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Full Name of Author — Nom complet de l'auteur

HENDERIKUS JOHANNES STAM

Date of Birth — Date de naissance

June 23, 1954

Country of Birth — Lieu de naissance

NETHERLANDS

Permanent Address — Résidence fixe

DEPT. of Psychology
University of CALGARY
CALGARY, ALBERTA

CANADA

T2N 1N4

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Name of Supervisor — Nom du directeur de thèse

Nicholas P. Spanos

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HYPNOTIC ANALGESIA AND THE PLACEBO EFFECT:
CONTROLLING ISCHEMIC PAIN

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A Dissertation presented to the Faculty of Graduate Studies,
at Carleton University in partial fulfillment of the
requirements of the Degree of
Doctor of Philosophy

OTTAWA, CANADA

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The undersigned recommend to the Faculty
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submitted by Henderikus J. Stam
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the degree of Doctor of Philosophy

Outside Examiner

Thesis Supervisor

Chairman, Department of
Psychology

Carleton University

July 1982

Abstract

The literature on hypnotic and suggested analgesia is reviewed under three topic areas. The first deals with the literature on the psychophysiological correlates of suggested analgesia and concludes that there is little, if any, relationship between these variables. The second section examines attempts to quantify reported pain followed by an analysis of the effects of numerous antecedent variables on reported pain in studies of suggested analgesia. In general, the use of a hypnotic induction procedure does not add to the reductions in reported pain produced by suggestions for analgesia alone. Finally, theories that have been proposed to account for hypnotic and suggested analgesia are presented. Special process accounts such as dissociation are found wanting. The most promising theoretical vantage point appears to be one that includes subjects' cognitions as mediator variables.

Two experiments were conducted to examine the relationship between hypnotic and placebo analgesia using ischemic pain. The first experiment was designed to elucidate a possible artifact in a previously used ischemic pain stimulus. Subjects either high or low in hypnotic susceptibility received two trials of ischemic pain. On each trial their task was to pump water with an occluded arm. Prior to the second trial all subjects received

hypnotically suggested analgesia. Results indicated that, following hypnotic analgesia, subjects pumped more water over a longer period of time. This was accompanied however by a reduced rate of "work" at which water was pumped. This artifact precludes the use of this technique as an uncontaminated measure of ischemic pain. Experiment 2 was an investigation of the relationship between hypnotic and placebo analgesia using a submaximum effort tourniquet technique to produce ischemic pain. This technique is not affected by the rate of "work". High and low susceptible subjects received hypnotic and placebo analgesia in counterbalanced order. High susceptible subjects who received placebo analgesia followed on a subsequent trial by hypnotic analgesia showed significant increases in tolerance from placebo to hypnotic analgesia. When presented in the reverse order, however, placebo analgesia and hypnotic analgesia led to equivalent high levels of tolerance in high susceptible subjects. These findings are discussed in terms of the experimental situation and the importance of control to the subject.

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The free market of ideas has never been free, but always a market.

R. Jacoby

TABLE OF CONTENTS

ABSTRACT	1
ACKNOWLEDGEMENTS	iii
INTRODUCTION AND LITERATURE REVIEW	
Introduction	1
Hypnotic Analgesia - Physiological Indicators	2
Endogenous Opioid Peptides and Hypnotic Analgesia	16
Experimental Studies of Hypnotic Analgesia -	
Verbal Reports	19
Category Ratings	20
The manipulation of antecedent variables	20
Between-subjects experiments	21
Within-subjects experiments	23
Magnitude Estimation	31
Signal Detection Theory	40
Functional Measurement	43
Theories of Hypnotic Analgesia	46
Special Processes	46
Negative Hallucinations	49
Cognitive Activity	51
Placebo and Suggestibility	56
Outline of Research	65

	PAGE
EXPERIMENT 1	
Introduction	69
Method	69
Results	74
Discussion	104
EXPERIMENT 2	
Introduction	111
Method	112
Results	119
Discussion	161
GENERAL DISCUSSION	168
REFERENCES	176
FOOTNOTES	204
APPENDICES	208

LIST OF TABLES

1. Summary Table for Susceptibility x Trial ANOVA on Duration to Threshold	76
2. Summary Table for Susceptibility x Trial ANOVA on Duration to Tolerance	77
3. Mean Time to Threshold and Time to Tolerance Scores in Sec	78
4. Summary Table for Susceptibility x Trial ANOVA on Volume to Threshold	79
5. Summary Table for Susceptibility x Trial ANOVA on Volume to Tolerance	80
6. Mean Volume to Threshold and Volume to Tolerance Scores in Ml	82
7. Summary Table for Susceptibility x Trial ANOVA on Work to Threshold	83
8. Summary Table for Susceptibility x Trial ANOVA on Work to Tolerance	84
9. Mean Ratio to Threshold and Ratio to Tolerance Scores	85
10. Summary Table for Susceptibility x Trial ANOVA on Catastrophizing VAS	86
11. Mean Catastrophizing VAS Scores	87
12. Summary Table for Susceptibility x Trial ANOVA on Coping VAS	89

	PAGE
13. Mean Coping VAS Scores for Susceptibility x Trial Interaction	90
14. Summary Table for Susceptibility x Trial ANOVA on Percent Time Imagining	91
15. Mean Percent Time Imagining Scores for Susceptibility x Trial Interaction	92
16. Correlations Among Performance and Cognitive Activity Measures	94
17. Summary Table for Susceptibility x Trial ANOVA on Arm-Tiredness VAS	95
18. Mean Arm-Tiredness VAS Scores	96
19. Mean Performance Scores for Subjects Indicating Ability to Continue vs. Subjects Indicating Maximum Pain	98
20. Summary Table for Susceptibility x Trial ANOVA on Estimated Duration of Continuation	99
21. Mean Estimated Duration of Continuation Scores for Susceptibility x Trial Interaction	100
22. Summary Table for Susceptibility x Trial ANOVA on Estimated Time	102
23. Summary Table for Susceptibility x Trial ANOVA on Estimated Volume	103
24. Summary Table for Susceptibility x Trial ANOVA on Estimated Work	105
25. Mean Estimated Time, Volume and Work Scores	106
26. Summary Statistics for Tolerance Scores	120

	PAGE
27. Summary Table for Group x Susceptibility x Trial ANCOVA on Duration to Tolerance	121
28. Summary Statistics for R^2 for Both Power and Linear Functions	127
29. Summary Tables for Group x Susceptibility x Trial ANOVAs on Slopes for Power and Linear Functions	129
30. Summary Tables for Group x Susceptibility x Trial ANOVAs on Intercepts for Power and Linear Functions	130
31. Correlations Between Tolerance and Slopes and Intercepts by Trial	131
32. Summary Table for Group x Susceptibility x Trial x Time ANOVA on Transformed Magnitude Estimates	133
33. Mean Adjusted Log Magnitude Estimates for Group x Trial Interaction, Trial Main Effect, and Time Main Effect	134
34. Summary Table for Group x Susceptibility x Trial ANOVA on Catastrophizing VAS	140
35. Catastrophizing VAS Means for Group x Trial Interaction	141
36. Summary Table for Group x Susceptibility x Trial ANOVA on Coping VAS	143
37. Mean Coping VAS Scores for Group x Trial Interaction	144
38. Summary Table for Group x Susceptibility x Trial	

	PAGE
ANOVA on Percent Time Imagining	145
39. Mean Percent Time Imagining Scores for Group x Trial Interaction	146
40. Correlations Between Cognitive Activities and Tolerance and Linear and Power Function Slopes	148
41. Summary Table for Group x Susceptibility x Trial ANOVA on State Anxiety	149
42. State Anxiety Means for Group x Trial Interaction	151
43. Summary Table for Group x Susceptibility x Trial ANOVA on Symptoms	152
44. Symptom Means for Group x Trial Interaction	153
45. Correlations Between Tolerance Difference Scores and Symptoms and Anxiety	155
46. Correlations Between Cognitive Activity Measures and Anxiety and Symptoms	157
47. Summary Table for Group x Susceptibility x Trial ANOVA on Expectancy	158
48. Expectancy Means for Group x Trial and Susceptibility x Trial Interactions	159
49. Summary Table for Group x Susceptibility x Trial ANOVA on Estimated Time	160A
50. Mean Estimated Time Scores for Group x Trial Interaction	160B

LIST OF FIGURES

1. Adjusted Mean Tolerance for Group x Susceptibility x Trial Interaction	123
2. Adjusted Mean Tolerance for Group x Trial Interaction	124
3. Mean Transformed Log Magnitude Estimates for Group x Trial Interaction	135
4. Mean Magnitude Estimates for Group B/P/H	137
5. Mean Magnitude Estimates for Group B/H/P	138
6. Mean Magnitude Estimates for Control Group	139

LIST OF APPENDICES

A. Informed Consent Agreement for Experiment 1	209
B. Instructions for Ischemic Pain Task in Experiment 1, Modified from McGlashan, Evans, and Orne (1969, p. 233)	211
C. Postexperimental Questionnaires	213
D. Hypnotic Induction Procedure	216
E. Suggestion for Analgesia	221
F. Instructions for Ischemic Pain Task Following Hypnotic Analgesia in Experiment 1 - From McGlashan, Evans, and Orne (1969, p. 234)	222
G. Informed Consent Agreement for Experiment 2	223
H. Symptom Checklist	225
I. Instructions for Magnitude Estimation Procedure	227
J. Physics of Lasers Lecture	229
K. Instructions Given Subjects Prior to Trial Three Modified from McGlashan, Evans, and Orne (1969, p. 234)	232
L. Mean Magnitude Estimates for Each Group and Trial Prior to Transformation to a Common Modulus and Function Fits Based on Average Data	233
M. Means and Standard Deviations for Catastrophizing, Coping and Time Imagining Scores in Experiment 2	239

Hypnotic Analgesia and the Placebo Effect:
Controlling Ischemic Pain

The use of hypnotic analgesia to alleviate pain has been reported since the beginning of the nineteenth century (see Ellenberger, 1970). Laboratory investigations of hypnotic analgesia however did not begin until the twentieth century and are still the subject of considerable controversy. It is the experimental literature that is the subject of this thesis and the following review.¹ The literature will be divided into the following three sections: (a) physiological concomitants of hypnotic analgesia, (b) measuring subjects' reports of reduced pain and the manipulation of antecedent variables, and (c) theories of hypnotic analgesia.

The definition of pain that will be adopted here is the definition proposed by the subcommittee on taxonomy of the International Association for the Study of Pain. Pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Bonica, 1979, p. 250). As will be noted later, there are several implications of accepting such a definition, the most important of which is that pain is not necessarily tied to a particular type of stimulus. Rather pain is viewed as belonging more to categories of experience such as hunger, thirst, nausea, vertigo, and so

on, than to such sensory systems as vision, hearing, and touch (Wall, 1979).

Suggestions for analgesia typically consist of brief verbal instructions to engage in some type of cognitive or imaginative activity aimed at alleviating the pain produced by a noxious stimulus (e.g., Barber, 1970; Hilgard & Hilgard, 1975). Hypnotic analgesia refers here to the presentation of a hypnotic induction procedure followed by suggestions for analgesia in a situation that is defined as "hypnosis." There is no convincing evidence for the existence of a hypnotic state (e.g., Barber, 1969). Subjects' reports of being hypnotized are themselves explicable in terms of situational demands and do not require the positing of an "hypnotic state" (Radtke & Spanos, 1981). Thus, "hypnotic" as used here refers to instructional procedures traditionally associated with the research area of hypnosis. For historical reasons these procedures typically consist of interrelated suggestions designed to (a) elicit cooperation and maximize performance, (b) produce relaxation, and (c) define the situation as hypnosis.

Hypnotic Analgesia - Physiological Indicators

The earliest experimental attempts to verify the efficacy of suggested analgesia focussed on physiological

indices that responded to painful stimulation. Physiological indices were believed to be involuntary responses to painful stimulation and thus were thought not to be subject to the response distortions inherent in verbal reports. Studies prior to 1930 generally involved a single subject exposed to a noxious stimulus, such as pinpricks, under hypnotic analgesia while the GSR was measured (e.g., Prince & Peterson, 1908). Seven such early studies were equivocal in their outcomes (see Shor, 1967).

Between 1930 and 1960 a total of seven more experiments were conducted to compare analgesia suggestions with various control procedures on responses to painful stimulation (Barber & Coules, 1959; Brown & Vogel, 1938; Doupe, Miller, & Keller, 1939; Dynes, 1932; Levine, 1930; Sears, 1932; West, Neill, & Hardy, 1952). These studies usually compared hypnotic analgesia to a control session on variables such as heart rate and GSR. As Shor (1967) and Barber (1970) have pointed out, however, these studies were fraught with methodological problems such as the lack of controls for order effects, unresponsive physiological measures, unreliable noxious stimuli, and so on. Such problems precluded conclusions regarding suggested analgesia (Barber, 1970; Shor, 1967).

Investigations assessing physiological changes in response to hypnotic analgesia subsequent to 1960 were

generally consistent in their findings. While physiological indices changed in response to painful stimulation, their modification by suggestions for analgesia was small or nonexistent.

For example, Shor (1962) found no differences on six psychophysiological variables (e.g., respiration rate, GSR) assessed after applications of shock across five counterbalanced conditions; (a) baseline, (b) hypnosis only, (c) "voluntary inhibition" of pain, (d) "voluntary inhibition" of pain following a hypnotic induction procedure, and (e) hypnotic analgesia. Shor's subjects had been exposed to the experimental setting prior to the experiment proper in order to minimize their anxiety. Even though the physiological measures were responsive to the painful stimulation per se, none of the conditions differed from one another. Shor attributed this failure to the fact that the subjects were relaxed and, that the relaxation alone had reduced their physiological responsivity.

Complementing Shor's findings, Sutcliffe (1961) reported no difference in the GSR to painful electric shocks in the following three groups of subjects: (a) baseline, (b) hypnotic analgesia, and (c) a simulation condition where subjects were asked to fake hypnosis.

To assess the effects of hypnotic analgesia using a stimulus more analogous to clinical pain than electric

shock, investigators turned to the "cold-pressor" or ice-water pain stimulus (Wolf & Hardy, 1941). Barber and Hahn (1962) selected the 48 most responsive subjects from 192 females who had been administered the Barber Suggestibility Scale and divided them into four groups; (a) hypnotic analgesia, (b) "waking" analgesia, (c) uninstructed condition, and (d) control condition. Control subjects had a hand immersed in water at room temperature for 3 min. Subjects in the remaining treatments had a hand immersed in 2 C water for 3 min. Subjects in the hypnotic analgesia condition were given a brief hypnotic induction procedure followed by suggestions for analgesia, while those in the waking condition were simply instructed to try and think of pleasant situations. Beginning 15 min. prior to stimulation, four physiological variables were monitored; (a) heart rate, (b) GSR, (c) frontalis muscle tension, and (d) respiratory irregularities.

The hypnotic and waking analgesia groups did not differ on any of the physiological measures. Muscle tension and respiratory irregularities were reduced for these two groups compared to the uninstructed group. These three groups in turn did not differ from one another on heart rate or skin resistance level. For these groups heart rate was faster and the GSR lower during than before stimulation.

Questionnaire responses following stimulation indicated that hypnotic and waking analgesia groups did not differ from one another but both groups reported less pain than the uninstructed group. The control group did not rate the stimulation as even uncomfortable.

In a further study Barber and Hahn (1964) divided 48 subjects who had scored in the upper 35% of the Barber Suggestibility Scale into four groups. One group received a hypnotic induction procedure and the other three groups received no induction or special instructions during a baseline test of responsivity to pain. Subjects immersed a hand in water at 2°C for 1 min. None of the groups differed on three physiological measures; all showed an increase in heart rate and frontalis muscle tension and a decrease in skin resistance. None of the groups differed on subjective pain reports. During a second trial, subjects in the hypnosis group and one of the control groups imagined they were receiving the painful stimulus while immersing their hand in water at room temperature. A third group received the actual stimulus again and the fourth group received an innocuous stimulus--water at room temperature. Following their imagined or real stimulation all subjects rated their degree of pain. The hypnotic plus imagine pain group, waking plus imagine pain group and the actual pain group did not differ from each other on the three physiological

measures, but each group had higher mean heart rates and greater frontalis muscle tension than the innocuous-stimulus control group. The hypnotic group's ratings of pain however were as high as the group receiving the actual pain stimulus. Suggestions for analgesia were not assessed in this study.

The notion that subjects exert some cognitive effort in achieving analgesia that has been suggested to them was supported in a study by Hilgard, Macdonald, Marshall, and Morgan (1974). Heart rate and blood pressure were higher just prior to a hypnotic analgesia condition than to a hypnotic nonanalgesia condition. In both conditions high susceptible subjects were about to have their arm placed in ice water for 1 min. These results correspond to those of other studies dealing with the anticipatory changes in autonomic response patterns (e.g., Lacey, 1967) and suggest that heart rate and blood pressure increase in response to cognitive effort (i.e., suggestions for analgesia) but remain relatively stable during the intake of environmental stimuli (i.e., nonanalgesia). They also indicate that physiological measures may not reflect subjects' pain experiences so much as their arousal level.

Barber and Calverley (1969) assessed heart rate, GSR, EMG, and respiratory irregularities in female subjects assigned to the following treatments: (a) no suggestion plus

pain stimulus, (b) suggestions for analgesia plus pain stimulus, (c) distraction (listening to and trying to remember an interesting story) plus pain stimulus, and (d) no pain stimulus - pressure alone. Within each of these treatments half of the subjects received a hypnotic induction procedure and half did not. The pain stimulus involved applying a 2,000 gm. weight to a finger for a period of 1 min (Forgione and Barber, 1971). All subjects, except those in the pressure alone group, were exposed to the pain stimulus twice--once as a baseline and once during treatment. Immediately following the stimulation subjects rated their responses to the stimulus on a 10-point category scale ranging from normal (0) to extremely painful (10).

Over all groups the correlation between pain reports and EMG was .33 during the experimental session and .36 during the baseline session. The correlation between pain reports and GSR was -.41 during the baseline session and -.46 during the experimental session. Heart rate and respiratory irregularities did not vary systematically with pain reports. Differences between groups were not reported on the physiological measures.

The subjective pain reports themselves indicated that suggested analgesia was equally effective in reducing felt pain when presented with or without a hypnotic induction procedure. Similarly, these two groups did not differ from

the two ~~distraction~~ groups (with or without a hypnotic induction procedure) in reductions of reported pain. The hypnotic induction procedure alone group reported greater reductions in pain than the no hypnosis - no suggestion group, and also did not differ from the above four groups.

These results replicate in part the Barber and Hahn (1962) study which found significant correlations between subjective reports of pain and skin resistance (-.46), EMG (.29), respiratory irregularities (.44), and heart rate (.29). In the Barber and Calverley (1969) study, heart rate and respiratory irregularities did not correlate significantly with pain reports. Differences between the two studies may reflect the different pain stimuli used - cold-pressor (Barber & Hahn, 1962) vs. finger pressure (Barber & Calverley, 1969). In neither of these studies however was there a significant correlation between the extent of change on physiological variables following suggestion and the extent of change on verbal report measures following suggestion. Overall, these results are consistent with those of earlier studies summarized by Beecher (1959) which suggest a possible relationship between the experience of pain and a decrease in GSR, and an increase in EMG, heart rate and respiratory irregularities.

An exception to the findings reported thus far comes from a study conducted by Zimbardo, Rapaport and Baron

(1969). These investigators tested three groups of subjects in an experiment designed to control for alternative explanations to a previous cognitive dissonance experiment. Two groups of subjects received extensive pre-experimental "hypnotic training." Subsequently, after baseline electric shock trials (and other tasks concerned with the cognitive dissonance aspects of the study) one of these groups received hypnotic analgesia suggestions. A second group received an hypnotic "achievement-motivation induction" designed to improve performance on a learning task. The third group was simply told to "pretend" that the shocks were not as painful as during the baseline trial. GSR, respiration rate, and category ratings of pain were measured. Respiration rate was inconsistently measured and was therefore equivocal. For both the GSR and category ratings however the hypnotic analgesia group showed decrements from pre- to posttest. This difference did not occur for the other two groups.

One reason for this outcome may lie in the extensive training procedures (up to 12 hrs.) given to these subjects. It is not clear what effect this had on subjects' perceptions of the experiment and their task. Furthermore, shocks were always delivered during a simultaneous word list learning task and hypnotic analgesia was not evaluated separately from suggestions for analgesia alone. Subjects

told to "pretend" do not constitute an appropriate control group. Finally, Zimbardo et al. (1969) did not test their subjects for hypnotic susceptibility. Thus firm conclusions cannot be made on the basis of these data.

Evans and Paul (1970) also assessed the relationship between physiological indices of pain and suggested analgesia. Sixty-four subjects were divided into four groups equated for susceptibility to hypnosis as measured by a modified version of the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A, Shor & Orne, 1962). These groups received (a) a hypnotic induction procedure plus suggestions for analgesia, (b) a hypnotic induction procedure alone, (c) waking self-relaxation instructions plus an analgesia suggestion, and (d) self-relaxation instructions alone. The pain stimulus was immersion of a hand in ice water ($0^{\circ}\text{C} - 2^{\circ}\text{C}$) for 1 min. Heart rate and pulse volume were measured before, during, and after stimulation.

Neither of these measures alone, or in combination, showed any difference in response to treatments, but did show a difference in response to the ice water when compared to a control immersion in water at room temperature. Susceptibility to hypnosis was unrelated to the physiological measures.

At the end of each ice water immersion subjects rated their experience on a 7-point scale ranging from pleasant to painful. These ratings indicated that subjects given suggestions for analgesia reported feeling less pain than subjects not given these suggestions, irrespective of the presence or absence of a hypnotic induction procedure. Susceptibility correlated .46 with the reduction in pain reported by subjects in the suggestion groups, but did not correlate significantly with the pain reports in the no-suggestion groups. Subjective reports of pain were not significantly related to the physiological measures.

In a study designed to assess the efficacy of various types of suggestions on cold pressor pain Johnson (1974) found that changes in physiological variables (pulse rate, blood pulse volume, skin temperature) following suggestions were unrelated to changes in verbal reports. Subjects given general relaxation instructions and those given instructions for imagined numbness and relaxation reduced reported pain more than the uninstructed control subjects. Reductions of reported pain could not have been due to differential changes in skin temperature when the hand was immersed in the cold water (Wolf & Hardy, 1941), since there were no differences between the two groups on this measure.

One further line of investigation into the physiological indices of suggested analgesia was reported by Hilgard and

his associates (Hilgard & Morgan, 1975). Both cold pressor pain and ischemic or tourniquet-exercise pain were used with heart rate and systolic blood pressure as the dependent measures. In a baseline condition, heart rate correlated .33 with subjective pain reports using cold pressor pain, but using ischemic pain the rise in heart rate was negligible. Blood pressure correlated only .16 (nonsignificant) with pain reports of cold pressor pain, but increased significantly as a function of time in ischemic pain (Hilgard & Morgan, 1975). Changes in heart rate and blood pressure were uncorrelated. Suggestions for analgesia did not alter these two physiological indices markedly (Hilgard, Morgan, Lange, Lenox, Macdonald, Marshall, & Sachs, 1974; Hilgard & Morgan, 1975). Lenox (1970) found reductions in heart rate and blood pressure associated with hypnotic analgesia for eight highly trained subjects. A further study failed to replicate this effect (Hilgard & Morgan, 1975). It is important to note that Lenox measured blood pressure relative to a relaxation baseline during which no pain stimulus was administered. He then compared hypnotic analgesia to a waking ischemic pain session in terms of the mean rise in systolic blood pressure from the relaxation baseline. Hilgard and Morgan (1975) point out that when measured this way, they were able to replicate Lenox's findings in their subsequent study. This was not

the case however when a waking pain session and a hypnotic analgesia session were compared directly.

