1972

Conditioned Inhibition In Mentally Retarded And Nonretarded Persons

Benjamin Carl Carruthers

CONDITIONED INHIBITION IN MENTALLY RETARDED AND NONRETARDED PERSONS

by

Benjamin Carl Carruthers

Department of Psychology

Submitted in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

Faculty of Graduate Studies

The University of Western Ontario

London, Canada

December, 1971

ABSTRACT

This research tested the hypothesis that certain differences between the behavior of mentally retarded individuals and that of nonretardates reflect an inhibition deficit in retardate learning. The evaluation consisted of a comparative examination of the conditioned inhibitory strength of a former CS^- used for differential GSR conditioning in both retarded and nonretarded subjects of similar age. Conditioned inhibition, defined as the acquired ability of a stimulus to control a response tendency opposite to excitation, was measured by two appropriate paradigms, summation and reacquisition.

The overall experimental design was a 3 x 2 x 2 factorial type involving Groups (mental retardates, a comparison group of nonretardates trained to the same criterion of differential conditioning, or, a comparison group of nonretardates equated on the number of training trials), Treatment (conditioning or control), and Inhibition Tests (summation or acquisition). Ninety-six subjects, 32 retardates and 64 nonretardates, completed three consecutive phases of training; habituation, differential conditioning (or control), and inhibition testing. Both the habituation and differential conditioning procedures were of a conventional nature, with a tone and ambient light diminution as CSs and a finger-shock UCS. The summation procedure consisted of the random presentation of five compound CS^-/CS^+ trials, five CS^+ alone trials, and five trials on which

the CS^+ was paired with the shock UCS. The reacquisition procedure involved the presentation of 20 CS^- trials; a random 50 per cent of which were paired with the UCS.

Assuming that retardates suffer a conditioned inhibition deficit, the following hypotheses were formulated and tested: (1) Given the same number of differential conditioning trials, or an equal degree of response differentiation, the CS^-, when presented in combination with the CS^+, will result in a smaller decrement in the responding of retardates than in that of their nonretarded peers; (2) Given the same number of differential conditioning trials, or an equal degree of response differentiation, the development of CRs to the former CS^-, when used as the CS in single-cue excitatory conditioning, will be retarded to a greater extent in nonretardates than in retardates. While the summation data clearly confirmed the former hypothesis, analysis of the reacquisition data failed to reveal a significant retardate-nonretardate difference and thus, did not confirm the latter. The reacquisition data did indicate, however, that the former CS^- acquired inhibitory control over the responding of both intelligence groups. Although the results were not in complete agreement with the predictions derived from the inhibition deficit formulation, an analysis of alternative interpretations of the data failed to provide a more parsimonious account. Implications of the findings and suggestions for further research were discussed.

ACKNOWLEDGMENTS

I wish to thank my thesis advisor, Dr. Harold Lobb, for the guidance, encouragement, and constructive criticism which he provided during all phases of this research. I appreciate, too, the advice of the members of my thesis committee: Dr. R. Gardner, Dr. H. Murray, Dr. S. Kendall, and Dr. F. VanFleet. In addition, I am extremely grateful to both Dr. Koegler and Dr. Mosely of the Ontario Hospital School, Cedar Springs, without whose help and cooperation the data collection phase of this study would have taken many more months to complete. I owe a special note of appreciation to my wife, Cheryl, whose constant encouragement and help greatly aided the completion of this thesis.

I should like to acknowledge the support given the thesis by Ontario Mental Health Foundation Grant #325 to Dr. Harold Lobb, and by London Psychiatric Hospital. During the 1970-1971 fiscal year, the author was supported as a Research Fellow of the Ontario Mental Health Foundation.

TABLE OF CONTENTS

	Page
Certificate of Examination	ii
ABSTRACT	iii
ACKNOWLEDGMENTS	v
LIST OF TABLES	viii
LIST OF FIGURES	ix
LIST OF PLATES	x
INTRODUCTION	1
Concepts of Inhibition	2
Inhibition Deficits in Retardate Learning	7
Theory	7
Experimental Evidence	8
Extinction	8
Differential Conditioning	9
Current Concept of Conditioned Inhibition	13
Definition	13
Measurement of Conditioned Inhibition	14
Summation	15
Reacquisition	17
Development of Conditioned Inhibition	20

Page

The Present Study 22

Hypotheses 24

Methodological Considerations 25

METHOD 29

Subjects 29

Apparatus 29

Design 31

Procedure 34

RESULTS 40

Habituation 41

Differential Conditioning 42

Conditioned Inhibition Tests 47

Summation 47

Reacquisition 50

DISCUSSION 53

REFERENCES 59

APPENDICES 64

APPENDIX A - Sequences of stimuli and schematic of the procedures used in the differential conditioning phase of training 64

APPENDIX B - Summaries of statistical analyses and corresponding tables of means 67

APPENDIX C - Individual GSR data 78

VITA xi

LIST OF TABLES

TABLE		Page
1	Schematic design of experiment	35

LIST OF FIGURES

FIGURE		Page
1	Habituation: Mean GSR magnitude as a function of Groups (PN, MR, & TN) and Trials	43
2	Habituation: Mean GSR magnitude as a function of Stimulus Type (tone or light diminution) and Trial sequence	44
3	Differential Conditioning: Mean GSR magnitude as a function of Treatment (conditioning or control) and Trial Type (CS^+ or CS^-) over the last two eight-trial blocks of training	46
4	Summation: Mean magnitude GSR as a function of Groups (PN, MR, & TN), Treatment (conditioning or control), and Trial Type (CS^-/CS^+ or CS^+)	49
5	Reacquisition: Mean magnitude GSR as a function of a Treatment Group (PN/con'd, MR/con'd, TN/con'd, PN/control, MR/control, & TN/control) and Blocks of two trials	51

LIST OF PLATES

PLATE		Page
I	Configuration of equipment in the control portion of the laboratory	32
II	The subject chamber of the laboratory	33

INTRODUCTION

Mental retardation is essentially a behavior syndrome. This is indicated by the following definition which is currently used by the American Association on Mental Deficiency:

> Mental retardation refers to subaverage general intellectual functioning which originates during the developmental period and is associated with impairment in adaptive behavior. (Heber, 1958, p. 3)

Expressed in terms of complex intellectual functioning and vague developmental criteria, the definition is somewhat imprecise. An overall objective of much psychological investigation in the area of mental retardation is the refinement of this description. Such refinement might be accomplished if differences in mental ability, presently given by scores on heterogeneous psychometric tests, were reduced to a description in terms of differences revealed by laboratory experiments in a small number of presumably more unitary and basic functions or capacities. In response to the criticism that this objective may be too ambitious or is unattainable at present, Estes (1970) has suggested that such an approach might at least lead to the replacement of the

> . . . description of mental [retardation] in terms of a broad cross section of performance on various tasks for which we have little knowledge of boundary conditions, with a description in terms of performance and rates of gain in performance in certain standard situations which are better understood and more amenable to manipulation and analysis. (p. 54)

The data obtained from empirical comparisons of retarded and nonretarded

individuals in these laboratory situations would be expected to contribute to the refinement of the description of mental retardation which, in turn, perhaps might eventually lead to improved diagnosis and therapy.

The approach taken in the present research was based largely on the assumption that the discovery of 'learning functions' that are correlated with intellectual or adaptive deficits would contribute to a more precise description of the abnormalities involved in mental retardation. The actual strategy employed was to compare the performance of retarded individuals with that of nonretarded individuals in a particular laboratory-learning situation. More specifically, the present experiment was designed to provide a comparative evaluation of 'conditioned inhibition' in both retardates and nonretardates; this being relevant to an hypothesized 'inhibition deficit' in retardate learning. The meaning of these terms is clarified in subsequent sections.

Concepts of Inhibition

In a scientific context, the term 'inhibition' originated within neurophysiology (Sechenov, 1935; Sherrington, 1906) and referred to hypothetical neural activity which opposed excitatory impulses. More recently, evidence for neural inhibition has actually been found at both the synaptic level (e.g., Eccles, 1964) and at the level of gross anatomical brain structures (see Kimble, 1968). It was Pavlov (1929), however, who first attempted to systematically relate a neurophysiological concept of inhibition to behavior. In so doing, Pavlov was responsible for the introduction of the term inhibition to psychological theory, in which it

came to be treated as an abstract hypothetical construct. In order to examine some of the major theoretical interpretations of inhibition which have arisen, a comparison of the role played by inhibition in Pavlovian theory with those played in the theoretical systems of some succeeding learning theorists is presented in the paragraphs which follow.

Pavlov's objective associationism was based primarily on his concept of the conditioned reflex which is today referred to as classical (or Pavlovian) conditioning. In his now famous experiments, Pavlov found that when meat powder (unconditioned stimulus--UCS) was placed in a dog's mouth, it resulted in a reflexive increase in salivation (unconditioned reflex--UCR); but if a 'neutral' stimulus such as the sounding of a bell (conditioned stimulus--CS) closely preceded the presentation of the meat powder several times, the sounding of the bell came to evoke salivation (conditioned reflex--CR) independent of the food. Pavlov viewed the development of such a simple conditioned reflex or response, as beginning with its 'acquisition', which was brought about by repeated 'reinforcement'; that is, the following of the CS repeatedly by the UCS, and therefore UCR, at an appropriate temporal interval. If reinforcement was discontinued and the CS presented alone, unaccompanied by the UCS, the CR gradually diminished and disappeared, a process Pavlov termed 'experimental extinction'. It was to explain the extinction process that Pavlov first employed the concept of inhibition.

Pavlov, greatly influenced by Sechenov (1935), initially discussed inhibition in terms of neurophysiological processes presumed to

occur in the cerebral hemispheres. In his theoretical interpretation of empirical results, however, Pavlov used the concept of inhibition in a somewhat different manner; that being, as a hypothetical intervening variable which had little or no reference to neurophysiological correlates. In his account of experimental extinction, for example, Pavlov theorized that CR magnitude (dependent variable) decreased with the number of nonreinforced trials (independent variable) due to the 'suppressive' effect of the inhibition (intervening variable) which accumulated. Neurophysiological terms were used only in speculating about the mechanism by which inhibition exerted its suppressive influence.

During the course of his experimentation on conditioned reflexes, Pavlov discovered numerous empirical relationships and determined the essential parameters involved in the establishment, maintenance, and extinction of CRs. Many such relationships were taken by Pavlov to reflect behavioral manifestations of inhibition, which he separated into three broad categories: external inhibition, disinhibition, and internal inhibition. External inhibition described the temporary decrement of a CR due to an extraneous stimulus (e.g., when conditioned salivation to a light is reduced by a sudden loud noise). Similarly, disinhibition described the temporary reappearance of an inhibited (e.g., extinguished) CR due to the presentation of an extraneous stimulus. More directly related to the present discussion, however, is Pavlov's concept of internal inhibition. Internal inhibition was presumed to develop slowly and progressively under a number of different experimental conditions, all of which involved either nonreinforcement

or delayed reinforcement. Experimental extinction was one such procedure which was postulated to reflect the development of internal inhibition. The development of 'differential inhibition' and 'conditioned inhibition', which were both subtypes of internal inhibition, occurred under specific experimental conditions. In differential conditioning, the reinforcement of one stimulus (CS^+) separately from the nonreinforcement of a second stimulus (CS^-) was postulated by Pavlov to result in the development of differential inhibition to the nonreinforced CS and the suppression of CRs elicited by that stimulus. The CS^- was thus said to become inhibitory. Similarly, conditioned inhibition was presumed to account for the case when a combination of stimuli was rendered ineffective (i.e., elicited no CR) through nonreinforcement, even though the combination included a stimulus which alone continued to evoke a CR. The other stimuli in the combination were said to be inhibitory. A final variety of internal inhibition, termed 'inhibition of delay' was presumed to develop if a regular interval of sufficient duration elapsed between CS onset and its reinforcement which occurred simultaneously with CS offset. During the early portion of its isolated action the CS became not only ineffective, but actively inhibitory of other intercurrent activities.

