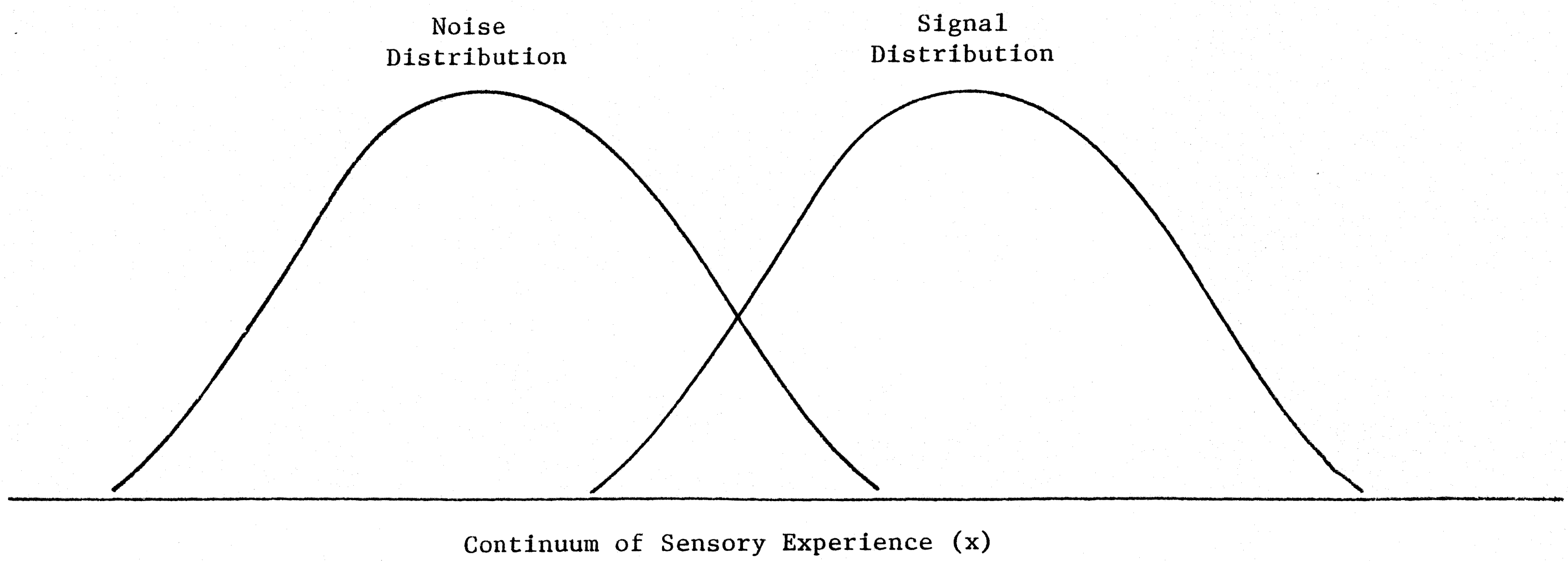

Figure 3. Hypothetical sensory distributions assumed in SDT.

noise plus the target stimulus, or signal, and is therefore termed the signal distribution. For the purpose of this explanation, it is further assumed that both distributions are Gaussian with unit variance ($s^2=1$).

Because subjects must make repeated judgments about the presence of confusing stimuli, signal detection theory is sometimes referred to as sensory decision theory. In any SDT experiment four decision outcomes are possible (Table 1). A subject could correctly identify the presentation of a stimulus, thereby scoring a "hit." Or he could correctly determine that no stimulus was presented (i.e., noise was presented), resulting in a "correct rejection." However, the subject could respond that a stimulus did occur when, in fact, it did not. This is called a "false alarm." Finally, a stimulus could be presented but it is not detected by the subject. This, of course, is a "miss."

By convention, only hit rates[3] and false alarm rates[4] are used in SDT calculations. Miss rates and correct rejection rates are their respective complements and could be used instead, if so desired. As complements, though, they pro-

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[3]The hit rate equals the number of times the stimulus presentation is correctly identified, divided by the total number of stimulus presentations.

[4]The false alarm rate equals the number of times the stimulus is said to have occurred when, in fact, it has not, divided by the total number of noise presentations.

Table 1. Four possible outcomes in any discrimination task.

Experimental Event	Subject's Response: Signal Present	Subject's Response: Noise Present
Signal	Hit	Miss
Noise	False Alarm	Correct Rejection

vide no additional information and need not be included in future discussion. By using the hit and false alarm rates, it is possible to determine both the subject's sensitivity and response bias.

Because the signal distribution represents the effects of a signal added to noise, subtracting out the contribution due to noise leads to a measure of the subject's sensitivity, called d' (Figure 4). By definition, d' equals the difference between the mean of the signal distribution, $\overline{X}_s$, and the mean of the noise distribution, $\overline{X}_n$, divided by the standard deviation of the noise distribution, s_n. The more confusing the stimulus is in terms of detectability, the smaller will be the difference between the means of the two distributions, and d' will take on a relatively low value. Similarly, the more apparent the stimulus the greater the difference will be, and d' will have a comparatively high value. When it is computed d' is usually measured in z-scores of the noise distribution, so that d'=[z(false alarm rate)-z(hit rate)]/s_n. In the case of Gaussian distributions with equal variance, this formula simplifies to d'=z(false alarm rate)-z(hit rate).

Response bias is represented by the point along the continuum where the subject has established his decision criterion. In Figure 4 any event that occurs which falls to

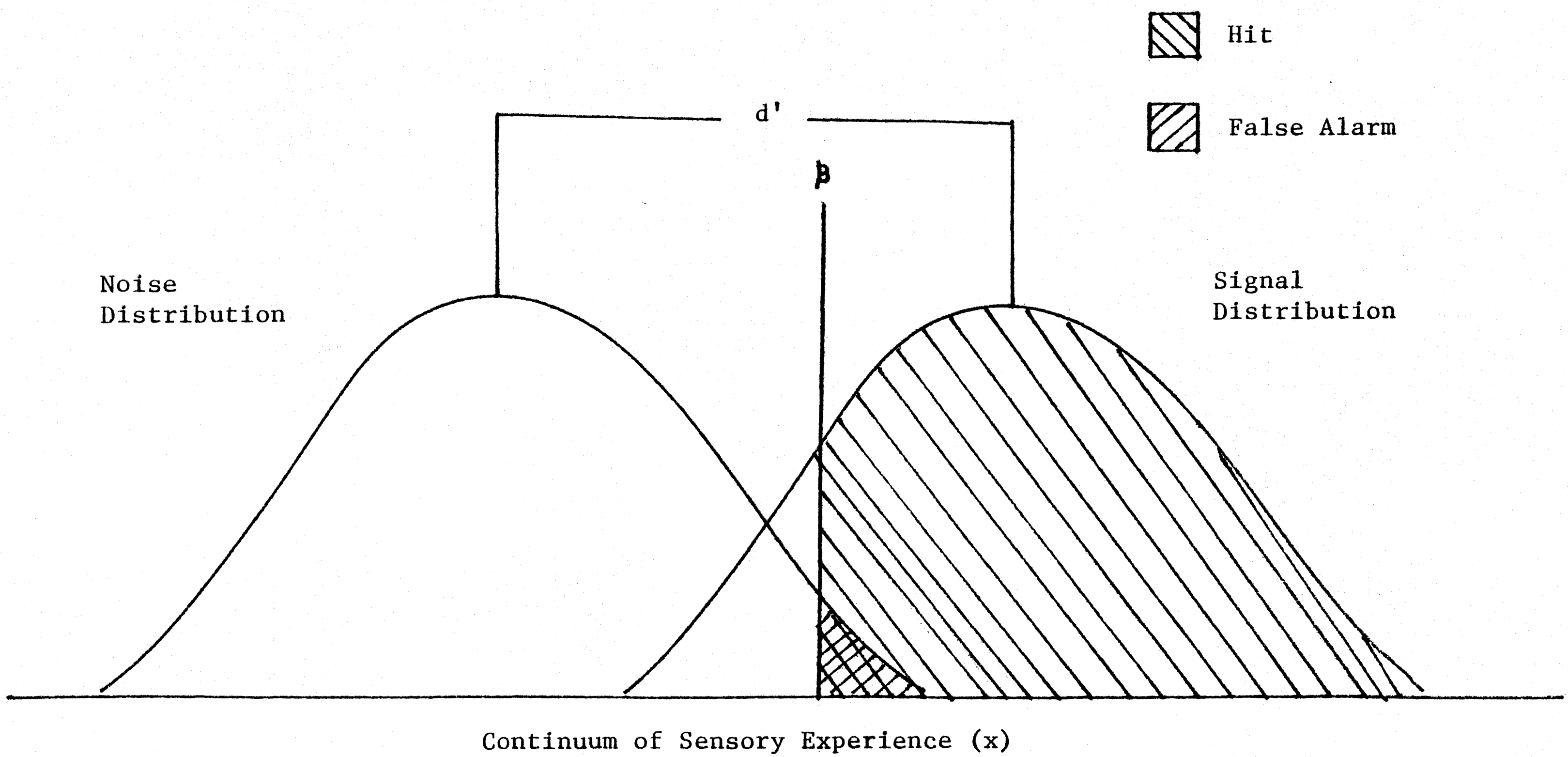


Figure 4. A graphical representation of sensitivity (d'), response bias (β), and hit and false alarm probabilities.

the right of B is judged to be a signal by the subject (hit or false alarm). Any event that falls to the left of β is deemed noise (correct rejection or miss). A β far to the right on the continuum represents a very conservative tendency to report a signal. Clark (1974) has offered as an example of this approach the stoic who will only report a noxious stimulus as painful if it is extremely intense. Conversely, a β far to the left is indicative of a liberal tendency to report the occurrence of a signal.

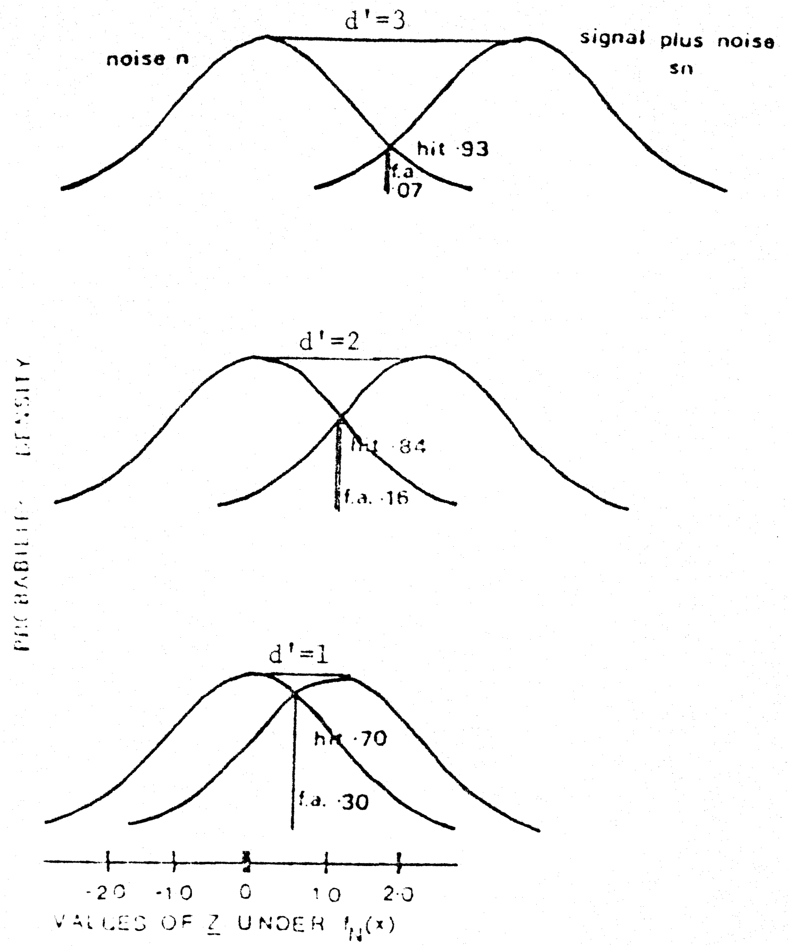
β equals the height of the signal distribution divided by the height of the noise distribution, given the associated hit and false alarm rates. As can be seen from Figure 4, the hit rate is that area to the right of the criterion which lies under the signal distribution. The false alarm rate is that area to the right of the criterion which lies under the noise distribution. When both distributions are Gaussian with equal variance, then ordinate values based on the hit and false alarm rates may be obtained from a normal curve table. β equals the signal ordinate divided by the noise ordinate.

Even though both sensitivity and response bias are calculated from hit and false alarm rates, Clark (1974) has demonstrated how the two measures can be independent. Figure 5 shows how d' can change while β remains constant.

Figure 6 depicts the opposite case where β moves and d' stays the same.

Up to this point, it has been assumed for the sake of illustration that both the noise and stimulus distributions are Gaussian with equal variance. In many instances, these assumptions are not justified and the consequences of violating them may be damaging to inferences drawn from the data. For example, unless a large number of noise and signal stimuli are presented d' is not a safe index of sensitivity (Green & Swets, 1966). In pain research, administering a large number of aversive stimuli may not be feasible. When the number of trials is prohibitive, a rating procedure is sometimes more desirable than the binary method described previously. Considerable experimental work has been done to compare the relative merits of the rating task with other psychological procedures (e.g., Egan, Schulman, & Greenberg, 1959; Pollack & Decker, 1958; Swets, Tanner, & Birdsall, 1961). The rating scale technique is really just a variation of the binary procedure in that adjacent <u>sets</u> of ratings are partitioned into noise and signal responses.

As it was presented, the binary decision method required the subject to make one of two choices: (1) yes, the stimulus occurred, or (2) no, it did not. For this reason, the binary decision procedure is sometimes called the "yes-no"



<u>Figure</u> <u>5</u>. The relation between hit and false alarm probabilities and sensory discriminability, d', when the likelihood ratio criterion remains constant at 1.0. d' is the distance between the means in terms of Z , the standard deviate. (Reprinted from Clark, 1974.)