Sachs (1970), using five subjects and a cold pressor pain stimulus, found that blood pressure was lower during a hypnotic analgesia condition than during a hypnotic relaxation condition. Both treatments however were administered consecutively within a 65 sec. session. The first 30 sec. comprised one treatment, followed by 5 sec. transition time and the last 30 sec comprised the second treatment. The order of treatments was counterbalanced between subjects, each subject returning for two sessions. On further analysis his findings held up only when the analgesia condition was the first of the two treatments and the relaxation condition was second. Subjective pain reports (0 to 10 scale) were lower during the analgesia than during the relaxation condition. All subjects were high susceptibles and had been trained over several sessions to respond to suggested analgesia.

Finally, several studies have examined the Electroencephalographic (EEG) correlates of hypnotic analgesia (Karlin, Morgan, & Goldstein, 1980; Karlin, Weinapple, Rochford, & Goldstein, 1979). One of these studies consisted of observing four chronic pain patients under hypnotic analgesia and the results were equivocal (Karlin et al., 1979). The dependent measure consisted of

the proportion of 5-sec epochs during which a R/L amplitude ratio deviated from an overall mean R/L amplitude ratio. In other words, this amplitude integration method expresses numerically the amplitudes cumulated over successive 5 sec epochs in homologous locations in the right and left hemispheres. These are converted to ratios. The ratios are then transformed so that zero is the transformed mean ratio for the entire recording period. Activation in the right hemisphere is then expressed by ratios below zero while activation in the left is expressed by ratios above zero (Goldstein, 1975). Proportions were then calculated expressing relative activation of one or the other hemisphere. No consistent results were found in these four subjects. Moreover no suggestion alone or relaxation control conditions were tested (see Sarbin & Slagle, 1979).

Karlin et al. (1980) used the proportion of 5-sec epochs in which the left hemisphere was more active to measure the effect of cold-pressor stimulation on six medium-high and five high susceptible subjects. High susceptible subjects did not show contralateral activation of EEG activity during single hand immersion under hypnotic analgesia whereas mediums did. Again, no relaxation control condition was used and hypnotic analgesia was not tested independent of suggested analgesia alone. Highs and mediums also received different training sessions. In an effort to convince

mediums. they could experience hypnotic analgesia, these subjects received slightly warmer ($4^{\circ}\text{C} - 5^{\circ}\text{C}$) water for a preliminary hypnotic analgesia session. Furthermore, the EEG findings may have been an artifact of the reduced hand movements of high susceptible subjects.

Endogenous Opioid Peptides and Hypnotic Analgesia

Endogenous opioid peptides were first isolated from brain extracts and on the basis of their pharmacological action were shown to have opiate-like properties. Other peptides were soon discovered leading to the generic term "endorphins" (see Goldstein, 1976; Sherman & Liebeskind, 1980; Snyder, 1980; for reviews). Endogenous opiates have been implicated in a variety of behaviors (e.g., exercise, sex, feeding) and have been found in most of the brain as well as in the cerebro-spinal fluid and blood plasma (e.g., Pert, 1981; Snyder, 1980). The role of endorphins in mediating various types of analgesia responses was inferred largely from the reversal of such responding by naloxone, an opiate antagonist. Using this methodology, three studies have been published to date examining the response of hypnotic analgesia to naloxone (Barber & Mayer, 1977; Frid & Singer, 1979; Goldstein & Hilgard, 1975).

Two of these found a clear lack of effect of naloxone on hypnotic analgesia (Barber & Mayer, 1977; Goldstein & Hilgard, 1975). The third found a reversal of hypnotic

analgesia, but only under conditions of "stress" (Frid & Singer, 1979). The stress manipulation consisted of a memory task in which subjects were to remember three lists of 12 nonsense syllables. Despite testing only 5 subjects in each of their stress and no-stress groups, Frid and Singer report a very large reversal of analgesia for the subjects receiving hypnosis and stress but not for the subjects receiving hypnotic analgesia alone.

The meaningfulness of this finding is in doubt however, because (a) order effects inherent in Frid and Singer's design may have contributed to their effect (stress subjects received five trials of ischemic pain; no-stress subjects received four trials), (b) naloxone alone has been shown to increase, decrease or not affect pain in man (e.g., Buchsbaum, Davis, & Bunney, 1977; El-Sobky, Dostrovsky, & Wall, 1976; Grevert & Goldstein, 1978), and (c) naloxone may act as a hyperalgesic (Henry, 1979). Consequently, a single study showing the naloxone reversal of hypnotic analgesia in five subjects must be viewed with caution and clearly needs replication.

Thus very few studies report significant correlations between reductions in reported pain and changes in physiological indices following suggestions for analgesia (e.g., Finer, 1974; Karlin et al., 1980; Lenox, 1970; Sachs, 1970; Zimbardo et al., 1969). All of these that do report

such correlations are difficult to interpret because of methodological weaknesses.

In sum, the studies reviewed above suggest the following conclusions with respect to psychophysiological indices of suggested analgesia:

1. Changes in "involuntary" physiological responses such as cardiovascular and skin resistance responses occur as a function of response to painful stimuli in laboratory settings and sometimes correlate to a moderate degree with reported pain. It is not clear whether these responses actually reflect the subject's pain or indicate his/her level of arousal.

2. These physiological responses are not modified in any consistent way with suggestions for analgesia and this appears to hold regardless of the presence or absence of a hypnotic induction procedure.

3. Physiological measures usually do not reflect the changes in verbal reports of pain intensity following suggested analgesia.

4. At least in high susceptible subjects anticipatory changes in heart rate and systolic blood pressure occur following a suggestion for analgesia but prior to the administration of a noxious stimulus.

Contemporary research indicates that (a) psychophysiological indices can not be assumed to reflect

accurately felt pain, and (b) that verbal reports are more sensitive indices of experimental conditions than are psychophysiological indices. The emphasis on verbal reports has led to a new and complex series of problems concerning the scaling of pain (e.g., Rollman, 1977). In general these issues are still the subject of some debate in the literature on experimental pain (e.g., Wolff, 1978). Consequently, their resolution will also influence the interpretation of experiments carried out on hypnotic analgesia.

Experimental Studies of Hypnotic Analgesia - Verbal Reports

Studies using verbal reports as a sole, or major index of felt pain can be assessed in terms of two issues; the adequacy of the psychophysical procedures used to assess pain and the manipulation of antecedent variables. The majority of studies assessing the effects of antecedent variables used category scales to scale painfulness and will therefore be discussed under that heading. Hypnotic and suggested analgesia have also been investigated using (a) magnitude estimation, (b) signal detection theory procedures, and (c) functional measurement procedures. Each of these will be discussed in turn below.

Category Ratings

The most common verbal report measure of pain consists of the simple category scale. Subjects are simply asked to partition their experience into equal intervals and to assign each interval a label. Categories are usually labelled as numbers, such as "1" to "10", or as words, such as "low," "medium," or "high" (later given a numerical value by the experimenter). The resulting numbers are then treated as interval scales. The biases inherent in such a procedure are numerous (see Poulton, 1979). For example, the spacing of the stimuli on the physical continuum typically influences the use of different categories. Nevertheless, their ease of administration has led to the use of category ratings in the majority of suggested analgesia studies. What in fact is being reduced when category ratings decrease will be discussed below under theories of suggested analgesia.

The manipulation of antecedent variables. The possible interactions of (a) the presence or absence of a hypnotic induction procedure, (b) the presence or absence of suggestions for analgesia, and (c) the pretested level of hypnotic susceptibility have only recently been assessed. Most studies of hypnotic analgesia have examined some but not all of these variables in differing experimental designs.

Between-subjects experiments. As noted above, studies done by Barber and Hahn (1962), Barber and Calverley (1969), and Evans and Paul (1970) found no differences in pain reports between groups given suggestions for analgesia with or without a hypnotic induction procedure. In two of these studies (Barber & Hahn, 1962; Evans & Paul, 1970) subjects in the two suggestion groups reported less pain than those in the two comparable no-suggestion groups. Evans and Paul (1970) reported a correlation of .46 between reductions in rated pain and hypnotic susceptibility in the two suggestion groups, and no relationship for these same measures in the two no-suggestion groups. Barber and Calverley (1969) and Evans and Paul (1970) used unselected subjects, whereas Barber and Hahn (1962) selected the upper quartile of subjects from a larger sample that was administered a susceptibility scale.

In a study by Spanos, Barber, and Lang (1974), 80 unselected subjects were pretested on the Forgione-Barber pain stimulator (Forgione & Barber, 1971) for 60 sec. Subjects rated their sensations from normal to extremely painful on a 10-point scale, for both the first and last 30 sec of stimulation. Following the pretest, subjects were randomly assigned to one of eight groups in a 2 X 2 X 2 design and retested on the stimulator. The three between-subjects variables were (a) the presence or absence

of a hypnotic induction procedure, (b) the presence or absence of suggested analgesia, and (c) the presence or absence of demands for honesty. Demands for honesty were fashioned after those of Bowers (1967) who argued that subjects' post-experimental verbal reports may be more accurate if they are obtained by a person other than the experimenter and this person admonishes subjects to be honest in their reporting. The results indicated that subjects given a suggestion for analgesia showed a reduction in pain regardless of the presence or absence of hypnotic induction procedure or demands for honesty. Neither of the latter two variables had any effect on pain reports.

To assess directly the relationship between susceptibility to hypnosis as measured on a pretest, hypnotic induction procedures and suggestions for analgesia, Spanos, Radtke-Bodorik, Ferguson, and Jones (1979) divided 96 subjects into high, medium and low susceptibles on the basis of scores on the HGSHS:A (Shor & Orne, 1962). All subjects were given a baseline test with 60 sec of cold pressor pain. Subjects rated their pain from 0 (no pain) to 10 (excruciating pain) at 30 and 60 sec. Following the pretest subjects were randomly assigned to four treatments within each susceptibility level; (a) hypnotic induction procedure alone, (b) hypnotic induction procedure plus suggestions for analgesia, (c) suggestions for analgesia

alone, and (d) no treatment control. Subjects were again tested with 60 sec. of cold-pressor pain and rated their pain as before.

Subjects given a suggestion reported significantly less pain from the first to the second trial compared to subjects not given a suggestion. High and medium susceptible subjects, but not low susceptible subjects, significantly reduced their reported pain on the second trial compared to the first trial. The hypnosis variable had no effect on reported pain. These findings replicate the results of those studies indicating that reductions in reported pain are correlated with pretested susceptibility level and that suggestions for analgesia produce decrements in reported pain (e.g., Barber & Hahn, 1962; Evans & Paul, 1970; Hilgard, 1969; Spanos, et al., 1974). The fact that the pain reduction was independent of the administration of a hypnotic induction procedure is consonant with the results of previous studies that have used either unselected subjects (Evans & Paul, 1970; Spanos, et al., 1974) or selected subjects (Barber & Hahn, 1962) in a between-subjects design.²

Within-subjects experiments. A number of studies have assessed the effects of hypnosis, suggestions, and susceptibility using within-subjects designs. Two studies using high susceptible females found that increases in

threshold and tolerance were greatest when subjects were given suggestions for analgesia following a hypnotic induction procedure, compared to "waking" imagery and relaxation control groups, respectively (Greene & Reyher, 1972; Stacher, Schuster, Baue, Lahoda, & Schulze, 1975).

Hilgard and his associates have also employed within-subjects designs to address the issue of the relationship between pretested susceptibility to hypnosis, suggested analgesia and the presence or absence of a hypnotic induction procedure (Hilgard & Hilgard, 1975).³

The results of these studies may be summarized as follows:

1. Compared to a baseline, high susceptible subjects reported reductions in pain during hypnotic analgesia with ischemic pain as well as with cold pressor pain (Lenox, 1970; Sachs, 1970).

2. Hilgard (1969) divided subjects into those who scored high or low on a susceptibility pretest. Each subject was then exposed to (a) a baseline with cold pressor pain, (b) a hypnotic induction procedure without suggestions for analgesia, and (c) a hypnotic induction procedure plus suggestions for analgesia. Sessions (b) and (c) were randomized. High susceptible subjects showed greater reductions in reported pain when given hypnosis plus suggestions than when given hypnosis alone. Low susceptibles did not differ across sessions.

3. Hilgard, Morgan, and Macdonald (1975) compared waking suggested analgesia with hypnotic analgesia in high susceptible subjects when these sessions were run on different days in a within-subjects design. The results were not reported at the time since they were deemed tangential to the main findings of the report. Hilgard (1977) discusses these results and reports that during hypnotic analgesia, pain was reduced from 14.2 to 4.2 compared to a baseline. That is, the pain was 30% of the baseline pain. When given suggestions for analgesia alone, the reduction only went from 14.2 to 8.5 or to 60% of the baseline pain. Hilgard and Hilgard (1975), briefly describe an unpublished study that appears to refer to these data. Subjects were exhorted by some means (not reported) to "stay out of trance." The effect of this demand on subjects' perceptions of the experiment is not known. Furthermore, it has been shown in several studies that high susceptible subjects can, if they so choose, withhold from experiencing the suggested events that follow a hypnotic induction procedure (e.g., Spanos, Radtke-Bodorik, & Stam, 1980, Experiment 3). It is likely then that Hilgard's procedure, while not only unnecessary, may have conveyed to the subjects the demand that they should feel less pain during the hypnotic analgesia condition than during the "waking" analgesia condition.

4. Hilgard, Macdonald, Morgan, and Johnson (1978) compared 12 high susceptible subjects to 12 low susceptibles asked to fake hypnosis (that is, to simulate; Orne, 1979). Subjects were tested in a within-subjects design using 30 sec of cold-pressor pain. All subjects received the same order of events; (a) baseline pain trial, (b) "waking" suggested analgesia, (c) hypnosis - no suggested analgesia, and (d) hypnosis plus suggested analgesia. Hypnotic subjects reported significantly less pain than simulating subjects during hypnosis alone and hypnotic analgesia trials. Notably hypnotic subjects but not simulator reported less pain during hypnotic analgesia than waking analgesia.

In summary, studies using within-subjects designs where each subject received all experimental conditions, have found that high susceptibles reported greater decrements in pain when suggestions for analgesia were preceded by a hypnotic induction procedure than when suggestions were presented alone. Subjects in these studies were aware of the order of testing or else were sophisticated hypnotic subjects who had participated in previous experiments. It is possible that these subjects formed beliefs regarding the outcome of such a procedure and responded accordingly. These results then may indicate that the within-subjects design has associated with it carry-over effects that

influence the outcome when the same subjects are tested with and without a hypnotic induction procedure (Barber, 1969; Orne, 1962). The within-subjects design has been criticized for this reason in other areas of psychology (Greenwald, 1976; Grice, 1966; Poulton, 1973; 1974; Rothstein, 1974).⁴

Hilgard, on the other hand, has interpreted these data to indicate that (a) between-subjects designs may be missing a small but significant treatment effect because of wide individual differences within treatments and (b) that high susceptible subjects may "slip into hypnosis" when given waking analgesia suggestions (Crawford, Macdonald, & Hilgard, 1979; Hilgard, 1977; 1979; Hilgard & Hilgard, 1975). As noted above, Hilgard's test of the notion that subjects inadvertently slip into hypnosis is equivocal. Furthermore this does not explain the absence of such an effect in within-subjects designs where hypnotic analgesia is a superior analgesic to waking analgesia.

In order to test the notion that hypnotic analgesia leads to less reported pain than waking analgesia in within-subjects designs because of carry-over effects, Stam and Spanos (1980) tested four groups of high susceptible subjects. Each group was exposed to three 60-sec. immersions of ice water. The first immersion was a baseline trial and was identical for all groups. The remaining two immersions were associated with hypnotic analgesia, waking

analgesia, or no treatment. The first group underwent a treatment sequence similar to that used by Hilgard et al. (1978); that is, waking analgesia for the second immersion followed by hypnotic analgesia for the last immersion (BSH). Subjects in the second group received two trials of suggested analgesia (BSS).. The third group received hypnotic analgesia for the second immersion and waking analgesia for the third immersion (BHS). They were told however that the experimenter expected waking analgesia to be more effective than hypnotic analgesia. Finally, the fourth group served as a no-treatment control group and received three identical immersions (BBB). After the baseline immersion subjects in each group were informed of the order of events to follow.

Magnitude estimates (obtained every 5 sec.) and category ratings (obtained at 60 sec.) of pain both indicated that (a) subjects in group BSH reduced pain somewhat with waking analgesia but more with hypnotic analgesia, (b) on immersion three subjects in group BSS reduced pain to the same low level as group BSH subjects, (c) group BHS subjects reduced their pain very little with either treatment after being told that hypnotic analgesia was less effective than waking analgesia, and (d) control (BBB) subjects did not change at all across immersions. Furthermore, subjects' coping cognitions were highly related to their category ratings of

pain. Subjects were posttested by a second experimenter under "demands for honesty" (Bowers, 1967) and almost all stated they reported pain "exactly" as they felt it.

In short, hypnotic analgesia was more effective, less effective or equally as effective as waking analgesia depending upon the expectations induced by varying treatment order. Thus there is nothing intrinsically superior in the action of hypnotic analgesia over and above that found in suggested analgesia.

The superiority of hypnotic analgesia to suggested analgesia alone is obtained only under conditions designed to maximize expectations. That is, it occurs when subjects are aware that their performance is being compared under the two conditions. Stam and Spanos (1980) interpreted these findings as indicating that subjects (a) modify their experience to conform to expectations inherent in within-subjects designs, (b) modify their cognitions in accord with these expectations, and (c) still define their responding as honest and exact. Subjects knowing they are to be hypnotized have, on other occasions, been shown to refrain from performing maximally during a waking condition in order to enhance their performance during hypnotic testing (e.g., Spanos, McPeake, & Carter, 1973; Zamansky, Scharf, & Brightbill, 1964).

Although a hypnotic induction procedure does not reduce reported pain above and beyond suggestions for analgesia alone, there are investigators who believe that hypnosis cannot be defined solely on the basis of antecedent variables (Orne, 1977). Rather, hypnosis is defined in terms of alterations in subjective experience such as those found when subjects are asked if they have been hypnotized (Orne, 1977).

Besides the obvious circularity of such a definition it does not acknowledge the possibility that phenomenological reports - for example of being hypnotized - are themselves the result of a manipulation of antecedent variables (Radtke & Spanos, 1981). To make them the final arbiter of the construct hypnosis renders the latter largely unnecessary. For example, assume two highly susceptible subjects were each given a suggestion for analgesia, one with a hypnotic induction and the other without. Each immersed a hand in ice-water for 60 sec before and again after their respective treatments. Both of these subjects would probably report less pain following the second immersion and they would likely not differ in the extent to which they reported less pain. According to Orne (1977), both of these subjects would be hypnotized since they both responded "to suggestions designed to elicit hypnotic phenomena" (p.16). In fact, both subjects would, upon further query, likely

attest to the subjective reality of their reduced pain. If, however, we can distinguish between these subjects and others (who do not report reduced pain) on the basis of their reported cognitions, saying they are hypnotized does not give us any additional information.

Finally, all investigators agree that, at least for category ratings, reductions in reported pain are correlated with hypnotic susceptibility. Regardless of the presence or absence of a hypnotic induction procedure, suggestions for analgesia are more effective with high susceptibles than with low susceptibles.

Magnitude Estimation

An alternative procedure to category scaling was proposed by S. S. Stevens (e.g., 1957). Stevens argued that for prothetic continua, category ratings of stimulus intensity are nonlinearly related to the actual intensity of the stimulus. One reason for this is that at the lower end of category scales discrimination is better than at the upper end. Magnitude estimation (ME) does not in general limit the subject to a predetermined number of categories. Rather subjects are asked to assign a number of their own choosing to the first stimulus (or are given a standard) and then assign numbers in proportion to the remaining stimuli.

A large number of experiments have shown that ME data are well fit by power functions of the sort

$$\psi = b\phi^a \quad (1)$$

where ψ refers to the reported sensation magnitude, ϕ refers to stimulus intensity, and a the exponent (or slope of the log-linearized function) and b is an arbitrary constant (Stevens, 1961; 1975). When applied to the scaling of pain following suggested analgesia, various outcomes are possible. These include a possible change in the rate of pain growth as reflected in the exponent, a , or a change in the constant, b . Changes in b may indicate that the rate of change in pain is constant while the subjects have shifted their response scale units.

In an attempt to determine the psychophysics of cold pressor pain using ME, Stam, Petrusic and Spanos (1981) tested subjects in two experiments. The first experiment used three immersions of 60 sec duration and the second experiment used two immersions. Subjects gave magnitude estimates every 5 sec. In the first experiment subjects were not provided with a standard while in the second experiment subjects were provided with a 10 sec immersion standard and a modulus of "100."

Linear, power, or additive constant functions fitted the data almost equally well. There appear to be no advantages to using power functions over linear functions to describe cold pressor pain data. Furthermore, as Stam et al. (1981) pointed out, magnitude estimates of cold pressor pain may be

affected by both an intensity-duration confound and the presentation of stimulus intensities in a single, ascending order. It is not clear then what power functions mean when calculated on the basis of these data.

Neither exponents (a), nor constants (b), derived from Equation 1 and calculated separately for each subject, differentiated between suggested analgesia, hypnotic analgesia and control conditions. These same data however when treated as scores in a factorial design and subjected to analyses of variance, did show that suggestion treatments were reported as less painful than nonsuggestion treatments (Spanos, Stam, & Brazil, in press; Stam & Spanos, 1980). Exponents may have failed to differentiate between treatments because the individual differences in exponents were relatively large. Alternatively, the rate of change in reported pain may not be affected by suggestions even though subjects report less pain numerically. These findings underscore the difficulties involved in using ME procedures to calculate exponents for cold pressor pain as well as in searching for so-called characteristic exponents.

In a further study of ME and cold pressor pain Spanos, Jones, Brown and Horner (1981) gave subjects a 10 sec standard and modulus of "100". They were then instructed to keep their arm in the water for as long as possible beyond the first minute. Subjects then received a second immersion

and prior to it half the subjects received a suggestion for analgesia. Subjects gave magnitude estimates every 10 sec. Results indicated that linear fits were generally superior to power function fits and that the exponents did not differentiate suggestion from no-suggestion subjects, replicating Stam et al. (1981). Subjects who were classified on the basis of their post-experimental testimony as "catastrophizers" showed a steeper slope than subjects classified as "copers." Furthermore, subjects with a high tolerance (>241 sec) had a lower slope than subjects with a low tolerance. In short, both of these studies demonstrate that linearity characterizes magnitude estimates of cold pressor pain equally well if not better than power functions.

Hilgard and his associates have also carried out a series of studies attempting to determine the psychophysics of hypnotic analgesia with cold pressor pain and ischemic pain (reviewed in Hilgard & Hilgard, 1975). In the cold pressor pain experiments to be discussed here subjects typically reported pain every 5 sec during a 60 sec immersion.

In an early report (Hilgard, Cooper, Lenox, Morgan, & Voevodsky, 1967) the pain reporting scale simply ranged from 1 to 10, where 10 was defined as "so painful that the subject could no longer keep his hand in the water" (p.

507). Hilgard argued that the use of a category scale creates a "ceiling" effect where the subject reports increasingly smaller increments in pain, while the actual pain may still be increasing. Thus the scale was redefined as one where 10 became the value "at which the subject would very much like to remove his hand from the water," but the subject was exhorted to "endure the pain beyond this and to count beyond 10 as the pain continued to mount" (Hilgard, Ruch, Lange, Lenox, Morgan, & Sachs, 1974).

Although Hilgard (1969) refers to this procedure as ME it is in fact a combination of category scaling and magnitude estimation procedures. In his instructions to the subject prior to stimulation, Hilgard (1972) states "I would like you to tell me the pain or discomfort that you are experiencing by stating some number between 0 and 10 If you are at pain state 10 and I ask you to leave it in a little longer, then if the pain continues to increase just go ahead and continue to count past 10." The subject is not given a standard nor is an iterative procedure used to allow the subject to choose his own modulus. Rather the subject is asked to equipartition the subjective pain continuum, with the upper end of the scale left ambiguous.