Both the preceding classification and terminology provided the background for a large number of subsequent experiments. Moreover, Pavlovian theory provided much of the foundation from which many influential learning theories were derived. Examples of such theories are that of Hull (1943; 1949), and the closely related theoretical system of Spence (1956; 1960).

By the time he had formalized his theory, Hull (1943; 1949) had rejected the Pavlovian basis of inhibition and offered a much different alternative. Hull's analysis assumed that the evocation of any response produced something like 'fatigue' which reduced the tendency of the organism to respond immediately again. This intervening variable, termed 'reactive inhibition', was postulated to dissipate rapidly with the passage of time, and was seen as a function of the amount of effort required to perform the response, the number of trials, and the frequency at which they occurred. His analysis further assumed that response fatigue was a 'drive' condition, and thus, since Hull was committed to a drive-reduction interpretation of reinforcement, ceasing to respond was considered to reduce this drive and hence be reinforced. This learned tendency not to respond ('conditioned reactive inhibition') also subtracted from the capacity of the organism to react to a given stimulus with a particular response. Like Pavlov, Hull postulated inhibitory influences which had a suppressive effect on responding, but unlike the Pavlovian formulation, the Hullian concept of inhibition was not in any way related to nonreinforcement.

In his major reorganization of Hullian theory, Spence (1956; 1960) assigned only a minor role to Hull's inhibition variables and returned to the traditional inhibitory factor which, as Pavlov had suggested, was presumed to develop in strength as a function of nonreinforcement. The suggested mechanism, however, through which nonreinforcement produced suppression of behavior was radically different in Spence's theory. The postulated mechanism was that of hypothetical competing (i.e., incompatible) responses which 'interfered' with CRs. Unlike

both Pavlov and Hull, Spence regarded inhibitory effects to be of an interference nature rather than of a suppressive nature, at least in instrumental reward conditioning.

As indicated by the preceding discussion, the term inhibition has received several different theoretical interpretations from the time it was introduced by Pavlov as an explanatory term. A significant commonality across these various theoretical treatments, however, is that all of the previously mentioned theorists employed the concept of inhibition as a hypothetical intervening variable which related independent and dependent variables in such a way as to offer an account of behavioral phenomena like extinction and differential conditioning.

Inhibition Deficits in Retardate Learning

Theory

Several investigators have attempted to relate a concept of inhibition to intellectual development. At one extreme, Diamond, Balvin, and Diamond (1963) have argued that inhibition is an overall manifestation of the neurophysiology of the organism which modulates intelligent behavior. Without any experimental support, these theorists postulate a direct relationship between neural inhibition and behavioral adequacy. On the other hand, others (Heal, Ross, & Sanders, 1966; Scott, 1969; Heal & Johnson, 1970) have completely avoided physiological speculation, while suggesting that retardate behavior, as shown by performance in various laboratory learning situations, reflects a deficiency in inhibitory functioning. These latter investigators have generally defined inhibition in purely psychological terms, in much the same manner as did

Hull, Spence, and others. For example, Heal and Johnson (1970) define inhibition as "withholding a response or suppressing stimulus input..." (p. 108). They point out that inhibition is a hypothetical construct which is defined in terms of antecedent and consequent events: "it must be inferred from a change in behavior that is occasioned by a change in the environment" (p. 108). Their definition further implies that there must be a baseline against which any inhibition of behavior is assessed.

Much of the empirical data which has been interpreted as supporting the notion of an inhibition deficit on the part of retardates as compared to nonretardates, has been obtained in comparative classical conditioning studies. These studies are reviewed in the following section.

Experimental Evidence

Extinction. To evaluate the notion of an inhibition deficit in retardate learning, a number of investigators have compared extinction of CRs in retarded individuals with that in nonretarded individuals. In a review of several early Russian studies which compared the performance of retarded and nonretarded children in classical conditioning of various responses ranging from salivation to instructed-bulb squeezing, Denny (1964) concluded that perhaps the most general finding was that retardates tended to exhibit a slower rate of extinction than did nonretardates. This finding that retardates persist in responding during extinction to a greater degree than nonretardates, coupled with the observation that retardates also often exhibit slower acquisition of CRs, has recently been replicated (Lobb & Nugent, 1966; Lobb, 1968; Ross, Koski, & Yaeger, 1964) in studies using aversive UCSs and more precisely measurable

responses, such as the galvanic skin response (GSR) and the eyeblink. In accordance with Pavlovian theory, the slower rate of extinction has been taken by some (e.g., Denny, 1964) as evidence of an inhibitory deficit in retardates. The observed differences in extinction between the two intelligence groups, however, is open to other interpretations. As Spence and Platt (1967) have suggested, rate of extinction may very well be a function of the strength of a 'negative cognitive set' based on a subject's recognition of nonreinforcement, rather than reflecting a process such as Pavlovian internal inhibition. Perhaps retardates do not form such a set as rapidly as do nonretardates, due to a failure to recognize stimulus change. Alternatively, in some cases differences in extinction rate between retardates and nonretardates may arise from acquisition differences. Furthermore, not all studies have found retardates to be inferior to nonretardates in terms of rate of extinction. For example, both eyelid conditioning data of Johnson (1968) and GSR conditioning data of Baumeister, Beedle, and Urquhart (1964) failed to indicate any significant retardate-nonretardate differences in extinction.

Differential Conditioning. In evaluating the notion that retardate learning is characterized by an inhibition deficit, a number of investigators have also compared the performance of retarded and nonretarded individuals in a differential conditioning situation. Differential eyelid conditioning performance of 32 nonretarded children ($\overline{CA}$ = 83 months) and 32 retarded adolescents ($\overline{CA}$ = 159 months) of similar 'mental age' (MA) was directly compared in a study by Ohlrich and Ross (1968). Using a separate-phase differential procedure, each subject was given 60 trials on which the stimulus that was to become the CS^+ was

paired with a corneal air-puff UCS, prior to the introduction of the CS^-. In the differential phase of training, each subject was given 90 random presentations of a tonal CS^+ or CS^- (0.8 and 2.5 kHz) over a two-day period. Half of the subjects in each group received an interstimulus interval (ISI--the temporal interval separating CS^+ onset and UCS onset) of 500 msec., while the other half received an 800 msec. ISI. Ohlrich and Ross found that at the longer ISI, regardless of which tone served as the CS^+, both retardates and nonretardates responded differentially to the positive and negative cues in terms of a percentage frequency measure. The response decrement to the CS^-, however, developed gradually in nonretardates, whereas the retardates exhibited a more sudden decrease in their response level on the first few trials after the introduction of the CS^-. The retardates' level of responding to both CS^+ and CS^- remained relatively constant thereafter. There was only minimal evidence for an increasing discrimination on the part of nonretarded children, as the change in their differential responding was quite small. The investigators concluded that the initial level of responding to the CS^- largely reflected the amount of generalized response strength from the positive to the negative cue upon the latter's introduction. On the basis of the retardates' failure to show gradual differentiation, they also suggested that whatever is necessary for differentiation is lacking in retardates relative to MA equated nonretardates, and to a lesser degree, lacking in children relative to adults.

A further series of related investigations was carried out by Ohlrich (1968). He examined the mixed-phase (i.e., CS^+ and CS^- both given from the start of acquisition training) differential eyelid

conditioning performance of retardates using tonal CSs and an air-puff UCS. Ohlrich found that 60 trials of such training at any of the ISI values used (500, 800, and 1100 msec.) was insufficient to produce differential conditioning in the retardates. Even when an extra 200 conditioning trials were given, the degree of response differentiation between the CS^+ and CS^- at the longer ISIs (800 and 1100 msec.) was inferior to that of college students who demonstrated good differential conditioning in 100 trials with the same parameters. Furthermore, Johnson and Heal (1967), in a similar study of differential eyelid conditioning using tonal CSs (1 kHz and 2 kHz) and an air-puff UCS, found little evidence of response differentiation in retarded adults within 100 trials, at several different ISIs.

In addition to the differential eyelid conditioning studies, a group of investigators (Grings, Lockhart, & Dameron, 1962; Lockhart & Grings, 1964) have also examined differential GSR conditioning in college students and retarded adults. Using a tone as the CS^+, a light as the CS^-, and an arm-shock UCS, Grings and his co-workers obtained successful differential conditioning in retardates, as measured by response magnitude on various test presentations of the CS^+ alone, compared to those responses elicited on CS^- trials. Differential responding was found to be an increasing function of the number of trials, as is generally the case in the establishment of an instrumental discrimination. Differential responding in nonretardates, however, developed much more quickly and remained fairly constant throughout training. Notwithstanding the fact that the required discrimination developed very quickly in nonretardates, these results indicate that differential GSR conditioning can

readily be established in both high grade (IQ 53-78) and low grade (IQ 20-43) mental retardates at either of two ISIs (.5 and 5 sec.).

In summary, the differential conditioning studies indicate that good response differentiation is more difficult to establish in retardates than in nonretardates, especially at short ISIs and within a limited number of trials. This general finding has been interpreted by some investigators (e.g., Heal & Johnson, 1970) as evidence of an inhibition deficit on the part of retardates. They have interpreted the retardates' inferior performance on such tasks as reflecting their inability to inhibit responding to the CS^-. Such an interpretation rests on the assumption that an important factor in differential responding is the accumulation of inhibition to the CS^-. A corollary of this assumption is that,

> . . . inhibition is not only evidenced by differentiation, but, as the CS^+ and CS^- are brought closer together, by a decrease in level of responding to the CS^+, presumably due to generalization of inhibition from the CS^-.
>
> (Heal & Johnson, 1970, p. 114)

Like the extinction data, however, the findings of the comparative differential conditioning studies do not preclude alternative interpretations. Although the data are consistent with an inhibition deficit hypothesis, some theorists have previously argued that rather than reflecting a deficit in inhibitory functions, such findings may reflect poor attention or high distractability on the part of retardates (e.g., Marinesco & Kreindler, 1933). More recently, Zeaman and House (1963) have advanced a similar hypothesis as a way to account for retardates' difficulty in instrumental discrimination learning. Denny (1964), on the other hand, poses the following questions:

> Is the inability to 'attend' caused instead by the inability to inhibit? May not the retardate's failure to inhibit the effects of extraneous stimuli account for this inability to attend to a particular task? (p. 103)

As Berger (1954) has suggested, perhaps the inhibition deficit is more basic.