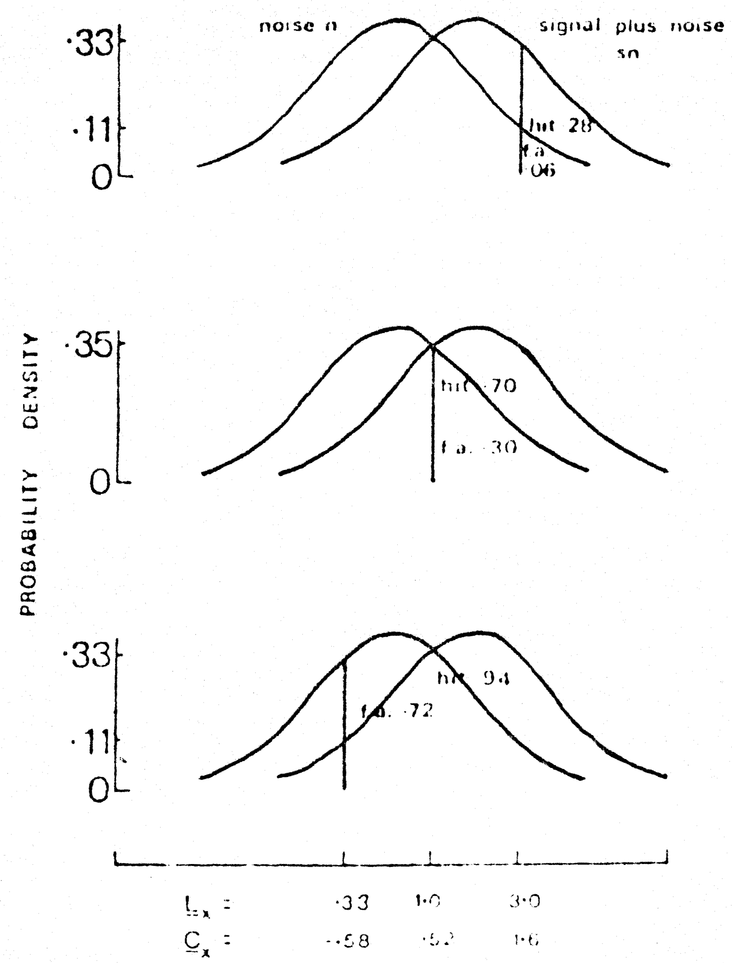


Figure 6. The relation between hit and false alarm probabilities and the response criterion, when discriminability remains constant at d'= 1.0. The decision axis may be interpreted logarithmically as the likelihood ratio criterion, L_x, or linearly as the sensory magnitude criterion, C_x. (Reprinted from Clark, 1974.)

task. Using a rating scale, the subject selects from a host of alternative responses. For instance, the subject might be asked to rate on a numeric scale how certain he is that a stimulus did or did not occur. Or the experimenter might ask the subject to rate the intensity of a number of different stimuli.

Suppose the subject is using a five point rating scale ("1" = low, "5" = high) to judge the intensity of a variety of randomly presented stimuli. When he adopts the strictest rating ("5"), he is saying, in effect, that all responses in that category are signals, and all responses in the other four categories are noise. Based on the intensity of the stimulus, it can be determined whether noise or a signal was presented. Hit and false alarm rates can then be calculated as described before. A second pair of hit and false alarm rates can be obtained using the next strictest rating category ("4"). The subject in this instance is saying, in effect, that all responses in this category ("4") or stricter categories ("5") are signals. Responses in the other three categories are considered to be noise. Logically, if an event is judged to be a signal when it is a "4," then any event which exceeds the criterion for being a "4" (i.e., an event rated a "5") will also be a signal.

Continuing in this fashion, three more pairs of hit and false alarm rates can be determined, making a total of five. Actually only the first four pairs contain any useful information, because the hit and false alarm rates for the fifth and final pair both equal 1.0. This final pair, viewed in the context of the "yes-no" task, is the same as <u>always</u> responding that a signal occurred irrespective of whether a signal or noise was, in fact, presented. The hit rate is perfect (i.e., 1.0), but so is the false alarm rate.

The number of meaningful response criteria is always equal to the number of rating categories minus one. In the example just described, four ßs could be computed on each subject. The number of d's for each subject is equal to the number of stimulus levels minus one, assuming that stimuli of zero intensity are also presented. (The zero intensity stimulus is more properly thought of as a noise presentation, but it still represents one of the stimulus levels.) Using pairs of hit and false alarm rates, d' and ß values can be calculated, yielding a set of sensitivity and response bias scores for each subject (Figure 7).

But as stated earlier, d' may not be a safe measure of sensitivity when the data do not support assumptions of normality and equal variance. A similar problem exists when ß is used as the measure of response bias (McNicol, 1972).

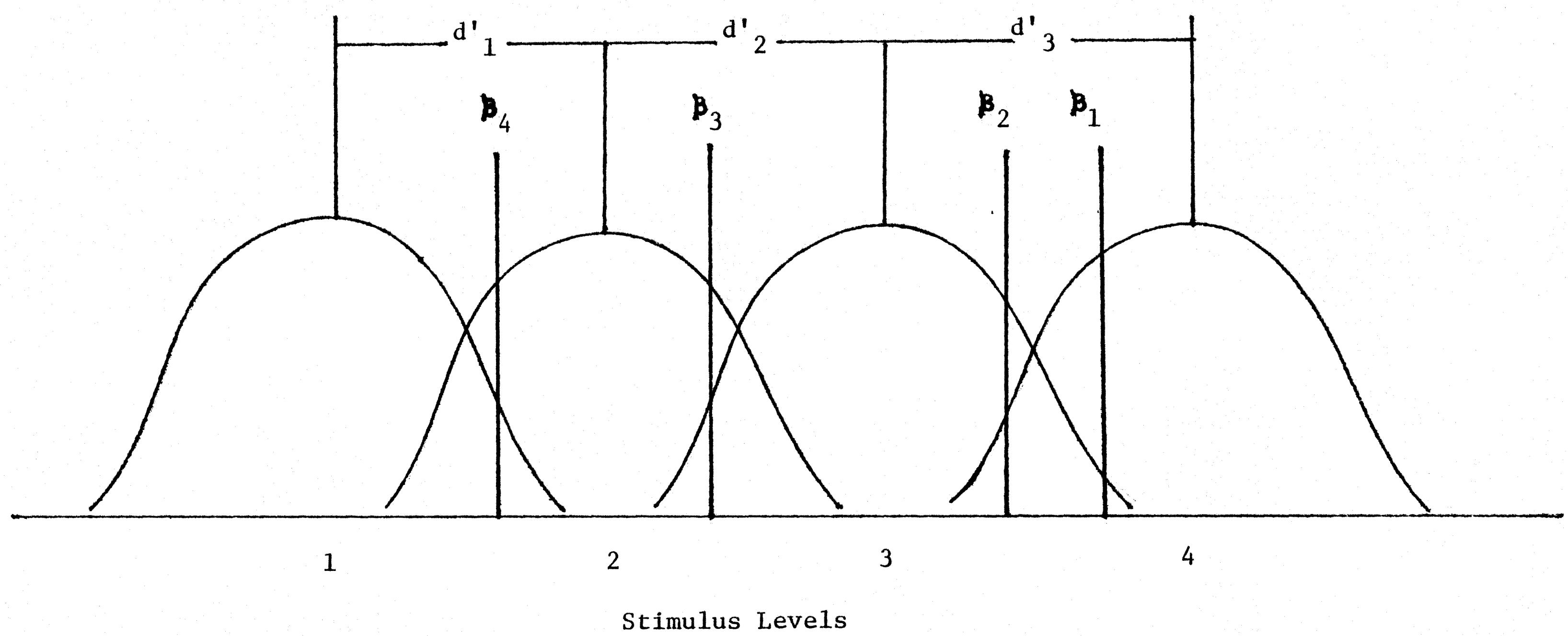


Figure 7. Hypothetical responses of a subject in a rating scale task using a 5-point scale and four stimulus levels. The first stimulus level is a zero intensity stimulus.

For this reason, nonparametric indices of sensitivity and response bias are sometimes preferred. These methods make no assumptions about the underlying statistical properties of the signal and noise distributions.

As has been shown, when a rating scale is employed a set of hit and false alarm probabilities is produced. By plotting on a graph the hit rates as a function of the false alarm rates, a Receiver Operating Characteristic (ROC) curve results (Figure 8). The area under the ROC curve is a nonparametric index of sensitivity called P(A) (Green & Swets, 1966).

To illustrate this point, refer to Figure 9a where the difference between the noise and signal distributions is represented by d'. Note that in Figure 9b, d' is larger and so is the hit rate, but the false alarm rate has not changed.

Refer back to Figure 8 and observe what happens when the hit rate increases but the false alarm rate remains constant (compare curve A with curve B). The increases in difference between the two means is reflected by an ROC curve having more area under it. It is important to note that the shape and variance of the two distributions have no effect on P(A).

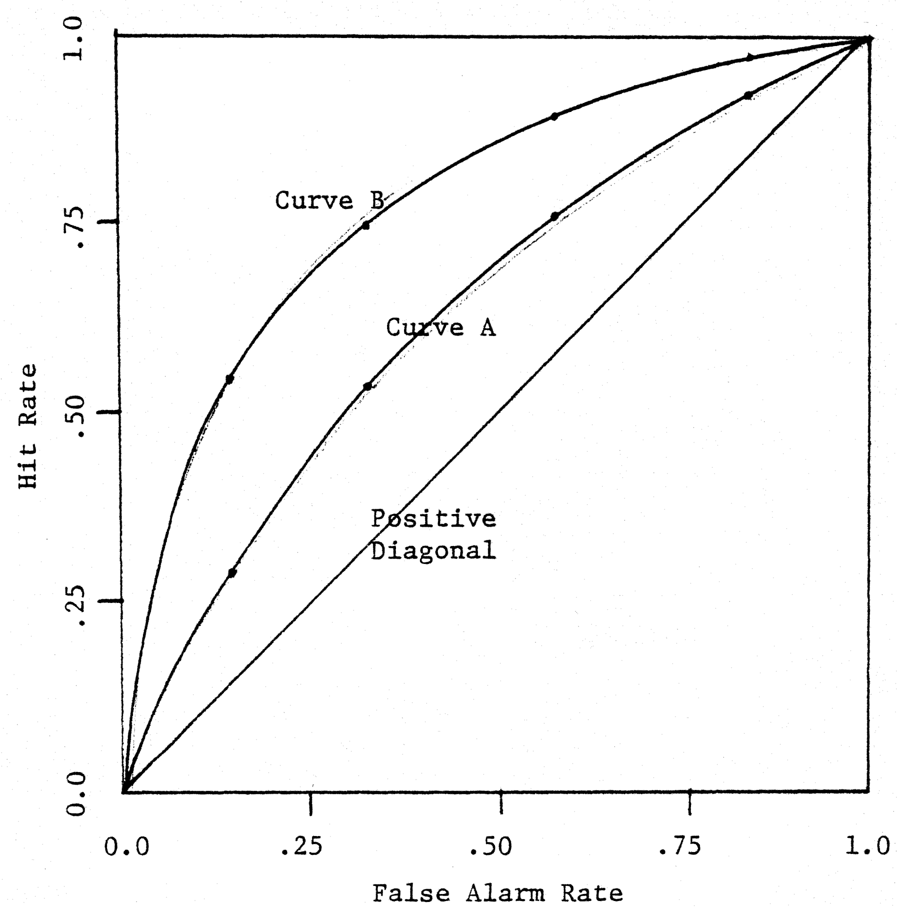
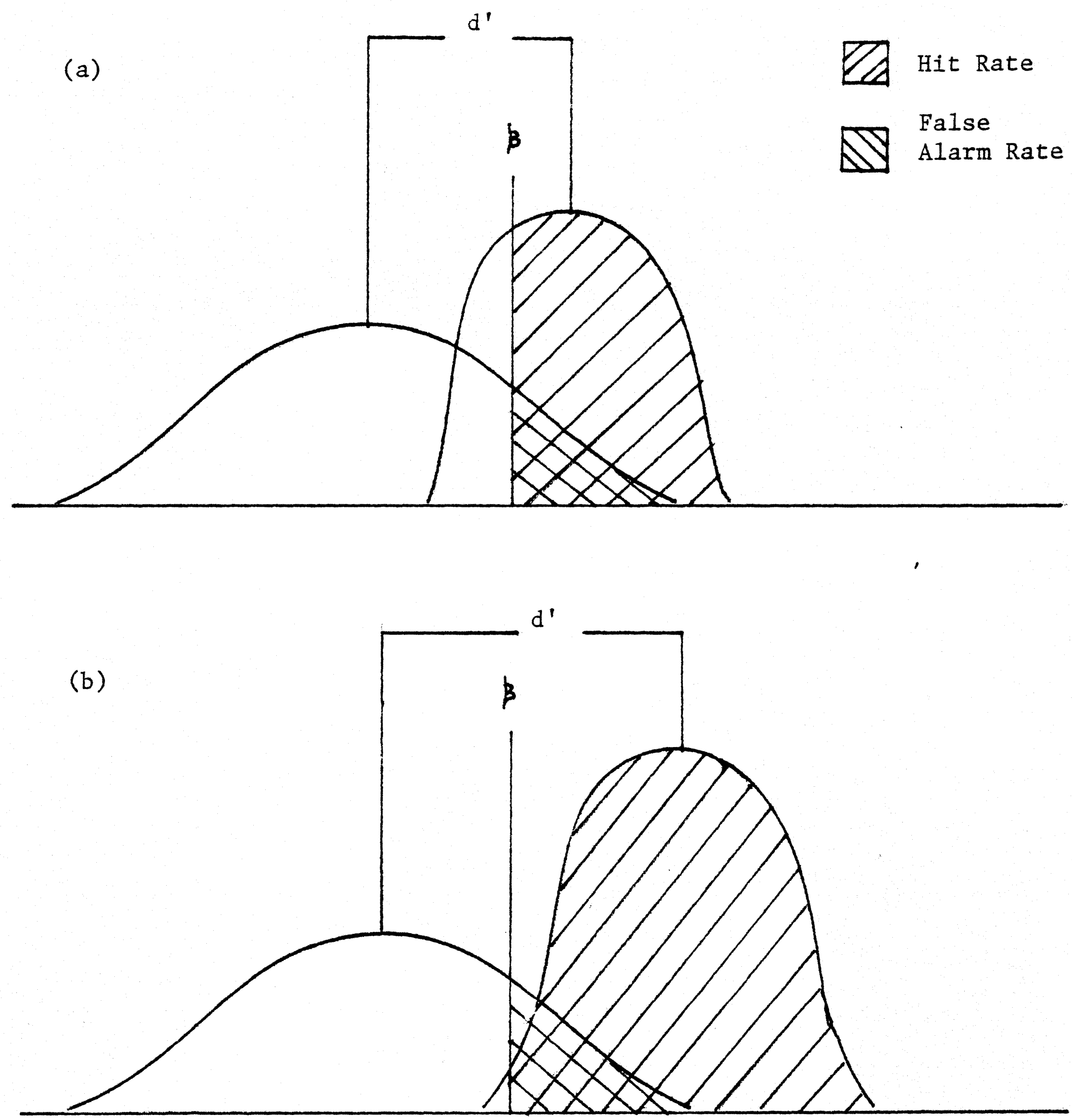


Figure 8. A Receiver Operating Characteristic (ROC) square showing two different sensitivity curves.