Despite these marked departures from ME, Hilgard et al. (1974) reported a linear function when pain reports were plotted logarithmically against either (a) time in ice

water, (b) temperature of the ice water, or (c) time in ischemia. Before accepting these findings however a closer observation of the data is warranted. The data provided by Hilgard et al. (1974) in the form of MES deals with only the first 30 sec. of immersion in ice water. Furthermore, when fitting the lines by least squares, the initial 5 sec. values were not used. In other words, the data that went into the plotted lines included only the five reports from the 10 sec to 30 sec interval. Hilgard et al. (1974) plotted separately the data obtained on 54 subjects as the logarithm of pain reports against the logarithm of time in ice water for the full minute immersion. Only 11 subjects' data when averaged and plotted produced a straight line from 0 sec. to 60 sec. duration. The data from the remaining subjects (43) produced either "two-slope" functions with an inflection at 10 on the pain scale or completely irregular curves. Despite these obvious departures from linearity, the authors used the data obtained from the 10 sec. to 30 sec. interval to calculate the slopes and intercepts of the line resulting when pain reports are plotted against time on a log-log graph. Not surprisingly since very few subjects reached the "critical" 10 on the pain scale by 30 sec., a straight line resulted for these five data points averaged over all subjects. Hypnotic analgesia, tested during a separate session for these 54 subjects, resulted in

a change in intercept from (a) 1.03 to .62 for high susceptibles, (b) 1.23 to 1.05 for medium susceptibles, and (c) 1.13 to 1.20 for low susceptible subjects. The slopes of the lines remained relatively constant across sessions (.71 for highs during both sessions, .67 and .65 for mediums and .64 and .59 for low susceptibles). Hilgard et al. (1974) interpreted these findings to mean that the rate of growth in pain remains unaltered but for high and medium susceptible subjects given hypnotic analgesia, the magnitude of pain is reduced by a constant amount. This is seen by Hilgard et al. (1974) as akin to placing a filter between the input and the response. The utility of such an analogy is questionable.

Since these differences in the log-log curves between conditions were found in the intercepts but not in the slopes, Hilgard et al. (1974) computed ratio scores in which subjective reports were expressed as a fraction of each other. That is, if $\psi = b\phi^a$ and $\psi_1 = b_1\phi^a$ and $\psi_2 = b_2\phi^a$, where b is constant, then $\psi/\psi_2 = b_1/b_2$. These ratio scores were then used to compare changes in reported pain over the 30-sec. interval between baseline immersions and hypnotic analgesia as well as between differing temperatures of ice water (0°C, 5°C, and 10°C).

The results of the experiments conducted by Hilgard and his associates may be summarized as follows:

1. The correlations between reductions in reported pain during hypnotic analgesia with ratio scores at 30 sec. and hypnotic susceptibility was .48.

2. Compared to a baseline session, high susceptible subjects reported significantly less pain at 30 sec. than medium or low susceptible subjects; medium and low susceptible subjects did not differ significantly. The difference between high and medium susceptible subjects was not significant when difference scores, as opposed to ratio scores, were used. Sixty-seven per cent of the high susceptible subjects, but only 17% and 13% of the medium and low susceptible subjects, respectively, reduced reported pain 33% or more.

3. Using difference scores, at 30 sec. pain was judged greater in water at 0°C than at 5°C, but both were judged more painful than 10°C water. Ratings in 15°C water were obtained from a different set of subjects and were not included in the analysis. In another report of the same data, Hilgard (1975) stated that changes in water temperature for 0°C, 5°C, and 10°C are associated with a change in intercept and not slope, therefore indicating that the pain is "filtered" in such a manner as to produce a constant ratio of the maximum pain between each temperature as the pain increased" (p. 215). When 15°C was included however Hilgard et al. (1974) stated that "differences in

reported pain at various temperatures are chiefly changes in the slope, a , especially with extreme values" (p. 29).

In summary, Hilgard's data are difficult to interpret. Furthermore, the linearity displayed by magnitude estimates of cold pressor pain (Spanos, et al., 1981; Stam et al., 1981) obviates the need to compute ratio or difference scores as Hilgard et al. (1974) have done.

Tursky and his colleagues have argued that ME is a scale of sensitivity while ~~the~~ threshold and tolerance measured aversiveness. Their evidence is based on the modulation of the latter (but not the former) by variables such as ethnic background and acupuncture when electric shock was used as a pain stimulus (e.g., Goldberger & Tursky, 1976; Tursky, 1976). That this is not the case with ME of cold pressor pain is illustrated by the finding that ME data subjected to analysis of variance does exhibit suggestion effects whereas exponents derived from the same data do not (Stam et al., 1981). Thus, the rate of change (but not the magnitude) of reported pain appears unaffected by suggested analgesia.

The notion that exponents reflect some intrinsic resolution power of the nervous system has been debated for some time (e.g., Anderson, 1970; Krantz, 1972; Luce & Galanter, 1963; Poulton, 1979; Shepard, 1978). There may be no a priori reason why an analgesic should affect one parameter but not another in ME. In this respect, ME

presents no real alternative to category ratings of reported pain. Elsewhere it has been demonstrated that the nonlinear relation between category scales and magnitude scales is probably an artifact of the restricted range of response categories in category scales (Gibson & Tomko, 1972).

Signal Detection Theory

Signal Detection Theory methodology allows the experimenter to calculate statistics which under specified circumstances can be taken to yield independent indices of sensitivity and decisional processes or "bias" (Green & Swets, 1966). According to the theory a subject in a SDT experiment behaves as a detection system having to decide (in the simplest case) between the presence or absence of a weak signal imbedded in "noise." In one variant of this procedure subjects make responses on a rating scale. In the rating scale task subjects respond to a single trial with a scale value reflecting their confidence in their decision on that trial. The scale might range from "positive noise" to "positive signal."

This latter procedure has been adapted by pain researchers (see Rollman, 1977 for a review). In short, subjects are presented with a discrete stimulus such as an electric shock and asked to rate it on a scale ranging from, for example, "1" to "5." These ratings are then transformed to proportions and used to estimate traditional SDT parameters of "sensitivity" and "response bias."

Two published experiments have evaluated the effects of "suggestion" on reported pain using a signal detection theory (SDT) methodology (e.g., Green & Swets, 1966). Clark and Goodman (1974), using radiant heat pain, gave subjects instructions to lower or raise their criterion for reporting pain. Likewise, Dougher (1979) gave subjects high or low in anxiety instructions to inhibit or facilitate pain reports to focal pressure pain. Clearly, these instructions were very different from typically used suggestions which ask subjects to engage in cognitive activities to reduce their pain.

Both Clark and Goodman (1974) and Dougher (1979) report that their manipulations changed response bias but not sensitivity. That is, subjects were still able to discriminate between intensity levels of the painful stimulus but raised their criteria for reporting pain. This led Clark and Goodman (1974) to argue that "suggestion had failed to produce an analgesia effect" (p.364).

That this interpretation is controversial is shown by the debate that surrounds the use of SDT measurement of pain (see Chapman, 1977; Jones, 1979; Rollman, 1977; 1979). Essentially this debate has focussed on the notion that alterations in pain are not equivalent to alterations in discrimination. What measures are in fact affected by any given analgesic is an experimental question (Jones, 1979).

Subjects' reduced pain ratings can not be considered simply "bias" if only because the subject cannot be said to be "wrong" if he reports less pain following an analgesic. That is, in calculating sensitivity and bias parameters, there is no a priori reason for assuming an analgesic is not in some sense a genuine analgesic if it does not change the sensitivity parameter (Jones, 1979; Rollman, 1977). Thus the meaning of Clark and Goodman's (1974) and Dougher's (1979) results must be considered equivocal. Further clarification of the role suggested analgesia plays in modifying various SDT parameters is still needed.

Planaş (1981) gave subjects high or low in hypnotic susceptibility the task of discriminating between members of consecutively presented pairs of shocks. The three pairs consisted of shocks of low, medium or high intensity. Subjects discriminated shocks in two sessions. In their second session subjects received either a hypnotic induction procedure, an induction with suggestions for analgesia, suggestions alone or no treatment.

Subjects who rated the high intensity pair as very painful (on a category rating task performed prior to the discrimination task) were less able to discriminate the high intensity pair than the medium pair. Discrimination accuracy was also lowered by the presence of any treatment variables. Thus, high painfulness may result in decreased

discrimination through a reduction in attentive capacities utilized for the discrimination task. Likewise, the type of cognitions elicited by the treatments (such as imagining events inconsistent with the pain) may have resulted in deflecting attention from the task of discriminating. Finally, following suggestions for analgesia subjects rated their pain as less on the category scale, a finding consistent with those studies that have manipulated antecedent variables.

Functional Measurement

According to Anderson (e.g., 1970; 1977) psychological scales are derivative from the stimulus integration function that governs the synthesis of separate stimulus components into a unitary perception or judgement. In general, functional measurement depends on the empirical validity of this integration function (Anderson, 1977). A large number of studies have provided support for algebraic models of stimulus integration pointing to substantial model generality (Anderson, 1974).

In an application of Anderson's functional measurement approach Jones (1980) fit an averaging model to average intensity ratings of painful electric shock. Jones (1980) argued that the psychophysical rule derived from this averaging function was essentially a power function with an exponent of 2. Nonpainful stimuli ratings resulted in a

linear function. Thus, one way an analgesic could be tested is by examining the derived psychophysical function before or after an analgesic.

Jones, Spanos and Anuza (1981) attempted such a test with hypnotic analgesia by assigning high susceptible subjects to three treatments. These consisted of hypnotic analgesia, suggested analgesia or a control condition. Subjects then averaged the painfulness of pairs of shocks ranging from 1mA to 4mA.

Relative to the control condition, neither procedure brought about any change in the integration process as indicated by the form of the psychophysical function and the relative weighting given each stimulus (the second of a pair of stimuli being weighted more heavily in non-painful processing). As with ME however (Stam et al., 1981) subjects gave lower average pain ratings in the two suggestion conditions than in the control conditions while the rate of pain growth remained constant across conditions.

One of the assumptions underlying much of the effort to determine the psychophysics of pain has been the fundamental equation of pain with nociception. This has led to confusion surrounding the usage of the term analgesia. Typically analgesia refers to the absence of pain during noxious stimulation. However, accepting a definition of pain that emphasizes its function as a psychological state

(even though associated with actual or potential tissue damage) implies that nociception may occur in the absence of pain, and vice versa. That is, activation of nociceptive pathways by noxious stimuli is not equated with pain even though pain is most often associated with some physical cause.

Defining pain in terms of nociception leads investigators to posit such processes as "affective and reactive components of pain" (e.g., Kruger & Kroin, 1978). The problem then becomes one of finding which "component" is affected by what analgesic (e.g., Gracely, McGrath, & Dubner, 1978). The meaningfulness of such an investigation is dubious.

It is probably more parsimonious to assume that pain is a psychological phenomenon closely associated with nociception but also influenced by a host of other, psychological variables. In this respect pain is more like the dysaesthesiae (e.g., nausea) than a separate sensory system such as touch (see Wall, 1979).

The implications of this point for psychophysical investigations are clear. Nociception, as measured by sensory integration functions for example, may remain unchanged while pain is reduced. Some support for this notion is found in studies that have indicated reduced pain but unaltered psychophysical indices following morphine

administration (e.g., Beecher, 1959; Chapman, Dingman, & Ginzberg, 1965). For example, Bromm and Seide (1982) administered electric shock to subjects who had received tilidine (an oral narcotic analgesic), prazepam (a tranquilizer) and a placebo on separate trials. Although tilidine resulted in reduced pain reports, reduced withdrawal reflexes and reduced skin resistance reactions, the exponents of fitted power functions remained unchanged from baseline. The constants or units of the subjects' scale however did diminish significantly following the administration of tilidine.

Theories of Hypnotic Analgesia

Special Processes

Although there have been some notable exceptions, investigators have traditionally posited special psychological processes to account for hypnotic behaviors (see Sarbin, 1962; Spanos, in press). Constructs such as hypnotic state, trance, or dissociation have been invoked as instrumental factors in eliciting hypnotic behaviors (Barber, 1969).

Recently, Hilgard (e.g., 1973; 1977; 1979) has reintroduced the notion of dissociation as an explanation of hypnotic analgesia in particular and other, mainly hypnotic, phenomena in general. According to Hilgard, events can

become functionally separated from awareness while nevertheless continuing to influence performance. In hypnotic analgesia, a "part" of the individual's "cognitive system" is supposedly registering and processing pain while the person is hypnotically analgesic. This pain is not being processed at a conscious level. Instead, Hilgard (1978) states that "the concealed or covert cognitive apparatus can be described as a 'hidden observer' who knows things that the overt cognitive apparatus in hypnotic analgesia is not aware of" (p. 231, italics in original).

Hilgard's experimental support for this hypothesis consists of an experiment wherein high susceptible subjects received several trials of painful stimulation (cold pressor or ischemic). The first trial consisted of a baseline trial and second or subsequent trials consisted of hypnotic analgesia trials. Subjects were also instructed however that there was a hidden part of them that was aware of experiences that their hypnotized part was unaware of. The instructions were very explicit:

When I speak with this hidden part of you, I will place my hand on your shoulder. . . . When I place my hand on your shoulder I will be in communication with this hidden part, and we can talk together; but the hypnotized part to which I am talking now, will not know that you are talking to me. It will not know what you

are saying, or even that you are talking. When I then remove my hand from your shoulder you will be back in the hypnotic state you are now in, and you will not know what you said or even that you were talking to me. . . .

(Hilgard, Hilgard, Macdonald, Morgan, & Johnson, 1978, p. 658).

Subjects were also trained in performing two tasks, supposedly simultaneously, with one out of consciousness (e.g., colour naming and finger tapping). When subjects were exposed to the pain stimulus under hypnotic analgesia they gave "overt" or spoken pain reports for that pain felt by their "hypnotized part" and "covert" or "hidden reports" (tapped out) for the pain felt by their "hidden part."

Under these conditions, Hilgard (e.g., Knox, Morgan, & Hilgard, 1974; Hilgard et al., 1978; Hilgard, Morgan, & Macdonald, 1975) has reported that hidden reports of pain are largely equivalent, or close to, baseline pain reports while both of these are substantially greater than hypnotic, overt, pain reports. Hilgard has taken this as evidence for the existence of dissociative processes brought about by hypnosis: Hidden reports are a function of dissociation and not suggestion.

Aside from the conceptual confusion created by Hilgard's language (Coe & Sarbin, 1977; Margolis & Margolis, 1979), there is also experimental evidence that vitiates an

explanation based on dissociated states. Spanos and Hewitt (1980) tested two groups of high susceptible subjects using hidden observer instructions. One group received instructions and training identical to Hilgard et al.'s (1975). These subjects were told in part that their hidden observer was more aware than their hypnotized self and would thus experience greater pain. The second group was told that their hidden selves were so deeply hidden, they would be even less aware of pain than their hypnotized parts.

The results indicated that subjects responded in accordance with these expectancies. They reported more or less "hidden" pain depending on whether they were told their hidden observers felt more or less pain. Consequently, Spanos & Hewitt (1980) argued that the hidden observer is an experimental creation, shaped and maintained by the experimental procedures. Thus so called hidden reports do not reflect the intrinsic characteristics of dissociated states.

Negative Hallucinations

Orne (e.g., 1974; 1980) has suggested that hypnotic analgesia may be the result of two different mechanisms. For most individuals this consists of the "placebo components" of hypnosis: The expectation and anticipation of relief, the redefinition of pain as less intense and the

presence of a presumably competent individual such as a physician. For a small minority however pain is reduced by a form of "negative somesthetic hallucinations." Thus subjects do not feel pain because supposedly they "hallucinate" that the pain is not there. Orne does not specify the processes involved. In fact, it is not clear what a "negative hallucination" is and how this process differs from a mechanism such as distraction. Orne (1974) only states that "under some circumstances, this may occur spontaneously (for example, the soldier who is unaware of being shot until after the battle is over)" (p. 569).

Orne is also not clear as to the relationship between negative hallucinations and Hilgard's neodissociation theory. Thus he states that,

. . . even though the patient reports feeling comfortable, may look comfortable, and will subsequently insist that he was comfortable at the time of surgery, there remains an appreciation of pain at another level.

. . . This, as well as many of the other paradoxes associated with hypnosis, ought not to lead us to dismiss the phenomenon either as trivial or unscientific. Rather, it should cause us to question many of our more general assumptions about the nature of pain (1974, 571 - 572).

It is clear that Orne's attempt to accomodate dissociation/theory to a negative hallucination position has led him into a theoretical cul-de-sac wherein he must conclude by discussing the "paradoxes" of hypnosis.

Cognitive Activity

An alternative theoretical approach to hypnotic analgesia has been to focus on subjects' cognitions. A large number of studies have demonstrated the pivotal role played by peoples' attitudes and cognitions in the manner in which they cope with pain (e.g., Barber & Cooper, 1972; Blitz & Dinnerstein, 1968; Beers & Karoly, 1979; Bobey & Davidson, 1970; Chaves & Barber, 1974; Chaves & Doney, 1976; Chaves & Scott, 1979; Girodo & Wood, 1979; Kanfer & Goldfoot, 1966; Klepac, Hauge, Dowling, & McDonald, 1981; Hackett & Horan, 1980; Horan & Dellinger, 1974; Johnson, 1974; Rosenbaum, 1980; Scott & Barber, 1977; Scott & Leonard, 1978; Spanos, Horton, & Chaves, 1975; Spanos et al., 1979; Worthington, 1978). In general these studies have shown that subjects given various coping strategies report significantly less pain and show heightened thresholds and/or tolerances for pain than do control subjects.

It is also clear however that subjects do not always engage in the suggested cognitions provided. A number of

studies have indicated that some subjects may either (a) fail to use the suggested strategies, (b) devise and utilize strategies of their own, or (c) focus on and exaggerate the negative aspects of the situation (i.e., catastrophize). These studies have also demonstrated that the amount of pain subjects report and the extent of the pain reduction following treatments is related to their ongoing cognitive activity (Barber & Cooper, 1972; Scott, 1978; Scott & Barber, 1977; Spanos, Brown, Jones, & Horner, 1981; Spanos, Jones, Brown, & Horner, 1980; Spanos et al., 1979; Spanos, Stam, & Brazil, in press; Stam & Spanos, 1980).

For example, Spanos, Stam, & Brazil (in press) found that subjects' scores on indices of coping and catastrophizing predicted 34% of the variance in pretreatment pain ratings. Spanos, Brown, et al. (1981) found that suggestions for analgesia were predominantly associated with reductions in reported pain only when it transformed pretest catastrophizers into posttest copers. Subjects who were already coping during the pretest immersion did not report pain reductions following the posttest. Furthermore, subjects who continued to report catastrophizing ideation, despite receiving suggestions for analgesia, reported the same high levels of pain on both immersions.

Since the same cognitive processes appear to correlate with reported pain reductions in hypnotic and nonhypnotic treatment groups, "dissociation" or "trance" accounts are probably unnecessary to account for reductions in reported pain following suggested analgesia (Spanos et al., 1979; Stam & Spanos, 1980). However, in order to account for the superiority of high susceptible subjects in reducing reported pain, it would have to be demonstrated that these subjects have a greater tendency to employ coping cognitions than low susceptible subjects. This was in fact demonstrated by Spanos et al. (1979) and is consistent with the notion that high susceptible subjects have a proclivity for becoming absorbed in imaginative activities (e.g., Barber, Spanos, & Chaves, 1974; J. Hilgard, 1979). Therefore, suggestions for analgesia, which typically instruct subjects to engage in various imaginative or distracting activities, are more likely to be successful with high susceptibles.

The focus on the cognitive correlates of suggested analgesia eliminates the need to propose special process explanations of their reported success. Nevertheless, it does not explain why suggestions for analgesia are effective in reducing reported pain.

One explanation for this could be that subjects fake or partially fake reduced pain. This is an unlikely

explanation however because subjects report elaborate cognitions following painful stimulation. Furthermore, the relationship between these cognitions, reported pain and hypnotic susceptibility would be difficult to account for in terms of faking. Finally, the large number of clinical reports of the use of hypnotic analgesia are likely not based on patients who faked pain relief.

As an alternative explanation Spanos (in press; Spanos, et al., in press) proposed that the intensity of subjects' "sensory events" remain unchanged but that the interpretation of these events is modified by suggestions. Thus subjects who were "observing themselves" engage in coping cognitions inferred they were feeling less pain than those subjects who catastrophized. This account is based on the assumption that subjects' pain experiences were in fact quite ambiguous and therefore led to a greater reliance on nonsensory information. Nonsensory information would include variables such as expectancies, presence of suggestions and coping or catastrophizing ideation.

Theoretical accounts of hypnotic analgesia continue to be controversial (Hilgard, 1977; Laurence & Perry, 1981; Spanos & Hewitt, 1980). The experimental evidence clearly indicates however that an explanation of suggested analgesia must account for the relationship between subjects' cognitions and reported pain. Although the exact nature of

this relationship is not clear, an examination of the experimental situation may elucidate it .

Subjects typically (a) agree to participate in a painful experience (b) for a benign and rational (i.e., scientific) purpose and (c) expect no long term harm to accrue from their experience. Furthermore, the pain stimulus is (a) under the control of the experimenter, (b) of a relatively short duration, (c) localized, and (d) predictable. Nevertheless, despite the long term innocuousness of the situation, the pain stimulus is typically severe enough and the rating tasks demanding enough that the subject will focus all his/her attention on the stimulus situation. Finally, the suggestion informs the subject (a) that it is probably possible to, in some sense, reduce one's pain, (b) that the experimenter expects him/her to try, (c) that the experimenter expects the subject to be at least partially successful (i.e., to report less pain), and (d) that the pain stimulus is probably quite harmless. If the subject is given a suggestion on a subsequent trial and he/she happens to be a high susceptible subject who is motivated to do well and who has a proclivity for becoming absorbed in and imagining suggested events, then removing attention from the stimulus is probably relatively easy. On the other hand, low susceptibles may have greater difficulty deploying their attention elsewhere. The result is a reduction in reported pain for highs but not for lows.

Pain can be viewed as an automatic intrusion into ongoing attentive processes. Attention diversion refers then to the attempts made by the subject to minimize these intrusions. Vigilance to alternate events or imaginings would aid such a process. Most subjects know they can safely (i.e., without fear of bodily harm) divert their attention and furthermore the experimenter has asked them to do so.

Placebo and Suggestibility

Perhaps the only other seriously tendered hypothesis for suggested analgesia consists of the placebo hypothesis. Briefly stated, this hypothesis usually equates suggested analgesia with placebo analgesia, both of which are due to "suggestibility" effects (e.g., Beecher, 1968; Brodeur, 1965; Melzack, 1973; Strupp, Levenson, Mamick, Snell, Hinrichsen, & Boyd, 1974; Trouton, 1957). Exactly what processes suggestibility refers to are rarely, if ever, specified (for reviews see Jospe, 1978; Shapiro & Morris, 1978). Furthermore, no single theory has satisfactorily explained the placebo effect although dozens of hypotheses have been tendered (see Evans, 1974; Jospe, 1978; Ross & Olson, 1981; Shapiro & Morris, 1978).⁵

According to Shapiro and Morris (1978), three general themes characterize the processes or mechanisms that have

been posited to account for the placebo effect. These are (a) social influence effects such as suggestion, persuasion, transference, role demands, operant conditioning, and guilt reduction; (b) expectancy effects such as those produced by classical conditioning, cognitive dissonance, and hope; and (c) evaluation effects such as response artifacts, labelling and misattribution. There is little empirical verification for any of these processes however.

In a series of papers Levine, Gordon and Fields (1978; 1979; Levine, Gordon, Bornstein, & Fields, 1979; Levine, Gordon, Smith, & Fields, 1981) have argued that placebo analgesia is mediated, at least in part, by endogenous opioid peptides and that placebo and morphine analgesia share a common mechanism. The evidence however is based on the injection of naloxone following the administration of a placebo post-operatively and is thus subject to all those criticisms noted earlier with regard to the evidence for endorphin mediated hypnotic analgesia. Furthermore, several studies have failed to replicate these results (Goldstein & Grevert, 1978; Mihic & Binkert, 1978; Sjolund & Ericksson, 1978). In short, the exact nature of the placebo response has yet to be determined.