Apart from theoretical considerations, the results of the comparative differential conditioning studies of both eyelid response and GSR, suggest a number of methodological generalizations. In order to maximize response differentiation between CS^+ and CS^- in retardates, for example, adult subjects rather than children should be used. Similarly, a relatively long ISI, an extended series of conditioning trials, and the use of clearly distinguishable CSs such as stimuli from different modalities, should all serve to increase response differentiation in retarded individuals.

Current Concept of Conditioned Inhibition

Definition

Rescorla (1969) has recently redefined Pavlovian conditioned inhibition in purely methodological terms. Although closely related to the original Pavlovian concept, Rescorla's description contains no reference whatsoever to any particular neurophysiological correlates or particular training paradigm. He defines conditioned inhibition as the acquired property of a stimulus to control a response tendency opposed to excitation. Typically, conditioned excitation is defined by the fulfillment of two conditions: an operation relating a conditioned stimulus and an unconditioned stimulus, such as the temporal pairing of the two; and secondly, a change in behavior as a result of this operation.

A stimulus is a conditioned inhibitor then, if, as a result of the experience of the organism with some operation relating that stimulus to the UCS, the stimulus comes to control a tendency opposite to that of the conditioned excitor. Rescorla has further emphasized two important aspects of this particular definition:

> 1. The experience must be with the same UCS as that forming the basis for the conditioned excitor. . .
>
> 2. The tendency controlled by the conditioned inhibitor must be opposite to that controlled by the conditioned excitor (cf. Jenkins, 1965). If the excitor produces an increased probability, decreased latency, and increased vigor of a particular response, then a conditioned inhibitor should decrease its probability, etc. Furthermore, the conditioned inhibitor should be specific to the behavior controlled by the excitor . . . . (p. 2)

Conditioned inhibition is thus a hypothetical construct defined in terms of both antecedent and consequent events. As Heal and Johnson (1970) point out, "Inhibition itself is not observable; it must be inferred from a change in behavior that is occasioned by a change in the environment" (p. 108). Whether or not, as Pavlov (1927) suggested, conditioned inhibitory tendencies are more fragile than excitatory tendencies, or quickly dissipate with time, are empirical questions concerning the properties of conditioned inhibitors; the definition, however, of a conditioned inhibitor is expressed purely in terms of the control of a response tendency directly opposite to that of a conditioned excitor.

Measurement of Conditioned Inhibition

In the vast majority of classical conditioning studies, a 'neutral' stimulus is selected as the CS; that is, a stimulus which elicits little or no responding prior to the administration of any conditioning trials. This neutrality, however, makes it difficult to

measure the degree to which a second stimulus acquires control over a tendency opposite to that of the CS. In order to solve the problem of detecting conditioned inhibitory effects, a variety of special techniques designed to measure conditioned inhibition have been developed. In his methodological article, Rescorla (1969) pointed out two such techniques as being the most suitable indicators of conditioned inhibition, since they directly monitor the ability of a stimulus to control a tendency opposite to excitation. The following paragraphs outline these two procedures and exemplify their use with reference to the experimental literature.

Summation. Pavlov's summation procedure is perhaps the most direct method of measuring conditioned inhibition. Given stimulus *S0*, which has been shown to be an excitor of a specific response, R, it is possible to determine if a second stimulus, *S1*, is a conditioned inhibitor of R, by comparing the magnitude (or some other appropriate measure) of R elicited by the combination of *S1* and *S0* with that elicited by *S0*. Generally, the *S1 S0* combination is arranged such that the onset of *S1* occurs simultaneously with that of *S0*. If this combination produces a smaller magnitude R than that elicited by *S0* alone, then *S1* is taken to be a conditioned inhibitor. *S1* is a *conditioned* inhibitor, however, only if it acquires such properties as the result of the experience of the organism with some operation relating *S1* to the UCS.

It should be noted that this measurement technique reflects the assumption that excitation and inhibition are algebraically additive, each producing directly opposite effects. This assumption, however, places no constraint on the direction of the behavior change. For

example, if an excitatory stimulus, *S0*, results in a decrement in responding, then *S1* is a conditioned inhibitor if the combination, *S1 S0* produces an increment in responding relative to *S0*.

The summation procedure has been used to demonstrate conditioned inhibition with a variety of different kinds of stimuli serving as the known elicitor of behavior. Conditioned inhibitory effects have been measured in terms of the reduction of behavior elicited by an excitatory CS (e.g., Szwejkowska, 1957); the reduction of behavior elicited by a UCS (e.g., Sergeev, 1961); the reduction in the general level of conditioned excitation (e.g., Rescorla & LoLordo, 1965); and, by stimulus generalization of inhibition (e.g., Jenkins, 1965). The technique which is of primary interest for the present purposes is the demonstration of inhibitory effects in terms of reduction in behavior elicited by an excitatory CS. Several demonstrations of this type are reported in Konorski's (1948) book, although the initial experiment was performed by Pavlov (1927). In one of a series of related experiments, Pavlov examined the reduction in salivation to an excitatory CS when it was presented in combination with a second stimulus which had been repeatedly presented to the dog but never followed by food (UCS). He found that the reduction, in terms of drops of saliva per unit time, was greater than that produced when the CS was presented in conjunction with a novel stimulus. This comparison of the suspected conditioned inhibitor plus the excitatory CS, versus a novel stimulus plus the CS, has become a common control procedure, providing a measure of conditioned inhibitory effects unconfounded with stimulus generalization decrement effects. More specifically, Rescorla (1969) notes that:

> A frequently raised difficulty is that the test presentation of the *S1 S0* combination simply involves the presentation of a stimulus complex which varies in similarity to the conditioned *S0*; such decrements as may be observed can then be attributed to stimulus generalization decrement without reference to inhibitory control of *S1*. It is clear that effects of *S1* can be assessed by appropriate control procedures designed to identify the treatment of *S1* necessary to make it an inhibitor. (p. 7)

The summation procedure has also been used with a wide variety of different responses and paradigms, and particularly in situations involving aversive UCSs. For example, Bull and Overmier (1968) have demonstrated conditioned inhibitory effects in terms of an increase in the latencies of avoidance responses of dogs to a CS^+ signalling shock, when the CS^+ was accompanied by the CS^-, a stimulus signalling no shock. Rodnick (1937) has used a summation procedure in a GSR (galvanic skin response) conditioning study with humans to detect conditioned inhibitory effects during the early portion of a long-delay CS for a shock UCS. Furthermore, a number of investigators working within an operant conditioning framework have developed a procedure similar to the summation technique for demonstrating stimulus control of nonresponding (i.e., inhibitory stimulus control). Brown and Jenkins (1967), for example, observed a significant decrease in the rate of pigeons' key pecking, when an S^- (a stimulus in the presence of which responses were never reinforced) was presented in conjunction with a discriminative stimulus (S^D) for food reinforced responding. They interpreted this reduction in response rate as evidence for the inhibitory control of S^-.

Reacquisition. The second procedure for measuring conditioned inhibition deals with the subsequent excitatory conditioning of a stimulus suspected to be a conditioned inhibitor of the response

elicited by the original UCS. If excitation and inhibition are taken as being subtractive from each other, then using a conditioned inhibitor for single-cue excitatory conditioning should retard the development of overt conditioned responses. For instance, Pavlov (1927) reported an investigation in which the acquisition of a salivary CR to a former CS^-, which previously signalled the absence of food, was greatly retarded. Later investigations dealing with the development of salivary CRs to a former CS^- for food have yielded similar findings (c.f., Szwejkowska & Konorski, 1959). A number of other workers such as Hammond (1968), and Carlton and Vogel (1967), using a conditioned emotional response procedure (CER: see Estes & Skinner, 1941) with rats, have reported that the development of conditioned suppression to a former CS^- for shock was much slower than that to a novel stimulus. These investigators have interpreted their findings as evidence that the CS^- was an effective conditioned inhibitor.

As Rescorla (1969) has pointed out, with both the summation and reacquisition procedures, there is the possibility that observed behavioral decrements may result from selective attention rather than from conditioned inhibitory effects. For instance, in the summation procedure, the treatment designed to endow *S1* with conditioned inhibitory properties may lead the organism to focus 'attention' on *S1*, with a resultant decrement in 'attention' to *SO*. Presentation of *S1* might then decrease the response to *SO*, when presented in combination, by causing a shift in 'attention' away from the stimulus normally controlling the response without actively controlling a tendency opposite to that of *SO*. Conversely, it is possible that, as a result of the

immediately preceding training designed to produce a conditioned inhibitor, an organism may not 'attend' to the suspected inhibitory stimulus. As a consequence, in the reacquisition test procedure, retardation of the development of excitatory CRs could occur without any effect of conditioned inhibition. The literature on the phenomenon of so-called 'latent inhibition' provides some support for this notion. A number of researchers (e.g., Lubow, 1965; Schnur & Ksir, 1969) working with different response systems, have reported that a series of CS alone trials, administered prior to any conditioning trials, often results in retardation of the development of CRs in single-cue conditioning. Such retardation is presumably due to the subject's initial inattention to a stimulus which previously had little significance.

The possible confounding of selective attention and conditioned inhibitory effects, however, may be controlled if both the summation and reacquisition procedures are employed in the same experiment. The rationale behind the use of both procedures to demonstrate conditioned inhibition is described by Rescorla (1969) as follows:

> It seems reasonable that a stimulus to which the organism does not attend will be retarded in acquisition of an excitatory CR but will produce little effect in the summation procedure. On the other hand, a stimulus which attracts attention might be expected to produce decrements in the summation testing procedure but to lead to facilitated acquisition of an excitatory CS. (p. 11)

Thus, a stimulus which results in the selective attention of the organism should not produce conditioned inhibitory effects in both the summation and reacquisition procedures. Since attentional accounts of mental retardation are popular, it appears necessary to measure conditioned inhibition with both procedures.

Development of Conditioned Inhibition

A variety of different procedures have been thought to endow a stimulus with inhibitory properties. In terms of the 'contingency' approach popularized by Rescorla (1967), however, the procedures which do in fact generate conditioned inhibitors are typically those in which the onset of the stimulus designed to acquire inhibitory control is negatively correlated with the onset of the UCS. Conversely, a stimulus which signals (i.e., is positively correlated with) UCS onset, gains excitatory control. Furthermore, to be termed a conditioned inhibitor, a stimulus must acquire its inhibitory power through an associative process resulting from a specific relationship arranged between CSs and UCSs.

Extinction of an excitatory CS has often been postulated to transform the CS into a conditioned inhibitor (Pavlov, 1927). Following an extensive review of the literature bearing on this question, however, Rescorla (1969) concludes that the evidence from studies using either summation or reacquisition test procedures, indicates that conditioned inhibitors are not generated by extinction. Moreover, the reacquisition studies reveal that a formerly excitatory CS, no matter how much extinction training intervenes, will recondition faster than a novel stimulus. Similarly, one additional procedure thought to lead to the development of conditioned inhibition, that in which a stimulus signals UCS termination, has received little experimental support.