Continuum of Sensory Experience (x)

Figure 9. Sensitivity increases from (a) to (b), but there is no change in the response criterion. Note that the hit rate increases from (a) to (b), but the false alarm rate remains constant.

In the measurement of response bias, unequal variances between the noise and signal distributions complicate the relationship between β and the stimulus event, x. When the variances are equal, the relationship is monotonic-- as x increases, β increases; as x decreases, β decreases (Figure 4). When the variances are not equal, monotonicity no longer exists. In Figure 10, the variance of the signal distribution is less than the variance of the noise distribution. Note that the two distributions intersect twice and that $\beta = 1$ at both of these points. To the left of point A, the height of the noise distribution is greater than the height of the signal distribution, so $\beta < 1$. From point A to point B, the height of the signal distribution exceeds the height of the noise distribution, so $\beta > 1$. But to the right of point B, the height of the noise distribution is larger than the height of the signal distribution, and $\beta < 1$ once again.

At least two major problems arise when the relationship between x and β is not monotonic. First, it makes the detector's job more difficult. The simple decision rule to respond "signal" when x exceeds β no longer applies. To be aware of simple changes in x is not sufficient any more. The detector must amend his rule to take into account nonmonotonic changes (e.g., when x exceeds point B in Figure 10).

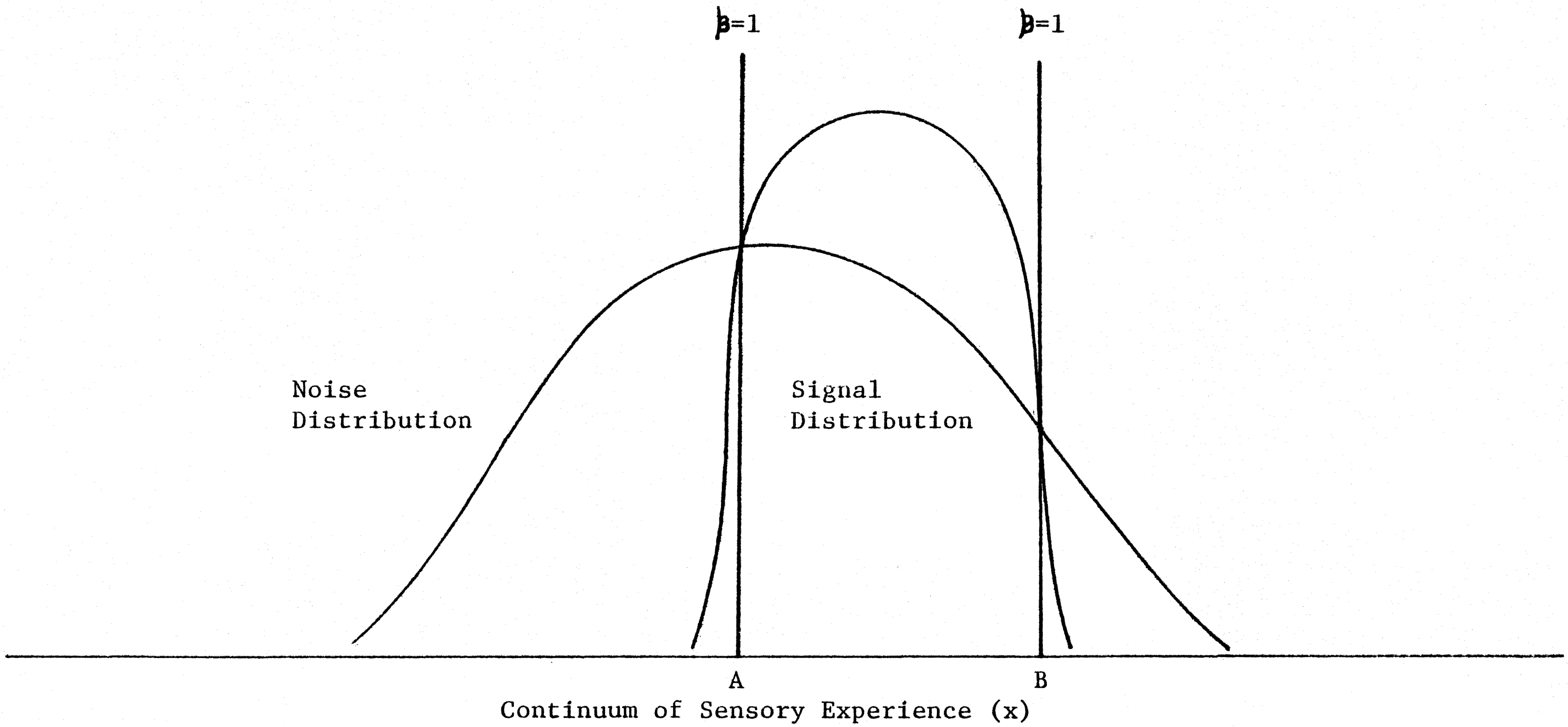


Figure 10. An example of unequal variance between the noise and signal distributions. At points A and B, the response likelihood ratio equals 1.0. To the left of point A, $\beta < 1.0$. From point A to point B, $\beta > 1.0$. To the right of point B, $\beta < 1.0$.

Whether, in fact, detectors are capable of operating with such sophistication is not yet clear (McNicol, 1972).

The second problem concerns the decision method that the detector chooses to employ. It has been assumed up to this point in the discussion that detectors base their decisions on the outcome of likelihood ratios. That is, detectors will be correct in their decisions the greatest percentage of time if they compare the heights of the two distributions at a given value of x and respond in favor of the larger distribution. However, when variances are equal, detectors are just as likely to be correct if they decide solely on the basis of the value of x. Because x and β are monotonic in the equal variance case, there is really no advantage to using one strategy over the other. Nor is there any guarantee which approach the detector will take. When the variances are unequal, however, the choice of method could make a great deal of difference theoretically. Using likelihood ratios to compute response bias would be misleading if the detector is really only concerned with the value of x.

Hodos (1970) has suggested a nonparametric measure of response bias which overcomes the difficulties associated with assumptions of normality and equal variance. Using a graphical estimation procedure, Hodos has developed formulas for computing percentage response bias. This method makes use of the fact that the negative diagonal of an ROC square represents zero bias and that points which deviate from it show varying degrees of bias (Figure 11). Points that lie in the area beneath the negative diagonal and above the positive diagonal demonstrate a tendency to report that no stimulus occurred (conservative approach). Points that lie in the area above the negative and positive diagonals indicate a tendency to report a stimulus did occur (liberal approach).

Grier (1971) has devised a functional expression for computing from data measures of response bias following Hodos' procedure: B'' =

$$\frac{y(1-y)-x(1-x)}{y(1-y)+x(1-x)}$$

where x equals the false alarm rate and y equals the hit rate. B'' values can range from -1 to +1, where negative numbers indicate a conservative approach and positive numbers signify a liberal approach.

Nonparametric measures of sensitivity, P(A), can be calculated by figuring out geometrically the area under the ROC curve (McNicol, 1972). Before tests of significance are performed the raw probabilities should be transformed to reduce the effects of possible skewness. The conventional procedure is to take 2 arcsin$\sqrt{P(A)}$ for statistical analysis, thus raising the upper limit of sensitivity to pi.

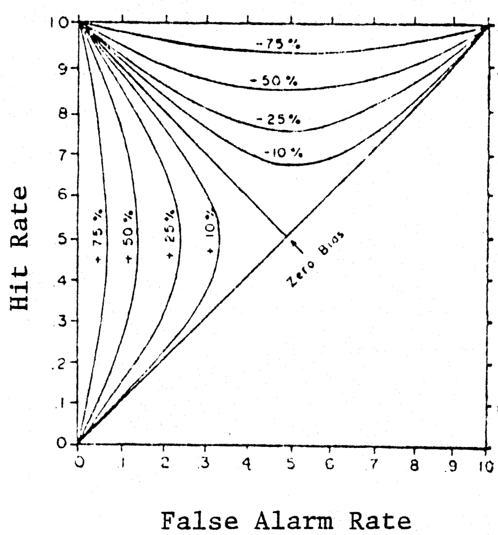


Figure 11. Two families of isobias contours. Each contour represents the locus of points having the same percentage bias. (Reprinted from Hodos, 1970.)

Now that the principles involved in SDT have been explained and the statistical groundwork has been laid, the findings from SDT research on pain can be presented and evaluated.

Chapter VI

PAIN RESEARCH USING SDT

The following review of the application of SDT to pain research is not intended to be exhaustive, but rather to be representative of the findings in this area. Changes in sensitivity and response bias have been the chief targets of interest in these investigations.

A number of studies have demonstrated decreases in sensitivity with accompanying alterations in response bias. Using radiant heat as the noxious stimulus, Chapman, Murphy, and Butler (1973) found that the administration of 33 percent nitrous oxide decreased an overall measure of sensitivity, but did not produce changes between adjacent stimuli. At the same time response criteria values significantly increased, demonstrating a reluctance on the part of subjects to report pain. Bloedel, Erickson, and McGreery (1974) found that stimulation at the dorsal column, anterior cord, or sciatic nerve resulted in decreased sensitivity and a concomitant increase in response criteria using radiant heat. Chapman, Gehrig, and Wilson (1975) found that both nitrous oxide and acupuncture attenuated sensitivity and increased the response criteria when electric stimulation was applied to the teeth. Chapman, Wilson, and Gehrig

(1976) using the same noxious stimulus demonstrated that transcutaneous stimulation also decreases sensitivity and raises the response criteria.

Often times changes in response bias are not associated with shifts in sensitivity. Using radiant heat, Clark (1969) and Feather, Chapman, and Fisher (1972) found that the administration of a placebo increased the response criteria, but did not change sensitivity. Clark (1974) and Clark and Goodman (1974) using radiant heat showed that a suggestion to tolerate more pain raised the response criteria but did not alter sensitivity. Clark and Goodman found, however, that a suggestion intended to decrease pain tolerance had no effect on either sensitivity or response bias. Bloedel, et al. found that transcutaneous stimulation increased the response criteria but had no significant effect on sensitivity when radiant heat was the painful stimulus. Investigating the analgesic properties of acupuncture, Clark and Yang (1974) arrived at similar findings.

Interestingly, the present author is unaware of any instance when a shift in mean sensitivity has not been accompanied by a concurrent change in response bias, suggesting that the two measures are not truly independent. Perhaps the tendency to report pain is functionally related to existing levels of sensitivity. Or more likely, interventions powerful enough to affect sensitivity are probably powerful enough to affect response bias.

Only two SDT studies have examined the effects of anxiety on the report of pain. Chapman and Feather (1973) found that diazepam (10mg) increased the length of time subjects would tolerate tourniquet pain compared to aspirin or a placebo. Furthermore, they found that diazepam reduced the anxiety associated with the most intense tourniquet pain in contrast to the placebo, but no more so than aspirin. The SDT analysis showed, however, that diazepam affected neither sensitivity nor response bias. Using focal pressure pain, Dougher (1979) found that both experimental instructions and the degree of subject trait-anxiousness influenced reports of pain. Telling subjects that a reluctance to report pain is a sign of emotional problems resulted in subjects reporting pain sooner. Telling subjects that a tendency to report pain too quickly is indicative of emotional problems resulted in subjects delaying reports of pain. In addition, high trait-anxious subjects reported pain sooner than low trait- anxious subjects. The SDT analysis revealed no differences in sensitivity, but the criteria for reporting pain were lower for high trait-anxious subjects than low trait-anxious subjects.

Unfortunately, few broad conclusions can be drawn from the data. It appears that, in general, treatments that act more or less directly on nervous system transmissions are necessary before shifts in sensitivity will occur. These manipulations are direct in the sense that they physically alter the electrochemical processes without requiring any cognitive mediation. It has been demonstrated that chemical analgesics such as nitrous oxide decrease overall measures of sensitivity. Electric stimulation has been shown to reduce sensitivity depending on the site and method of application. The ability of acupuncture to attenuate sensitivity has been reported, but findings are mixed. The effects of diazepam are especially puzzling. Diazepam has been shown to increase tolerance and to reduce the anxiety associated with pain, but it does not appear to modify either sensitivity or response bias.

More cognitively-oriented approaches using placebos or suggestion seem to have no effect on sensitivity (e.g., Clark, 1969; Feather, Chapman, & Fisher, 1972). They have been shown, however, to have a profound influence on response bias. Telling someone that he will feel less pain often results in his reporting less pain, even though the physiological basis for the pain has not changed.

Dougher's (1979) findings support the view that trait anxiety operates primarily on the emotional-attitudinal aspects of pain for individuals high or low in trait-anxiousness. However, recent studies reported in the anxiety literature have highlighted the importance of situational influences in the prediction of behavior. Increasing evidence suggests that situation variables interact with person variables in determining how individuals behave under anxious conditions (e.g., Breen, Prociuk, Endler, & Okada, 1978; Endler & Magnusson, 1977). Traditionally, trait anxiety has been characterized as a unidimensional construct. For example, individuals who were diagnosed as high in trait anxiety were thought to have a predisposition to respond anxiously in all situations. Recent research suggests, however, that a unidimensional concept of anxiety is inadequate. People respond differently in different situations. A measure of anxiety, therefore, must be multidimensional. This consideration will now be examined in order to provide the rationale for the choice of anxiety measure intended for use in the present study.