One reason for this inability to find the locus of the placebo effect may reside in the large range of situations where placebos are administered. Investigators have

attempted to propose a single mechanism to account for such diverse placebos as pills, injections, pseudo-psychotherapy, "subliminal" noise, false biofeedback, false-loci acupuncture, and so on; which are administered from laboratories to clinics to all manner of hospital wards; and which are administered to people radically differing in age, patient status, subject status, and presenting pathology or lack thereof. It may be somewhat unrealistic to expect a single process to account for the placebo effect across all these situations.

The experimental evidence suggests that there is no relationship between placebo responding and suggestibility as measured by various tests of suggestibility as devised by different investigators (see Evans, 1967; 1974; Jospe, 1978; Shapiro & Morris, 1978). One obvious problem underlying the attempt to relate suggestibility with placebo responsiveness lies in the assumption that the latter is a consistent personality trait. While suggestibility measures are often reported to have test-retest correlations up to .9, (e.g., Evans, 1967), the evidence for the existence of a consistent placebo personality or responder is almost nonexistent (Buckalew, Ross, & Starr, 1981; Jospe, 1978; Shapiro & Morris, 1978).

The relationship between placebo responsiveness and hypnotic analgesia has been explicitly studied in several

experiments. In the first of these Bentler, O'Hara, & Krasner (1963) tested 19 female nursing students on hypnotic susceptibility after their participation in an experiment in which they received a placebo. No relationship was found between subjects' susceptibility scores and their increased pain thresholds and tolerances following placebo ingestion.

In two experiments Saletu, Saletu, Brown, Stern, Sletten and Ulett (1975) and Stern, Brown, Ulett, and Sletten (1977) conducted 9 and 14 experimental pain sessions, respectively, comparing hypnosis, placebo, acupuncture and a variety of pharmacological analgesics. Twenty subjects participated in every session in each experiment making it likely that complex order effects were involved. Furthermore, Saletu et al. (1975) used "not really painful" brief electrical stimuli. Stern et al. (1977) used ice water immersion of the fingers only to produce cold-pressor pain and inflated a blood-pressure cuff for only 5 min. to produce ischemic pain. Thus none of these stimuli were as severe as those typically used in laboratory investigations of pain. Also, a great number of comparisons were conducted largely relying on multiple t-tests. Finally, placebo administration did not significantly affect reported pain in either experiment whereas hypnotic analgesia did. The lack of an effect precludes any direct comparison between the treatments.

In an oft-cited paper McGlashan, Evans and Orne (1969) explicitly compared the responses of high and low susceptible subjects to hypnotic and placebo analgesia. These authors wished to demonstrate in particular that (a) hypnotic analgesia and placebo responding could not be predicted by standard tests of non-hypnotic suggestibility, (b) that the reduction of anxiety was not the main factor mediating both hypnotic analgesia and placebo responding, and (c) that high susceptible subjects' reductions in pain following hypnotic analgesia is not due to any "placebo effects of the hypnotic treatment situation" (p.229). That is, McGlashan et al. (1969) wished to argue that hypnotic analgesia could not be equated with placebo analgesia but, for high susceptible subjects, also consisted of a "pain-reducing sensory alteration" (p.229).

McGlashan et al. (1969) tested 24 subjects who had scored consistently in the upper or lower 5% of standard susceptibility scales (12 highs and 12 lows). In session 1 all subjects completed a medical history and short physical examination and then underwent a baseline ischemic pain trial. In their particular procedure, McGlashan et al. applied a tourniquet to the upper arm and then had subjects pump water by way of a double-valved rubber bulb. Subjects reported when they first felt pain and continued pumping until they could no longer do so. Using this procedure the

subject had control over the rate of work and therefore, to some degree, the amount of pain he/she experienced.

Subjects rated the intensity of their pain on a 10-point category scale at the point at which they stopped pumping. McGlashan et al. thus confounded the rating of pain with the length of time subjects pumped. (Despite the extensive use made of these ratings by the authors for secondary analyses, this confound precludes any sound conclusions.) Subjects then underwent a hypnotic analgesia trial with electric shock which was carried out by a second experimenter. In this trial all subjects received a shock well above threshold, before and after hypnotic analgesia. Low susceptibles however were administered a shock lower than their pre-hypnotic shock in order to convince them that they were able to experience hypnotic analgesia. Although this was done ostensibly to motivate low susceptible subjects for their subsequent hypnotic analgesia session by engendering expectancies for analgesia, it may also have had an opposite effect. Low susceptibles may have had unrealistic expectations about the effects of hypnotic analgesia and thus may have been surprised at the actual level of pain they experienced in their subsequent session.

In their second session all subjects received an hypnotic induction and analgesia suggestions before their ischemic pain was reintroduced. When session two was

completed they were informed that session 3 would involve taking an experimental pain-relief pill and they were asked to sign a consent form. Subjects underwent a third ischemic pain trial during session 3 after taking the placebo. The experimenter was not aware all subjects were receiving a placebo but rather believed that half the subjects were receiving a moderate strength analgesic. Thus, the usual double-blind conditions were operative despite the fact that all subjects received placebos.

For each session, four measures of pain were calculated. The amount of time pumped and the volume of water pumped at each of threshold and tolerance. Because of session 1 differences between high and low susceptible subjects (highs exhibited greater thresholds than lows) all session 2 and 3 results are based on change scores. These indicated that overall, subjects showed a placebo effect and that low susceptibles showed equivalent reductions in pain with hypnotic analgesia and placebo analgesia as measured on all four measures.

High susceptible subjects showed significant increases on all four measures during hypnotic analgesia and these were in turn significantly greater than their placebo performance. Highs also showed greater increases than lows on three of the four measures during hypnotic analgesia. In general hypnotic susceptibility and placebo analgesia were

unrelated. A relationship between hypnotic and placebo analgesia was present for the low susceptibles but not for the highs.

Evans (1969) also reported on three general "suggestibility" measures taken during the experiment. These confirmed earlier findings that they were in the main unrelated to placebo responsiveness or hypnotic susceptibility.

In a further report from the same experiment, Evans (1974) presented the results of trait and state anxiety tests administered to these subjects. Following ingestion of the placebo, state anxiety was positively related to increases in pain threshold. Trait anxiety was positively related to increases in pain tolerance. The latter also related to an intensity of side effects measure and reported psychosomatic symptoms. In a later report, however, Evans (1981) stated that state anxiety, but not trait anxiety, was related to tolerance. This is in contradiction to the data reported by Evans (1974). Thus the manner in which placebo responsiveness was related to subjects' anxiety levels is unclear.

The major problem with this experiment however lies in the nature of the task.⁶ As noted above, subjects have some control over the pain they experience. In fact, in a separate report Evans & McGlashan (1967) noted that "it was

apparent to [the subject] that he was controlling the rate at which the intensity of pain increased" (p. 794). McGlashan et al. (1969) noted that "most" subjects' tolerance levels were limited by fatigue, not maximum pain.

An alternative way of summarizing the data was originally proposed by Evans & McGlashan (1967). By forming a ratio of the amount of water pumped to the amount of time pumped (ccs/sec) the "rate of work" could be calculated. According to this calculation high susceptibles pumped significantly less water than low susceptibles "per squeeze of the rubber bulb" (McGlashan et al., 1969, p.238). The authors interpreted this to mean that, during the hypnotic analgesia session, despite the fact that both groups pumped relatively equivalent amounts of water, high susceptibles took longer to reach this point. They also argued that highs could not have pumped more water because of the fatigue factor.

An alternative interpretation however can be derived from the same data. That is, by pumping slower, highs were able to tolerate pain longer because it simply did not build as fast. Lows on the other hand worked harder and consequently felt greater pain sooner. On the basis of the written reports however there is no possibility of distinguishing between these interpretations. Later researchers employing ischemic pain have typically avoided

this problem by setting an upper limit on the amount of exercise performed by the occluded arm and measuring the time to tolerance (Smith, Egbert, Markowitz, Mosteller, & Beecher, 1966).

Finally, a note on the comparability of hypnotic and placebo analgesia: When subjects receive a placebo (which they believe to be an active drug) they typically wait relatively passively for the effects to occur. Subjects given suggestions for analgesia however engage in various cognitive strategies to reduce their pain (see literature cited above). Furthermore, reduced pain following suggested analgesia is correlated with hypnotic susceptibility - a relatively stable attribute variable whereas placebo responsiveness does not appear to be related to any reliable personality measure. Thus the two types of analgesia may be mediated by two different cognitive mechanisms (cf. Jospe, 1978; Shapiro & Morris, 1978).

Outline of Research

The present studies were aimed at elucidating and comparing the processes underlying hypnotic and placebo analgesia. Two experiments were conducted: The first addressed issues raised concerning the interpretation of McGlasnan et al.'s (1969) dependent measures. The second examined in greater detail subjects' experiences following hypnotic analgesia and placebo analgesia.

Both experiments utilized ischemia as the pain stimulus. The first used the procedures of McGlashan et al. (1969) and the second used the submaximum effort tourniquet technique (SETT; Smith et al., 1966). The SETT is a revision of an earlier ischemic pain method originally developed by Lewis, Pickering, and Rothschild (1931) in which pain was produced in normal limbs by occluding the blood flow in exercising muscles. Smith et al. (1966) standardized the amount of exercise for precisely the reasons outlined above, namely, that the subject can control pain by limiting exercise. Subjects in the Smith et al. study had a tourniquet inflated around their upper arm and then squeezed a hand exerciser 20 times. Pain was measured as the length of time it took subjects to reach levels of pain labelled as "slight," "moderately distressing," "very distressing," and "unbearable."

A large amount of research has been conducted using this technique (e.g., Adler, Gervasi, Holzer, & Hemmeler, 1974; Adler & Lomazzi, 1974; Chapman & Feather, 1973; Liberman, 1964; Smith, Lowenstein, Hubbard, & Beecher, 1968; Smith & Beecher, 1969; Sternbach, Deems, Timmermans, & Huey, 1977; Von Graffenried, Adler, Abt, Nüesch, & Spiegel, 1978). High test-retest reliabilities have been reported (Postlethwaite, Grieve, Santacroce, Renfree, Wilson, & Peck, 1980) and although there have been reports of failures (Sternbach et

al., 1977) the SETT has generally been responsive to a wide range of pharmaceutical analgesics. Anxious and non-anxious individuals have also been shown to respond differently to the test (Adler et al., 1974; Adler & Lomazzi, 1974; Chapman & Feather, 1973). Reported pain increases monotonically as a function of time, and the majority of experimental subjects report pain in a negatively accelerated fashion (Fox, Steger, & Jennison, 1979; Moore, Duncan, Scott, Gregg, & Ghia, 1979). Studies that have examined reported pain over time in ischemia however have always used category scales, visual analogue scales or verbal descriptor scales (e.g., Fox et al., 1979; Hilgard & Hilgard, 1975; Moore et al., 1979; Smith et al., 1966). Thus the negatively accelerating function may in part be a function of bounded response scales (Stevens, 1975).

In the Smith et al. (1966) study subjects squeezed a hand spring exerciser 20 times for 2 sec where each squeeze was followed by a 2 sec rest. Although most subjects (or patients) had no difficulty in making the necessary 20 squeezes, the amount of work expended varied between individuals. Since work is a crucial determinant of ischemic pain, this procedure may add further variability to tolerance and pain reports. Moore et al. (1979) varied the percentage of maximum effort subjects exerted (a) by determining subjects' maximal grip strength, (b) by having

subjects squeeze an exerciser 20 times at 30% or 50% of maximum effort and (c) by varying the duration of each squeeze at an instantaneous release or at 2 sec. Subjects' range of maximal effort varied greatly from 12 g/in² to 45 g/in². Both duration and percentage of effort appeared to affect pain growth rate and time to tolerance. Percentage of effort did not reach statistical significance, however, possibly due to the small range used (30% - 50%). Thus, when referred to with regard to Experiment 2 below, the SETT will consist of the modification made by Moore et al., namely, the use of a 50% force of maximum strength held for 2 sec.

EXPERIMENT 1

McGlashan et al.'s data were ambiguous because differences in threshold and tolerance (in time and volume of water pumped) may have been influenced by subjects' control of their pumping or rate of work. The present experiment attempted to replicate the findings of McGlashan et al. with respect to hypnotic analgesia. The use of the pumping measure allowed for an evaluation of the rate of work conducted by highs and lows.

Design

This experiment consisted of a 2 X 2 split-plot design. Subjects were (a) either high or low in hypnotic susceptibility and (b) tested on ischemic pain before and after hypnotic analgesia.

MethodSubjects

Sixteen high susceptible and 16 low susceptible subjects volunteered to participate in an experiment on hypnosis and pain perception. Each susceptibility group consisted of eight males and eight females. All subjects were drawn from a larger pool of introductory psychology students at Carleton University who had been tested on the Carleton University Responsiveness to Suggestion Scale (CURSS; Spanos, Radtke, Hodgins, Stam, & Bertrand, 1982) in session

1. Subjects scoring six and seven constituted the high susceptible group and subjects scoring zero made up the low susceptible group. Subjects reporting any of the following were excluded: histories of diabetes, coagulopathies, heart disease, blood pressure disorders, recent venipuncture, and presently using any type of medication. Furthermore, subjects over 30 were not asked to participate and all subjects had their blood pressure taken to determine if it was within normal limits. Mean ages were 19.6 years (18-22) for males and 19.2 years (18-28) for females. One subject was replaced because she failed to follow instructions.

Apparatus

A closed 4 liter flask filled with water had two rubber tubes attached to it. One tube was connected to a double-valved rubber bulb. Squeezing the bulb resulted in water being forced from the flask to the second tube. The water was collected and measured in a separate beaker.

A blood pressure cuff from an Avenue (#12-300) portable mercury sphygmomanometer was placed on the subjects' upper arm. Subjects then held their arm above their head for 1 min, at the end of which the cuff was inflated to 200 mm Hg. At this point subjects were instructed to lower their arm, they were given the bulb and began squeezing in time to a metronome moving at 40 beats/min.

Procedure

Session 1. All subjects were pretested on the CURSS in this session. The CURSS is typically administered in small groups of 5 - 10 subjects and takes approximately 30 - 40 min to complete. Subjects obtain three scores - one for each of the three subscales which comprise the CURSS. The first is the objective (CURSS:O) score, the traditional measure of hypnotic susceptibility which is based on the number of items the subject completed behaviorally. The CURSS:O score can range from 0 to 7. The second and third scales, respectively, are the subjective (S) and involuntariness (I) scales. For each item on the scale subjects rate the degree of their subjective involvement (S scale) and involuntary responding (I scale). The S and I scales range from 0 to 21.

Those subjects who volunteered to participate further in a study on pain and hypnosis, and who scored either 6 - 7 (highs) or 0 (lows) on the O scale, returned for a second session.

Session 2: Baseline. Subjects were first given an informed consent agreement to sign (Appendix A). The agreement asked them to participate in two sessions of ischemic pain from which they could withdraw at any time without penalty. Subjects were then asked to remove any watches and jewelry from their arms. Next, the procedures

were described and subjects were told they would recognize four stages of sensation as they pumped. These were (from McGlashan et al., 1969, p. 233), "(1) no change in sensation; (2) paresthesia, or the awareness of sensations that were not painful; (3) pain threshold, or the moment that sensations first became painful; [and] (4) the maximum tolerable pain, or the point at which he [she] could not possibly continue pumping." Once subjects understood the four stages they were instructed that the experimenter wished them to pump and say "now" when they reached "stage 3" or that point at which they began to feel pain. They were told to signal tolerance by saying "now" again and dropping the bulb. As in McGlashan et al. (1969) subjects were exhorted to continue "pressing almost to the limits of your endurance" and to "continue squeezing as hard as you can until it hurts so much that you cannot continue for even one more second" (p. 233). Complete instructions are in Appendix B.

Subjects had the cuff placed on an arm (right and left were counterbalanced) and squeezed the bulb as described. When the subject said "now" to indicate his/her pain threshold, the experimenter switched the outlet tube to another beaker while the subject continued pumping. When the subject dropped the bulb and indicated he/she had reached tolerance, the cuff was immediately deflated.

Subjects were unable to see the amount of water being pumped. Time to threshold and tolerance and volume of water pumped (in ml) were both recorded and the subjects were asked to estimate how much water and for how long they thought they had pumped.

Subjects then rated the percentage of time during the immersion that they thought about or imagined events inconsistent with pain. Two further questions asked subjects to rate on visual analogue scales (VAS) the amount of time they engaged in coping ideation or catastrophizing ideation. Finally, subjects were asked to rate the extent to which their arm had become tired, whether they could have continued pumping had their arm not been so tired, and, if so, for how long (see Appendix C).

Session 2: Posttest. Following a 10 min rest, all subjects were reassured that they were excellent subjects for this particular experiment and that, since they were now going to be hypnotized, they would not feel as much discomfort this time as last time. These assurances were taken from McGlashan et al. (1969) to motivate low susceptibles. Furthermore, the experimenter conducting session 2 was blind to subjects' hypnotic susceptibility levels.

A standard 10 min hypnotic induction procedure was presented via tape recorder to all subjects. The induction

consisted of interrelated suggestions designed to (a) elicit cooperation and maximize performance, (b) define the situation as hypnosis, and (c) produce relaxation (see Appendix D). The induction procedure was followed by a standard analgesia suggestion asking subjects to imagine their arm as numb and insensitive (see Appendix E). The ischemic pain task was reintroduced to subjects with instructions taken verbatim from McGlashan et al. (p. 234) which emphasized that this time the task would be easy and painless (Appendix F).

Upon reaching tolerance, the cuff was deflated and subjects were given standard "waking" instructions. They then estimated the volume of water pumped and the duration of pumping and filled in the same questionnaires they received after the first trial. When these were completed, subjects were debriefed and thanked for their participation.

Results

Time

The durations (in seconds) subjects pumped to reported threshold and to tolerance were each subjected to a 2x2 split-plot analysis of variance (ANOVA). The ANOVAs included one between-subjects variable (High/Low susceptibility) and one within-subjects variable (Trials). The analyses for threshold and tolerance both resulted in a significant Trials main effect, $F(1,30) = 9.42, p < .05$, for

threshold, and $F(1,30) = 9.79$, $p < .05$, for tolerance. No other effects were significant in either ANOVA. Table 1 presents the ANOVA for threshold and Table 2 presents the ANOVA for tolerance.

Thus, regardless of hypnotic susceptibility level, subjects pumped longer to threshold following suggested analgesia ($M = 65$ sec, $sd = 24$ for Trial 1; $M = 76$ sec, $sd = 28$ for Trial 2). Likewise, the total length of time pumped increased from Trial 1 ($M = 123$ sec, $sd = 29$) to Trial 2 ($M = 138$ sec, $sd = 41$). The means for each cell of the ANOVAs are presented in Table 3.

Volume

The volume of water pumped (in ml) was likewise subjected to split-plot ANOVAs for both threshold and tolerance with susceptibility and trials as factors. These results were identical to those for duration. The analysis for threshold resulted in a significant main effect for Trials, $F(1,30) = 5.29$, $p < .05$. Total volume at tolerance was also associated with a significant Trials main effect, $F(1,30) = 5.83$, $p < .05$. No other effects were significant. These ANOVAs are presented in Tables 4 and 5.

Subjects pumped significantly less water by the time they reported they had reached threshold on Trial 1 ($M = 987$ ml, $sd = 403$) than on Trial 2 ($M = 1107$ ml, $sd = 442$). Tolerance increased from a mean 1978 ml ($sd = 616$) on Trial

Table 1

Summary Table for Susceptibility x Trial ANOVA
on Duration to Threshold

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	873.9	<1
Subjects Within Groups	30	1185.9	
Trial (B)	1	2075.9	9.42*
A x B	1	243.8	1.11
B x Subjects Within Groups	30	220.4	

* $p < .05$

Table 2
Summary Table for Susceptibility x Trial
ANOVA on Duration to Tolerance

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	114.5	<1
Subjects Within Groups	30	2205.7	
Trial (B)	1	3872.0	9.79*
A x B	1	541.7	1.37
B x Subjects Within Groups	30	395.6	

* $p < .05$

Table 3

Mean Time to Threshold and Time to
Tolerance Scores in Sec
Susceptibility

Trial	High		Low	
	Threshold		Tolerance	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	66	25	63	24
2	82	33	70	22
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	118	23	127	35
2	140	39	137	44

Note. n = 16 for each mean.

Table 4

Summary Table for Susceptibility x Trial
ANOVA on Volume to Threshold

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	41769	<1
Subjects Within Groups	30	324750	
Trial (B)	1	231000	5.29*
A x B	1	16416	<1
B x Subjects Within Groups	30	43636	

* $p < .05$

Table 5

Summary Table for Susceptibility x Trial

ANOVA on Volume to Tolerance

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	494380	<1
Subjects Within Groups	30	801780	
Trial (B)	1	198580	5.83*
A x B	1	20129	<1
B x Subjects Within Groups	30	34076	

* $p < .05$

1 to a mean 2090 ml ($sd = 668$) on Trial 2. The means for each cell of the ANOVAs are presented in Table 6.

Work

For each subject the rate of work was calculated as the ratio (ml/sec). Again, the ratios at threshold and the ratios at tolerance were each subjected to split-plot ANOVAs with susceptibility and trials as factors. The ANOVA for the ratios at threshold resulted in no significant main effects or interactions. These results are presented in Table 7.

A significant Trials main effect was obtained from the ANOVA on the ratios at tolerance, $F(1,30) = 4.11$, $p = .05$. Higher ratios were obtained on Trial 1 ($M = 16.02$ ml/sec, $sd = 2.45$) than on Trial 2 ($M = 15.14$ ml/sec, $sd = 2.03$). No other effects approached significance (see Table 8). Table 9 presents the means for each cell of the two ANOVAs.

Cognitive Activities

Catastrophizing. Subjects' scores on the catastrophizing ideation visual analogue scale (in mm) were subjected to a 2x2 split-plot ANOVA. As above, the factors were Hypnotic Susceptibility and Trials. A Trials main effect was the only significant result from this ANOVA, $F(1,30) = 16.03$, $p < .05$. The analysis is presented in Table 10, and the means for each cell of the analysis are presented in Table 11. Overall, subjects reported engaging

2

Table 6

Mean Volume to Threshold and Volume
to Tolerance Scores in Ml

Susceptibility

Trial	High		Low	
	Threshold		Tolerance	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	997	390	978	428
2	1149	526	1066	352
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	1873	423	2084	763
2	2020	510	2160	806

Note. n = 16 for each mean.

Table 7

Summary Table for Susceptibility x Trial

ANOVA on Work to Threshold

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	16.93	2.64
Subjects Within Groups	30	6.42	
Trial (B)	1	4.04	1.36
A x B	1	7.63	2.58
B x Subjects Within Groups	30	2.96	

Table 8

Summary Table for Susceptibility x Trial
ANOVA on Work to Tolerance

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	7.09	<1
Subjects Within Groups	30	7.14	
Trial (B)	1	12.27	4.11*
A x B	1	1.64	<1
B x Subjects Within Groups	30	2.99	

*p = .05

Table 9.

Mean Ratio to Threshold and Ratio to
Tolerance Scores

Susceptibility

Trial	High		Low	
	Threshold		Tolerance	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	14.85	2.12	15.18	2.35
2	13.65	1.89	15.37	2.27
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	15.85	2.20	16.19	2.73
2	14.65	1.90	15.64	2.09

Note. n = 16 for each mean.

2

Table 10

Summary Table for Susceptibility x Trial

ANOVA on Catastrophizing VAS

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	2665	3.33
Subjects Within Groups	30	799	
Trial (B)	1	10176	16.03*
A x B	1	722	1.14
B x Subjects Within Groups	30	635	

*p < .05

Table 11
Mean Catastrophizing VAS Scores
Susceptibility

Trial	High		Low	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	44.06	32.76	50.25	28.22
2	12.13	19.51	31.75	24.86

Note. n = 16 for each mean.

in less catastrophizing ideation after the suggestion ($M = 21.94$, $sd = 24.14$) than before the suggestion ($M = 47.16$, $sd = 30.24$).