Perhaps the surest evidence for the development of conditioned inhibition comes from differential conditioning studies, and closely related procedures, in which a stimulus is negatively correlated with

the UCS. There is only meager evidence that 'inhibition of delay' procedures, in which a long delay CS precedes the UCS, result in the inhibitory control of the early portion of the CS. Consequently, in the present research emphasis was placed on the most reliable and straightforward procedure of developing conditioned inhibition; that of Pavlovian differential conditioning.

There is a large body of evidence indicating that a former CS^- of a differential conditioning sequence is a strong conditioned inhibitor. As indicated earlier, Pavlov was first to use differential conditioning and a summation procedure to demonstrate that a CS^- reduced the salivation elicited by the CS^+ to a greater degree than that occasioned by a novel stimulus presented in conjunction with the CS^+. Also, Hammond (1967) has found that with a CER procedure, a CS^- reduced the suppression of ongoing operant behavior normally elicited by the CS^+, to a greater degree than that by a novel stimulus plus the CS^+. Rescorla and LoLordo (1965) report that a CS^- inhibited fear, in the sense of disrupting ongoing Sidman avoidance behavior in rats, while in novel CS, 'truly random' CS, and CS alone controls (i.e., animals receiving no separate differential conditioning) presentation of the CS resulted in very little disruption. In a GSR conditioning study with humans, Grings and O'Donnell (1956) using a summation procedure, found that a CS^- for a shock UCS, when coupled with the CS^+, reduced the GSR magnitude normally elicited by the CS^+ to a greater degree than that by a novel stimulus. There is also evidence that the CS^-, following differential conditioning, is a conditioned inhibitor as measured by the retardation of the development of CRs procedure. As mentioned previously, Hammond

(1968) reported that in an avoidance learning situation, a former CS^- for shock was much more difficult to establish as a CS^+ for shock than was a control stimulus. In summary, then, it may be concluded that discriminative stimuli which signal the nonoccurrence of the UCS, such as the CS^- in differential conditioning, do satisfy the criteria for conditioned inhibitors. They do so largely because of a negative contingency between the stimulus and the UCS, as Rescorla (1969) specifies.

The Present Study

In order to accurately compare the strength and development of the conditioned inhibitory properties of a former CS^- between retardates and nonretardates, it is particularly necessary to ensure that retardates learn to respond differentially to the positive and negative cues. Significant differential responding may be guaranteed if each retardate is trained to some specific criterion of differentiation prior to receiving any tests of conditioned inhibition. Given this procedure, there are two comparative strategies open to an investigator. One such strategy is to equate the amount of training across both nonretarded and retarded subjects prior to testing the conditioned inhibitory properties of the former CS^-, by yoking nonretarded subjects to individual retardates in terms of the number of differential conditioning trials. The alternative strategy would be to equate the performance of the two groups prior to the administration of the conditioned inhibition tests, by training the nonretarded subjects to the same differentiation criterion as that used with the retardates. Each procedure used alone, however, suffers a limitation. If, for example, the amount of training

were equated in both groups of subjects, and as the evidence suggest (e.g., Lockhart & Grings, 1964), the retardates attained a lesser degree of response differentiation than the nonretardates, the CS^- could have become a stronger conditioned inhibitor in nonretardates simply due to the fact that they had overlearned the differentiation. Similarly, if only criterion performance was equated across the two groups of subjects, the conditioned inhibition tests would provide information concerning the development of conditioned inhibition but not necessarily its ultimate strength. Suppose that performance was equated across both groups, and, as expected, the nonretardates achieved an equivalent degree of response differentiation with much less training, but no differences occurred between the two groups of subjects in the strength of the conditioned inhibitory properties of the former CS^-. It could hardly be concluded that no inhibitory deficit exists in retardates. Nonretardates might merely require more training for the CS^- to achieve a higher degree of inhibitory control than in the case of retardates.

The use of both nonretardate comparison groups, one equated with the retardates on the amount of training and the other on the degree of response differentiation attained, would provide much information as to the effects of these variables on the strength of conditioned inhibition. Moreover, if the two procedures were used and the retardates were shown to be inferior to both groups of nonretardates, in terms of the inhibitory strength of the CS^-, then substantial evidence for a conditioned inhibition deficit in retardate learning would have been provided. Thus, in the present study, each subject from one group of nonretardates was

individually yoked to a retardate, in terms of an equal number of differential conditioning trials, while another group of nonretardates was equated with the retarded subjects in terms of differential conditioning performance.

Hypotheses

If retardates suffer from a deficit in conditioned inhibition, the following hypotheses should be confirmed:

(1) Given the same number of differential conditioning trials, or an equal degree of response differentiation, the CS^-, when presented in combination with the CS^+, will result in a smaller decrement in the responding of retardates than in that of their nonretarded peers, on the basis of comparisons with appropriate control groups.

(2) Given the same number of differential conditioning trials, or an equal degree of response differentiation, the development of CRs to the former CS^-, when used as the CS in single-cue excitatory conditioning, will be retarded to a greater extent in nonretardates than in retardates, according to comparisons with appropriate control groups.

Assuming that the CS^- does become an effective conditioned inhibitor, differing only in degree between retardates and nonretardates, the response decrement occasioned by the CS^-, when presented in combination with the CS^+, will be greater in both groups than that produced by a novel or control stimulus. Similarly, during subsequent single-cue excitatory conditioning, the development of CRs in both types of subjects will be retarded to a greater extent when the former CS^- is used than when a novel or control stimulus is used. If, however, the CS^- fails to

gain any inhibitory control over the responding of retardates whatsoever, then, of course, there would be no difference between the CS^- and the control stimulus in either the summation or reacquisition procedures.

Methodological Considerations

The response system studied was the GSR since the evidence indicates that both retarded adults and college students can be readily conditioned to respond differentially to clearly distinguishable positive and negative cues without prolonged training (Grings, Lockhart, & Dameron, 1962; Lockhart & Grings, 1964). As previously indicated, the same cannot be said for differential eyelid conditioning.

The GSR is defined as a rapid decrease in the electrical resistance measured between two points on the skin. Differing from the slow tide-like changes in the so-called 'basal' or 'tonic' resistance level, the GSR is a rapid, temporary decrease of the electrical skin resistance occurring in response to a wide variety of types of stimulation. A loud noise, a brief electrical shock, an itch or a thought may act as an effective eliciting stimulus. The GSR is an indirect result of an autonomic response; that response being a reflexive increase in sweat gland activity regulated by the sympathetic nervous system. The GSR has been regarded (e.g., Sokolov, 1960) as one component of a more general, nonspecific reflex termed the 'orienting reflex' (OR), which is evoked by any sufficiently large quantitative or qualitative change in the stimulus field. The OR is a constellation of reflexes consisting of autonomic components such as the GSR, vasomotor reactions, change of heart rate and respiration; central components such as alpha-blocking in the EEG; and somatic components. Perhaps the most widely used method of

recording the GSR is a constant current technique, in which a slight, steady DC current is continuously passed through the skin by means of appropriate electrodes while the voltage developed across the electrode-skin circuit is recorded (see Montagu & Coles, 1966). As the voltage is in direct proportion to the circuit resistance, a simple transformation yields the subject's skin resistance.

Due to the long latency (2-5 sec.) characteristic of the GSR, it was necessary to employ a number of 'test' trials, or CS^+ alone trials, so as to permit measurement of CRs to the CS^+ during the conditioning phase of training. Moreover, a relatively long, but optimal ISI was used. Longer ISIs have been reported to result in better differential GSR conditioning in college students by Kimmel and Pennypacker (1963) who, studying four such intervals (0.25, 0.50, 1.00, and 2.00 sec.), found the degree of differential responding to be an increasing function of the delay between stimulus onsets.

Due to the extreme sensitivity of the GSR to both internal and external stimulation, the use of a 'novel' stimulus against which to compare CS^- effects would confound such effects with those resulting from stimulus novelty. This problem may be overcome, however, if CS^- elicited GSRs are compared to those elicited by a 'noncontingent' CS^- of an appropriate control group, rather than a novel stimulus (cf., Rescorla, 1969). In both the summation and reacquisition test procedures, the comparison of responses elicited by the former CS^- of a group having received differential conditioning with those elicited by the CS^- of a control group which received similar training, except for the absence of any systematic relationship between the CS^- and UCS, would

provide an accurate measure of conditioned inhibition. The control procedure used in the present study consisted of the presentation of a series of CS^- alone trials, equal in number to that used for differential conditioning, which overlapped the conditioning sequence of CS^+ - UCS and CS^+ alone trials. This procedure, which is described in detail in the next chapter, was designed to provide a baseline of responding to the CS^-, in the absence of any contingency, against which to measure conditioned inhibitory effects.

A further methodological consideration lies in the rationale for using a nonretardate comparison group of similar chronological age (CA) rather than, for example, an MA-matched group. Upon theoretical analysis of the issues related to this question, Estes (1970) offers the following considerations and conclusion:

> If previous opportunities to learn are in fact equated for all the groups of subjects being compared, then among groups having the same CA, those with higher IQs will differ with respect to parameters having to do with rate of learning as well as with respect to present levels of the skills and knowledge sampled on the test. This is so, since the higher-IQ groups would have profited more from equal opportunities to learn over the same period of time. One might expect, then, by means of these comparisons, to localize the differences in learning rates with respect, for example, to particular types of tasks or intellectual functions, and to investigate conditions that might modify the differential learning rates in laboratory learning situations. (p. 56)

Similarly, both Ellis (1969) and Heal (1970), in reference to retardate-nonretardate comparisons, have suggested using a procedure in which CA and all nonintellectual variables are held constant.

A final consideration concerns the argument against lumping together different diagnostic types of mental retardation. Both Denny (1964) and Lipman (1963) have pointed out that the exclusion of

etiological category as a variable, which is commonly done in mental retardation research, is unfortunate, since it is possible that some specific deficit or combination of deficits is more basic with different etiological classifications. In accordance with this consideration, the subnormal subjects used in the present experiment were subdivided into two broad etiological categories: 'functional' retardates and 'structural' retardates. A functional retardate is defined as one in which there is no detectable pathology of the central nervous system, while a structural retardate exhibits evidence of such pathology. Although such a dichotomy is far from the refined medical classification that may be possible, it is nevertheless a realistic step in that direction.

METHOD

Subjects

A total of 103 persons served as subjects (Ss). Thirty-seven were mentally retarded, ranging in age from 18 to 25 years (mean = 20.6 years). They were selected from the resident population of the Ontario Hospital School, Cedar Springs. Only those retardates whose recorded IQ was between 30 and 65 and who did not have a hearing disability, a severe visual defect, or any neurological impairment of the limbs, and who were not psychotic, epileptic, or currently receiving medication were selected as Ss. In addition, the retardate sample was divided with respect to sex and etiological category ('structural' or 'functional'), as were all treatment groups derived from this sample.

Volunteers from the students enrolled in the Mental Retardation Counsellor certificate course, which was given at the same institution, served as nonretarded Ss. Their age was also restricted to the 18 to 25 year range (mean = 20.4 years) and their 'normal' intelligence established by a record of at least Grade 11 education. Sixty-six were selected as Ss in the same sex ratio as for the mentally retarded Ss.