Chapter VII

A MULTIDIMENSIONAL APPROACH TO ANXIETY ASSESSMENT

Dougher (1979) in his study of the pain responses of high and low trait-anxious subjects used a forced choice version of the Taylor Manifest Anxiety Scale (Kabrick, 1954) in order to assess trait anxiety. Although this measure controls for the social desirability of responses, it evaluates primarily those aspects of anxiety due to ego-threatening, interpersonal situations (Endler & Okada, 1975). Because this approach is limited to only one possible source of anxiety it is termed unidimensional assessment. Recent data, however, support the superiority of tests that assess anxiety across a variety of situations.

Generally, situation-specific trait tests have been shown to be more accurate in predicting behavior under anxious conditions than broad trait measures (Zuckerman, 1979). Mellstrom, Zuckerman, and Cicala (1978) found that situation-specific trait measures of fear were equal to or better than global trait measures in predicting responses to a rat, a test, and social anxiety. Mellstrom, Cicala, and Zuckerman (1976) found that situation-specific trait measures of fear were better able to predict subject responses to a snake, a high place, and a dark room as compared to broad trait measures, but not as well as state measures.

The multidimensional approach to the assessment of anxiety can also include the measurement of qualitative differences in the manifestation of anxiety. Hamilton (1959) had psychiatrists interview and rate anxiety neurotics on 13 categories of anxiety symptoms. The correlations between ratings were subjected to a factor analysis that yielded a general factor of anxiety and a biopolar factor contrasting cognitive with somatic symptoms. Cognitive symptoms consisted of tension, fears, insomnia, anxious mood, cognitive changes, depression, and behavior at interview. The somatic items included gastro-intestinal, genito-urinary, respiratory, cardiovascular, general somatic, and autonomic symptoms. In another factor analytic study with psychiatric patients having varying diagnoses, Buss (1962) found two anxiety factors that he termed autonomic overreactivity and conditioned anxiety. Autonomic overreactivity consisted of observed and reported physiological concomitants, subjective feelings, somatic complaints, and distractibility. Conditioned anxiety was comprised of responses concerning restlessness, worry, and muscular tension.

Barrett (1972) performed an item analysis of anxiety items from a large battery of commonly used scales. He found two major subsets, awareness of somatic changes (e.g., blushing), and conscious awareness of unpleasant feelings about self or external stimuli (e.g., worrying).

Schwartz, Davidson, and Goleman (1978) constructed a dual component scale designed to assess cognitive and somatic trait anxiety separately. This scale was then administered to subjects who were already participating in either a physical exercise (somatic) or meditation (cognitive) program. As expected, subjects practicing physical exercise reported less somatic and more cognitive anxiety than meditators. As the authors have noted, the retrospective nature of the design restricts what firm conclusions can be drawn. Since the subjects were not randomly assigned to programs, it may be that individuals who are attracted to meditation versus physical exercise differ in their patterning of cognitive and somatic anxiety. Furthermore, the subjects may have differed in their expectations. Despite these methodological limitations, this study does appear to support the view that anxiety is not a global, undifferentiated internal state.

Schalling, Cronholm, and Asberg (1975) developed a self-report measure of somatic and cognitive trait anxiety and applied it to psychiatric patients. Unlike previous researchers, Schalling _et al_. did not find strong evidence for the existence of different anxiety factors. Schalling and her colleagues found a rather high degree of correlation (r=.81) between somatic and cognitive subscales. Schwartz *et al*. have noted, however, that the face validity of items assigned to the two subscales is questionable. The somatic anxiety scale consisted of items concerning autonomic disturbances, disquietable and mental discomfort of a diffuse kind, panic attacks, concentration difficulties, and distractibility. The cognitive anxiety scale included items related to worrying, anticipation, post-stress reactions, increased muscular tension, nervousness, and lack of self-confidence in social situations. Schwartz and his colleagues have attributed the high degree of correlation to the apparent item content overlap between the two subscales.

Since the main purpose of the proposed study is to examine the effects of induced anxiety on the report of pain, it seemed important to consider the nature of anxiety that is experimentally produced. Qualitatively different kinds of anxiety may not have identical effects on the report of pain. Accurately measuring the different components of anxiety poses an equally challenging problem.

Unfortunately, there does not exist at present a widely accepted self-report measure of somatic and cognitive state anxiety. Hamilton (1959) and Buss (1962) both used interviewers' ratings to assess state anxiety. Conventional measures of state anxiety such as Spielberger's State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970) do not explicitly delineate between somatic and cognitive aspects of anxiety. Instead, such measures typically yield a single score representing global anxiety. For these reasons, an additional purpose of the proposed investigation will be to try to develop an adequate way of objectively distinguishing between somatic and cognitive state anxiety.

At this point it is now possible to ennumerate the hypotheses under test in the current study. As these hypotheses are presented, empirical justification will be provided primarily from the research previously discussed.

Chapter VIII

HYPOTHESES UNDER TEST IN THE PRESENT STUDY

It has been shown repeatedly that anxious individuals tend to report pain sooner and to tolerate less pain than normals or psychiatric patients not regarded as pathologically anxious (e.g., Dougher, 1979; Hall & Stride, 1954; Hemphill, Hall, & Crookes, 1952; Hare, 1965; Merskey, 1965a and b; and Schalling & Levander, 1964). The results of laboratory investigations indicate that the induction of anxiety lowers pain threshold levels (Haslam, 1966; Hill, Kornetsky, Flanary, & Wikler, 1952; Lepanto, Moroney, & Zenhausern, 1965).

The effect induced-anxiety has on pain tolerance is less certain. The tranquilizer diazepam was found to decrease the anxiety associated with pain and to increase pain tolerance (Chapman & Feather, 1973). On the other hand, Bobey and Davidson (1970) found that tolerance levels were higher for female subjects who were presumably made anxious by listening to a tape of women in child labor compared to a control group of females who heard a neutral tape on study habits. It should be noted, however, that no attempt was made to measure the amount of anxiety actually induced by the experimental procedures.

In the only SDT study to date on pain and trait anxiety, Dougher (1979) found that threshold differences between high and low trait-anxious subjects were due to differences in the response criteria and not sensitivity. High trait-anxious individuals tended to set lower criteria for reporting pain and, thus, admitted to feeling pain sooner than low trait-anxious subjects. The effects of experimentally-induced state anxiety on the report of pain have not yet been investigated using SDT.

In general, researchers have not carefully distinguished between the different kinds of anxiety responses that have been produced in the laboratory. Until recently, anxiety was measured primarily in global terms and not much attention was paid to qualitative differences in the manifestation of anxiety. In an effort to induce anxiety, pain investigators have used a variety of methods including threat of electric shock (Haslam, 1966), lack of perceived control over the aversive stimulus (Hill _et al_., 1952; Lepanto _et al_., 1965), and audio tapes of women in child labor (Bobey _et al_., 1970). Outside the realm of pain research, a host of other techniques have been employed in order to produce anxiety, such as: films of surgery or serious accidents, skills testing, negative feedback on performance, and many forms of social pressure. Even though these methods are likely to be effective in producing what is generally referred to as anxiety, it is doubtful that they result in similar cognitive and somatic reactions.

Increasing evidence suggests that there are at least two distinctly different ways in which anxiety is exhibited (Schwartz *et al.*, 1978). Somatic anxiety is characterized by autonomic overreactivity and is distinguished from cognitive anxiety, which is chiefly associated with feelings of restlessness and worry. Considering the various experimental techniques for inducing anxiety, it seems reasonable to expect that viewing gory films would increase somatic anxiety more so than negative feedback on performance, which would probably increase cognitive anxiety. In examining the effects of anxiety on pain, it seemed worthwhile to distinguish between the two forms of anxiety in that they may differentially influence the report of pain.

In that somatic anxiety is associated with autonomic changes, it was hypothesized that somatic anxiety would be associated with lower levels of sensitivity. Conversely, it was hypothesized that cognitive anxiety would affect the interpretation of noxious stimulation and lower the criterion for reporting pain.

Based on the evidence presented here, it was hypothesized that: (1) subjects who are exposed to a condition designed to induce cognitive anxiety will set lower response criteria than those who are not, and that (2) subjects who receive an experimental treatment designed to increase somatic anxiety will demonstrate lower levels of pain sensitivity than those who do not receive this treatment.

Because of the difficulties inherent in any study involving anxiety induction, special steps should be taken to preserve the power of the design. Checking the effectiveness of the anxiety manipulations was considered to be of utmost importance. Therefore, specific attention was paid to the amount and quality of state anxiety reported by the subjects. For the manipulation to be considered effective, it was considered necessary to demonstrate that experimental subjects report more state anxiety than control subjects at various times throughout the course of the experiment.

Chapter IX

METHOD

Subjects

Subjects were 39 female undergraduate volunteers enrolled in an introductory psychology class. Three subjects were dropped from the experiment due to equipment failure, experimenter error, and subject withdrawal. Subjects were assigned randomly to one of three experimental conditions: (1) a somatic plus cognitive anxiety group, (2) a cognitive anxiety group, and (3) a no-anxiety control group. Each group contained 12 subjects. For their participation, subjects received extra course credit.

Apparatus

Pain was produced by a modified Hardy-Wolff-Goodell (1952) dolorimeter which emits radiant heat. A 150-watt floodlight was housed in the dolorimeter, so that radiant heat passed through a 1.0 cm aperture at one end. The dolorimeter was connected to a circuit board and timer which allowed stimuli to be delivered in a predetermined order and rate. During the experiment, radiant heat stimuli were presented semi-randomly 30 times at each of the following intensities: 0, 98, 155, 215, 256 $mcal \cdot sec^{-1} \cdot cm^{-2}$. In addition, 20 practice stimuli were presented at the outset, making a total of 170 stimulus presentations for each subject. Stimuli were presented semi-randomly in that the order was repeated after every 50 presentations. The output of the floodlight was calibrated daily at each of the stimulus intensities by means of a 450B Optical Power Meter.

The stimuli were applied to six 3.0 cm patches of India ink painted on the middle of the volar surface of each subject's right arm. Each stimulus was presented for 3 sec., starting at the patch closest to the wrist and moving toward the elbow. The subject's arm was placed against the aperture by an experimental assistant. The assistant removed his hold prior to the onset of the stimulus, so that the subject was free to withdraw from the dolorimeter. So that the subject's pain ratings would not be influenced by the amount of light emitted, a dark opaque curtain blocked the subject's view of the dolorimeter.

The interval between stimuli was 15 sec. to allow time for the stimulated patch to return to its initial temperature before being stimulated again.

Procedures

Upon entering the laboratory, subjects were informed that the experiment entailed the rating of heat stimuli, some of which could be painful. Written permission was obtained from those subjects who agreed to participate. (See Appendix A for a copy of the Consent Form.) Subjects were advised that they could withdraw from the experiment at any time without penalty.

Subjects were told that the experiment has two purposes. One is to examine the effects of visual stimulation on sensory discrimination, and the other is to see how performing certain tasks influences the way in which people rate different intensities of heat. Specifically, subjects were told that the experiment involves watching two short films and a session in between films where they would rate the intensity of various heat stimuli.

Subjects were then shown a short film (about 15 min. long). Subjects in the somatic plus cognitive anxiety group viewed a film on cattle slaughtering, while subjects in the other two groups saw a neutral film about the National Arboretum. After watching the film, subjects completed Endler's Present Affect Reactions Questionnaire (PARQ; See Appendix B) in order to obtain measures of somatic and cognitive anxiety.

In all, the PARQ was administered three times throughout the course of the experiment: (1) after the subjects viewed the film (Pre-test), (2) after the 100th heat stimulus trial (During-test), and (3) after the 170th heat presentation (Post-test).

After viewing the film, subjects in the somatic plus cognitive anxiety group were told that at the end of the rating task they would see a similar film (on eye surgery). The other two groups were told that they would see another neutral film (on soil erosion) at the end of the rating task. This was done in order to maintain the differences in anxiety expected to occur in response to the films. No additional film was actually shown to any of the subjects.

Following the film and the first administration of the PARQ, subjects rated the intensity of the heat stimuli, while they took an oral vocabulary test. The two anxiety groups were told that the test is easy and that college-bound, high school seniors typically get considerably more than half of the items correct. (See Appendix C for the anxiety groups' instructions.) The no-anxiety group was told that the test is difficult and that college seniors, on the average, get considerably less than half the items right. (See Appendix D for the control group's instructions.)

The 150 experimental stimuli presentations were preceded by 20 practice presentations in order to familiarize subjects with the stimuli, the rating scale, and the vocabulary test. In order to allow time to answer questions and to prevent fatigue, several 1-min. breaks occurred throughout the rating period: (1) after the 10th and 20th practice presentations and (2) after the 60th, 100th, and 140th presentations (counting practice).

After every 20 stimulus presentations including practice trails, subjects were provided with predetermined feedback with respect to their performance on the vocabulary test. Subjects in the two anxiety conditions were told that they answered about 20 percent of the items correctly, regardless of their actual performance. The subjects in the anxious groups were thus led to believe that they were performing worse than the average college-bound, high school senior. The control subjects were told that they answered approximately 90 percent of the items right. The subjects in the no-anxiety group were therefore led to believe that they were performing better than the average college senior. In actuality, both groups were given the same test, one that was developed using words similar to those found in the Graduate Record Examination. (See Appendix E for Vocabulary Test items.) One test question was presented with time to answer during each 15-sec. interval between stimuli. Figure 12 illustrates the sequence of events for all three groups.