Coping. The data from the visual analogue coping scale were also subjected to a 2x2 split-plot ANOVA. As can be seen from Table 12, the Susceptibility x Trial interaction is significant, $F(1,30) = 6.72$, $p < .05$. The means are presented in Table 13. Newman-Keuls post hoc multiple comparisons indicated that high and low susceptible subjects did not differ on reported coping during Trial 1 but following Trial 2, high susceptibles reported significantly more coping than low susceptibles. Highs also increased significantly on Trial 2 over their Trial 1 reports, whereas lows did not.

Time imagining. Subjects' ratings of the percent of time they spent imagining events inconsistent with the pain were also subjected to a 2x2 split-plot ANOVA. The Susceptibility x Trial interaction was significant, $F(1,30) = 12.16$, $p < .05$ (see Table 14). Newman-Keuls post hoc multiple comparisons on the means involved in the interaction (see Table 15) indicated both high and low susceptible subjects reported increased time imagining on Trial 2 than on Trial 1. Highs however reported significantly more time imagining than lows on Trial 2 but not on Trial 1.

Table 12
Summary Table for Susceptibility x Trial
ANOVA on Coping VAS

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	6622	8.28*
Subjects Within Groups	30	799	
Trial (B)	1	16224	26.53*
A x B	1	4112	6.72*
B x Subjects Within Groups	30	612	

*p < .05

Table 13
Mean Coping VAS Scores for Susceptibility x
Trial Interaction
Susceptibility

Trial	High		Low	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	30.25	27.66	25.94	27.11
2	78.13	24.22	41.75	27.13

Note. n = 16 for each mean.

Table 14

Summary Table for Susceptibility x Trial

ANOVA on Percent Time Imagining

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	5148	5.93*
Subjects Within Groups	30	868	
Trial (B)	1	23409	42.59*
A x B	1	6683	12.16*
B x Subjects Within Groups	30	550	

* $p < .05$

Table 15
Mean Percent Time Imagining Scores for
Susceptibility x Trial Interaction
Susceptibility

Trial	High		Low	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	18.44	24.54	20.94	24.51
2	77.13	26.96	38.75	30.08

Note. n = 16 for each mean.

Cognitive activities and pain measures. The performance measures were intercorrelated with the measures of cognitive activities. These correlations are presented in Table 16. There is little interrelationship between the two sets of measures. Nevertheless, within each set of measures there are relatively large intercorrelations. Thus, while the measures of cognitive activities were responsive to treatment effects they were not related to ischemia performance measures.

Arm-Tiredness

Subjects' ratings of the extent to which their arm was fatigued at the end of each trial were subjected to a 2x2 split-plot ANOVA. As can be seen in Table 17, there were no significant differences between levels of susceptibility or trials. The means are presented in Table 18.

Among high susceptibles 15 (94%) answered "yes" on both trials to the question asking subjects if they could have continued to pump before reaching maximum pain. Thirteen (81%) and twelve (75%) low susceptibles answered "yes" to the same question on Trials 1 and 2, respectively. Thus, overall four subjects answered "no" following Trial 1 and five did so following Trial 2.

The point-biserial correlation between arm-tiredness and subjects' reports of continuing was .29 (ns) following Trial 1 and .54 ($p < .01$) following Trial 2. Thus, following

Table 16
Correlations Among Performance and
Cognitive Activity Measures

	1	2	3	4	5	6	7	8	9
1	-----	.73**	.93**	.75**	.63**	.31	-.42*	-.10	-.02
2	.62**	-----	.56**	.88**	.65**	.65**	-.15	-.16	-.09
3	.95**	.56**	-----	.73**	.33	.01	-.44*	-.02	-.01
4	.49**	.90**	.54**	-----	.38**	.22	-.20	-.01	-.05
5	.33	.25	.08	-.06	-----	.73**	-.24	-.19	-.05
6	.40*	.40*	.15	-.02	.73**	-----	.02	-.37*	-.17
7	-.17	-.02	-.12	.09	-.12	-.25	-----	-.29	-.26
8	-.28	-.09	.00	-.11	-.03	.08	-.44*	-----	.69**
9	.07	-.13	.06	-.14	.00	.05	-.43*	.96**	-----

Note. Correlations above the diagonal are those for Trial 1;
Correlations below the diagonal are those for Trial 2.
1 = Volume to Threshold, 2 = Volume to Tolerance, 3 = Time to
Threshold, 4 = Time to Tolerance, 5 = Work to Threshold,
6 = Work to Tolerance, 7 = Catastrophizing, 8 = Coping,
9 = Percent Time Imagining.

* $p < .05$

** $p < .01$

Table 17

Summary Table for Susceptibility x Trial

ANOVA on Arm-Tiredness VAS

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	236	<1
Subjects Within Groups	30	379	
Trial (B)	1	244	1.05
A x B	1	338	1.45
B x Subjects Within Groups	30	233	

Table 18

Mean Arm-Tiredness VAS Scores

Susceptibility

Trial	High		Low	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	85.38	12.37	86.13	9.91
2	86.06	20.61	77.63	23.40

Note. n = 16 for each mean.

Trial 2 subjects who reported being unable to continue also reported their arms as being ~~less~~ tired. These subjects had presumably reached their "true" pain tolerance point and stopped pumping because of pain, not exhaustion. This is supported by the point-biserial correlation between the amount of work done at tolerance and subjects' answers to the above question, $-.14$ (ns) following Trial 1 and $-.35$ ($p < .05$) following Trial 2. Subjects who reported that they could not continue even if their arm was less tired also squeezed more water per second than did those who reported they could have continued in the absence of tiredness. Table 19 presents the mean performance measures for subjects who reported being able to continue versus those who did not. Only the work ratio at tolerance on Trial 2 discriminated between these two groups of subjects, $t(30) = 2.08$, $p < .05$.

Subjects' estimates of the time they could have continued pumping if their arm had not been exhausted were also subjected to a 2×2 split-plot ANOVA. The Susceptibility $\times$ Trials interaction was significant, $F(1,30) = 5.62$, $p < .05$ (see Table 20). Newman-Keuls post hoc multiple comparisons indicated (see Table 21) that high susceptibles did not differ from lows on Trial 1 but gave higher estimates on Trial 2. Lows did not change from Trial 1 to Trial 2 whereas highs increased their estimates

Table 19

Mean Performance Scores for Subjects Indicating Ability
to Continue vs. Subjects Indicating Maximum Pain

	Could Continue			Could Not Continue		
Trial	Time to Threshold					
	<u>M</u>	<u>sd</u>	<u>n</u>	<u>M</u>	<u>sd</u>	<u>n</u>
1	66	25	28	55	25	4
2	75	30	27	79	13	5
	Time to Tolerance					
1	124	28	28	115	40	4
2	141	43	27	122	21	5
	Volume to Threshold					
1	1007	402	28	850	442	4
2	1085	473	27	1226	205	5
	Volume to Tolerance					
1	1975	593	28	2000	872	4
2	2095	709	27	2063	433	5
	Work to Threshold					
1	15.03	2.22	28	14.93	2.49	4
2	14.33	2.36	27	15.48	1.03	5
	Work to Tolerance					
1	15.89	2.50	28	16.90	2.12	4
2	14.84	1.99	27	16.78	1.41	5

Table 20

Summary Table for Susceptibility x Trial
ANOVA on Estimated Duration of Continuation

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	21719	6.08*
Subjects Within Groups	30	3572	
Trial (B)	1	4744	3.02
A x B	1	8813	5.62*
B x Subjects Within Groups	30	1569	

* $p < .05$

Table 21
Mean Estimated Duration of Continuation Scores
for Susceptibility x Trial Interaction

Trial	Susceptibility			
	High		Low	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	37.38	32.15	24.00	25.47
2	78.06	89.45	17.75	24.49

Note. n = 16 for each mean.

significantly over the two trials. There were no significant relationships between subjects' estimates and the performance measures on either trial.

Estimated Time and Volume

Following each trial subjects estimated the duration and volume pumped on that trial. Separate 2x2 split-plot ANOVAs performed on these measures revealed a Trials main effect for both estimated time, $F(1,30) = 21.30$, $p < .05$, and estimated volume, $F(1,30) = 22.54$, $p < .05$ (see Tables 22 and 23). Subjects' estimates of time increased from 82 sec ($sd = 33.3$) on Trial 1 to 106 sec ($sd = 44.6$) on Trial 2. Likewise, estimated volume increased from 357 ml ($sd = 235$) to 563 ml ($sd = 432$) over trials. Although subjects substantially underestimated both duration and volume, their estimates of time were significantly correlated with measured time (Trial 1, $r = .46$, $p < .05$; Trial 2, $r = .39$, $p < .05$) but their estimates of volume were not significantly related to measured volume (Trial 1, $r = .33$, ns; Trial 2, $r = .07$, ns). Estimated time and volume were not significantly correlated on Trial 1 ($r = .18$) or Trial 2 ($r = .13$). There was also no relationship between these estimates and measures of subjects' cognitive activities.

Estimates of volume were divided by estimated time, and the resulting ratio provided an indirect measure of estimated work performed. A 2x2 split-plot ANOVA on these

Table 22

Summary Table for Susceptibility x Trial

ANOVA on Estimated Time

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	13	<1
Subjects Within Groups	30	2750	
Trial (B)	1	9049	21.30*
A x B	1	763	1.80
B x Subjects Within Groups	30	425	

* $p < .05$

Table 23

Summary Table for Susceptibility x Trial

ANOVA on Estimated Volume

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	89625	<1
Subjects Within Groups	30	216530	
Trial (B)	1	679590	22.54*
A x B	1	1016	<1
B x Subjects Within Groups	30	30151	

* $p < .05$

ratios resulted in a significant Trials main effect, $F(1,30) = 4.85$, $p < .05$ (see Table 24). Estimates of work were significantly greater following Trial 2 ($M = 6.26$, $sd = 6.34$) than following Trial 1 ($M = 4.67$, $sd = 3.07$). The means for each cell of the ANOVAs on estimated time, volume and work are presented in Table 25.

Discussion

The results indicate that subjects increased both the duration and volume of pumping following hypnotic analgesia. That this increase was independent of hypnotic susceptibility levels constitutes a failure to replicate McGlashan et al.'s (1969) results. These authors reported a larger increase in duration to threshold and tolerance and a larger amount of water pumped to threshold for high susceptible than for low susceptible subjects. High and low susceptibles did not differ in the amount of water pumped to tolerance. They also reported a difference in baseline threshold between high and low susceptible subjects (highs pumping longer and more water) that was not replicated here.

The reasons for this failure to replicate the susceptibility differences is unclear. Since this experiment is the first reported replication of McGlashan et al. (1969), any attempts to account for this finding remain highly speculative. Nevertheless, one obvious difference between the two experiments lay in subject selection.

Table 24

Summary Table for Susceptibility x Trial
ANOVA on Estimated Work

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>
Susceptibility (A)	1	35.3	<1
Subjects Within Groups	30	41.5	
Trial (B)	1	40.2	4.85*
A x B	1	5.9	<1
B x Subjects Within Groups	30	8.3	

* $p < .05$

Table 25
Mean Estimated Time, Volume
and Work Scores

Susceptibility

High

Low

Trial	Estimated Time			
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	79	30	85	37
2	110	48	102	41
	Estimated Volume			
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1	324	166	391	290
2	522	322	605	527
	Estimated Work			
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
1		2.18	5.11	3.78
2		3.59	7.30	8.23

Note. n = 16 for each mean.

McGlashan et al.'s (1969) subjects were very familiar with the laboratory and had participated in at least four previous hypnotic susceptibility testing sessions wherein they performed consistently high or low. Although the experimenter was blind to susceptibility (as was the experimenter in this study), subjects may still have known they were being evaluated as "high" or "low" susceptibles.

Despite this failure to replicate, the finding of greatest importance concerns the rate of work done. Following hypnotic analgesia, despite the overall increase in volume and duration pumped, the rate of work showed a significant decline. Thus, subjects pumped fewer ml/sec after hypnotic analgesia than before. Given a physiological upper limit to the amount of time and water a subject could pump, one manner of complying with the suggestion was simply to pump less water per squeeze of the bulb. Subjects could easily have lengthened their pumping time in such a manner without defining the situation to themselves or the experimenter as faking. Rather, by paying less attention to the task and distracting themselves in some manner they could increase their duration while simultaneously reducing their output.

The main importance of this finding is that it confirms the problems raised with regard to the McGlashan et al. (1969) study. Namely, it is clear that volume and duration

of pumping do not measure pain per se. Whether any measure ever does is a moot point. Nevertheless, the present measures have clear upper limits determined by fatigue and not pain alone.

Further evidence in support of this notion comes from two sources. The first is subjects' cognitive activities, and the second is subjects' ratings of arm-tiredness. Subjects' responses to the catastrophizing, coping and percent time imagining scales were similar to those reported in previous studies (e.g., Spanos, Stam, & Brazil, in press; Stam & Spanos, 1980). High susceptibles typically report more coping cognitions following suggestions for analgesia than do low susceptibles. In previous studies however there were clear relationships between reported pain and these measures. In the present study there was a clear absence of any relationship between the performance measures and the cognitive activity measures. Each set of measures was internally consistent however. Thus, one interpretation of this outcome is that the performance measures are unrelated to the cognitive measures because the former are not directly related to experienced pain. Rather they are complicated by the fatigue inherent in the task.

This becomes evident as well in the results of subjects' ratings of arm-tiredness. Subjects did not differ in ratings of tiredness before or after the suggestion. High

and low susceptibles also did not differ. Overall, 88% and 84% of subjects indicated they had not reached maximum pain by Trials 1 and 2, respectively. High susceptibles estimated they could have continued well over a minute on Trial 2 had their arm not become fatigued. These results are clearly grounds for skepticism in evaluating the McGlashan et al. results.

Evans (1969) reported that subjects had been asked to estimate the amount of water they had displaced. A partial analysis of these results was presented and indicated that highs and lows did not differ in their estimates (at least following their placebo trial). Subjects in the present study were more accurate in estimating time than volume. Both estimates however increased following suggestion reflecting the actual increases in duration and volume pumped. Indirect estimates of work based on these measures indicated that subjects rated themselves as working harder following hypnotic analgesia than prior to it while, in fact, subjects' performance dropped significantly.

In summary, these results indicate that the performance of a continuous exercise task during ischemia is not necessarily painful. The measures are confounded by fatigue and appear not to be reliable estimates of experienced pain. Furthermore, subjects quite readily acknowledged their inability to continue because of fatigue. Consequently, the

nature of the relationship between placebo analgesia and hypnotic susceptibility cannot be determined from the McGlashan et al. study.

EXPERIMENT 2

The relationship between hypnotic and placebo analgesia was specifically addressed in this experiment. As was noted in the introduction, subjects receiving the two treatments are asked to perform different tasks that engender different expectations. High susceptible subjects receiving hypnotic analgesia appear to engage in a variety of cognitive strategies with some facility. It was hypothesized that these same subjects would not engage in any such manoeuvres when they received a placebo but would, instead, wait for "something" to occur. Low susceptibles, on the other hand, may approach both situations with a passive attitude. That is, for them hypnotic and placebo analgesia, both, are manipulations which do not require anything of them. Their low score on the susceptibility scale likely precludes any high expectations of hypnotic analgesia. Thus, a wait-and-see approach to both analgesia situations is the most likely strategy of low susceptibles.

Experiment 2 also evaluated the relationship between anxiety and ischemic pain. The relationship between trait and state anxiety and ischemic pain is still confused at best. Finally, this experiment allowed for an assessment of the psychophysics of ischemic pain.

Design

This experiment consisted of a 2 X 2 X 3 split-plot design. Subjects (a) were high or low in hypnotic susceptibility, (b) received hypnotic analgesia and placebo or no treatment, and (c) were tested on three ischemic pain trials.

MethodSubjects

Thirty high susceptible and 30 low susceptible subjects volunteered to participate in an experiment on pain perception. Each susceptibility group consisted of 15 males and 15 females. All subjects had completed the CURSS in their first session and thus constituted a sample from a larger pool of introductory psychology students. High susceptibles scored five and above on the CURSS:0 whereas lows scored zero. The same exclusion criteria used in Experiment 1 were used here. All subjects had their blood pressure taken to determine if it was within normal limits. Mean ages were 19.7 (18-26) for males and 19.2 (17-23) for females. Six subjects were replaced; three for keeping their arm in the pressure cuff beyond 20 min on the baseline trial, and three for keeping their arm in the cuff less than 2.5 min during the baseline trial. All six were low susceptibles (5 females and 1 male). They were not deviant with respect to any other variable.

Apparatus

Maximal grip was measured using a Lafayette (#78010) hand dynamometer calibrated from 0 to 100 kilograms. Subjects squeezed the dynamometer twice with each hand. A blood pressure cuff from an Avenue (#12-300) portable mercury sphygmomanometer was placed on an arm (left and right were counterbalanced). An Esmarch bandage was wrapped from the fingers to the elbow while the arm was extended upward to drain it of venous blood. The cuff was then inflated to 200 mm Hg. Duration was measured by a stopwatch beginning at the point of inflation. The arm was lowered and tape-recorded instructions told subjects to squeeze the dynamometer to the point at which the marker was located (half their maximal strength). This was done every 4 sec and subjects held their hands clenched for 2 sec for a total of 20 times. Subjects rested their arms for the remainder of the ischemia trial. If subjects had not reached tolerance after 20 min the trial was terminated by the experimenter.

For their placebo session, subjects inserted their hand and forearm into a 10 cm X 15 cm opening in a large instrument panel. This "machine" measured approximately 100 cm X 45 cm and was 55 cm deep. It consisted of an array of lights, switches, wires, plugs, and a tachistoscope eye piece. When the various components were turned on, the "machine" produced a low level noise and various panels lit

up. A red light bulb was on in the space where subjects rested their arm. None of these units served any functional purpose however beyond legitimating the claim of using a technologically advanced piece of equipment. It was also perfectly safe to the subject despite a "danger - high voltage" sign placed on the front. The entire equipment bank was assembled inside a sound ~~attenuated~~ audiometric booth. A concentric ring electrode was attached to the subject's arm to "measure" the effects the machine was having. This electrode was a dummy electrode attached to a clearly visible polygraph outside the booth.

Procedure

Session 1. All subjects were ~~pre~~tested on the CURSS in this session following the procedures described in Experiment 1.

Session 2: Baseline. During this session subjects were first given an informed consent agreement to sign (see Appendix G). This agreement asked subjects to participate in three sessions of ischemic pain from which they could withdraw at any time without penalty. Subjects then filled out the State-Trait Anxiety Inventory (STAI; Spielberger, Gorusch, Lushene, 1970), and a symptom checklist (Appendix H). The reason for presenting the check-list at this time was to obtain baseline data. Following this they had the procedures described to them and were taught a magnitude

estimation procedure taken from Stam et al. (1981, Experiment 1).

A familiarization task was used to acquaint subjects with the general ME procedure. Various lines were assigned a number of the subjects own choosing and all other lines were judged relative to the first. Subjects were then instructed that when asked, they were to report a number that corresponded to the intensity of the pain they were feeling. The first number could be any number of their own choosing while successive numbers were to reflect their subjective impression (see Appendix I). This procedure allowed a test of the hypothesis that findings of negatively accelerating functions of reported pain over time are an artifact of bounded response scales.

Once the procedures were understood, subjects removed any jewelry and watches and then underwent the ischemic pain procedure described above. They were instructed beforehand that they were to tell the experimenter the point at which their arm hurt "so much that you cannot continue for even one more second." These instructions were modified from Experiment 1 by simply removing any references to pumping water. Every 30 sec following the inflation of the cuff subjects were asked to "report" and give a numerical estimate of their pain.

When the subject indicated that he/she had reached tolerance the cuff was deflated. Subjects then estimated their tolerance time and completed the same series of questionnaires as those used in Experiment 1 with the exception of the questions concerning arm-tiredness.

Session 2: Trial 2 & 3. Within each susceptibility level (a) 10 subjects received hypnotic analgesia during trial 2 and placebo analgesia during trial 3, (b) 10 subjects received placebo analgesia during trial 2 and hypnotic analgesia during trial 3, and (c) 10 subjects received no treatment during either trial 2 or trial 3. All subjects completed the state form of the STAI and the symptom checklist just prior to their second and third trial. Subjects about to receive an hypnotic or placebo analgesia trial also filled in a visual analogue scale asking them to rate the extent to which they thought the treatment they had received would reduce their pain. The scale was bounded by "no reduction - I expect no pain relief" on one end and "full reduction - I expect complete pain relief" on the other end.

Hypnotic analgesia. The procedures for hypnotic analgesia were almost identical to those in Experiment 1. Subjects rested for 10 min, were assured they were excellent subjects (the experimenter was blind to hypnotic susceptibility), were given a standard induction procedure

and suggestions for analgesia. The only change in procedure from Experiment 1 consisted of having subjects fill out the state form of the STAI, the symptom checklist, and the VAS following the induction and suggestions. This was done with the arm opposite from the arm to be used in this trial. When subjects had completed this relatively brief task they were given further suggestions for relaxation and analgesia lasting approximately 1 - 2 min. Then the ischemic pain task was completed. Subjects again estimated their tolerance time and completed the questionnaires.

Placebo analgesia. All subjects underwent a brief, sham medical "history" to determine the presence of any muscular or dermatological disorders. The placebo "machine" was then described to subjects as a "cold laser." Subjects received a brief lecture on the physics of lasers and were shown a newspaper clipping describing the effectiveness of this equipment in combating arthritic pain (see Appendix J). They were then told that this "laser" was capable of substantially reducing the pain they would experience with the tourniquet..

One concentric ring electrode was then attached to the subject's upper arm with the explanation that they would monitor the action of the "laser." Ostensibly to protect their eyes from the laser, subjects had prism goggles (set at 20 diopters, base right) placed over their eyes for the

duration of their exposure. This was done to accentuate any aftereffects of the placebo.

Subjects were led into a sound attenuated audiometric booth with a window. The experimenter controlled the operation of the "laser" from outside the booth. The arm to be used for ischemia on that trial was placed in the opening of the machine and the machine was turned on. Subjects sat silently in front of the machine for 10 min.

Following exposure to the "laser," the goggles were removed and the symptom check-list and STAI were filled out using the arm not placed in the machine. Subjects were told that the "laser" would take a few minutes to take effect and that they would fill in these forms while they waited. The ischemic pain task was then re-introduced and completed as before. Finally, subjects estimated their time to tolerance and completed the questionnaires.

Although subjects knew they would receive three trials, they were never informed of the nature of the treatments until treatment administration. This was necessary to avoid carry-over effects (see Stam & Spanos, 1980). Prior to Trial 3, all experimental subjects were read an explanation taken from McGlashan et al. (see Appendix K). It informed subjects that the experimenter was interested in comparing the "laser," a known physiological process to a psychological process like hypnosis.

All subjects were questioned with regard to their belief in the reality of the "laser" following their third trial. Although subjects were not always sure how much pain relief they would obtain (as discussed below), no subject expressed doubts about the reality of the laser. Finally, subjects were thoroughly debriefed and thanked for their participation.

Results

Tolerance

Tolerance scores (in sec) varied considerably between subjects on all three trials. Table 26 presents the mean tolerance scores for each group as well as the means, medians, standard deviations, and the range for each trial. Skewness was less than 1 for any trial. Because of this variability, the tolerance scores were subjected to an analysis of covariance (ANCOVA) with tolerance on the baseline trial as the covariate (see Table 27). Susceptibility (high vs. low) and Group were between-subjects variables and Trials (2 and 3) was a within-subjects variable. The Groups variable consisted of three levels: (a) the treatment order Baseline - Placebo - Hypnosis (B/P/H); (b) the order Baseline - Hypnosis - Placebo (B/H/P); and (c) Control or no treatment.