Apparatus

The GSR conditioning and recording apparatus consisted of a Grason-Stadler Psychogalvanometer (Model E664) used in conjunction with a Fels Dermohmmeter (Model 22-A) and a two-channel direct inkwriting

oscillograph (Beckman Dynograph, Type RS). The Dermohmmeter produced a constant DC current of 70μA which was delivered to S through two recording electrodes which consisted of zinc discs, 2 cm. in diameter, set in Plexiglas cups. These electrodes were positioned on the volar and back surfaces of S's left hand. Electrode jelly composed of zinc sulphate, bacto-agar and distilled water was used between the zinc electrodes and the place of application. The Dermohmmeter provided a continuous measure of S's electrical skin resistance between the two recording electrodes which was recorded on one channel of the Dynograph operating at a chart speed of 1 mm/sec.

The UCS used for conditioning was a 1.0 mA electric shock of 0.5 sec. duration, generated by an electric shock source housed in the Psychogalvanometer. The shock was delivered through two zinc electrodes, 10 mm. in diameter, attached by adhesive tape to the index and third fingers of S's right hand.

One CS was an 8k Hz tone, presented over a loud speaker positioned directly in front of the seated S. The measured intensity of the tone was 70 db (re: 0.0002 dynes/cm^2), with a duration of 800 msec. The other CS was an 800 msec. diminution of the ambient light in the experimental chamber, from approximately 12.0 ft. candles to 0.07 ft. candles of illumination. For one-half of Ss in each treatment group receiving a differential conditioning sequence of trials, the pure tone served as the CS^+ and the visual stimulus as the CS^-. For the other half of such Ss, this arrangement was reversed. On trials in which the CS^+ was paired with the UCS, the ISI was 800 msec.; that is, UCS onset was coincident with the CS^+ offset. All stimuli as well as the ISIs

were sequentially controlled by timers incorporated in the Psychogalvanometer. The duration of both the tone and the UCS was also controlled by the Psychogalvanometer, while the duration of the visual stimulus was controlled by a Hunter decade interval timer (Model 111-C). Both the presentation sequence of all stimuli and the intertrial intervals (ITIs) were controlled by a Gerbrands three-channel punched-tape programmer. An event-marker of the Dynograph recorded CS onset on each trial.

The experiment was performed in a mobile laboratory in which a one-way window separated S from E and the bulk of the equipment. Plate I shows the general configuration of equipment and the experimental control portion of the laboratory, while Plate II illustrates the S chamber. Ss were seated in a comfortable chair throughout the experimental session and were shown commercially available motion pictures depicting animal wildlife and life in foreign countries. The super-8 mm. movies were projected by a Bolex projector to a reflectance mirror which directed the image to a viewing screen positioned about 1.5 m. directly in front of the seated S. The technique of showing movies to low grade retardates during eyelid conditioning was first reported by Ross, Koski, and Yaeger (1964) to distract Ss so they would tolerate the experimental situation.

Design

The overall experimental design was a 3 x 2 x 2 factorial type, involving Groups [mental retardates (MR), performance equated nonretardates (PN), or training equated nonretardates (TN)], Treatment

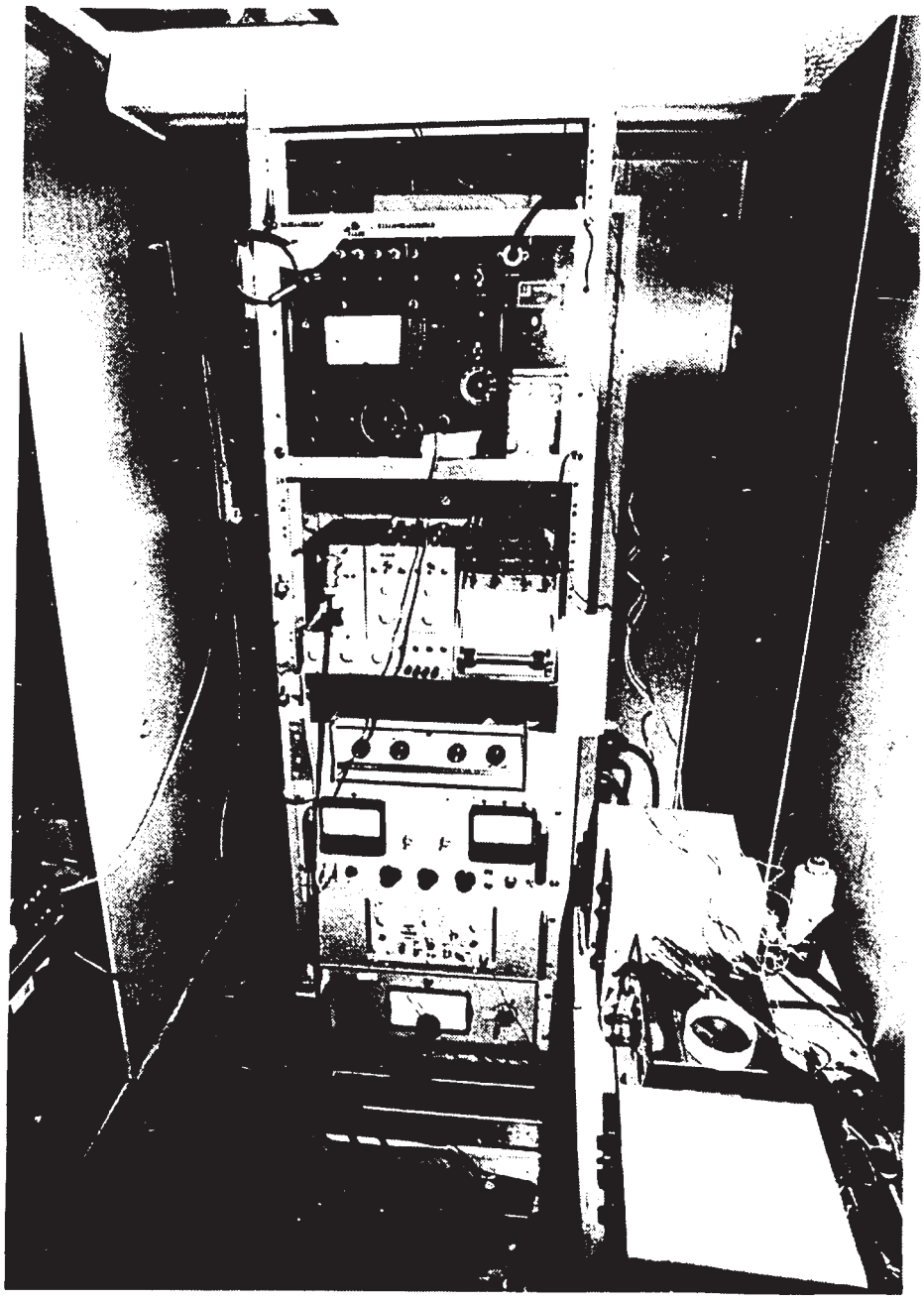

PLATE I. Configuration of equipment in the control portion of the laboratory.

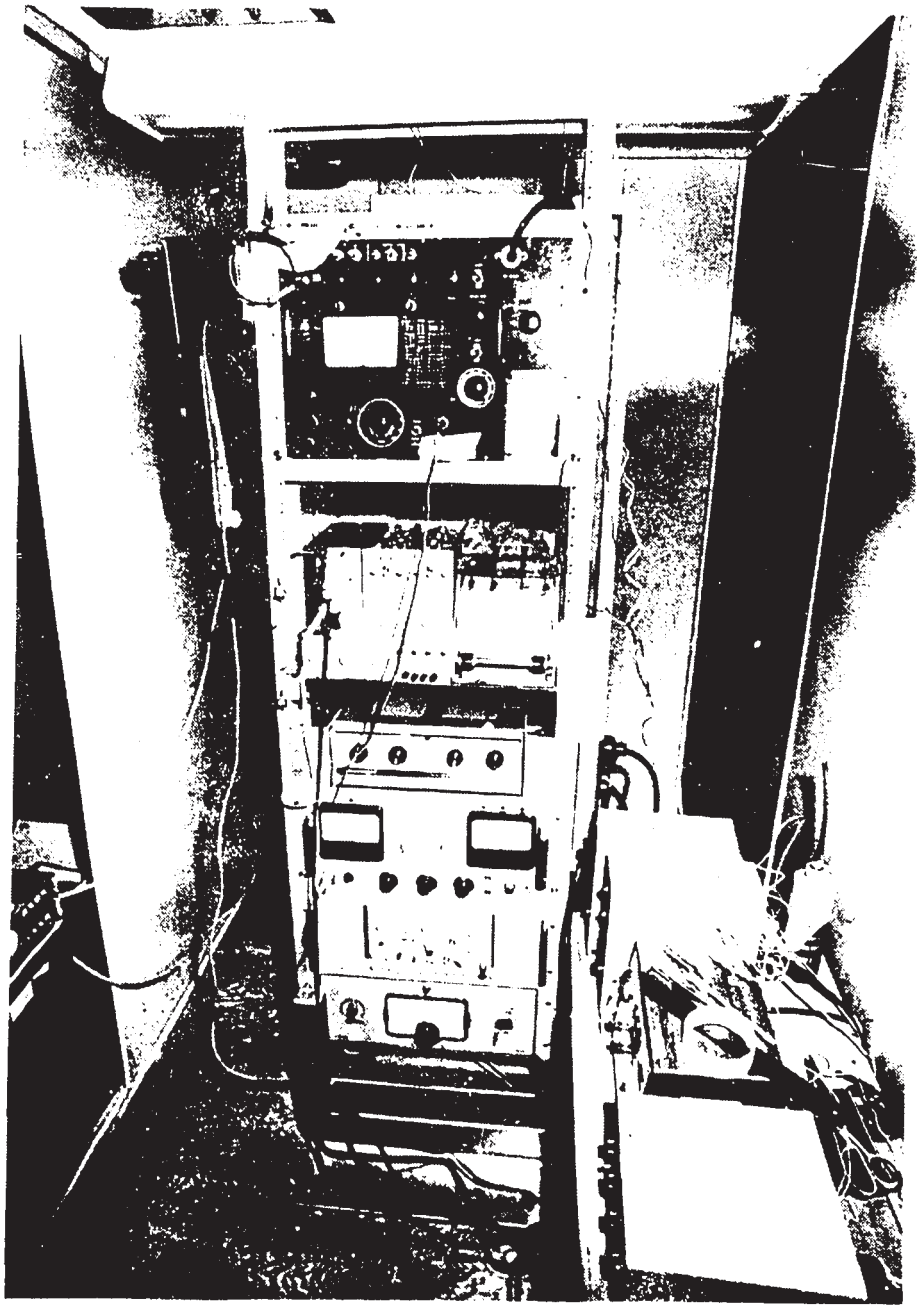

PLATE I. Configuration of equipment in the control portion of the laboratory.