Subjects rated the intensity of the radiant heat stimuli aloud using the following 7-point scale: 0=nothing, 1=something, 2=warm, 3=hot, 4=faint pain, 5=painful, 6=very

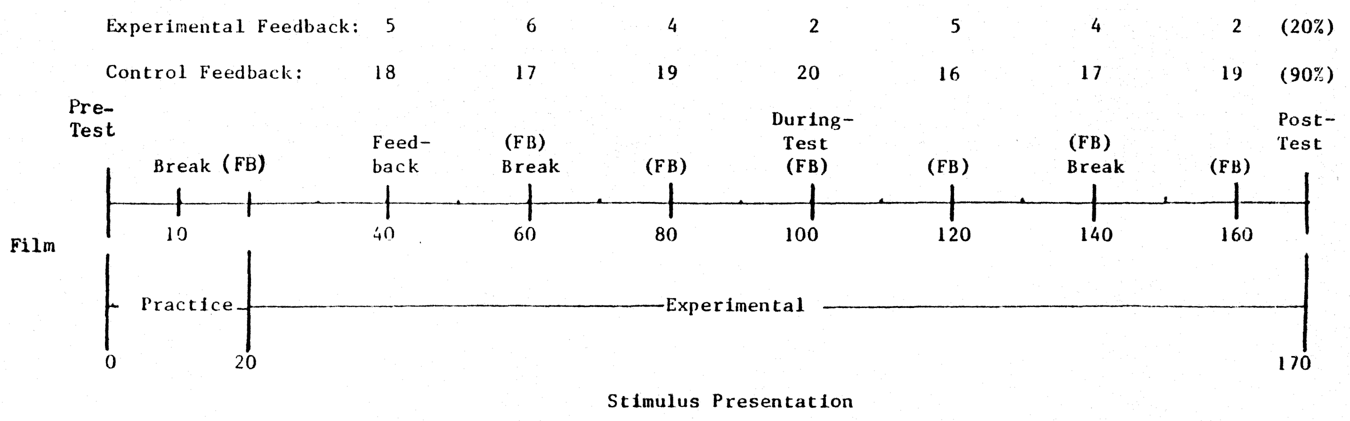


Figure 12. Schedule of experimental procedures. Feedback is the number of correct responses out of 20 on the vocabulary test. The experimental subjects are told that they have answered about 20 percent of the items correctly. The control subjects are told that they have answered approximately 90 percent of the items right. Test periods indicate times when the PARQ was administered. (FB = Feedback on word test.)

painful. Regardless of the rating assigned by the subjects, withdrawal from the dolorimeter was scored a "7" for the purpose of analysis.

Once the subjects completed the Post-test, they were informed that the experiment was over. Subjects were then debriefed and asked to keep the nature of the experiment confidential.

Chapter X

RESULTS

Manipulation Check

To check the effectiveness of the anxiety manipulation, a 3 X 3 repeated measures analysis of variance (ANOVA) was performed on the PARQ data. The results are presented in Table 2. There were significant main effects across experimental groups and across PARQ administration times. Their interaction was not significant.

Additional planned comparisons were carried out in order to examine more specific differences between groups. These data are presented in Table 3. As expected, subjects in each of the two anxiety conditions reported more overall anxiety than subjects in the control condition. Furthermore, these differences were apparent at about half way through the experiment and at the end. There were no significant differences, however, in the total amount of anxiety reported between subjects in the two anxiety groups. Nor were there any significant differences between subjects who saw the neutral film and those who viewed the anxiety film on anxiety measured immediately following the film presentation. See Figure 13.

Table 2

Summary of Analysis of Variance for the

Effects of the Anxiety Manipulation on PARQ Scores

Source	SS	df	MS	*F*
Exp. Group	9.61	2	4.81	5.66*
S(Exp. Group)	28.01	33	0.85	
Time	7.34	2	3.67	27.06**
Time X Exp. Group	0.63	4	0.16	0.34
Time X S(Exp. Group)	8.95	66	0.14	

*$p < .01$

**$p < .0001$

Table 3

Planned Comparisons of PARQ Means

Group Comparison[a]	*t*	Group With Greater Anxiety	As Predicted?
Grp. 1 with Grp. 2	2.77*	Grp. 2	yes
Grp. 1 with Grp. 3	2.95*	Grp. 3	yes
Grp. 2 with Grp. 3	0.16	------	---
Grps. 1-2 with Grp. 3 at Time 1[b]	1.16	------	no
Grp. 1 with Grp. 2 at Time 1	1.44	------	yes
Grp. 1 with Grp. 2 at Time 2[c]	3.05*	Grp. 2	yes
Grp. 1 with Grp. 3 at Time 2	2.85*	Grp. 3	yes
Grp. 1 with Grp. 2 at Time 3[d]	2.93*	Grp. 2	yes
Grp. 1 with Grp. 3 at Time 3	3.09*	Grp. 3	yes

[a]Subjects in Group 1 viewed a "neutral" film (on flowers) and received positive feedback concerning their performance on a word test. Subjects in Group 2 saw the "neutral" film and received negative feedback on the word test. Subjects in Group 3 saw an "anxiety" film (on cattle slaughtering) and received negative feedback on the word test.

[b]Time 1 occurred immediately following the film presentation and just prior to exposure to the pain stimuli.

[c]Time 2 occurred following presentation of the 80th pain stimulus.

[d]Time 3 occurred at the conclusion of the word test and immediately following the final (150th) pain stimulus presentation.

*$p < .01$

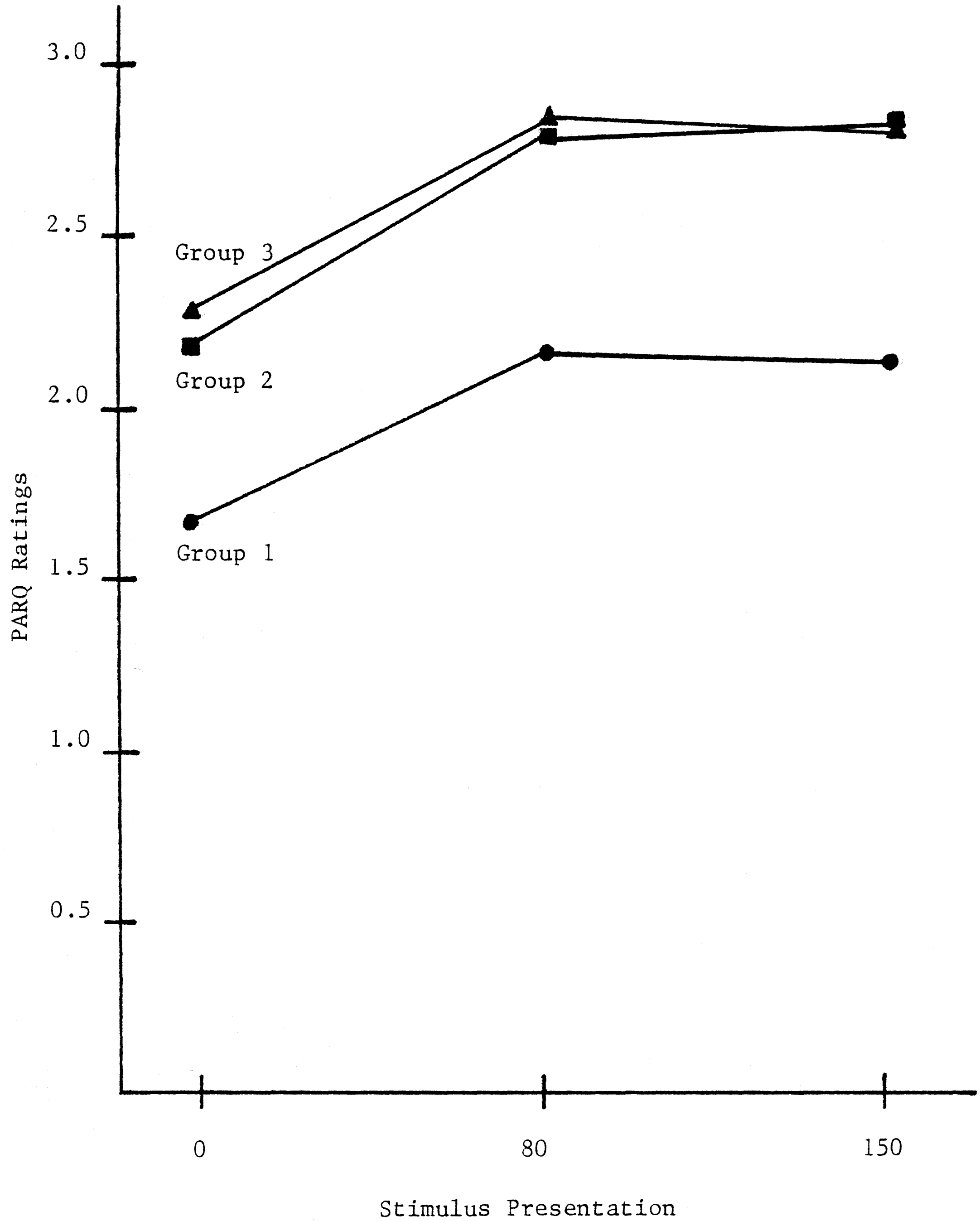


Figure 13. Mean PARQ ratings for the three experimental groups.

Taken together these results demonstrate the effectiveness of the experimental manipulation in producing and maintaining anxiety. However, it appears that the anxiety film produced no more anxiety than the neutral film and that the differences in anxiety between groups can primarily be attributed to the nature of feedback on the word test. The actual scores of subjects on the vocabulary test appear in Table 4.

Because there were no differences between groups in the amount of anxiety reported following the film presentations, it may be inferred that these different experimental conditions had essentially the same overall effect on anxiety responses. Therefore, it was considered possible to submit the PARQ ratings of all subjects for the first administration period to a factor analysis without doing serious harm to the underlying assumption of independence between questionnaire items and experimental treatment effects.

In order to examine the possible existence of cognitive and somatic components of anxiety, a principal axis factor analysis was performed and subjected to a varimax rotation. The results of the factor analysis are presented in Table 5. Two factors were specified. Sixteen of the 24 PARQ items loaded highly ($r > .40$) on one factor and seven loaded highly on the other. Two of these items loaded highly on

Table 4

Mean Number of Correct Responses

to Vocabulary Test Items

	Group					
	1		2		3	
Vocabulary Items	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
1 - 80	36.92	12.18	34.33	9.50	42.33	11.99
81- 150	35.83	11.30	32.58	8.27	41.08	11.67
All	72.75	22.98	66.92	17.21	83.42	23.15

Table 5

Factor Analysis of PARQ Scores

PARQ Item	Factor I[a]	Factor II[b]
Hands feel moist	.103	.744*
Feel relaxed	.854*	.129
Hands feel unsteady	.393	.461*
Feel self-confident	.735*	.277
Stomach feels tense	.632*	.149
Enjoy this situation	.505*	.351
Heart beats faster	.674*	.307
Feel calm	.833*	.166
Perspire	.317	.721*
Feel comfortable	.602*	.367
Mouth feels dry	.395	-.058
Unable to focus thoughts	.227	.285
Feel pleasant	.528*	.351
Feel nervous	.756*	.160
Feel throbbing in head	-.427	.473
Feel secure	.747*	.108
Feel upset	.468*	.379
Hands feel cold	.018	.365
Feel good	.652	.462
Feel anxious	.705*	.032
Breathing is irregular	.484*	.334
Feel uneasy	.740*	.246
Want to avoid this situation	.318	.492*
Feel lump in throat	.038	.664*

Note. A principal axis factor analysis was performed using a varimax roation. Two factors were specified.

[a]Cognitive anxiety

[b]Somatic anxiety

*$|r| \geq .40$ on this factor only

both factors and three additional items did not load on either factor. Neither factor is purely characterized by items thought to represent cognitive or somatic anxiety, but there is some slight similarity.

Of the 14 items predicted to load highly on just the cognitive factor, 11 did in addition to three items that were not expected to. However, of the ten items that were predicted to load just on the somatic factor, only four did, along with one additional item that was not expected to. Given the lack of association between prediction and outcome, the small number of observations upon which the factor analysis was based (N= 36), and the possible confounding effects of treatment, it was not considered appropriate to use these two factors to examine the effects of cognitive and somatic anxiety on the report of pain.

<u>Analysis of Pain Data</u>

Sensitivity values were calculated for each subject using the nonparametric measure, P(A), described by McNicol (1972). This method produces a single sensitivity value for adjacent stimulus intensity levels. Total sensitivity for each subject was determined by averaging sensitivity values across stimulus levels.

Response bias values were calculated for each subject by determining the cumulated conditional probabilities of the stimulus responses (Clark, 1974). Adjacent stimulus level probabilities associated with the lowest pain rating response (Faint Pain) were compared, with the one associated with the higher stimulus level being treated as the "hit rate" probability and the other as the "false alarm" rate. Each of these pairs of hit and false alarm rates was then used in a formula devised by Grier (1971) for calculating a nonparametric measure of response bias, B''. The total measure of response bias for each subject was computed by averaging response bias values across stimulus intensity levels.

Mean pain ratings were calculated by averaging each subject's ratings to all 150 radiant heat stimuli. A summary of these calculations is presented in Table 6. In addition, mean stimulus ratings at each of the stimulus intensity levels were computed. The results are presented in Table 7.

To determine the effects of the manipulation on the report of pain two multivariate analyses of variance (MANOVAs) were performed, one on individual sensitivity and response bias values and one on the averages. Neither was significant.

A similar pair of MANOVAs was conducted after dividing subjects into extreme groups of High and Low Anxiety. The ten subjects scoring highest across all three administrations of the PARQ were assigned to the "High Anxiety" group.

Table 6

Mean Pain Measures by Experimental Groups

	Experimental Group		
Pain Measure	1	2	3
Sensitivity	2.20	2.12	2.09
Response Bias	.132	.166	.223
Stimulus Rating	3.48	3.07	3.01

Table 7

Mean Stimulus Ratings

Stimulus Intensity (in mcal·sec^{-1}·cm^{-2})	Group 1		Group 2		Group 3	
	$\bar{x}$	SD	$\bar{x}$	SD	$\bar{x}$	SD
256	5.87	0.71	5.18	1.28	5.03	1.07
215	4.98	0.94	4.50	1.30	4.46	1.08
155	3.70	0.61	3.36	0.91	3.13	0.87
98	2.30	0.71	1.84	0.61	1.87	0.59
0	0.53	0.49	0.45	0.38	0.54	0.44

The remaining 26 subjects were assigned to the "Low Anxiety" group.

In order to determine whether the division into High and Low groups actually produced groups that differed on these measures, a t-test was conducted on mean anxiety scores. The results are presented in Table 8. The means of the High and Low groups were significantly different, suggesting that the high and low distinction is at least statistically meaningful.

The two MANOVAs examining the effects of High and Low Anxiety were both significant. The results are presented in Table 9.