On the basis of McGlashan et al.'s results clear predictions had been made regarding the relationship between

Table 26

Summary Statistics for Tolerance Scores

Susceptibility	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
	B/P/H					
High	458.8	236.0	473.1	252.6	585.7	248.5
Low	550.5	172.7	544.8	194.2	577.5	152.1
	B/H/P					
High	433.2	256.8	634.1	281.9	576.5	290.2
Low	375.1	145.2	427.2	172.5	386.3	119.7
	Control					
High	615.2	163.3	604.5	116.2	561.1	176.8
Low	460.1	238.0	548.5	241.8	433.5	186.7
Overall M	482.2		538.7		520.1	
Overall sd	213.0		219.7		210.9	
Overall Md	483.5		522.5		483.5	
Overall Range	934.0		1032.0		1005.0	

Table 27

Summary Table for Group x Susceptibility x Trial
ANCOVA on Duration to Tolerance.

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	44353.09	1.53
Susceptibility (B)	1	92628.94	3.19
A x B	2	82906.34	2.85
Subjects Within Groups	53	29081.46	
Trial (C)	1	10378.80	<1
A x C	2	64691.73	5.26*
B x C	1	15142.53	1.23
A x B x C	2	7165.56	<1
C x Subjects Within Groups	54	12296.58	

*p < .01

placebo and hypnotic analgesia and hypnotic susceptibility. It was expected that low susceptibles would show equivalent reductions in pain for hypnotic and placebo analgesia whereas high susceptibles would show greater reductions following hypnotic analgesia than placebo analgesia. To test these predictions orthogonal a priori t-tests on the adjusted means from the ANCOVA (depicted in Figure 1) were carried out. Two effects achieved significance: the difference between placebo and hypnotic analgesia in high susceptibles in group B/P/H, $t(54) = 3.23$, $p < .05$, and the difference between Trials 2 and 3 for low susceptibles in the control group, $t(54) = 3.29$, $p < .05$. In other words, high and low susceptibles in group B/H/P and low susceptibles in group B/P/H did not differ from hypnotic to placebo analgesia. High susceptibles in the latter group did show an increase however. Thus, the results expected on the basis of McGlashan et al.'s findings were obtained only in group B/P/H.

The ANCOVA resulted in a significant Group x Trial interaction, $F(2,54) = 5.26$, $p < .01$. No other effects achieved statistical significance. The interaction is presented in Figure 2. Post hoc comparisons carried out using t-tests indicated that group B/P/H significantly increased in tolerance from placebo to hypnotic analgesia trials whereas control subjects significantly decreased from

Figure 1
Adjusted Mean Tolerance for Group X Susceptibility X Trial Interaction

Figure 2
Adjusted Mean Tolerance for Group X Trial
Interaction

Trial 2 to Trial 3. Group B/H/P did not change significantly from the hypnosis to the placebo trial however. On Trial 2 the two treatment groups differed significantly from one another (but not from the control group). On Trial 3 the two treatment groups did not differ from one another but both differed from the control group. Thus, hypnotic and placebo analgesia were equivalent between groups on Trial 3 but not on Trial 2.

To examine the relationship between hypnotic and placebo analgesia more closely, subjects were divided into those who did or did not exhibit a placebo response. In group B/P/H, 10 subjects increased their time to tolerance from baseline following placebo analgesia while 10 did not. In group B/H/P, 15 subjects responded positively while 5 did not. This difference was not significant, $\chi^2(1) = 2.67$. Among high susceptibles, 15 subjects responded positively to the placebo manipulation whereas 5 did not. Low susceptibles, on the other hand, were divided equally into those who responded (10) and those who did not (10). This difference was also not significant, $\chi^2(1) = 2.67$.

The difference between each subjects' baseline trial and their placebo and hypnotic analgesia trials were calculated and intercorrelated. For high susceptible subjects in group B/P/H this correlation was high and statistically significant, $r = .75$, $p < .05$. Thus, subjects who increased

their tolerance following placebo analgesia were also those subjects who increased their tolerance following hypnosis. The relationship between increases in tolerance for the two treatments was not significant for lows in group B/P/H, $r = .39$, highs in group B/H/P, $r = .18$, or lows in group B/H/P, $r = .28$. Collapsing across susceptibility, the correlations were, $r = .60$, $p < .01$, for group B/P/H and $r = .40$, $p < .08$, for group B/H/P.

Magnitude Estimates

Parameters. For each subject and for each trial the slope and the intercept were calculated from the magnitude estimates (ME). Similarly, after converting all MEs and durations to logarithms slopes and intercepts were calculated for the resultant log-linearized functions. These parameters are equivalent to the exponents and constants of Equation 1 - the power function. Mean squared coefficients of determination are presented in Table 28. The two sets of coefficients are roughly identical and highly correlated ($r = .93$, $.95$, and $.96$ for Trials 1 to 3, respectively). Thus the MEs are fairly well described by linear as well as power functions.

The slopes and intercepts from the linear and log-linearized functions were each subjected to a 3 (Groups) x 2 (Susceptibility) x 3 (Trials) split-plot ANOVA. The results of these four ANOVAs are presented in Tables 29 and

Table 28
Summary Statistics for R^2 for Both Power
and Linear Functions

	Trial					
Susceptibility	1	2	3	1	2	3
	Power-M			Lin-M		
	B/P/H					
High	.8305	.8235	.7430	.8339	.8185	.7856
Low	.9232	.9401	.8990	.9065	.9208	.8943
	B/H/P					
High	.9337	.8305	.9277	.9152	.8438	.9538
Low	.8938	.9422	.9475	.8940	.9582	.9510
	Control					
High	.9055	.9040	.9165	.9171	.9224	.9572
Low	.8722	.9134	.9326	.8622	.9287	.9460
Overall M	.8931	.8923	.8944	.8881	.8987	.9146
Overall Md	.9345	.9516	.9523	.9321	.9579	.9657

30. As is clear from these tables, there were no statistically significant effects. The parameters of the linear and power functions were not affected by any of the treatment variables.

The slopes and intercepts derived from the linear and power functions were correlated with time to tolerance. These correlations are presented in Table 31. Although the slopes are negatively correlated with tolerance, their magnitude is small, the largest correlation accounting for less than 7% of the variance. The correlations between the intercepts and tolerance are equally small.

Common modulus. Subjects MEs were reduced to a common modulus using the procedure outlined by Stevens (1975). This was necessary because subjects were not assigned a modulus. As a consequence, they exhibited large individual differences in their use of numbers. Every subject whose tolerance time equalled or exceeded 5 min (300 sec) on all three trials was included in the analysis. On this basis, 3 and 2 subjects from the high and low susceptible B/P/H groups, respectively, 4 subjects from each of the B/H/P groups, and 1 subject from each control group were excluded. Thus, 45 or 75% of the subjects were included in the analysis. Only MEs up to 5 min were used.

After all scores had been transformed to logarithms, the difference between a subject's mean and the grand mean was

Table 29

Summary Table for Group x Susceptibility x Trial ANOVAs
on Intercepts for Power and Linear Functions

Source	<u>df</u>	<u>Pow-MS</u>	<u>Pow-F</u>	<u>lin-MS</u>	<u>lin-F</u>
Group (A)	2	.64	1.55	.0412	2.24
Susceptibility (B)	1	.35	<1	.0052	<1
A x B	2	.56	1.35	.0339	1.84
Subjects Within Groups	54	.42		.0185	
Trial (C)	2	.10	2.08	.0040	2.10
A x C	4	.01	<1	.0023	1.20
B x C	2	.09	2.02	.0007	<1
A x B x C	4	.03	<1	.0002	<1
C x Subjects Within Groups	108	.05		.0019	

Table 30

Summary Table for Group x Susceptibility x Trial ANOVAs
on Intercepts for Power and Linear Functions

Source	<u>df</u>	<u>Pow-MS</u>	<u>Pow-F</u>	<u>lin-MS</u>	<u>lin-F</u>
Group (A)	2	2.58	1.05	2363.25	3.16
Susceptibility (B)	1	2.56	1.05	262.69	<1
A x B	2	3.06	1.25	2374.96	3.18
Subjects Within Groups	54	2.45		747.94	
Trial (C)	2	.98	2.65	36.10	<1
A x C	4	.16	<1	77.76	<1
B x C	2	.68	1.84	39.73	<1
A x B x C	4	.23	<1	48.89	<1
C x Subjects Within Groups	108	.37		111.32	

Table 31
Correlations Between Tolerance and Slopes
and Intercepts by Trial

Function	Trial		
	1	2	3
Slopes			
Linear	-.23	-.12	-.09
Power	-.16	-.26*	-.24
Intercepts			
Linear	.29*	.18	-.05
Power	.16	.26*	.16

* $p < .05$

Note. $n = 60$

added to each of the subject's individual judgements. This procedure equated the mean estimates across the three trials for all subjects.

When reduced to a common modulus, the MEs were subjected to a 3x2x3x8 split-plot ANOVA with two between-subjects variables (3 Groups; High/Low Susceptibility) and two within-subjects variables (3 Trials; 8 Time periods of 30 sec). The results are presented in Table 32.

Three effects achieved statistical significance: the main effects for Trials, $F(2,78) = 7.35$, $p < .01$, and Time, $F(7,273) = 93.87$, $p < .01$, and the Group x Trial interaction, $F(4,78) = 6.39$, $p < .01$. The means for each of these effects are presented in Table 33 and the Group x Trials interaction is plotted in Figure 3. Newman-Keuls post hoc multiple comparisons on the means involved in the interaction indicated that hypnotic analgesia in group B/P/H resulted in significantly lower MEs than either the baseline or placebo trial. No other within-subjects comparisons approached significance. Analyses of the between-subjects comparisons indicated that the hypnotic analgesia trial in group B/P/H was rated as significantly less painful than the third trial of the remaining two groups. No other differences approached significance. Thus, the only significant treatment effect for the transformed MEs occurred in group B/P/H. Subjects in this group reported

Table 32

Summary Table for Group x Susceptibility x Trial x Time
ANOVA on Transformed Magnitude Estimates

Source	df	MS	F
Group (A)	2	2.1×10^{-6}	<1
Susceptibility (B)	1	1.6×10^{-7}	<1
A x B	2	6.3×10^{-7}	<1
Subjects Within Groups	39	3.2×10^{-5}	
Trial (C)	2	1.577	7.35*
A x C	4	1.371	6.39*
B x C	2	.258	1.20
A x B x C	4	.288	1.34
C x Subjects Within Groups	78	.215	
Time (D)	7	5.152	93.87*
A x D	14	.047	<1
B x D	7	.013	<1
A x B x D	14	.019	<1
D x Subjects Within Groups	273	.055	
C x D	14	.006	<1
A x C x D	28	.007	<1
B x C x D	14	.013	1.42
A x B x C x D	28	.011	1.24
CxDxSubjects Within Groups	546	.009	

*p < .01

Table 33

Mean Adjusted Log Magnitude Estimates for Group x Trial
Interaction, Trial Main Effect, and Time Main Effect

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	1.13	.26	1.16	.23	.84	.35
B/H/P	1.12	.26	1.01	.27	1.00	.36
Control	1.02	.27	1.04	.25	1.06	.27
Overall	1.09	.27	1.07	.25	.97	.31
Time (sec)	<u>M</u>	<u>sd</u>				
90	.67	.29				
120	.86	.19				
150	.98	.16				
180	1.06	.19				
210	1.12	.20				
240	1.18	.20				
270	1.22	.22				
300	1.26	.23				

Figure 3

Mean Transformed Log Magnitude Estimates for Group X Trial

Interaction

less pain during hypnotic analgesia than during the previous two trials. They also reported less pain than the other groups during Trial 3.

To examine the change in transformed MEs over time, the MEs were plotted separately for each group and susceptibility level. They are presented in Figures 4 to 6. It is clear from these plots (on linear axes) that any treatment effects were in fact localized to group B/P/H (Figure 4). The slight trend for changes over trials evidenced in group B/H/P (Figure 5) was not strong enough to achieve statistical significance. Appendix L presents the average data, prior to transformation to a common modulus, for each group and trial.

Cognitive Activities

Catastrophizing. Subjects' scores on the visual analogue catastrophizing scale were subjected to a 3 (Groups) x 2 (Susceptibility) x 3 (Trials) split-plot ANOVA. The summary table is presented in Table 34. Two effects achieved statistical significance; the main effect for Groups, $F(2,54) = 4.40$, $p < .05$, and the Group x Trial interaction, $F(4,108) = 4.49$, $p < .01$. The means for this interaction are presented in Table 35. Newman-Keuls post hoc multiple comparisons indicated that the two treatment groups reported a relatively constant low level of catastrophizing across trials while the control group reported significantly greater catastrophizing by Trial 3 than on Trial 1 or 2.

Figure 4

Mean Magnitude Estimates for Group B/P/H

Figure 5
Mean Magnitude Estimates for Group B/H/P

Figure 6

Mean Magnitude Estimates for Control Group

Table 34

Summary Table for Group x Susceptibility x Trial

ANOVA on Catastrophizing VAS

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	4417.01	4.40*
Susceptibility (B)	1	68.45	<1
A x B	2	777.05	<1
Subjects Within Groups	54	1003.96	
Trial (C)	2	308.96	<1
A x C	4	1706.67	4.49*
B x C	2	144.27	<1
A x B x C	4	277.52	<1
C x Subjects Within Groups	108	380.00	

* $p < .01$

Table 35
Catastrophizing VAS Means for
Group x Trial Interaction

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	26.05	21.08	28.25	18.78	12.55	12.54
B/H/P	28.65	20.72	18.80	24.92	27.15	23.27
Control	35.65	28.42	30.60	27.30	48.55	32.76

Coping. A 3x2x3 ANOVA on the visual analogue coping scale resulted in a statistically significant Susceptibility main effect, $F(1,54) = 4.25$, $p < .05$, and a significant Group x Trial interaction, $F(4,108) = 7.56$, $p < .05$. The ANOVA summary table is presented in Table 36 and the means for the significant interaction in Table 37. The Susceptibility main effect indicated that in general highs ($M = 46.8$) reported greater coping than lows ($M = 33.7$). Newman-Keuls post hoc multiple comparisons on the interaction indicated that subjects in group B/P/H reported significantly greater coping on the third trial than on the first or second trial. No other differences approached significance.

Percent time imagining. Subjects' ratings of the percentage of time spent imagining events inconsistent with pain were subjected to a 3x2x3 split-plot ANOVA (see Table 38). The main effect for Trials achieved statistical significance, $F(2,108) = 4.47$, $p < .05$, as did the Group x Trial interaction, $F(4,108) = 5.28$, $p < .01$. The means involved in the interaction are presented in Table 39. Newman-Keuls post hoc multiple comparisons indicated that subjects in group B/P/H scored higher on Trial 3 than either Trial 1 or 2. No other effects were statistically significant. These results are identical to those found with the coping scale.

Table 36

Summary Table for Group x Susceptibility x Trial
ANOVA on Coping VAS

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	222.62	<1
Susceptibility (B)	1	7644.05	4.25*
A x B	2	121.72	<1
Subjects Within Groups	54	1798.54	
Trial (C)	2	1427.55	3.07
A x C	4	3520.87	7.56**
B x C	2	148.12	<1
A x B x C	4	211.13	<1
C x Subjects Within Groups	108	465.54	

* $p < .05$

** $p < .01$

Table 37
Mean Coping VAS Scores for
Group x Trial Interaction

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	36.75	29.19	27.60	27.01	60.55	33.11
B/H/P	37.10	34.33	52.45	27.87	33.65	31.92
Control	31.45	30.13	42.50	30.29	40.20	29.50

Table 38

Summary Table for Group x Susceptibility x Trial
ANOVA on Percent Time Imagining

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	324.29	<1
Susceptibility (B)	1	2960.46	1.81
A x B	2	3213.16	1.96
Subjects Within Groups	54	1637.64	
Trial (C)	2	2093.49	4.47*
A x C	4	2472.80	5.28**
B x C	2	80.69	<1
A x B x C	4	436.21	<1
C x Subjects Within Groups	108	468.61	

* $p < .05$

** $p < .01$

Table 39

Mean Percent Time Imagining Scores For
Group x Trial Interaction

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	33.85	25.79	32.00	25.86	58.65	32.33
B/H/P	34.95	34.38	46.15	28.40	30.30	31.27
Control	28.80	28.32	41.25	30.30	43.75	29.10

The means and standard deviations of the catastrophizing, coping and percent time imagining scores for each cell of the design are presented in Appendix M.

Cognitive activities and pain measures. Table 40 presents the intercorrelations among the three scales of cognitive activities and subjects' time to tolerance, the slopes from their linear functions, and the slopes from their power functions. Only the catastrophizing scale was related to the pain measures. On all three trials there was a positive relationship between the slopes obtained from the power functions and subjects' ratings of the extent of catastrophizing ideation. Also, tolerance on Trial 3 was negatively related to catastrophizing.

Correlations were also calculated among the cognitive activity measures and tolerance difference scores. There were no statistically significant correlations between differences in tolerance for Trial 2 and these measures. Difference scores for Trial 3 however were significantly related to catastrophizing, $r = -.35$, $p < .01$, coping, $r = .29$, $p < .05$, and percent time imagining, $r = .28$, $p < .05$.

Anxiety and Symptoms

Subjects' scores on the State Anxiety Inventory were subjected to a 3x2x3 split-plot ANOVA (see Table 41). The only statistically significant effect was the Group x Trial interaction, $F(4,108) = 5.10$, $p < .01$. The means involved

Table 40

Correlations Between Cognitive Activities and Tolerance
and Linear and Power Function Slopes

	Trial								
	1			2			3		
	Tol	Lin	Pow	Tol	Lin	Pow	Tol	Lin	Pow
	Slope	Slope	Slope	Slope	Slope	Slope	Slope	Slope	Slope
Catastr.	-.05	0.20	.25*	0.04	.28**	.30**	-.28**	0.12	.25*
Coping	-.02	-.12	0.04	-.02	-.03	0.09	0.18	-.04	-.06
Time Imag.	-.17	-.11	0.04	0.05	-.18	-.13	0.15	-.13	0.00

*p < .06

**p < .05

Table 41

Summary Table for Group x Susceptibility x Trial

ANOVA on State Anxiety

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	54.17	<1
Susceptibility (B)	1	33.80	<1
A x B	2	25.32	<1
Subjects Within Groups	54	89.69	
Trial (C)	2	82.22	2.74
A x C	4	152.96	5.10*
B x C	2	64.80	2.16
A x (B x C)	4	4.77	<1
C x Subjects Within Groups	108	30.00	

* $p < .01$

in this interaction are presented in Table 42. Newman-Keuls post hoc multiple comparisons indicated that subjects in group B/P/H reported significantly less anxiety prior to Trial 3 than prior to either Trial 1 or 2.

The number of reported symptoms from the checklist were also subjected to a 3x2x3 split-plot ANOVA and the results are presented in Table 43. Again, the only statistically significant effect was the Group x Trial interaction, $F(4,108) = 4.52, p < .01$. Newman-Keuls post hoc multiple comparisons on the means involved in the interaction indicated that subjects in group B/P/H reported significantly fewer symptoms just prior to their third trial than prior to either Trial 1 or 2. The control group however reported significantly more symptoms on Trials 2 and 3 than on the first trial. Trials 2 and 3 did not differ significantly. The means involved in the interaction are presented in Table 44.

State anxiety scores, reported symptoms and subjects' scores on the trait anxiety version of the STAI were correlated with time to tolerance and the slopes derived from the linear and power magnitude estimation functions for each trial. The correlations between the anxiety measures and the pain measures were small and statistically nonsignificant. Reported symptoms however correlated significantly with state anxiety on Trial 1 ($r =$

Table 42

State Anxiety Means for Group x Trial Interaction

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	34.10	10.72	33.20	6.62	27.20	5.09
B/H/P	32.45	6.30	28.15	5.25	30.95	5.76
Control	31.55	7.09	32.75	6.15	32.95	8.02

Table 43

Summary Table for Group x Susceptibility x Trial

ANOVA on Symptoms

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	29.04	1.93
Susceptibility (B)	1	9.34	<1
A x B	2	20.54	1.37
Subjects Within Groups	54	15.04	
Trial (C)	2	3.84	2.00
A x C	4	8.70	4.52*
B x C	2	2.44	1.27
A x B x C	4	.91	<1
C x Subjects Within Groups	108	1.92	

* $p < .01$

Table 44

Symptom Means for Group x Trial Interaction

Group	Trial					
	1		2		3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	2.30	3.31	2.70	1.92	1.05	1.61
B/H/P	2.60	2.01	2.30	1.56	2.30	2.18
Control	2.75	2.86	3.60	2.93	3.75	3.40

.70, $p < .01$), Trial 2 ($r = .47$, $p < .01$), and on Trial 3 ($r = .50$, $p < .01$). Symptoms also correlated significantly with trait anxiety on Trial 1 ($r = .56$, $p < .01$), Trial 2 ($r = .35$, $p < .01$), and Trial 3 ($r = .28$, $p < .05$).

Subjects' baseline tolerance scores were subtracted from their Trial 2 and 3 tolerance scores and these differences were also correlated with symptoms, state anxiety and trait anxiety. These correlations are presented in Table 45 and indicate that increases in tolerance were associated with lower state anxiety on Trial 2 and 3 and fewer reported symptoms on Trial 3. There was no statistically significant relationship between increases in tolerance and trait anxiety however.

To further examine the relationships among state anxiety, symptoms and placebo responding subjects who exhibited increased tolerance following placebo analgesia were compared with nonresponders on these variables. Responders in group B/P/H reported significantly less state anxiety prior to the placebo trial ($M = 29.8$, $sd = 3.6$) than subjects who did not respond ($M = 36.6$, $sd = 7.3$), $t(18) = 2.63$, $p < .05$. Responders in group B/H/P ($M = 30.4$, $sd = 6.3$) however did not differ from nonresponders ($M = 32.6$, $sd = 3.7$), $t(18) = .73$, $p = ns$, on state anxiety. In both groups responders did not differ significantly from nonresponders on reported symptoms or trait anxiety.

Table 45

Correlations Between Tolerance Difference Scores
and Symptoms and Anxiety

	Trial 2	Trial 3
Symptoms	-.14	-.30*
State Anxiety	-.36**	-.32*
Trait Anxiety	.09	-.03

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

State anxiety, trait anxiety and reported symptoms were correlated with the cognitive activity measures (see Table 46). State anxiety was negatively related to percent time imagining on Trials 2 and 3 and reported symptoms were related to catastrophizing on Trial 3. None of the other correlations were statistically significant.

Expectancy

Subjects in the two treatment groups rated the extent to which they expected the treatment to reduce their pain prior to Trials 2 and 3. These ratings were subjected to a 2 (Groups) x 2 (Susceptibility) x 2 (Trials 2 and 3) split-plot ANOVA. The results are presented in Table 47. Three effects achieved statistical significance; the Susceptibility main effect, $F(1,36) = 7.83, p < .01$, the Group x Trial interaction, $F(1,36) = 6.30, p < .05$, and the Susceptibility x Trial interaction, $F(1,36) = 5.95, p < .05$. The means involved in the two significant interactions are presented in Table 48.