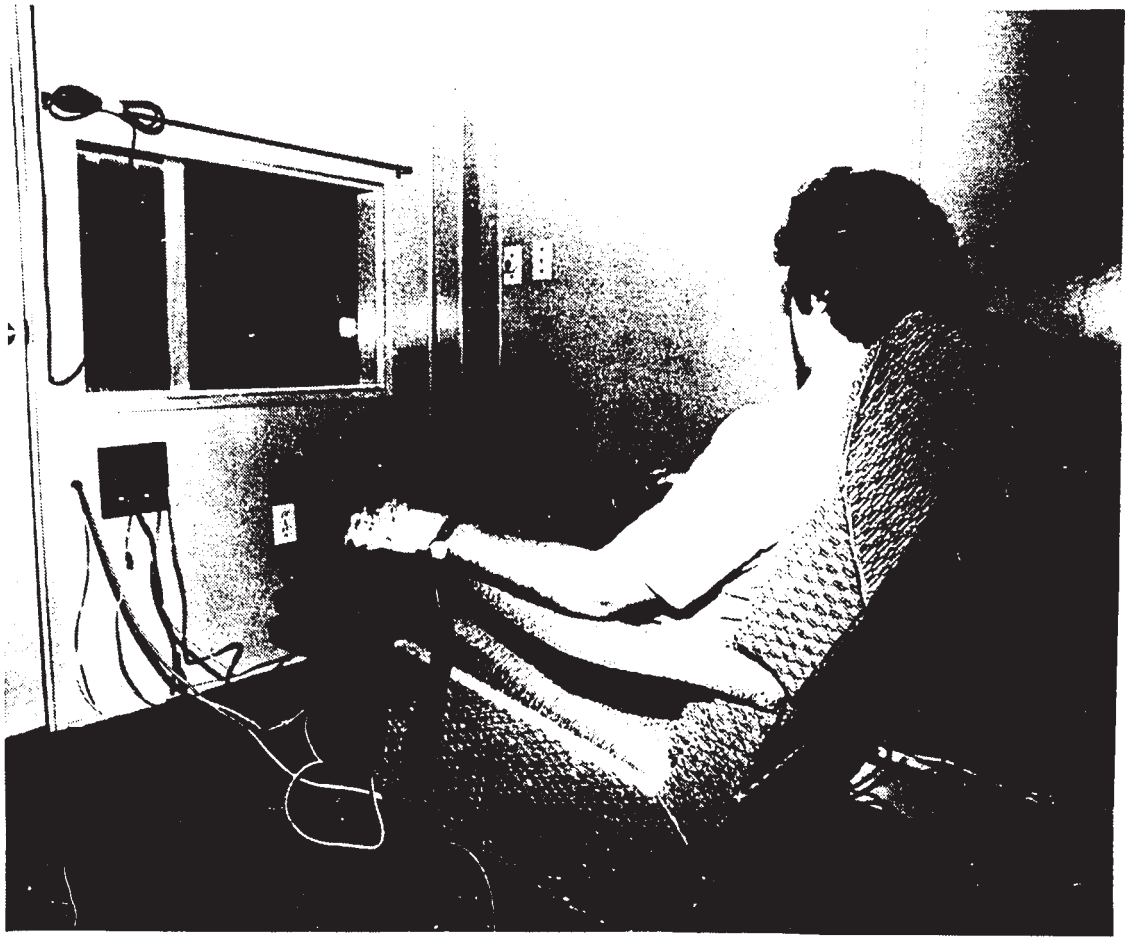

PLATE II. The subject chamber of the laboratory.

PLATE II. The subject chamber of the laboratory.

(conditioning or control), and Inhibition Tests (summation or reacquisition). Of the initial 103 Ss, 96 (32 retardates and 64 nonretardates) completed the experiment which consisted of three phases of training: habituation, differential conditioning, and inhibition testing. While the procedures used in each of these phases of training are detailed in the following section, a schematic summary of both the experimental design and the sequence of training used in this study is presented in Table 1.

As stated previously, it was hypothesized that the former CS^- used in the differential conditioning sequence of trials would have a greater suppressive effect for the nonretardates than it would for the retardates as measured by both the summation and reacquisition test procedures. Furthermore, the average magnitude of such suppressive effects could be determined within each of the three major groups of Ss by comparing the inhibition test data of Ss who had previously been differentially conditioned, with those of Ss who had received the control sequence of trials.

Procedure

Each S was individually tested by the same E. After S had been escorted to the laboratory and seated in the reclining chair, both the GSR recording electrodes and the fingershock electrodes were properly attached. During this period, nonretardates were instructed that the purpose of the experiment was to measure changes in their skin conductivity to various stimuli such as sudden darkness, noise, and/or mild electrical stimulation, and to compare such changes to those of mentally

TABLE 1. Schematic design of experiment.

	PHASES OF TRAINING		
	1st Phase (Habituation)	2nd Phase (Differential Conditioning)	3rd Phase (Inhibition Test)
MR	Habituation	Conditioning -- to criterion	Summation
			Reacquisition
		Control -- trial yoked	Summation
			Reacquisition
TN	Habituation	Conditioning -- trial yoked to MR Ss	Summation
			Reacquisition
		Control -- trial yoked	Summation
			Reacquisition
PN	Habituation	Conditioning -- to criterion	Summation
			Reacquisition
		Control -- trial yoked	Summation
			Reacquisition

retarded individuals of the same age. Retarded Ss were told essentially the same thing but such words as 'conductivity' and 'stimulation' were replaced with simpler words (e.g., 'skin messages' and 'tingles'). Following this, each S was asked to relax and pay attention to the movies. The S's electrical skin resistance was recorded throughout this five or six-minute relaxation period so as to allow for stabilization of S's basal resistance level. The first phase of training was then initiated. Each S received a series of habituation trials consisting of five presentations of the visual stimulus (L) and five presentations of the auditory stimulus (T). The sequence of stimuli was random (LTLTTLTLLT). The ITI varied between 20 and 60 sec. As in all phases of training, stimuli were presented according to an alternating sequence of 20 sec. periods of time. Periods of time in which a trial was randomly presented at any point within the period alternated with periods of time having equal duration in which a trial was never presented.

Immediately following the habituation trials, each S received a mild, subthreshold electrical fingershock of approximately five sec. duration which enabled E to determine the appropriate setting of the Psychogalvanometer stimulator, so as to deliver a 0.5 sec. current of approximately 1.0 mA. A similar procedure was repeated after every 16 training trials so as to permit readjustment in accordance with changes in the electrical resistance of the fingershock electrode-skin circuit. As outlined in Table 1, a series of either differential conditioning or control trials was then presented (Phase two).

One half of the retardate sample received a series of differential conditioning trials, 37.5 per cent of which were single CS^- trials,

37.5 per cent of which were paired CS^+ - UCS trials, and 25 per cent of which were CS^+ test trials. Moreover, each block of eight trials consisted of three CS^+ - UCS trials, three CS^- trials, and two CS^+ test trials presented in random order, apart from the restriction that no two CS^+ test trials occurred adjacently. The actual sequence of CS^-, CS^+, and CS^+ - UCS trials used in the differential conditioning series is presented in Appendix A. Each retardate in the conditioning group received such training until the following criterion[1] was reached: three consecutive GSRs elicited by the CS^- with a magnitude of not more than 50 per cent of those elicited on each of the two CS^+ test trials, in any eight trial block. If S failed to reach this criterion after 72 conditioning trials, he was replaced. Of the 37 retardates, two refused to participate and three who received differential conditioning training failed to reach the criterion.

The same type of differential conditioning training was given to two nonretarded groups (N = 16). Each S in one of these groups was individually yoked, in terms of identical trials, to one retardate who had received the differential conditioning sequence. Ss in this group were termed training equated. The other group of nonretarded Ss were trained to the same criterion of differentiation as were the retardates. These Ss were thus termed performance equated. Only one nonretarded S failed to reach criterion but one other was lost to the experiment through

---

[1]This criterion was based jointly on (1) pilot data obtained from college students with the same conditioning procedure, and (2) differential GSR magnitude displayed by mental retardates in the report of Grings, Lockhart, and Dameron (1962).

equipment breakdown.

Three corresponding control groups (one of retardates and two of nonretardates) also contained 16 Ss. Each S was individually trial-yoked to one S from the corresponding conditioning group. The procedure used with each of these groups was what might be described as a restricted 'truly random' CS^- control. This procedure involved the presentation of a series of CS^- alone trials which overlapped a sequence of paired CS^+ - UCS and CS^+ test trials identical to that used for differential conditioning. Three CS^- presentations occurred per eight trial block, the same as in the conditioning procedure. In the control procedure, however, CS^- presentation was programmed so that it could occur randomly at any point in time during any three of the eight 20 sec. periods of possible stimulus occurrence per block. A further specification was that the CS^- could occur only once in any such 20 sec. interval. As the CS^- could occur alone, or just precede, follow, or coincide with either CS^+ or paired CS^+ - UCS presentations, it bore no direct or obvious relationship with either the CS^+ or UCS. The actual stimulus sequence used in the control procedure is presented in Appendix A. Also presented in this Appendix is a schematic comparison of the conditioning and control procedures.

Following the completion of the differential conditioning or control phase of training, inhibition testing was initiated. As outlined in Table 1, each S received one of two randomly assigned treatments. One treatment (summation) consisted of the presentation of 15 trials: five CS^+ alone trials (+), five paired CS^+ - UCS trials (P), and five trials on which the CS^- and CS^+ were presented with simultaneous onset and

offset (±). The sequence of trials was random (±, ±, P, +, P, +, +, ±, P, P, +, ±, P, +, ±). The other treatment (reacquisition) consisted of 20 presentations of the former CS^-, a random 50 per cent of which were paired with the UCS. The sequence of paired (P) and unpaired (-) trials was as follows: P, -, P, P, -, -, P, -, P, -, P, -, P, -, -, P, P, -, P, -. In both inhibition tests, the stimulus parameters remained identical to those of the second phase of training.

RESULTS

The GSR was defined by the amount, measured in mm., by which the maximum positive deflection of the recording pen during the five sec. period, immediately following stimulus onset, exceeded the maximum positive deflection occurring in the five sec. period immediately preceding stimulus onset. This is a conventional scoring interval, necessitated by the considerable amount of variability in GSR latencies (see Gormezano, 1966). On the basis of calibration/conversion charts, each scored lineal difference (mm.) was converted to a value of resistance loss (ΔR) in ohms. At the single sensitivity setting of the Dermohmmeter used throughout the experiment, the minimum measureable ΔR was estimated to be 200 ohms. To reduce the effect of any extreme scores and possible nonnormality of the distribution of response magnitude, ΔR values were transformed to common logarithims. In order to avoid negative characteristics, any ΔR of zero or negative magnitude was assigned an initial value of 1.0 ohm.

Prior to performing any statistical analyses (detailed procedures for each analysis performed can be found in Winer, 1971) of performance differences Letween treatment groups, a preliminary analysis evaluated the so-called 'law of initial values'. According to this law, a positive relationship should exist between a subject's skin resistance and GSR magnitude. Pearson product-moment correlation coefficients (N = 96) were computed between initial resistance level (log R) and GSR ($\log \Delta R$)

on each of five trials selected from the habituation sequence, namely, trials one, three, four, seven, and nine. None of the coefficients attained statistical significance at the .05 level, a finding which replicates previous findings with the same and different equipment (e.g., Lobb, 1968; Carruthers, 1969; Lobb & Kaplun, 1970). Moreover, a one-way analysis of variance across the major subject groupings: performance equated nonretardates (PN), mental retardates (MR), and training equated nonretardates (TN), failed to reveal any significant differences in mean skin resistance taken over the same five habituation trials. In view of this evidence for the general independence of resistance level and GSR magnitude, the resistance measure (log ΔR) was retained. The correlation coefficients, mean GSR magnitudes, and resistance levels are presented in Appendix B[2].