The subsequent ANOVAs revealed significant differences in total sensitivity, response bias and mean stimulus ratings between the High and Low Anxiety groups. Overall, the High Anxiety group reported lower stimulus ratings and exhibited lower sensitivity and higher response criteria. The differences in sensitivity were consistent with that which had been expected, but the differences in response bias and mean stimulus ratings were not. On the average, subjects in the Low Anxiety group reported significantly greater amounts of heat stimulation than subjects in the High Anxiety group. The means are presented in Table 10.

Table 8

Mean Anxiety Scores

for Subjects in High and Low Anxiety Groups

Group	n	t	Mean
Total Anxiety		3.70*	
High	10		222.50
Low	26		151.08

*p $<$ .05

Table 9

Multivariate Analyses of Variance for the Effects of Total Anxiety on the Report of Pain

Individual Pain Measures[a]

Anxiety	Sensitivity								Response Bias							
	1		2		3		4		1		2		3		4	
	F^c	p	F	p	F	p	F	p	F	p	F	p	F	p	F	p
Total Anxiety	0.53	.473	3.46	.071	5.93	.020	7.03	.012	0.52	.477	9.02	.005	0.00	.988	0.12	.728

Average Pain Measures[b]

Anxiety	Sensitivity		Response Bias		Stimulus Ratings	
	F^c	p	F	p	F	p
Total Anxiety	9.06	.005	5.17	.029	4.62	.039

[a]Multivariate $F = 2.46$, $p < .038$

[b]Multivariate $F = 3.46$, $p < .028$

[c]F = Univariate F

Comparisons between High and Low Anxiety groups on the individual pain measures revealed that subjects in the High Anxiety group showed significantly lower sensitivity for the two pairs of lowest stimulus intensity. In addition, they also showed significantly higher response criteria at the second highest pair of stimulus levels. No other comparisons between the High and Low Anxiety groups were significant.

A Pearson Product-Moment Correlation was performed in order to assess the possible linear relationship between anxiety and the pain measures. The correlation between the PARQ scores and the average measures of sensitivity and response bias were -.26 and .16 respectively. The correlation between the PARQ scores and the mean stimulus ratings was -.08. None of these correlations was significant.

In order to attain a more balanced distribution of subjects into groups, subjects were divided into equal thirds on the basis of their PARQ scores. As before, a pair of MANOVAs was performed to examine the effects of anxiety on individual and average pain measures. Neither was significant.

In order to assess the possibility of a curvilinear relationship between the mean pain measures and anxiety, eta squared values were calculated. None was significant.

Table 10

Sensitivity Response Bias, and Stimulus Rating

Means for High and Low Total Anxiety Groups

Individual Pain Measures

Total Anxiety	Sensitivity				Response Bias			
	1	2	3	4	1	2	3	4
High[a]	1.92	1.97	2.05	2.24	-0.15	0.11	0.43	0.64
Low[b]	1.88	2.14	2.21*	2.48*	-0.24	-0.34**	0.43	0.71

Average Pain Measures

Total	Sensitivity	Response Bias	Stimulus Ratings
High	2.04	0.26	2.80
Low	2.17**	0.14*	3.33*

Note. Comparisons were made between means in the same column.

[a] $n = 10$

[b] $n = 26$

*$p < .05$

**$p < .01$

The 150 stimulus ratings for each subject were divided into thirds according to order of presentation, and mean ratings for each third were computed. The results are illustrated in Figure 14. In order to assess the effects of the anxiety manipulation over time a 3 X 3 ANCVA was performed on the mean stimulus ratings. The results are presented in Table 11. There was a significant main effect for time and a significant manipulation X time interaction. There was no significant main effect for the manipulation.

Duncan's Multiple Range Test was performed to assess overall differences between groups and times. The results are presented in Table 12.

There were no overall differences between any of the experimental groups, but mean stimulus ratings from each of the three time periods all differed significantly from one another with ratings increasing over time.

Newman-Keuls Test was performed in order to examine the specific effects of the interaction. The results are presented in Table 13.

There were no significant differences in mean stimulus ratings over time for subjects in Group 1, the no-anxiety condition. This suggests that stimulus ratings did not change merely as a function of time in the experiment. For subjects in Group 2, mean stimulus ratings increased signi-

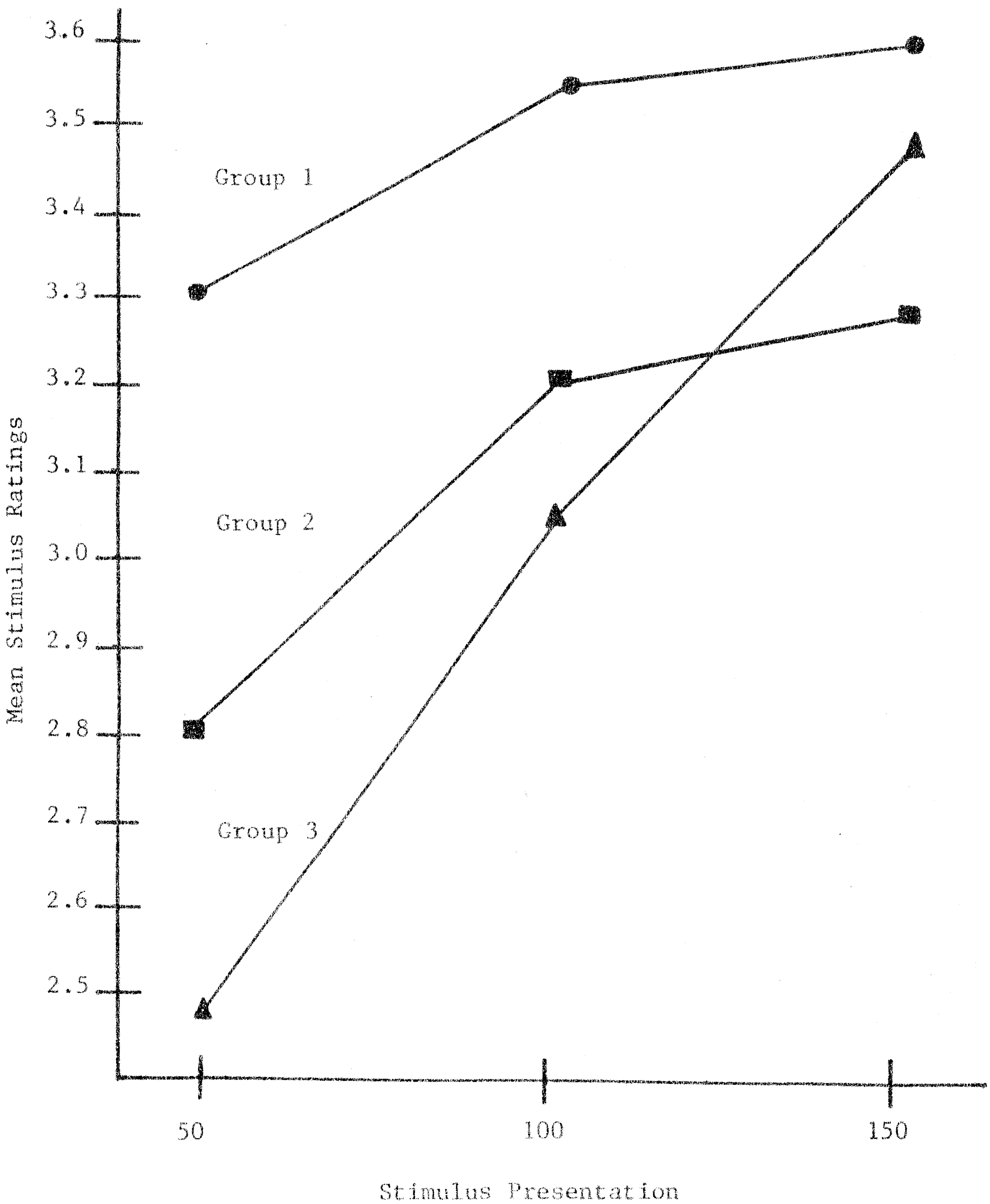


Figure 14. Plot of the effects of the anxiety manipulation and time on mean stimulus ratings.

Table 11

Summary of Analysis of Variance for the

Effects of the Anxiety Manipulation and Time on Stimulus Ratings

Source	SS	df	MS	*F*
Exp. Group	4.70	2	2.35	1.71
S(Exp. Group)	45.43	33	1.38	
Time	5.72	2	2.86	27.31**
Time X Exp. Group	1.80	4	0.45	4.29*
Time X S (Exp. Group)	6.91	66	0.10	

*$p < .005$

**$p < .0001$

Table 12

Duncan's Multiple Range Test for Differences

in Mean Stimulus Ratings

Time in Thirds	Mean[a]	Grouping[b]
First	2.87	A
Second	3.25	B
Third	3.43	C
Experimental Group		
Group 1	3.48	A
Group 2	3.07	A
Group 3	3.01	A

[a]n=36

[b]Means with different letters in each grouping are significantly different at $p < .05$.

Table 13

Newman-Keuls Test for Differences Between Experimental

Groups Across Time

							Number			
		1	2	3	4	5	6	7	8	9
No.	Group 1									
	Time									
1)	1	---			*			*		
2)	2		---		*			*	*	
3)	3			---	*	*	*	*	*	
	Group 2									
	Time									
4)	1				---	*	*	*		*
5)	2					---		*		
6)	3						---	*		
	Group 3									
	Time									
7)	1							---	*	*
8)	2								---	*
9)	3									---

*$p < .05$

ficantly from the first to second third of the experiment, but did not change from the second to final third. For subjects in Group 3, mean stimulus ratings increased significantly throughout the experiment. Although subjects in Group 3 reported the lowest stimulus ratings of all during the first third of the experiment, there were no differences between ratings of subjects in Group 3 and the other two groups during the final third of the experiment.

Chapter XI

DISCUSSION

The results of the data analysis support the conclusion that the experimental manipulation was effective in inducing and maintaining anxiety in female undergraduates. The two groups receiving the anxiety manipulations reported significantly more anxiety than the control group, and these differences were maintained throughout the course of the experiment. There were no differences, however, in the overall amount of anxiety reported between subjects in the two anxiety conditions. It appears that differences in the amount of anxiety reported are primarily attributable to the anxiety-arousing effects of negative feedback on the word test and not to viewing a gory film. Due to a lack of available data concerning the psychometric properties of the PARQ, the results must be interpreted somewhat cautiously, however.

It was originally hypothesized that increases in cognitive anxiety would be associated with a reduction in the response criterion. In addition, increases in somatic anxiety were expected to result in a decrease in sensitivity. However, a factor analysis of the anxiety responses failed to produce factors that could clearly be regarded as representing cognitive and somatic anxiety. This may have occurred because the factor analysis had to be performed under conditions of relatively low anxiety, as the design of the experiment prohibited a statistically sound analysis of the high anxiety data. Therefore, the pain responses were analyzed solely in terms of the general effects of anxiety and not specifically in relation to cognitive or somatic effects. Anxiety, in general then, was expected to be associated with decreases in sensitivity and a reduction in the response criterion. In other words, anxious subjects were expected to demonstrate poorer discriminability of heat stimuli and to tend to report pain at lower levels of stimulation.

Despite its general effectiveness, the experimental manipulation produced no differences between groups on measures of pain sensitivity, response bias, or mean stimulus ratings. This suggests that either the manipulation was not powerful enough to produce differences in pain ratings or that even significant amounts of induced anxiety do not necessarily affect the report of pain. Dividing the subjects into extreme groups of High and Low Anxiety in order to enhance the potential effects of anxiety did yield significant differences on the pain measures. Overall, the High Anxiety group reported lower stimulus ratings and showed lower sensitivity and higher response criteria. The differences in sensitivity were consistent with that which had been expected, but the differences in response bias and mean stimulus ratings were in the opposite direction to what was anticipated. In other words, subjects in the High Anxiety group showed poorer discriminability of heat stimuli but tended to be more <u>conservative</u> in reporting a stimulus as painful. On the average, subjects in the High Anxiety group actually reported <u>less</u> heat stimulation than those in the Low Anxiety condition.

Dividing subjects into groups of equal thirds on the basis of their anxiety scores also failed to demonstrate specific differences between the groups on reports of pain. Furthermore, none of the correlational analyses assessing a possible linear or curvilinear relationship between anxiety and the pain measures was significant. Taken together, these results suggest that the effects of induced anxiety on the report of pain are at best weak, and under certain conditions, situational anxiety may even be associated with a reduction in the self-report of pain to noxious stimulation.

These findings call into question previous research, which overwhelmingly supports the position that anxiety exacerbates the report of pain. Unfortunately, much of this earlier work suffers in terms of methodological soundness and due to inadequate analyses of effects. Most pain researchers (e.g., Bobey & Davidson, 1970; Hall & Stride, 1954; Hill, Kornetsky, Flanary, & Wikler, 1952; Lepanto, Moroney, & Zenhausern, 1965) have failed to demonstrate the actual induction of anxiety and have only inferred that the experimental manipulation has had the intended effects. The experimental manipulation, such as lack of control over noxious stimulation, may produce differences in the report of pain, but to attribute these differences to anxiety is unjustified without some kind of corroborating evidence. Furthermore, until recently, threshold and tolerance measures have been the primary means for assessing differences in pain reports. Because these measures are incapable of sorting out the effects due to sensitivity from those due to response bias, they provide no way of determining to what extent "anxious" subjects are reporting an increase in pain due to differences in discriminability or response tendencies. "Anxious" subjects in these experiments may not be feeling more pain than controls, but they might be biased to report more pain. This response bias effect might occur for a couple of reasons.

First, anxiety might be mislabelled as pain by subjects, particularly if the experiment entails the reporting only of pain and not anxiety as well. This is the usual case when manipulation checks for anxiety are absent. The assumption of this researcher is that although anxiety can be discriminated from concomitant pain, when anxiety responses are not measured there is a tendency to report total amount of negative affect experienced rather than just that due to pain. Second, under certain conditions subjects may be predisposed to report more pain whether they feel it or not. Take, for example, the patient who is nervous about receiving an inoculation. He is ready to yell "Ouch!" long before he feels the prick of the needle. Unfortunately, threshold and tolerance measures cannot distinguish between the physical experience of pain and attitudes affecting the reporting of it. Signal detection analysis provides a partial solution in that it examines differences in the ability of subjects to discriminate noxious stimuli independently from the tendency of subjects to report pain. SDT analyses allow us to determine whether differences in the pain reports of "anxious" subjects are due to perceptual factors, higher cognitive functions, or both.