Newman-Keuls post hoc multiple comparisons indicated that none of the pairwise differences on the Group x Trial interaction were statistically significant at the .05 level. The difference between groups B/P/H and B/H/P at Trial 3 approached significance ($p < .10$) indicating that the former group expected greater pain reduction with hypnosis than the latter group expected with placebo analgesia. The two

Table 46

Correlations Between Cognitive Activity
Measures and Anxiety and Symptoms

	Trial		
	1	2	3
	Catastrophizing		
State Anxiety	-.08	.15	.20
Trait Anxiety	-.21	-.13	-.08
Symptoms	.08	.23	.33**
	Coping		
State Anxiety	-.19	-.19	-.12
Trait Anxiety	-.18	-.19	-.02
Symptoms	-.21	.02	-.13
	Percent Time Imagining		
State Anxiety	-.11	-.24*	-.24*
Trait Anxiety	-.16	-.15	-.11
Symptoms	-.23	-.01	-.12

*p < .06

**p < .05

Table 47

Summary Table for Group x Susceptibility x Trial
ANOVA on Expectancy

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	1	1051.2	1.31
Susceptibility (B)	1	6265.8	7.83**
A x B	1	18.05	<1
Subjects Within Groups	36	800.35	
Trial (C)	1	0.00	<1
A x C	1	3050.45	4.6.30*
B x C	1	2880.0	5.95*
A x B x C	1	1674.45	3.46
C x Subjects Within Groups	36	484.34	

* $p < .05$

** $p < .01$

Table 48
Expectancy Means for Group x Trial and
Susceptibility x Trial Interactions

Group	Trial 2		Trial 3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	33.75	28.98	46.10	31.07
B/H/P	38.85	27.38	26.50	21.19
Susceptibility				
High	51.15	28.71	39.15	31.44
Low	21.45	17.76	33.45	24.72

groups expected equivalent reductions on Trial 2. Post hoc comparisons on the Susceptibility x Trial interaction indicated that high susceptibles expected a greater pain reduction on Trial 2 than low susceptibles. They did not differ significantly on Trial 3. In general, highs expected greater pain reductions ($M = 45$) than lows ($M = 27$).

Correlations among expectancy, and tolerance, tolerance difference scores and slopes from the magnitude estimation functions were all small and nonsignificant. Subjects who responded to the placebo manipulation did not differ from nonresponders on expected pain relief.

Estimated Time

Subjects' estimates of the duration of ischemia were subjected to a 3x2x3 split-plot ANOVA (see Table 49). Two effects achieved statistical significance; the Trial main effect, $F(2,108) = 3.77$, $p < .05$, and the Group x Trial interaction, $F(4,108) = 2.76$, $p < .05$. The means involved in this interaction are presented in Table 50. Newman-Keuls post hoc comparisons indicated that subjects in group B/P/H reported higher estimates following Trial 3 than following Trial 2. No other differences achieved statistical significance. Subjects' estimates of duration were highly correlated with their actual time to tolerance for Trial 1 ($r = .61$, $p < .01$), Trial 2 ($r = .62$, $p < .01$), and Trial 3 ($r = .67$, $p < .01$). Estimated time was not significantly related to any of the cognitive activity measures.

Table 49

Summary Table for Group x Susceptibility x Trial

ANOVA on Estimated Time

Source	<u>df</u>	<u>MS</u>	<u>F</u>
Group (A)	2	96805.14	<1
Susceptibility (B)	1	182723.47	1.61.
A x B	2	91825.14.	<1
Subjects Within Groups	54	113569.58	
Trial (C)	2	64985.97	3.77*
A x C	4	47511.39	2.76*
B x C	2	36610.97	2.12
A x B x C	4	12053.89	<1 ✓
C x Subjects Within Groups	108	17235.14	

*p < .05

Table 50
Mean Estimated Time Scores For
Group x Trial Interaction

Group	Trial 1		Trial 2		Trial 3	
	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>	<u>M</u>	<u>sd</u>
B/P/H	351.00	225.84	337.00	209.87	457.00	360.38
B/H/P	256.25	160.45	340.50	239.66	325.50	189.81
Control	341.50	160.31	417.00	198.50	354.00	192.86

Discussion

The order of presentation of placebo and hypnotic analgesia had a significant effect on the outcome of Experiment 2. The time to tolerance exhibited by group B/H/P was equivalent for hypnotic and placebo analgesia. Group B/P/H however increased in tolerance from placebo to hypnotic analgesia, especially in high susceptible subjects. Furthermore, examining between-group differences, hypnotic analgesia was superior to placebo analgesia on Trial 2 but equivalent to it on Trial 3.

High susceptibles who received placebo analgesia following hypnotic analgesia did not differ in tolerance between the two trials. This is not consistent with the results reported by McGlashan et al. These investigators found a significant decrease in tolerance following placebo analgesia. Recall that McGlashan et al. always administered placebo analgesia on the trial following hypnotic analgesia. An explanation of the present results can be phrased in terms of carry-over effects similar to those found when hypnotic and suggested analgesia are compared within-subjects (Stam & Spanos, 1980). Subjects in the present experiment were never aware of the treatment they would receive until they were at that point of the experiment where the treatment was administered. Thus, subjects in group B/P/H were unaware of the hypnotic analgesia treatment to follow the placebo

analgesia trial. Note that their placebo analgesia response did not differ from that of the control group. Hypnotic analgesia led to significant increases in tolerance however, at least in high susceptibles. High susceptible subjects in group B/H/P already had high tolerance scores by their second or hypnosis trial. Maintaining these high levels of tolerance following exposure to a "laser" may have legitimized their performance following hypnosis. That is, if these subjects reduced their tolerance to the placebo to the level of subjects in group B/P/H, they would be implicitly acknowledging that (a) the "laser" did not work and the experimenter was lying, or (b) the "laser" did not work despite their belief that it would. Since they did not acknowledge publicly any disbelief in the "laser", it may be assumed that at least most subjects believed in the efficacy of the placebo manipulation. Maintaining a high tolerance level thus indicated a belief that their hypnotic analgesia tolerance was as legitimate or "real" as that following the physical "laser" manipulation. On the other hand, since group B/P/H had shown little increase in tolerance during the placebo trial, it was consistent with the high susceptible role for these subjects to improve following hypnotic analgesia.

One reason that these results may differ from those of McGlashan et al. is that these investigators tested subjects

with at least 48 hours between sessions. It may have been difficult for subjects to compare their responses from one session to another. A further contributing factor may have been the short duration of the ischemic pain task (as used in Experiment 1) and its confounding with tiredness of the arm. The lack of a relationship between hypnotic and placebo analgesia that is often propounded may be dependent in part on the manner in which this relationship is tested.

The absence of differences in slopes and intercepts between the groups is not surprising. These parameters were calculated on MES that varied in number depending on subjects' tolerance. Thus, some slopes were based on as few as 8 to 10 scores whereas others were based on as many as 38 scores. Within-group variability was sufficient to preclude any treatment effects on these variables.

The transformed MES indicated that subjects in group B/P/H reported less pain during hypnotic analgesia than during their first two trials. This was the only treatment effect to attain statistical significance. One reason for this may be the relative insensitivity of MES to susceptibility differences in treatments. Spanos, Stam, and Brazil (in press) also found no differences in MES between high and low susceptibles given a suggestion for analgesia. Category ratings however differentiated between these subjects. The underlying reason for such findings is not

clear. Of importance here maybe the difference between generating a large number of pain reports and generating a single overall rating or responding to tolerance. Single ratings and tolerance, in particular, may be more susceptible to expectancy effects in the experimental situation since they are more global pain behaviours.

The absence of susceptibility effects on most of the other variables was common in this experiment. In fact, the interaction between Group and Trials, independent of susceptibility, was statistically significant for each of the dependent variables.

The analyses of the cognitive activity measures indicated an increase in reported coping and percent time imagining events inconsistent with pain for subjects in group B/P/H during Trial 3. The catastrophizing scale indicated greater catastrophizing in the control group by Trial 3. Neither coping nor percent time imagining were directly related to the pain measures. Trial 3 tolerance difference scores were related to all three measures however. Catastrophizing was also related to ME slopes. Exponents of the power ME functions increased with increasing catastrophizing scores. Spanos, Jones, Brown, and Horner (1981) also reported that subjects classified as catastrophizers had higher exponents fitted to MEs obtained during cold-pressor pain stimulation. This relationship was

apparent on a posttest but not on a pretest trial. Overall, the interrelationships between the cognitive activity measures and the independent variables and pain measures were not strong in the present experiment.

State anxiety and reported symptoms were also significantly different over trials in group B/P/H. Prior to Trial 3 subjects in this group had lower scores on both of these measures. The lack of a susceptibility effect on anxiety or symptoms reported following placebo analgesia replicates McGlashan et al. as reported by Evans (1969). It is not clear in what way the relationship between anxiety and the pain measures are related to McGlashan et al.'s experiment however. Evans (1981) contradicts the conclusions he presented in 1974, and in neither case does he present any data for evaluation.

In the present experiment symptoms were significantly related to both state and trait anxiety. State anxiety was negatively related to whether subjects increased in tolerance on the second and third trials. Symptoms were negatively related to increases in tolerance on the third trial. Trait anxiety however was not related to increases in tolerance. Interestingly, only placebo responders in group B/P/H reported less state anxiety than nonresponders. Thus, the negative relationship between anxiety and placebo responding may have been mitigated by the intervening hypnotic analgesia trial in group B/H/P.

While in general there was a relationship between state anxiety and increases in tolerance there was little, if any, relationship between the anxiety measures and the cognitive activity measures and between the cognitive activity measures and the pain measures. The absence of anxiety and catastrophizing ideation were more predictive of the pain measures than the presence of any coping responses. This effect has been found in several other experiments. Spanos et al. (1981) found that suggestions for analgesia typically exerted an effect only to the extent that they transformed baseline catastrophizers into posttest copers. Subjects already engaged in coping ideation did not reduce reported pain or increase their time to tolerance. Baseline catastrophizers however who reported coping on the posttest also reported significantly less pain on the posttest. Spanos et al. (in press) found a similar pattern of results.

Presenting subjects in a laboratory situation with any credible manipulation may be sufficient to reduce anxiety and catastrophizing responses in subjects reporting high pain and low tolerance on a baseline trial. In the present experiment this relationship occurred regardless of the manipulation, i.e., hypnotic or placebo analgesia.

Surprisingly, subjects' expectancy ratings were not related to their subsequent performance on an ischemia trial. That subjects have expectancies about the efficacy

of a treatment is not in doubt. Asking subjects to rate their expectancies however may bias the ratings in some manner. Alternatively, this particular measure may not be useful.

GENERAL DISCUSSION

Experiment 1 provided evidence that the particular task used by McGlashan et al. (1969) confounded pain with fatigue. In fact, the majority of subjects explicitly stated that they could have tolerated higher levels of pain, but were too fatigued to continue pumping. McGlashan et al.'s (1969) conclusions were framed in terms of the effects of placebo and hypnotic analgesia on pain. The present findings indicated that those conclusions were unwarranted, and that firm statements about pain tolerance should not be proffered on the basis of the dependent variables assessed by McGlashan et al.

Experiment 2 attempted to determine the relationship between placebo and hypnotic analgesia using an ischemia task not dependent on the amount of work subjects performed. The variation in the order of presentation of hypnotic and placebo analgesia resulted in the most notable finding. Namely, when placebo analgesia followed hypnotic analgesia the two treatments did not differ. They resulted in equivalent increments in tolerance. When hypnotic analgesia followed placebo analgesia however, the increments for hypnosis were significantly greater than those for placebo in high susceptibles. The placebo manipulation resulted in lower tolerance than hypnotic analgesia when these treatments were presented on the second trial. Placebo and

hypnotic analgesia were equivalent however when compared on the third trial.

These results underscore the lability of placebo responding. Consistent with other investigations (Buckalew et al., 1981; Jospe, 1978; Shapiro & Morris, 1978) they indicate that placebo responding is more situationally determined than dependent on a "placebo personality." This is also corroborated by the negative relationship between anxiety and placebo responding in group B/P/H and the absence of such a relationship in group B/H/P. Subjects presented with the "laser" following a baseline trial were more likely to respond with increased tolerance if they reported relatively lower anxiety just before their trial. The same "laser" manipulation was unrelated to anxiety however when it followed hypnotic analgesia.

The question posed at the beginning of this thesis does not appear then to have a simple answer. The relationship between hypnotic and placebo analgesia is not clearcut but rather is determined by complex situational variables. Subjects' responses may differ if they have undergone other treatments and know that their performance in different treatments is being compared. It has been shown, for example, in clinical trials that placebo responding fades if repeated placebo injections for pain relief are not reinforced with active drugs (see Wall, 1979). Likewise,

Evans (1974) has noted that the strength of the placebo response is often dependent on the strength of the drug with which it is compared. The relative effectiveness of placebo responding needs to be examined further when compared to such experimental manipulations as hypnotic analgesia.

One criticism of Experiment 2 is that it was carried out single-blind. The experimenter was obviously aware that the "laser" was a placebo manipulation. Although this is certainly a valid criticism, it does not explain the dependence of the placebo effect on the order of presentation of the independent variables. It could be argued that these order effects might not occur if the experiment was double-blind. This is an experimental question for which, unfortunately, there is no answer at present. There is no a priori reason however for assuming order effects in single-blind but not double-blind experiments.

An issue that was raised by both experiments and deserves further examination is the notable absence of effects of hypnotic susceptibility on a number of dependant variables. In Experiment 1 there was no effect for susceptibility on any of the ischemia performance measures. In Experiment 2 there was no effect on the ME, catastrophizing, time imagining, and anxiety measures. Several explanations present themselves. The first is

simply that subjects were not selected for hypnotic susceptibility with sufficient rigor and that the CURSS is an ineffective screening instrument for this purpose. This is unlikely because the CURSS has been shown to be a reliable and valid measure of hypnotic susceptibility (Spanos et al., 1982).

A second possible explanation emerges from an examination of the differences between the present experiments and those that have consistently found differences in responding in highs and lows. The main difference consists in the pain stimulus. Susceptibility differences have been observed in a large number of experiments where exposure to a cold pressor or pressure pain stimulus was limited to 60 sec but where all subjects were urged to endure the full minute of stimulation and subsequently did so (e.g., Barber & Hahn, 1962; Evans & Paul, 1969; Hilgard & Hilgard, 1975; Spanos et al., 1974, 1979). Ischemic pain however is measured in terms of the length of time a subject keeps his/her arm in the pressure cuff. Even after eliminating the fatigue artifact by using the SETT technique described in Experiment 2, it is still the subject that controls the duration of any trial. Unlike the studies using a stimulus of fixed duration, subjects in the present experiments were aware that duration was under their control. In other words, subjects had behavioral

control or the availability of a response that would terminate the painful event. Subjects undergoing pain of a shorter but fixed duration such as cold pressor pain do not have behavioural control in the same sense. They can terminate the experiment prematurely if they so desire but they have been requested to endure stimulation for a fixed time. In this case subjects may be more likely to resort to some form of cognitive control or the use of some cognitive strategy aimed at diverting attention from the event.

This analysis is of course speculative and assumes the importance of control over aversive events in human behaviour (see Averill, 1973; Miller, 1979; Thompson, 1981). Nevertheless, it does appear to be consistent with the literature on control and stress. Both behavioural and cognitive control are effective means of coping with aversive events (e.g., Averill, 1973; Thompson, 1981). Aversive situations typically lead individuals to seek out a means to minimize future danger (Miller, 1979). If one has behavioural control, then it is not necessarily advantageous to engage in cognitive control as well. In fact, distracting oneself may lead one not to exercise behavioural control at an appropriate time. Thus vigilance in the face of ischemic pain may be more controlling when one has behavioural control. Nonvigilance may be more controlling (i.e., reduce the aversiveness of the event) when the pain

stimulus is of a short, fixed duration. Consistent with this is a study by Averill, O'Brien, and deWitt (1977) indicating that under conditions of avoidable shock, vigilant subjects experienced less stress than nonvigilant subjects. Under conditions of unavoidable shock however nonvigilant subjects experienced less stress than vigilant subjects. These results suggest that behavioural and cognitive control are interactively and not linearly related.

If the above analysis is correct, then the absence of susceptibility effects may be the result of the lowered need for cognitive control associated with the ischemic pain tasks. The presence of behavioural control mitigated the need for subjects to distract themselves or construct imagery inconsistent with pain. Since these cognitive variables are precisely the ones related to hypnotic susceptibility effects in previous experiments (e.g., Spanos et al., 1979), their relative absence may account for the absence of differences between high and low susceptible subjects on some variables.

A test of this hypothesis could be carried out using ischemic pain. Subjects exposed to a limited period of ischemia (e.g., 5 min) could be compared to those given typical tolerance instructions. Following a baseline, these subjects would then be given a suggestion for analgesia.

Subjects asked to endure ischemia for a fixed period of time should report higher coping cognitions. High susceptibles given suggestions in this group should report less pain than lows. Comparing these subjects to a tolerance instruction group at 5 min should indicate that the latter report fewer coping cognitions and do not show a susceptibility effect following suggestion. A similar test could be conducted by comparing ischemic pain and cold pressor pain in the same subjects.

Notice that this argument does not assume that behavioural control precludes the use of cognitive coping strategies. Subjects in Experiment 2 did increase tolerance following suggested analgesia and, at least in group B/P/H, reported increased coping. This line of reasoning does predict however the presence of stronger effects for cognitive variables and for hypnotic susceptibility in the absence of behavioural control.

The ischemic pain stimulus itself is also an atypical laboratory pain when compared to stimuli such as ice water and electric shock. The pain produced by SETT takes a relatively long time to become intense and the first minutes are not at all painful. Thus, measures that ask subjects the percentage of time they imagined events inconsistent with pain are not going to reflect accurately the extent of coping while subjects were in fact experiencing pain. With

a stimulus such as ice water however, the pain begins within the first 10 to 15 seconds and will reach a maximum at 60 to 90 seconds. Therefore measures of the extent of coping and catastrophizing will more accurately reflect the extent to which subjects engage in these events while they experience pain.

In a similar vein, Chaves and Brown (1978) assessed coping cognitions in patients during stressful dental procedures. Coping and catastrophizing ideation were related to rated stress but not rated painfulness. The latter measure however indicated that the procedures were not very painful. Thus, in the absence of unambiguous painful stimulation, cognitive strategies may not be related to reported pain.

The relatively long duration of ischemic pain, with its nonpainful early phase, may also have contributed to the finding of order effects in Experiment 2. Several other investigators have reported order effects when using the SETT (e.g., Chapman & Feather, 1973; Von Graffenried et al., 1978). The combination of nonpainful and painful periods as well as the provision of behavioural control may make ischemic pain particularly susceptible to expectancies that would result in order effects. Ischemic pain is, overall, a less predictable pain stimulus than cold pressor pain.

For the above reasons, the effects found in Experiment 2 need to be replicated. It may be that the relationship between hypnotic and placebo analgesia is quite different for stronger, more punctate pain stimuli. More importantly, it would then have to be determined what this relationship is in clinical pain patients.

To summarize, Experiment 2 found that hypnotic and placebo analgesia were related depending on the nature of the experimental situation in which they were administered. Hypnotic analgesia was both superior or equivalent to placebo analgesia as a function of the order of testing. Thus placebo responding may not be related to hypnotic susceptibility in an absolute manner. Unlike previous reports, this experiment indicates placebo responding may interact with hypnotic susceptibility and the experimental situation.

The ischemic pain stimulus as it is currently used provides subjects with behavioural control. This may account for the absence of susceptibility effects insofar as behavioural control promotes vigilance and monitoring rather than distracting cognitive activities. Thus hypnotic analgesia and related cognitive strategies may be most effective for pain that is not amenable to behavioural control.

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Footnotes

¹Clinical reports of the use of hypnosis as an analgesic agent are numerous (see Hilgard & Hilgard, 1975). Reviews of this literature indicate that the critical variables in these studies are (a) the suggestions for pain relief, (b) the presence of an interpersonal relationship between the patient and the clinician, and (c) a patient's hypnotic susceptibility level (Barber, 1970; 1979; Chaves & Barber, 1976; Elton, Burrows, & Stanley, 1977; Hilgard & Hilgard, 1975). With a small percentage of patients suggestions for analgesia, regardless of hypnotic induction procedures, appear to ameliorate the experience of pain and reduce the anxiety associated with painful procedures such as surgery (Barber, 1970). In a recent review of the clinical literature Turner and Chapman (1982) argued that more rigorous research is needed to evaluate the clinical use of hypnosis.

²An exception to the finding that reductions in reported pain are correlated with hypnotic susceptibility has been reported by J. Barber (1977). He stated that no correlation was found between susceptibility and reductions in experimental dental pain in 17 student subjects when using

his own particular hypnotic induction procedure ("indirect induction"). He also reported near perfect success with this procedure in reducing pain in a dental patient sample. Unfortunately Barber does not indicate how pain was measured, and provided no quantitative data whatsoever. Two informal attempts to replicate his findings have been unsuccessful however (Hilgard, 1978; Orne, 1980). In fact, Orne (1980) reports a relationship between reduced pain and susceptibility resulted in his laboratory even with J. Barber conducting the induction procedures.

³Hilgard and his associates have also examined the relationship between several of these variables and the perceptual styles of "augmenting" and "reducing" (Hilgard, Morgan, & Prytulak, 1968; Morgan, Lezard, Prytulak, & Hilgard, 1970). According to Petrie (1967), augmenters are those who magnify sensory input while reducers are those who minimize sensory input. Augmenters and reducers are chosen on the basis of their performance on a kinesthetic aftereffects task. Petrie (1967) argued that augmentation is predictive of over-reaction to pain while reducing is predictive of a high tolerance for pain. Morgan et al. (1970) however found that augmenters and reducers did not differ in their responsiveness to cold pressor pain or hypnotic susceptibility. While augmenters reported less

pain than reducers after hypnotic analgesia, the entire experiment is of questionable validity due to the finding (later reported by others as well) that augmentation and reduction are unreliable individual difference measures.

⁴Occasionally investigators have found that high susceptible subjects will report more pain during a baseline pain trial than low or medium susceptible subjects (Crawford, 1978; Hilgard, et al., 1978; Karlin et al., 1980; Shor, 1964). Probably the most parsimonious explanation for this effect would be to consider the high susceptibles increased baseline reports as attempts to report pain consistent with their expectancies. That is, high susceptibles expect to reduce pain with hypnotic analgesia and, therefore, allow themselves room to decrease by reporting greater pain during the pretest (cf., London & Fuhrer, 1961; Stam & Spanos, 1980).

⁵Investigators have long speculated on the nature of the relationship between placebo, acupuncture and hypnotic analgesia (e.g., Chaves & Barber, 1976; Frost, 1978; Knox, Handfield-Jones, & Shum, 1979; Moore & Berk, 1976). Although the analgesic activity of acupuncture is the center of a great deal of controversy, a recent review attributed its persistence as a tool for pain relief to "(1) the

tremendous role played by the high level psychological factors in the maintenance or suppression of the symptom, and (2) our meager understanding of the whole process at all levels" (Sweet, 1981, p. 304). A tendency for high susceptibles to be more responsive to acupuncture than low susceptibles was reported by Knox and Shum (1977) and Knox, Shum and McLaughlin (1978) in experimental subjects and Katz, Kao, Spiegel and Katz (1974) in pain patients. A number of studies, however, have reported no relationship between these two variables or a lack of difference between placebo and acupuncture treatments when these were compared to a hypnotic analgesia group (Knox, Gekoski, Shum, & McLaughlin, 1981; Li, Ahlberg, Lansdell, Gravitz, Chen, Ting, Bak, & Blessing, 1975; Moore & Berk, 1976). Although a relationship may exist between placebo and acupuncture responsiveness, the correlation between hypnotic susceptibility and acupuncture responsiveness is probably nil.

⁶ Thanks to Miguel Planas for pointing out the problems with this task (see Planas, 1981).

APPENDICES

Appendix A

Informed Consent Agreement for Experiment 1

This project has been reviewed and approved by the Department of Psychology Committee on Research with Human Subjects as preserving safeguards of subjects' privacy, welfare and civil liberties. The Chairperson of the Committee may be reached through the Department of Psychology, Carleton University.

Because this experiment may involve some discomfort, we would like you to be aware of the procedures involved before you agree to participate further.

During the experiment you will have a blood pressure cuff inflated on your arm on two successive occasions. You will then be asked to pump water with your arm. Each time your arm is in the cuff you will be asked to estimate when you feel pain. You will also be asked to keep your arm in the cuff until you can no longer endure any pain.

It is important to the success of this experiment that subjects keep their arm in the cuff for as long as they can for both tests. We have found in previous experiments that almost all subjects are able to do this. However, if you should find that your arm becomes so uncomfortable that you do not wish to continue in the experiment then you may stop at that point at no penalty or loss of credit to you.

I hereby agree to cooperate and participate in the research project entitled "Cognitive activity, pain perception and hypnotic susceptibility" to be conducted by Hank Stam of the Department of Psychology as principal investigator.