Habituation

An overall 3 x $\overline{5 \times 2}^{3}$ x 4 analysis of variance with Groups (PN, MR or TN), Trials, Stimulus Type (tone or light diminution), and Treatment (conditioning/reacquisition, control/reacquisition, conditioning/summation, control/summation) as factors, was computed on GSR magnitude over the ten habituation trials. This analysis showed that there were no significant differences between any of the Groups or Treatment conditions, nor were there any significant interactions involving either of

---

[2]Appendix B also contains complete summaries of each major analysis performed, in order of mention, along with accompanying tables of cell means (unless otherwise presented).

[3]The superscribed symbol, $\overline{\quad}$, denotes within-subject variables.

these factors. There was, however, a significant Trials effect (F = 46.64, df = 4/336, $p < .001$) resulting from the general decrease in response magnitude across trials for all Groups. This effect is clearly seen in Figure 1 which presents mean habituation responses on each trial for each of the three major Groups (PN, MR, & TN). The analysis also yielded a significant main effect of Stimulus Type (F = 6.99, df = 1/84, $p < .025$), which indicated a slight superiority of the visual stimulus for GSR elicitation.

Both significant main effects of Trials and Stimulus Type were further qualified by a significant Trials x Stimulus Type interaction (F = 2.45, df = 4/336, $p < .05$). This two-way interaction is diagrammed in Figure 2. Additional analyses (t tests) indicated that the only points during the habituation phase of training at which the two types of stimuli resulted in a significant difference in response magnitude were the second and final presentations of each, the visual stimulus producing the larger responses ($p < .05$).

To determine if etiological category (and its possible interaction with sex) was a significant variable which resulted in initial systematic differences, a subsequent analysis was performed on the habituation data of the retardate sample (N = 32). A 2 x $\overline{5 \text{ x } 2}$ x 2 analysis of variance having Sex, Trials, Stimulus Type, and Etiological Class (structural or functional) as factors failed to show, however, any significant effect or interaction involving etiology.

Differential Conditioning

In order to compare the rate at which the criterion of differentiation (three consecutive GSRs elicited by the CS^- which had a

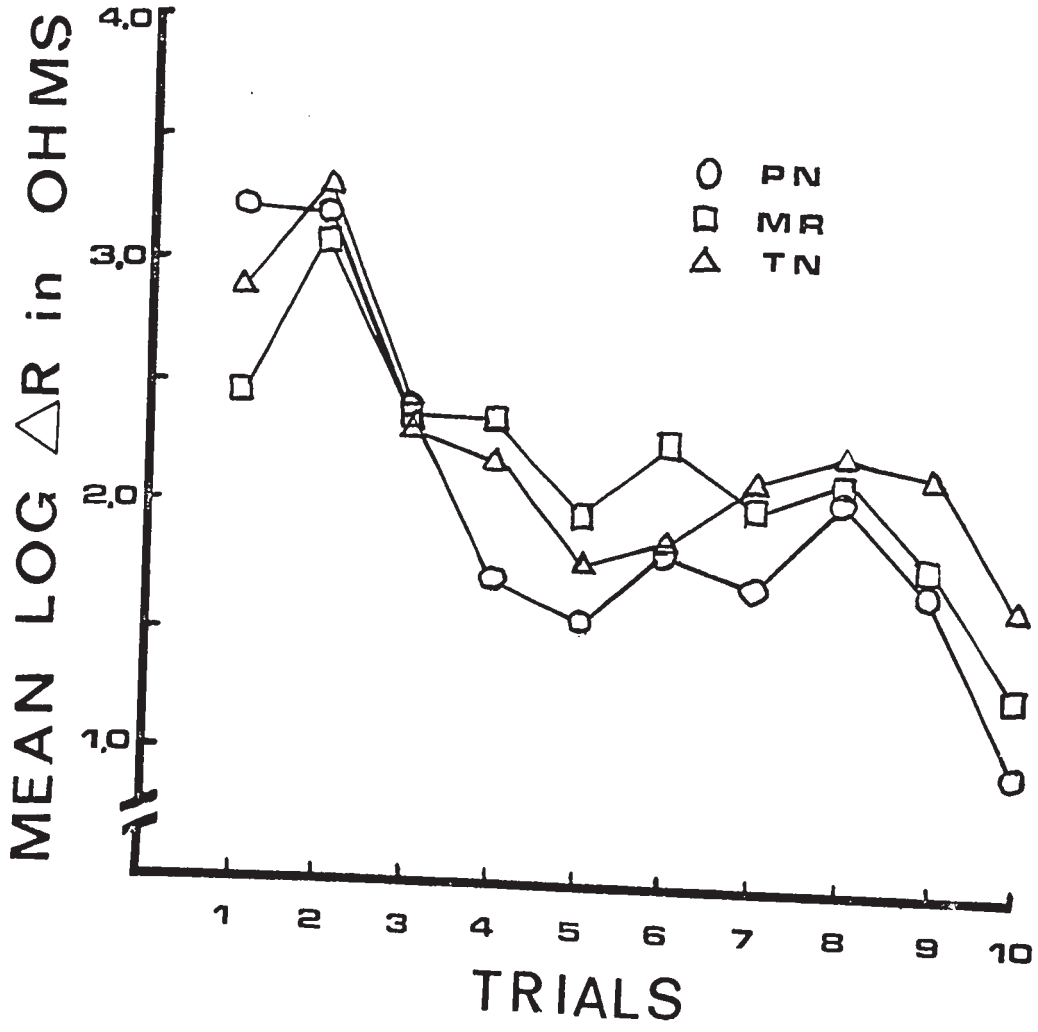

Figure 1. Habituation: Mean GSR magnitude as a function of Groups (PN, MR, & TN) and Trials.

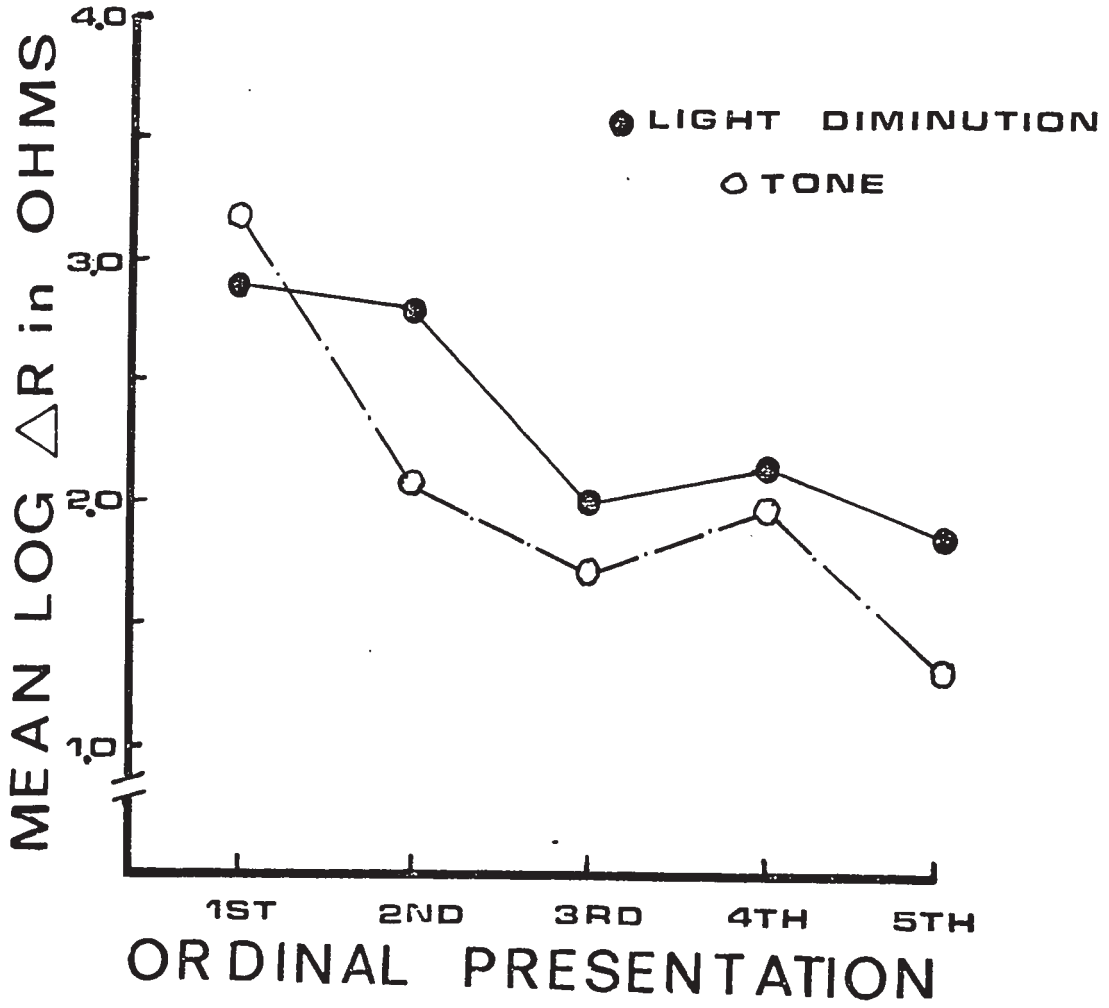

Figure 2. Habituation: Mean GSR magnitude as a function of Stimulus Type (tone or light diminution) and Trial sequence.

magnitude of not more than 50 per cent of those elicited on the two CS^+ test trials in any eight trial block) was reached by the PN and MR subjects, a one-way analysis of variance was performed on the number of trials to criterion. This analysis showed there was a significant difference (F = 53.28, df = 1/30, $p < .001$) between the PN subjects who required a mean of 27 trials (3.4 blocks) and the retarded subjects who required a mean of 46 trials (5.7 blocks). This difference clearly indicates the nonretardates' superiority on this particular task.

The major analysis of the differential conditioning phase of training compared the degree of response differentiation attained by the TN subjects (who were not trained to criterion but were trial-yoked to the retardates) with that of the PN and MR subjects, as well as group performance during the control sequence of trials. A 3 x $\overline{2 \times 2}$ x 2 analysis of variance with Groups (PN, MR, or TN), Blocks of Trials, Trial Type (CS^- or CS^+), and Treatment (conditioning or control) as factors, was computed on the mean GSR magnitudes on the final two blocks of training. This analysis revealed two significant main effects, that of Treatment (F = 4.77, df = 1/90, $p < .05$) and of Trial Type (F = 48.06, df = 1/90, $p < .001$). Respectively, these main effects reflected the fact that the average level of responding was greater in those subjects who had received the conditioning sequence than it was in those who had received the control procedure, and that the average level of responding was greater to the CS^+ than to the CS^- when all groups were combined.