Many of the results from previous research on anxiety and pain indicating a positive relationship between the two can be questioned solely on the basis that manipulation checks have been weak (e.g., Haslam, 1966) if not absent altogether (e.g., Bobey et al., 1970; Hall et al., 1954; Hill et al., 1952; Lepanto, et al., 1965). An experimental manipulation is implemented, and it is assumed that state anxiety has been significantly changed in the intended direction. This may or may not be the case. Various "anxiety" manipulations have included lack of control over noxious stimulus presentations (Hill *et al.*, 1952; Lepanto *et al.*, 1965) and instructions alluding to forthcoming pain (Hall *et al.*, 1954). Both of these manipulations have resulted in significantly greater reports of pain, but in no case have differences in actual anxiety levels been demonstrated. Plausible alternative explanations include differences in tendencies to label noxious stimulation as painful and differences in degree of stimulus control. In neither case is it necessary to infer that anxiety functions as a mediating variable. None of these studies has shown that it was actually anxiety which affected reports of pain.

Haslam asked her subjects post-experimentally whether they had felt anxious or not under conditions of threat of electric shock. The results for nine of the 29 experimental group subjects were discarded because they reported not feeling anxious. Even though she found significantly lower threshold values for "anxious" subjects, it is conceivable, if not probable, that subjects decided whether or not they were anxious based on self-observations of performance in the pain rating task. Subjects may have labelled themselves as anxious if they felt that they reported pain at lower than "average" levels of stimulation.

Chapman and Feather (1973), using the Spielberger STAI (State Anxiety) form as a manipulation check, found that diazepam and aspirin when compared to placebo reduced the anxiety associated with intense tourniquet pain. Although diazepam was shown to be more effective than aspirin or placebo in extending tolerance time, there were no differences in either sensitivity or response bias. Since diazepam affects anxiety, Chapman and Feather's results may be interpreted as providing no support for the relationship between anxiety change and SDT measures of pain. Chapman and Feather's findings are in line with those of the present study, which found no relationship between measures of experimentally manipulated anxiety and measures of pain sensitivity or response bias. An exception to this generalization was found when a <u>post hoc</u> analysis was done on extreme groups of High and Low Anxiety. High Anxiety in this analysis was related to lower pain ratings, higher response criteria, and lower sensitivity.

It would appear, then, that manipulations which have been shown to be effective in altering levels of state anxiety have no effect on sensitivity and response bias measures of pain or an effect opposite to that predicted. Although there is some evidence that reduced state anxiety tends to increase tolerance to pain (Chapman <u>et al</u>., 1973), experimental levels of state anxiety do not appear to affect ratings of pain intensity. When differences between levels of state anxiety have been increased by post-experimentally reassigning subjects into extreme groups of high and low state anxiety, it appears that subjects exhibiting greater amounts of anxiety tend to show poorer discriminability of noxious stimuli and a higher criterion for reporting pain. In other words, high state-anxious subjects may even be less likely to report a stimulus as painful.

A similar phenomenon has been observed in surgical patients. Martinez-Urrutia (1975) found that sensory pain ratings of high fear of surgery patients were consistently lower prior to and after surgery than those of the low fear of surgery patients. In general, however, findings have been mixed. While some researchers (e.g., Wolfer & Davis, 1970) have found no relationship between state anxiety and pain measures before or after surgery, others (e.g., Martinez-Urrutia, 1975) have found a significant positive correlation between state anxiety and pain reported postsurgery.

One possible explanation for the results of the present study is that the "anxiety" manipulation may have served to distract subjects from the pain rating task. "Anxious" subjects might have been so worried about their "negative" performance on the word test that they failed to attend as closely to the heat stimuli. This seems plausible in that the subjects were all college students who expect to perform well on "achievement" tasks. Many, if not all, of the subjects in the "anxiety" conditions made some excuse for their poor performance. This indicates a concern for doing well, one which may have overridden their concern about occasional painful stimulation. Perhaps, the word test drew the attention of "anxious" subjects away from the noxious stimuli so that the heat was not rated as being so painful. Bobey and Davidson offered a similar explanation when they found that female subjects listening to an audio tape of women in child labor showed more tolerance for pain than female subjects who listened to a rather boring tape on study habits. A reasonable conclusion is that state anxiety does not always adversely affect reports of pain, particularly if the procedure which induces anxiety directs attention away from the painful stimulation. In fact, under these conditions reports of pain may even be _reduced_ despite the presence of anxiety.

Another possible explanation, albeit less intuitively appealing, is that state anxiety, itself, inhibits reports of pain (Bolles, 1980) by reducing sensitivity and raising the response criteria. However, closer inspection of the data challenges the viability of this hypothesis. Figure 14 and Table 12 illustrate that mean stimulus ratings tended to increase throughout the course of the experiment. In addition, Table 2 and Figure 13 show that anxiety ratings also increased over time. If both anxiety and mean stimulus ratings increased as the experiment progressed, one might expect high levels of anxiety to be associated with high pain ratings but certainly not low pain ratings as was the case. Furthermore, Figure 14 shows that mean stimulus ratings for the three groups tended to converge over time. Table 13 indicates that stimulus ratings for subjects in the two anxiety groups "climbed" significantly during the experiment, while the stimulus ratings of the control subjects remained constant. However, anxiety ratings increased for all groups across time. Therefore, one would expect the stimulus ratings of the control subjects to increase as well if anxiety were mediating the pain responses. This did not occur, however, casting doubt on the anxiety hypothesis.

If state anxiety does not affect the perception or rating of acute pain, then it would seem from a theoretical perspective that separate neural pathways are involved. In terms of Melzack's theory, state anxiety apparently has no effect on the neural gating mechanism that is thought to regulate the experience of pain. If certain cognitive activities such as distraction effectively inhibit reports of pain as this study tentatively suggests, then descending fibers from higher cognitive centers may be involved in closing the gate. This may have the effect of reducing the upward transmission of neural impulses that are perceived as painful. Another way of viewing the function of the neural gate is to think of closing the gate as reducing sensitivity or discriminability. When the gate closes, it may prevent sufficient input about noxious stimuli from reaching the brain where it is interpreted as painful or not. In other words, closing the gate may have the effect of making stimulus inputs ambiguous enough so that discriminability is poor. Opening the gate may permit input to flow freely and allow for increased discriminability. Whether or not the stimuli are interpreted as painful or not may depend on previous experience, expectations toward feeling pain, or the levels of sensitivity involved. Stimuli that elicit high levels of sensitivity may be regarded as painful, because of extreme differences between them and ambient levels of stimulation. Lower levels of sensitivity may be reported as painful if cognitive factors (i.e., response biases) mediate. The preceding interpretation is entirely speculation, however, and was offered only as one possible way to integrate signal detection analysis with current pain theory.

In terms of clinical applicability, the present study suggests that state anxiety and pain are two different symptoms and that both problems may need to be targeted for treatment. In other words, it cannot be assumed, as it has been, that alleviating anxiety will automatically ameliorate the pain associated with it (Clum, Scott, & Burnside, 1979). It seems likely that anxiety treatments will reduce reports of pain only if they involve patient responses that directly counteract the cause of pain. For example, progressive muscle relaxation training might be provided to women preparing for natural childbirth. Not only should it help relieve the anxiety associated with delivery, but it should reduce the amount of pain involved by minimizing muscle contractions in the abdominal and pelvic regions. Didactic instruction and preparatory exposure (e.g., via films) may alleviate the anxiety associated with delivery, but theoretically these procedures should have little effect on pain because they do not counteract the source of the pain--excessive muscle contraction. The point is that state anxiety and pain are separate symptoms and treating one will not necessarily result in improvement of the other.

Because the present study is the first to demonstrate that induced anxiety has at best a minimal effect on the report of pain, additional support from further research is needed before the arguments made here can carry much empirical weight. One valuable contribution of the present study is that it demonstrated an effective way of inducing and maintaining anxiety for rather long periods of time (about 90 minutes). Future research may need to address the role of cognitive and somatic anxiety in the report of pain. Because of design limitations, the present study was only minimally informative in this regard. In any case, the present study highlights the importance of using manipulation checks in future research on anxiety.

The extent to which these results generalize to clinical populations is also a major concern. Are the responses of female undergraduates in a laboratory situation comparable to those of acute or chronic pain sufferers? In addition, the proposed clinical applicability of these results needs to be tested. Is it more efficacious to devise treatments that address symptoms of both anxiety and pain? Or will treating one symptom sufficiently alleviate the other? The present study and common sense suggest that interventions should be matched to the symptoms they affect, but this approach has largely been untried and untested in the treatment of anxiety and pain.

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Appendix A

Consent form

Participant: ________________________ Date: ______
Date of Birth: ________________________ Sex: ______

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 tasks on the response to noxious stimuli.

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

   a) Answer a number of questionnaires.
   b) Take a test that will measure my verbal abilities
   c) Have different intensities of radiant heat applied to points on my arm darkened with India ink
   d) View two films, one on ____________ and one on ____________

4. I understand that some of these heat stimuli will produce a painful sensation. I have been assured that even though a temporary redness of skin may result, there is no danger of being burned.

5. I understand that my participation is voluntary and that I may terminate my participation at any time without penalty. I have been told that if I have any questions concerning this project I may call either:

   Project Director--Dr. George A. Clum (961-1697)
   or
   Chairman, Institutional Review Board--Dr. Martin Stombler (961-5283)

6. Although I understand that the procedure is safe and has been used by previous researchers at Virginia Tech and elsewhere, I further acknowledge 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: __________
____

Phone: ________________ Social Security Number: ____________

Appendix B

Date____________________

PRESENT AFFECT REACTIONS QUESTIONNAIRE (PARQ)

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

"HOW YOU FEEL AT THIS PARTICULAR MOMENT"

1.	Hands feel moist	1 Not at all	2	3	4	5 Very moist
2.	Feel relaxed	1 Very relaxed	2	3	4	5 Not at all
3.	Hands feel unsteady	1 Not at all	2	3	4	5 Very unsteady
4.	Feel self-confident	1 Very much	2	3	4	5 Not at all
5.	Stomach feels tense	1 Not at all	2	3	4	5 Very tense
6.	Enjoy this situation	1 Very much	2	3	4	5 Not at all
7.	Heart beats faster	1 Not at all	2	3	4	5 Much faster
8.	Feel calm	1 Very calm	2	3	4	5 Not at all
9.	Perspire	1 Not at all	2	3	4	5 Very much
10.	Feel comfortable	1 Very much	2	3	4	5 Not at all
11.	Mouth feels dry	1 Not at all	2	3	4	5 Very dry

Item	1	2	3	4	5
12. Unable to focus my thoughts	1 Able to focus	2	3	4	5 Unable to focus
13. Feel pleasant	1 Very pleasant	2	3	4	5 Not at all
14. Feel nervous	1 Not at all	2	3	4	5 Very nervous
15. Feel throbbing in my head	1 Not at all	2	3	4	5 Very much so
16. Feel secure	1 Very secure	2	3	4	5 Not at all
17. Feel upset	1 Not at all	2	3	4	5 Very upset
18. Hands feel cold	1 Not at all	2	3	4	5 Very cold
19. Feel good	1 Very good	2	3	4	5 Not at all
20. Feel anxious	1 Not at all	2	3	4	5 Very anxious
21. Breathing is irregular	1 Not at all	2	3	4	5 Very irregular
22. Feel uneasy	1 Not at all	2	3	4	5 Very uneasy
23. Want to avoid this situation	1 Not at all	2	3	4	5 Very much
24. Feel lump in throat	1 Not at all	2	3	4	5 Very much

PARQ-75

Appendix C

INSTRUCTIONS FOR THE ANXIETY GROUPS

The purpose of this experiment is to see what effect performing certain tasks has on the way in which people discriminate different intensities of heat. Your task is to answer items from a multiple-choice vocabulary test, as you rate the intensity of various heat stimuli.

Heat will be applied to areas of skin on your right arm that have been darkened with India ink. The heat source is a floodlamp housed inside the gray metal box on the table in front of you. Heat will be presented for a few seconds at a time at regular intervals.

The intensity of the heat will vary. Sometimes it will be great enough to produce a painful sensation. Other times, the heat will be less intense, and sometimes no heat will be presented at all. A buzzer will sound after the stimulus has been presented. After you hear the buzzer, you are to rate the intensity of the heat using the following scale. A rating of "0" means that you felt "nothing" at all. A "1" means that you felt "something." A "2" means that the stimulus was "warm." A "3" means that the stimulus was "hot." A "4" means that the stimulus was "faintly painful." A "5" means that the stimulus was "painful." And a "6" means that the stimulus was "very painful."

So that your ratings are not influenced by the amount of light you see, a dark curtain will block your view of the equipment. The experimental assistant will position your arm against the apparatus for you. Please understand that this experiment is designed to see how people rate different levels of heat intensity, not how much pain they can endure. If the stimulus becomes too painful, feel free to remove your arm from the heat.

As soon as you rate the intensity of the heat, an item from the vocabulary test will be read to you. These items are all multiple-choice with four alternatives to choose from. You are to select the letter of the answer which is nearest in meaning to the word that was presented first. For example, if you are told: "Apple: a) building b) telephone c) fruit d) coat," you would answer "c." You must respond using the appropriate letter. The answer fruit would be marked "wrong." Each item will only be read once, so listen carefully. Furthermore, you must answer before the next heat stimulus is presented. If you fail to answer in this time, that item will be marked as wrong. If you rate the intensity of the heat as soon as you hear the buzzer, you should have about ten seconds to answer the test item once it has been presented. Please note that the order of the heat intensities is random and that neither your ratings nor your test responses influence how much heat is presented.

The vocabulary test you will be taking has been used on a national level with high school seniors preparing to go to college. On the average, college-bound high school seniors get considerably more than half of the items correct.

To summarize, you are to rate aloud the intensity of each heat stimulus after you hear the buzzer. You will then be presented with a test item which you must answer before the next heat stimulus comes (in about ten seconds). Remember to say the letter of the apprpriate answer and not the word, itself.

Throughout the experiment, there will be several short breaks. During some of these you will be asked to fill out a questionnaire.

Any questions? In order to familiarize you with the procedure, we will now try 20 practice trials.