I acknowledge that the principal investigator has informed me about my participation in the experiment. I also acknowledge that my participation in the experiment has been fully explained to me, and that should my participation in the experiment be altered in any way, I will be duly informed by the principal investigator of the changes.

I understand that I have the freedom to withdraw from the experiment at any time.

Participant Signature

Principal Investigator

Date

Appendix B

Instructions for Ischemic Pain Task in Experiment 1,
Modified from McGlashan, Evans, and Orne (1969, p. 233)

In this experiment your task will be to pump water with the arm that has the pressure cuff on it. As you are pumping you will recognize four stages of sensations: (1) no change in sensations; (2) paresthesia, or the awareness of sensations that are not painful; (3) pain threshold, or the moment that sensations first become painful; and (4) the maximum tolerable pain, or the point at which you could not possibly continue pumping.

As you carry out this task, I want you to estimate for me when you have reached the beginning of stage 3, that is, the moment when you first feel painful sensations. You are to squeeze the bulb in time to the metronome at the rate of one squeeze for every beat. Squeeze the bulb until you have reached the first sign of pain. When you have reached this point, which we call the threshold of pain, or the very least amount of pain that is discernible to you, say "Now" but continue squeezing the bulb. As you continue pumping through stage 3, the pain will increase in intensity until it becomes very unpleasant, and you will reach a point where you don't want to go on any further. Even then, I want you to make a maximum effort to go beyond this point. Whenever

you feel you want to give up, keep trying a little bit more. It is absolutely crucial for the success of the experiment that you continue pressing almost beyond the limits of your endurance. When you have reached stage 4, the end point, or that point where under no circumstances can you go on no matter how badly you may wish to do so, say "Now" again, and drop the bulb. Your arm will get very tired, but continue squeezing as hard as you can until it hurts so much that you cannot continue even one more second.

Appendix C

Post-experimental questionnaires

1. During the last time your arm was in the pressure cuff what percentage of the time did you think about or imagine anything to help you experience less pain. Make up a fraction that best describes how long you thought about or imagined this from the time I placed your arm in the cuff until I took it out. For example, if you imagined or thought about anything to help you experience less pain for the complete period, write 100; if you did this for half the time write 50; if for twenty percent of the time, write 20; if you did not imagine or think about anything to help you experience less pain, write 0; and so on. Please place the fraction you decide on in this space _____.

2. This question is concerned with what goes on in people's minds while their arm is in the pressure cuff. Sometimes in this situation people become fearful and anxious; focus on how much pain they are feeling; keep thinking about how bad the pain is; think about how much they want to get out of the situation and think about or imagine other unpleasant and painful situations this reminds them of. Please indicate below by placing a mark on the line, the extent to which you became absorbed in, imagined or thought about things of this sort.

I was engaged in thinking or imagining things of this sort:

Not at all +-----+ All of the time

3. The last question was concerned with unpleasant or fearful thoughts. This question is concerned with non-fearful thoughts or imaginings that people have while their arm is in the pressure cuff. Frequently in this situation people think of things other than pain. For example, some people imagine a pleasant scene; some imagine that their arm is separated or dissociated from their body; other people think of nonpainful events of other kinds; some people try to distract themselves from the pain. Please indicate below by placing a mark on the line, the extent to which you became absorbed in, imagined or thought about things of this sort. I was engaged in thinking or imagining about things of this sort:

Not at all +-----+ All of the time

(Questions 1 and 3 were slightly modified when subjects had received a suggestion for analgesia on the particular trial in question. In that case they asked about the extent of subjects' time engaged in imagining the suggested events).

4. Please answer the following question by placing a mark on the line:

During the last time the cuff was inflated around my arm, when I dropped the bulb to indicate maximum pain, my arm was: (Subjects placed a mark on a visual analogue scale bounded on one end by "NOT AT ALL TIRED" and on the other end by "COMPLETELY EXHAUSTED: I COULD NO LONGER SQUEEZE".)

5. If your arm had not been so tired could you have continued to pump further before reaching maximum pain?

YES _____ NO _____

If yes, how much longer could you have continued? Place your estimate in the space _____.

Appendix D

Hypnotic Induction Procedure

Close your eyes. Your ability to be hypnotized depends entirely on your willingness to cooperate. It has nothing to do with your intelligence. As for your will power; if you want to, you can pay no attention to me and remain awake all the time. In that case, you might make me seem silly, but you are only wasting time. On the other hand, if you pay close attention to what I say, and follow what I tell you, you can easily learn to fall into a hypnotic sleep. In that case you will be helping this experiment and not wasting any time. Hypnosis is nothing fearful or mysterious. It is merely a state of strong interest in some particular thing. In a sense you are hypnotized whenever you see a good show and forget you are part of the audience, but instead feel part of the story. Your cooperation, your interest, is what I ask for. Your ability to be hypnotized is a measure of your willingness to cooperate. Nothing will be done that will in any way cause you the least embarrassment.

Now relax and make yourself entirely comfortable.

Relax completely. Relax every muscle of your body. Relax the muscles of your legs. Relax the muscles in your arms. Make yourself perfectly comfortable. Let yourself be

limp. Relax, more and more, more and more. Relax completely. Relax completely. Relax completely.

Your legs feel heavy and limp. Your arms are heavy, heavy as lead. Your whole body feels heavy, heavier, and heavier. You feel tired and sleepy, tired and sleepy. You feel drowsy and sleepy, drowsy and sleepy. Your breathing is slow and regular, slow and regular.

Your feel pleasantly drowsy and sleepy as you continue to listen to my voice. Just keep your thoughts on what I am saying. You are going to get much more drowsy and sleepy. Soon you will be deep asleep but you will have no trouble hearing me. You will not wake up until I tell you to.

Remember that the dangers of hypnosis are a myth. Nothing will be done that is in any way harmful to you. I shall now begin to count. At each count you will feel yourself going down, down, down, into a deep, comfortable, a deep restful sleep. A sleep in which you will be able to do all sorts of things I ask you to do. One - you are going to go deeply asleep . . . Two - down, down, into a deep, sound sleep . .

. Three - four - more and more, more and more asleep . . .

Five - six - seven - you are sinking, sinking into a deep, deep sleep. Nothing will disturb you. Pay attention only to my voice and the things I tell you. . . Eight - nine - ten - eleven - twelve - deeper and deeper, always deeper asleep . . . Thirteen - fourteen - fifteen. You will

always hear me clearly no matter how deeply asleep you may be . . . Sixteen - seventeen - deep asleep, fast asleep. Nothing will disturb you. You are going to experience many things that I will tell you to experience . . . Nineteen - twenty. Deep asleep! You will not awaken until I tell you to do so. You will wish to sleep and have the experiences I shall presently describe.

You are feeling comfortable, relaxed, thinking of nothing, nothing but what I say, your eyes are closed, comfortably closed, you are thinking of nothing, nothing but what I say, your arms and legs feel heavy, your arms and legs feel heavy and you are relaxed, relaxed, your whole body feels relaxed, your whole body feels relaxed, the muscles of your face, arms and legs are relaxed, your whole body is relaxed. Drift deeper! It feels as though you are going backward into the darkness, backward - into the darkness, and as you go backward into the darkness you are more and more relaxed, more and more comfortable, you are going backward, backward, and backward into the darkness and as you go backward you feel more and more and more comfortable, more and more relaxed, you are listening only to my voice, only to my voice, thinking of nothing, absolutely nothing, concentrating only on my voice, listening only to what I say, listening only to my voice, you are feeling comfortable and relaxed, comfortable and

relaxed, and as you go backward and backward into the darkness you feel drowsy, very drowsy, and you are thinking of nothing, nothing but the sound of my voice, you feel comfortable and relaxed, comfortable and relaxed, breathing regularly and deeply, regularly and deeply, regularly and deeply. . . thinking of nothing, nothing but the sound of my voice - breathing regularly and deeply, regularly and deeply, regularly and deeply, and you are in a sleep, a deep, sound comfortable sleep - a deep sound comfortable sleep, breathing regularly and deeply, regularly and deeply, regularly and deeply - your sleep is getting deeper, deeper, deeper, as you go backward into the darkness your sleep is getting deeper and deeper - deeper and deeper - deeper and deeper - you feel comfortable and relaxed - listening only to my voice - breathing regularly and deeply, regularly and deeply, regularly and deeply, in a deep, sound sleep - deep, sound, sleep - deep, sound, sleep, and your sleep is getting deeper and deeper, deeper and deeper, deeper and deeper - you are in a deep sound sleep, deep sound sleep, deep sound sleep, breathing regularly and deeply, regularly and deeply, regularly and deeply, and you are in a deep, sound, sleep, a deep sound sleep, sleep, sleep, sleep, sleep.

"Wake-up" Procedure

You are going to wake up in a few minutes. You will feel refreshed, wide awake, and in a good mood. I will

count from 5 to 1 and with each count you will
be more fully awake.... 5 - 4 - 3 - 2 - 1.
Wide awake. Open your eyes. Wide awake.

Appendix E

Suggestion for Analgesia

I want you to succeed in not being disturbed by the pain in your arm by doing the following. Try to the best of your ability to imagine and think of your arm as numb and insensitive. Think of your arm as unable to sense any pain or discomfort. Please try to think of your arm as numb and insensitive as if it were a piece of rubber, until I deflate the pressure cuff. Other students were able to think of their arm in this way and it isn't as hard as it seems. What I want you to do is to control your thoughts and think continuously that your arm has no feeling. Keep thinking that it is unable to feel any pain or discomfort. Continue to think of your arm as without pain, discomfort, or feeling of any kind. Please try to the very best of your ability to think continuously and to imagine vividly that your arm is numb, insensitive, and like a piece of rubber until I deflate the pressure cuff. Now keep thinking and vividly imagining that your arm is becoming more and more numb and insensitive.

Appendix F

Instructions for Ischemic Pain Task Following Hypnotic

(Analgesia in Experiment 1

From McGlashan, Evans, and Orne (1969, p. 234)

When I say start - begin squeezing the bulb to the click of the metronome. If you happen to feel any unpleasant sensation, say "Now" if it is clearly discernible, just like the last time. Then keep pressing as long as you possibly can, and continue to squeeze to the rhythm of the metronome up to the very limit of your tolerance - painlessly. If you come to that limit and cannot keep pressing, say "Now" and drop the bulb, just like last time. It will be easier this time though, very easy, for there will be no pain to bother you. Your arm will not become tired or fatigued. It is deeply anesthetized, and you will remain alert, but deeply hypnotized.

Appendix G

Informed Consent Agreement for Experiment 2

This project has been reviewed and approved by the Department of Psychology Committee on Research with Human Subjects as preserving safeguards of subjects' privacy, welfare and civil liberties. The Chairperson of the Committee may be reached through the Department of Psychology, Carleton University.

Because this experiment may involve some discomfort, we would like you to be aware of the procedures involved before you agree to participate further.

During the experiment you will have a blood pressure cuff inflated on your arm for three successive occasions. You will then be asked to perform a limited amount of exercise with the occluded arm. Each time your arm is in the cuff you will be asked to estimate how much pain you feel. You will also be asked to endure as much pain as you can stand.

It is important to the success of this experiment that subjects keep their arm in the cuff as long as they can for all three tests. We have found in previous experiments that almost all subjects are able to do this. However, if you should find that your arm becomes so uncomfortable that you do not wish to continue in the experiment then you may stop at that point at no penalty or loss of credit to you.

I hereby agree to cooperate and participate in the research project entitled "Cognitive activity, pain perception, and hypnotic susceptibility" to be conducted by Hank Stam of the Department of Psychology as principal investigator.

I acknowledge that the principal investigator has informed me about my participation in the experiment. I also acknowledge that my participation in the experiment has been fully explained to me, and that should my participation in the experiment be altered in any way, I will be duly informed by the principal investigator of the changes.

I understand that I have the freedom to withdraw from the experiment at any time.

Participant Signature

Principal Investigator

Date

Appendix H
Symptom Checklist

Below are a number of statements that describe bodily sensations. Read each statement and then check the appropriate space to the right of the statement to indicate which of these statements apply to you right now, that is, at this moment. For each statement place a checkmark under the response indicating whether you are experiencing (a) not at all, (b) somewhat, or (c) a great deal of what is described in each statement.

	<u>Not</u>	<u>At</u>	<u>All</u>	<u>Somewhat</u>	<u>A Great Deal</u>
Headache		0		0	0
Indigestion		0		0	0
Aching in arm		0		0	0
Stomach cramps		0		0	0
Sleepiness		0		0	0
Tension in jaw		0		0	0
Buzzing in the ears		0		0	0
Blurry vision		0		0	0
Stomach pain		0		0	0
Nausea		0		0	0
Tingling in arm		0		0	0
Itchy		0		0	0

Hypnotic Analgesia

226

Dizziness	0	0	0
Cold & sweaty hands	0	0	0
Fast heart beat	0	0	0
Shortness of breath	0	0	0
Flushed	0	0	0
Shoulder & back tension	0	0	0
Trembling hands	0	0	0
Pain in or around eyes	0	0	0

Appendix I

Instructions for Magnitude Estimation Procedure

In this study, I will be asking you to judge the intensity of the pain you feel while you have your arm in the pressure cuff. In order to do this I will first teach you a technique for estimating the pain intensity. To show you what I mean I have drawn up an example using line lengths. I want to show you how you can use numbers to label changes in line lengths. I want you to judge the length of one line relative to another line. I will present you with a series of lines in irregular order. Your task is to tell how long they seem by assigning numbers to them. Call the first line any number that seems appropriate to you. Then assign successive numbers in such a way that they reflect your subjective impression. There is no limit to the range of numbers that you may use..... You may use whole numbers, decimals or fractions. Try to make each number match the length as you perceive it.

(When the subject had successfully completed this task he or she was given the following instructions:)

OK. Now I am going to place your arm in the pressure cuff. Every so often I will say report. Whenever I say report, I want you to give me a number that corresponds to the intensity of the pain you are feeling at that moment.

{ That is, I want you to tell me how much pain you feel by assigning a number to it. The first time I say report, call the pain any number that seems appropriate to you. Then assign successive numbers in such a way that they reflect your subjective impression. There is no limit to the range of numbers that you may use. You may use whole numbers, decimals, or fractions.. Try to make each number match the intensity as you perceive it. It is important that you give me a number for the pain intensity you feel at the moment I say report, and not a number that corresponds to the amount of time you think you've had your arm in the cuff. OK, now can you tell me what the procedure will be in your own words?

(If the subject did not understand or was not clear about any part of the instructions, these were explained to her/him by rephrasing the above instructions.)

Appendix J

Physics of Lasers Lecture

Modified from R. Stevenson and R. B. Moore. Theory of Physics.

Saunders: Philadelphia, 1967, pp. 730 - 732.

Light sources can conveniently be categorized by the wavelength region in which they produce useful radiation, or according to whether the radiation they produce is coherent or incoherent. Coherence implies some definite relationship among phases of all the electromagnetic waves or photons leaving the source. Incoherence means that photons emitted from different parts of the source have random phase relationships. A coherent source is inherently more powerful than an incoherent source, because all the radiation is emitted in a specific direction.

A coherent light source can be produced by a device known as a laser which is what you will be exposed to. It consists of a collection of atoms enclosed in a cavity with partially transmitting mirrors at the end. Pumping radiation is applied to the system of atoms in order to excite them into higher energy states. The pumping has the effect of having more atoms or molecules in the higher energy states than in the ground state.

Laser action occurs when the probability of stimulated emission is greater than the probability of absorption.

This occurs where there is a population inversion - when more atoms are in the excited than in the ground state - accomplished here by the existence of metastable excited helium levels. As more photons are reflected in phase between mirrors, more emission is stimulated until an equilibrium condition comes about. If the mirrors are partially transmitting some of the beam can be removed from the system. This then is the laser beam.

(Subjects were shown the newspaper article at this point.)

This laser, which you will be exposed to for several minutes, will work on your muscular tissue. It takes some time to take effect so when I have finished exposing you to it you will fill in some forms while we are waiting for it to take full effect. Its effects should last for a good half hour.

At first you may not feel anything but eventually you should feel a numbness in your arm or a tingling sensation. When we place your arm in the pressure cuff again you should be able to withstand the pressure for a much longer period of time. In fact you should feel no discomfort for quite some time.

Appendix J (Continued)

Newspaper Article Shown Subjects Prior to Placebo Manipulation

Restructured Article from Globe (Tabloid)

NEW TREATMENT FOR ARTHRITIS PAIN

A MIRACULOUS new laser treatment can eliminate agonizing arthritis pain forever without drugs or surgery.

The fantastic treatment was developed by Dr. Norman Beals, director of the King Family Medical Center in Anaheim, California.

"It's a sort of space-age acupuncture," Beals told **GLOBE**.

"We use a low-intensity laser, which is focused on the appropriate acupuncture points. The treatment can relieve all types of pain, from arthritis to colitis."

Beals explains that the laser beam triggers the release of endorphins — the body's natural painkilling chemicals.

"We use a 'cold' laser, which generates no heat, so the treatment is completely painless," he says.

"Conventional 'hot' lasers have a potential to cause pain and even severe injuries."

Patients normally receive 10 to 15 treatments over a period of about two weeks, says Beals, who has helped hundreds of people with his laser therapy over the last few years.

The treatments are relatively inexpensive, and take between 15 and 25 minutes apiece, he adds.

And once the treatments are completed, a patient will not have to receive them again for up to 18 months — much longer than is the case with most other techniques, Beals claims.

"Our treatment is nine times more effective than traditional needle acupuncture and newer methods using electric arc machines," he says.

Acupuncture stimulation by electric arc has been in use for a number of years, and is considered by many to be much more effective than using needles.

However, Beals claims he was disappointed when he first began experimenting with electric arc machines.

"We did some work with them, but found them not always up to our expectations and requirements," he says.

"The laser technique is vastly superior."

Beals began using the cold laser treatment in 1977, "with some really remarkable results."

One of his patients experienced immediate pain relief after only one treatment.

Remarkably, patients invariably feel more content and energetic — as well as pain-free — after receiving the treatment. Beals admits doctors come to him, "usually in desperation, after they have been unsuccessful in treating a patient with surgery or drugs."

Frank McKinney of the Food and Drug Administration says the FDA is adopting a "wait-and-see" attitude.

"We have had some glowing claims from patients concerning the treatment," says McKinney.

Beals adds: "There are an awful lot of people in the United States with so-called 'untreatable' pain who are not accepting the pessimistic advice of their doctors to learn to live with it."

"And, to tell you the truth, there isn't any reason why they should."

Appendix K

Instructions Given Subjects Prior to Trial Three

modified from McGlashan, Evans, and Orne (1969, p. 234)

Why are we using [this laser/hypnosis] then? Because hypnosis is psychological and therefore prone to all the variations that are inherent in a psychological phenomenon. This laser, on the other hand, is physiological and works on a physical or cellular level. Consequently, it is much more stable, reproducible, and uniformly more effective than hypnosis in producing pain relief or increasing performance. This is why we want to use it as a standard for measuring the efficacy of hypnosis, because we know how good it is and know its effects both quantitatively and qualitatively.

Appendix L

Mean Magnitude Estimates for Each Group and Trial
Prior to Transformation to a Common Modulus and
Function Fits Based on Average Data

High Susceptible - B/P/H ($n = 7$)

Time	Trial 1	Trial 2	Trial 3
90	11.9	13.6	7.3
120	13.6	16.4	7.6
150	15.5	19.6	6.2
180	17.7	22.3	6.9
210	21.4	24.1	8.1
240	23.6	25.9	9.1
270	25.6	28.0	10.1
300	26.1	29.3	11.7
Linear R^2	.9813	.9831	.7266
Power R^2	.9819	.9950	.5628
Log R^2	.9594	.9962	.5769
Power Exponent	.716	.645	.382

Appendix L (Continued)

Low Susceptible - B/P/H ($n = 8$)

Time	Trial 1	Trial 2	Trial 3
90	7.8	6.3	4.3
120	9.5	9.0	6.8
150	11.9	10.7	8.9
180	14.0	13.0	11.1
210	15.6	15.3	12.3
240	19.3	17.9	13.0
270	21.6	19.9	15.6
300	23.9	20.9	20.0
Linear R^2	.9925	.9932	.9656
Power R^2	.9915	.9952	.9792
Log R^2	.9447	.9781	.9283
Power Exponent	.953	1.012	1.160

Appendix L (Continued)

High Susceptible - B/H/P ($n = 6$)

Time	Trial 1	Trial 2	Trial 3
90	7.8	5.8	5.2
120	10.0	10.3	8.5
150	13.2	16.3	14.3
180	18.7	25.3	19.2
210	23.2	31.2	21.8
240	28.6	37.2	28.9
270	35.2	46.5	33.4
300	44.5	53.0	38.6
Linear R^2	.9674	.9951	.9948
Power R^2	.9845	.9928	.9907
Log R^2	.8848	.9505	.9553
Power Exponent	1.466	1.849	1.669

Appendix L (Continued)

Low Susceptible - B/H/P ($\underline{n} = 6$)

Time	Trial 1	Trial 2	Trial 3
90	5.2	3.5	3.3
120	9.2	5.8	6.3
150	11.6	7.3	8.1
180	14.2	9.3	11.2
210	15.5	11.3	12.9
240	16.5	13.1	14.7
270	17.9	14.6	16.9
300	19.2	15.8	19.2
Linear R^2	.9457	.9953	.9951
Power R^2	.9367	.9899	.9791
Log R^2	.9942	.9849	.9816
Power Exponent	1.016	1.241	1.389

Appendix L (Continued)

High Susceptible - Control ($n = 9$)

Time	Trial 1	Trial 2	Trial 3
90	7.7	7.7	7.7
120	8.9	10.0	11.2
150	12.2	13.3	13.7
180	15.0	16.2	18.2
210	15.6	17.3	21.7
240	18.0	20.4	24.7
270	20.1	23.4	28.4
300	21.8	25.9	30.8
Linear R^2	.9864	.9948	.9968
Power R^2	.9845	.9950	.9963
Log R^2	.9730	.9690	.9725
Power Exponent	.899	1.008	1.169

Appendix L (Continued)

Low Susceptible - Control ($n = 9$)

Time	Trial 1	Trial 2	Trial 3
90	5.1	5.3	6.4
120	8.8	9.9	10.9
150	16.1	15.4	15.2
180	15.7	20.4	20.0
210	20.1	23.6	24.1
240	24.3	26.8	30.3
270	25.6	29.7	33.7
300	28.8	35.9	37.6
Linear R^2	.9677	.9892	.9975
Power R^2	.9504	.9715	.9933
Log R^2	.9740	.9811	.9710
Power Exponent	1.390	1.509	1.460

Appendix M

Means and Standard Deviations for Catastrophizing,
Coping and Time Imagining Scores in Experiment 2

		Catastrophizing					
Group	Susceptibility	T1		T2		T3	
B/P/H	High	27	(27)	25	(15)	10	(9)
	Low	25	(13)	31	(22)	16	(15)
B/H/P	High	32	(23)	18	(27)	35	(27)
	Low	25	(19)	20	(24)	19	(17)
Control	High	29	(23)	29	(24)	47	(31)
	Low	43	(33)	33	(31)	50	(36)
		Coping					
B/P/H	High	44	(33)	42	(32)	75	(26)
	Low	30	(24)	14	(10)	47	(35)
B/H/P	High	40	(39)	58	(31)	41	(27)
	Low	34	(31)	47	(25)	27	(36)
Control	High	36	(36)	47	(31)	40	(25)
	Low	27	(23)	38	(31)	41	(34)
		Time Imagining					
B/P/H	High	41	(30)	48	(28)	74	(25)
	Low	27	(21)	17	(9)	43	(32)
B/H/P	High	34	(37)	43	(32)	33	(28)
	Low	36	(33)	49	(26)	27	(36)
Control	High	32	(30)	43	(34)	40	(28)
	Low	26	(28)	40	(27)	48	(31)

Note. Standard deviations are in parentheses (T1 = Trial 1, T2 = Trial 2, T3 = Trial 3).

END

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FIN