Appendix D

INSTRUCTIONS FOR THE CONTROL GROUP

The instructions for the control subjects were identical to the instructions for the subjects in the anxiety conditions, except for the paragraph describing the average national performances on the vocabulary test. In place of that section, the following paragraph was inserted:

The vocabulary test you will be taking has been used on a national level with college seniors. On the average, college seniors get considerably less than half of the items correct.

APPENDIX E

Vocabulary Test

(c) 1. regime: a) military group b) summary c) rule d) manor
(a) 2. tenacity: a) persistence b) large town c) indifference d) ecstasy
(d) 3. cogent: a) geared b) formidable c) strong d) convincing
(c) 4. condiment:a) vegetable b) salad c) relish d) sugar
(a) 5. cryptic: a) obscure b) written c) copied d) puzzled
(b) 6. dormant: a) active b) hibernating c) vigorous d) birdlike
(c) 7. altercation: a) adjustment b) repair c) quarrel d) echo
(a) 8. bombastic: a) inflated b) explosive c) meek d) enraged
(c) 9. inundate: a) overwhelm b) surrender c) flood d) conquer
(b) 10. morose: a) calm b) gloomy c) damp c) conquer
(d) 11. surmise: a) plan b) unexpected c) tragedy d) guess
(d) 12. attrition: a) addition b) regret c) attitude d) abrasion
(b) 13. chagrin: a) chin b) mortification c) elation d) intuition
(c) 14. hamlet: a) actor b) small rodent c) village d) introvert
(a) 15. pneumatic: a) pertaining to air b) automatic c) sick d) elastic
(d) 16. tepid: a) enraged b) transported c) embarrassed d) lukewarm
(d) 17. panacea: a) praise b) inactivity c) talk d) cure-all
(a) 18. celibate: a) single b) double c) married d) bald
(a) 19. chasten: a) discipline b) pursue c) sanctify d) stop
(d) 20. requiem: a) recess b) assignment c)profanity d) dirge
(b) 21. effigy: a) boldness b) dummy c) exit d)proxy
(a) 22. disparity: a) difference b) harmony c) discord d) argument
(c) 23. filch: a) pretend b) dirty c) steal d) embarass
(c) 24. infinite: a) verbal b) indefinite c) endless d) vague
(b) 25. demise: a) residence b) death c) accident d) act
(c) 26. fastidious: a) speedy b) precise c) squeamish d) hungry
(a) 27. vestige: a) trace b) undergarment c) hallway d) swamp
(d) 28. perfunctory: a) thorough b) impossible c) lively d) listless
(d) 29. indigence: a) nativity b) tolerance c) eating d) poverty
(b) 30. chicanery: a) aroma b) trickery c) chastity d) poultry
(a) 31. vindictive: a) revengeful b) strategic c) triumphant d) bigoted
(a) 32. haggard: a) gaunt b) irascible c) sluggish d) witty
(c) 33. staid: a) weary b) remaining c) sedate d) afraid
(d) 34. expedient; a) precise b) expert c) expendable d) advisable
(a) 35. nepotism: a) favoritism b) pool c) philosophy d) hedonism
(a) 36. propensity: a) inclination b) intelligence c) dishonesty d) act
(b) 37. mollify: a) sweeten b) appease c) worry d) discourage
(b) 38. baroque: a) polished b) highly ornate c) constant d) transformed
(c) 39. benign: a) peaceful b) blessed c) favorable d) wavering
(c) 40. barrister: a) specialist b) teacher c) attorney d) professor
(b) 41. bland: a) meager b) mild c) soft d) uncooked
(a) 42. bellicose: a) war-like b) navel c) amusing d) piecemeal
(d) 43. condone: a) stop b) evaluate c) piece d) overlook
(b) 44. prodigal: a) wandering b) thrifty c) compatible d) consistent

(b) 45. misdemeanor: a) felony b) peccodillo c) indignity d) illiteracy
(d) 46. dogmatic: a) canine b) impatient c) petulant d) arbitrary
(b) 47. tenuous: a) vital b) thin c) dangerous d) necessary
(a) 48. remiss: a) lax b) lost c) foolish d) ambitious
(c) 49. hybrid: a) product b) species c) mixture d) genus
(b) 50. impunity: a) violation b) liability c) joke d) appropriate
(a) 51. allegory: a) fable b) poem c) essay d) novel
(d) 52. tentative: a) prevalent b) certain c) mocking d) experimental
(a) 53. pert: a) impertinent b) moral c) deliberate d) stubborn
(c) 54. mundane: a) futile b) spiritual c) worldly d) heretic
(c) 55. garnish: a) paint b) banish c) adorn d) abuse
(b) 56. glib: a) slippery b) articulate c) fashionable d) old
(a) 57. dour: a) sullen b) grizzled c) speechless d) international
(b) 58. insinuate: a) resist b) suggest c) report d) lecture
(b) 59. cursive: a) avowing b) running c) flashy d) lewd
(b) 60. amazon: a) native b) female warrior c) responsive d)jungle
(b) 61. remedial: a)therapeutic b) corrective c)traumatic d)psychotic
(a) 62. robust: a)vigorous b) violent c) vicious d) voracious
(d) 63. modish: a) sentimental b) contrary c) humid d) stylish
(b) 64. mirage: a) dessert b) illusion c) water d)mirror
(c) 65. franchise: a) subway b) reason c)license d)fashion
(a) 66. construe: a) explain b) promote c) reserve d) block
(a) 67. brazen: a) shameless b) quick c) modest d) melodramatic
(b) 68. acrid: a) sour b) bitterly pungent c) sweetish d) very hard
(c) 69. infallible: a) final b) unbelieveable c) perfect d) inaccurate
(a) 70. abysmal: a) bottomless b) eternal c) diabolic d) internal
(b) 71. innocuous: a) not capable b) not dangerous c) not eager
(b) 72. insipid: a) witty b)flat c) talkative d)lucid
(b) 73. jurisprudence: a) caution b) law c) grand jury d) haste
(c) 74. kinetic: a) cinematic b) polar c) motion d) foreign
(b) 75. prophylactic: a) toxic b) preventive of disease c)antagonistic
(d) 76. reprisal: a) assessment b) loss c)nonsense d) retaliation
(c) 77. motley: a) active b)disguised c) variegated d) somber
(a) 78. mentor: a)guide b) genius c) talker d) philosopher
(a) 79. enervate: a) weaken b) sputter c) arrange d) supervise
(b) 80. contrite: a) smart b) penitent c) restful d) recognized
(d) 81. foster: a) speed b) fondle c) roll d) raise
(d) 82. germane: a) excitement b) prominent c) warlike d) relevant
(a) 83. musty: a)flat b) necessary c) indifferent d) vivid
(a) 84. retrograde: a) receding b) inclining c) evaluating
(d) 85. tenacious: a) running b) intentional c) obnoxious d) holding fast
(b) 86. succor: a) juicy b) aid c)fanciful d) filth
(c) 87. moot: a) insignificant b) strong c) debatable d) wet
(b) 88. contusion: a)abrasion b) bruise c) mistake d) hazy
(d) 89. dubious: a)unrefined b) thin c) correct d)doubtful
(a) 90. grandiose: a) impressive b) happy c) free d)smooth
(b) 91. indolence: a) wholeness b) baziness c) indisputable d)bankruptcy
(a) 92. officious: a) meddlesome b) pompous c) offensive d)gloomy
(c) 93. anomaly: a) yearly allowance b) charm c)irregularity d) summit

(a) 94. astute: a) shrewd b) dull c) stupid d) deceitful
(b) 95. oblique: a) dark b) slanting c) stingy d) odorous
(c) 96. sanguine: a) angry b) satiated c) cheerful d) regretful
(b) 97. ruse:a) misuse b) trick c) shadow d) bloody
(b) 98. transgression: a) diversion b) sin c) proceedings d) conversion
(d) 99. zephyr: a) four-legged b) tidbit c) ghost c) breeze
(a)100. venal: a) dishonest b) tarnished c) clear d) ignorant
(b)101. strident: a)loved b) loud c) loose d) long
(b)102. pastoral: a) scarce b)rural c) religious d) perfect
(d)103. inimical: a) unequaled b) paltry c) unbelieveable d) hostile
(c)104. foist: a) raise b) clamor c) palm off d)brawl
(a)105. salient: a) prominent b) salty c)cynical d) profane
(d)106. eschew: a) captivate b)reprimand c) call forth d) avoid
(a)107. depredation: a) plundering b) decay c) annihilation d) degrading
(b)108. potable: a) spotted b) suitable for drinking c) old-fashioned d) presumptuous
(c)109. hyperbole: a) deduction b) induction c) exaggeration
(b)110. boorish: a) tiresome b) rude c) youthful d) eccentric
(a)111. tirade: a) denunciation b) tortuous c) local government
(a)112. terse: a) abrupt b) poisonous c) irritable d) docile
(b)113. portend: a) babble b) foretell c)purge d) appease
(d)114. pragmatic: a) confused b) striking c)necessary d) practical
(c)115. novice: a)pedestal b) writer c) beginner d)departure
(a)116. incorrigible: a) uncorrectable b)poor c) listless d) hateful
(c)117. insidious: a) tasteless b) haughty c) sly d) rebellious
(c)118. condescent: a) digress b) cancel c) bestow courtesies d) assemble
(b)119. bogus: a) extra b) counterfeit c) spoiled d) useless
(a)120. abdicate: a) renounce b) separate c) pacify d) urge
(d)121. supple: a)tight b) smooth c)slippery d) flexible
(c)122. refurbish: a) bend b) mortgage c) renovate d) drench
(b)123. throttle: a) whip b) strangle c) gnash d) stretch
(b)124. precarious: a) empowered b) risky c) harmful d) mysterious
(b)125. innuendo: a) disguise b) hint c) conclusion d) restraint
(d)126. hapless: a) furthest behind b) grisly c) disgusting
(c)127. enamored: a) efficient b) worn out c) in love d) attired
(b)128. droll: a) boring b) amusing c) weighty d) simple
(a)129. nocturnal: a) night b) forever c) medical d) western
(a)130. subterfuge: a) pretense b) assistance c) metallic d) strength
(b)131. sundry: a) lavish b) various c) binding d) playful
(b)132. homily: a)danger b) sermon c) domestic d) gap
(d)133. avid: a) reluctant b) good-natured c) mature d) greedy
(b)134. lackey: a) liberal b) footman c) druggist d)rascal
(a)135. eulogy: a) praise b) mourning c) calmness d) giant
(a)136. precept: a) rule b)juncture c) disfigured d) gem
(a)137. tainted: a)contaminated b) still c) dry d)rich
(c)138. stymie: a) conquer b) verify c) stump d) allot
(b)139. exonerate: a) tax b) acquit c) glorify d) exclaim
(a)140. dank: a) damp b) flip c) slope d) lure
(a)141. collusion: a) comspiracy b) injury c) remark d) knowledge

(d)142. cleft: a) security b) competition c) courtesy d) split
(b)143. amiable: a)lively b) agreeable c) manlike d) fruitless
(b)144. adamant: a) artificial b) inflexible c) skillful d) profound
(b)145. lampoon: a) lance b) ridicule c) lust d) misbehave
(c)146. impetuous: a) unprepared b) faulty c) violent d) silky
(a)147. devout: a) pious b) dainty c) untidy d) humble
(c)148. convene: a) depart b) revoke c) assemble d) forgive
(b)149. caprice: a) honor b) whim c) quick d) brutal
(c)150. frigid: a) unhealthy b) deceitful c) cold d) mad
(a)151. paucity: a) scarcity b) clarity c) hesitancy d)frailty
(c)152. simile: a) greeting b) criticism c) comparison d) sedation
(a)153. urbane: a) suave b) industrial c) careless d) empty
(d)154. trident: a) sugar b) star c) super d) spear
(b)155. undulate: a) argument b) wave-like motion c) archives d)lender
(c)156. ruminate: a)board b) rescue c) ponder d) battle
(d)157. secular: a) frisky b) prophetic c) reduced d) temporal
(a)158. lurid: a) wild b) tearful c) pertinent d) criminal
(d)159. dawdle: a) trinket b) flame c) infant d)loiter
(c)160. equivocal: a) superior b) delicious c) doubtful d) small-minded
(d)161. putrid: a) in bloom b) political c) nautical d) foul
(b)162. fusion: a)proposal b) union c) hook d) denial
(c)163. engender: a) eat away b) captive c)cause d)vex
(b)164. labyrinth: a)tiger b) maze c) lie d) jeopardy
(b)165. intrepid: a) traveled b) fearless c) wasted) sincere
(c)166. jaded: a) sharp b) lonely c) fatigued d) oriental
(a)167. maxim: a) proverb b) exotic c) plentiful d) royalty
(d)168. motif: a)necklace b) decorum c) formula d) theme
(c)169. import: a) product b) lunacy c) significance d) glassware
(c)170. titanic: a) frozen b) silent c) gigantic d) experimental

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THE EFFECTS OF EXPERIMENTALLY-INDUCED ANXIETY ON THE REPORT OF PAIN: A SIGNAL DETECTION ANALYSIS

by

Scott Nelson Mohler

(ABSTRACT)

Previous research on pain threshold and tolerance suggests that induced anxiety tends to increase the report of pain, but rarely have actual levels of anxiety been monitored. Furthermore, threshold and tolerance measures of pain have been shown to be highly susceptible to cognitive and attitudinal factors which affect response bias. The present study attempted to investigate the effects of experimentally-induced anxiety on the report of pain, taking these problems into account. Thirty-six female undergraduates were assigned to one of three experimental conditions. Subjects in the two anxiety groups received negative feedback on a vocabulary test and saw a neutral film or a gory film. Control subjects were given positive feedback on the word test and viewed the neutral film. It was expected that anxious subjects would exhibit poorer discriminability of noxious heat stimuli (lower sensitivity) and tend to report pain at lower levels of stimulation (lower response criteria). Self-reports of state anxiety were higher for both anxiety conditions. However, a signal detection analysis found no

differences between groups on pain sensitivity or response bias. Nor were there any differences in mean stimulus ratings. When subjects were post-experimentally divided into extreme groups of High and Low Anxiety, High Anxiety subjects showed lower sensitivity. But contrary to expectations, they exhibited higher response criteria and lower mean stimulus ratings. These results suggest that the relationship between situational anxiety and pain is at best weak, and under certain conditions, state anxiety may be associated with reduced reports of pain.