Both main effects of Treatment and Trial Type were qualified by a significant two-way interaction (F = 9.26, df = 1/90, $p < .01$) involving these same two factors. Figure 3, which graphically displays the

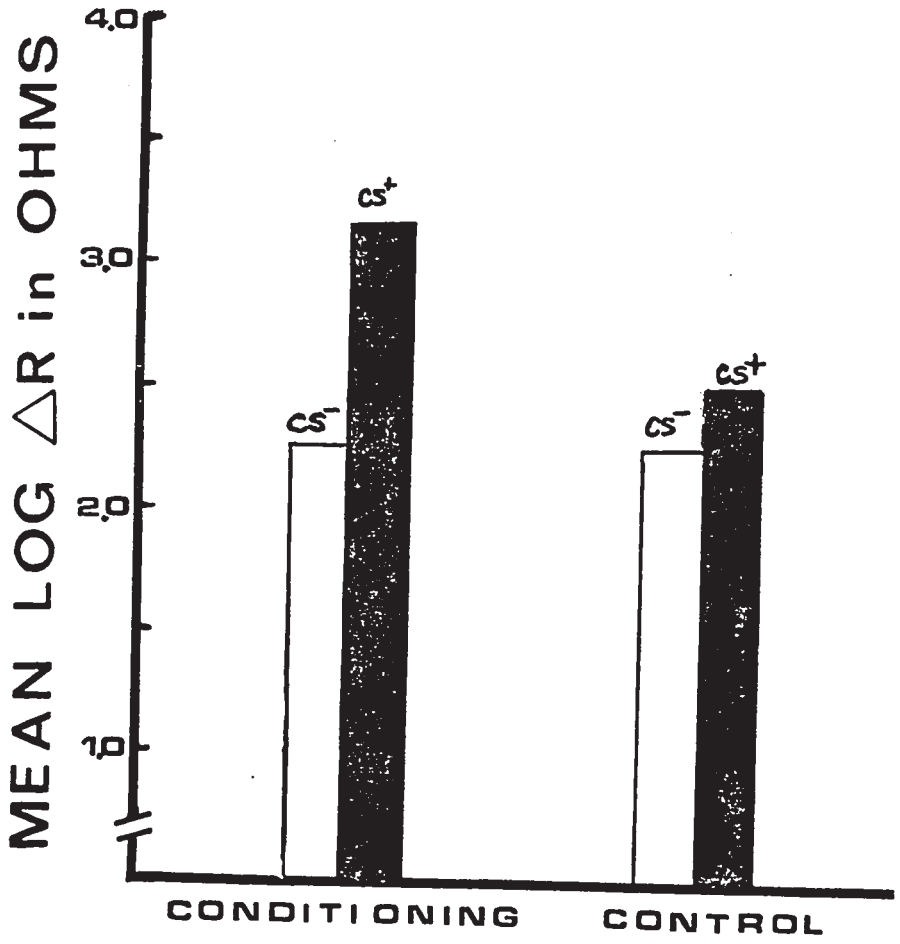

Figure 3. Differential Conditioning: Mean GSR magnitude as as function of Treatment (con'd or control) and Trial Type (CS^+ or CS^-) over the last two eight-trial blocks of training.

Treatment x Trial Type interaction, illustrates the fact that while there was a substantial difference in the magnitude of GSRs elicited by the CS^+ between conditioning and control groups, there was no difference in CS^- responding between the two groups. Subsequent analyses (t tests) confirmed that both the conditioning and control procedures resulted in a significant degree of response differentiation ($p < .05$), and hence, conditioning. Finally, a significant Blocks x Trial Type interaction ($F = 8.65$, $df = 1/90$, $p < .01$) simply reflected the fact that response differentiation increased over the last two blocks of training, and is not shown graphically. A similar analysis, with Stimulus Type (tone or light diminution) as an additional factor, failed to reveal any significant main effect or interactions involving this factor.

As the analyses of the differential conditioning phase of training yielded neither a significant Group difference, nor any significant interaction involving this factor, it may be concluded that in terms of the degree of response differentiation attained, the retarded subjects who received conditioning training did not differ from the nonretardates trained to the same criterion (PN), or from nonretardates who received an identical number of trials. Similarly, there was no significant difference between any of the three subject groups which received the control sequence of trials, in responses to CS^+ or CS^-. Although the mean number of trial blocks differed greatly between the two nonretarded groups (PN = 3.4 and TN = 5.7), the two groups did not differ significantly in the amount of response differentiation at the completion of the differential conditioning phase of training. Furthermore, the

additional training received by the TN subjects did not apparently affect absolute GSR levels.

Conditioned Inhibition Tests

Summation. GSR magnitude on each of the five CS^+ alone trials was directly compared with that elicited on each of the five CS^-/CS^+ compound trials for subjects who received summation testing. A $3 \times \overline{5 \times 2} \times 2$ analysis of variance with Groups (PN, MR, or TN), Trials, Trial Type (CS^+ or CS^-/CS^+), and Treatment (conditioning or control) as factors, showed a significant main effect of Groups ($F = 3.02$, $df = 2/42$, $p < .05$) as well as three significant interactions.

Figure 4 illustrates the significant triple interaction of Groups x Trial Type x Treatment ($F = 4.98$, $df = 2/42$, $p < .05$) and also shows the average magnitude of group responding during summation testing combined across trials. It can be seen in Figure 4 that while all of the major subject groups (PN, MR, & TN) which had previously received the control sequence of trials exhibited GSRs of greater magnitude on summation trials than on CS^+ alone trials, this situation was reversed in every group which had received conditioning training, except the retardates. This interpretation was confirmed by t tests. The CS^-, when presented in combination with the CS^+, resulted in significantly smaller responses than did the CS^+ alone in both the PN and TN conditioning groups, but produced significantly larger responses than did the CS^+ in the retardate conditioning group. Whereas the former CS^- suppressed the responding of nonretarded subjects, it tended to augment the responding of mental defectives as it did in all control groups.

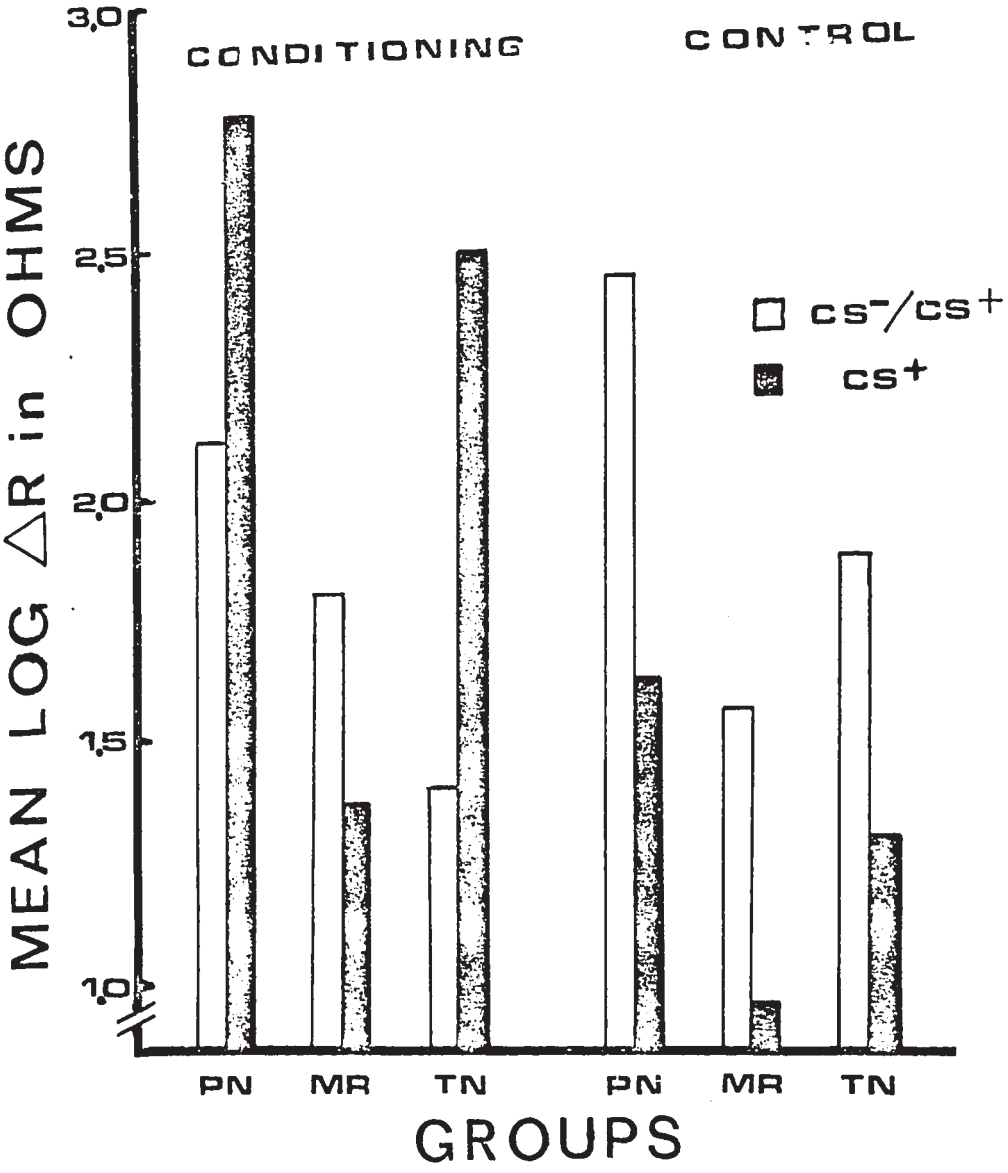

Figure 4. Summation: Mean magnitude GSR as a function of Groups (PN, MR, & TN), Treatment (con'd or control), and Trial Type (CS^-/CS^+ or CS^+).

Of the two remaining significant interactions of the summation analysis, the Group x Trial Type (F = 4.60, df = 2/42, $p < .05$) interaction was closely related to the triple interaction just discussed. Although confounded with Treatment, this interaction indicated that while both groups of nonretarded subjects yielded GSRs of only a slightly greater magnitude than did the retardates on the summation trials, their GSRs were much greater than retardates' on CS^+ trials. Responses of the retardates to the CS^+ were greatly reduced in comparison to their summation responding. Finally, the significant Trial Type x Treatment interaction (F = 26.85, df = 1/42, $p < .001$) indicated that in terms of CS^+ responding the control and conditioning treatments resulted in a divergence in opposite directions, but in terms of responses to the compound stimulus there was no significant difference between conditioning and control treatments for combined PN, MR, and TN groups.

Reacquisition. The ten CS^- trials of the reacquisition sequence were divided into five two-trial blocks for the purpose of analysis. A 3 x $\overline{5}$ x 2 analysis of variance with Groups (PN, MR, or TN), Blocks, and Treatment (conditioning or control) as factors, was computed on mean magnitude GSRs per block. This analysis yielded a significant effect only for Treatment (F = 4.95, df = 1/42, $p < .05$). This effect indicates that reacquisition of CRs to the former CS^- was retarded in all groups of subjects which had been differentially conditioned in comparison to the performance of those subjects who had previously received the control procedure. Figure 5 illustrates Group responding as a function of Treatment and Trials on the reacquisition test. It can be seen that the retardates previously given differential conditioning training

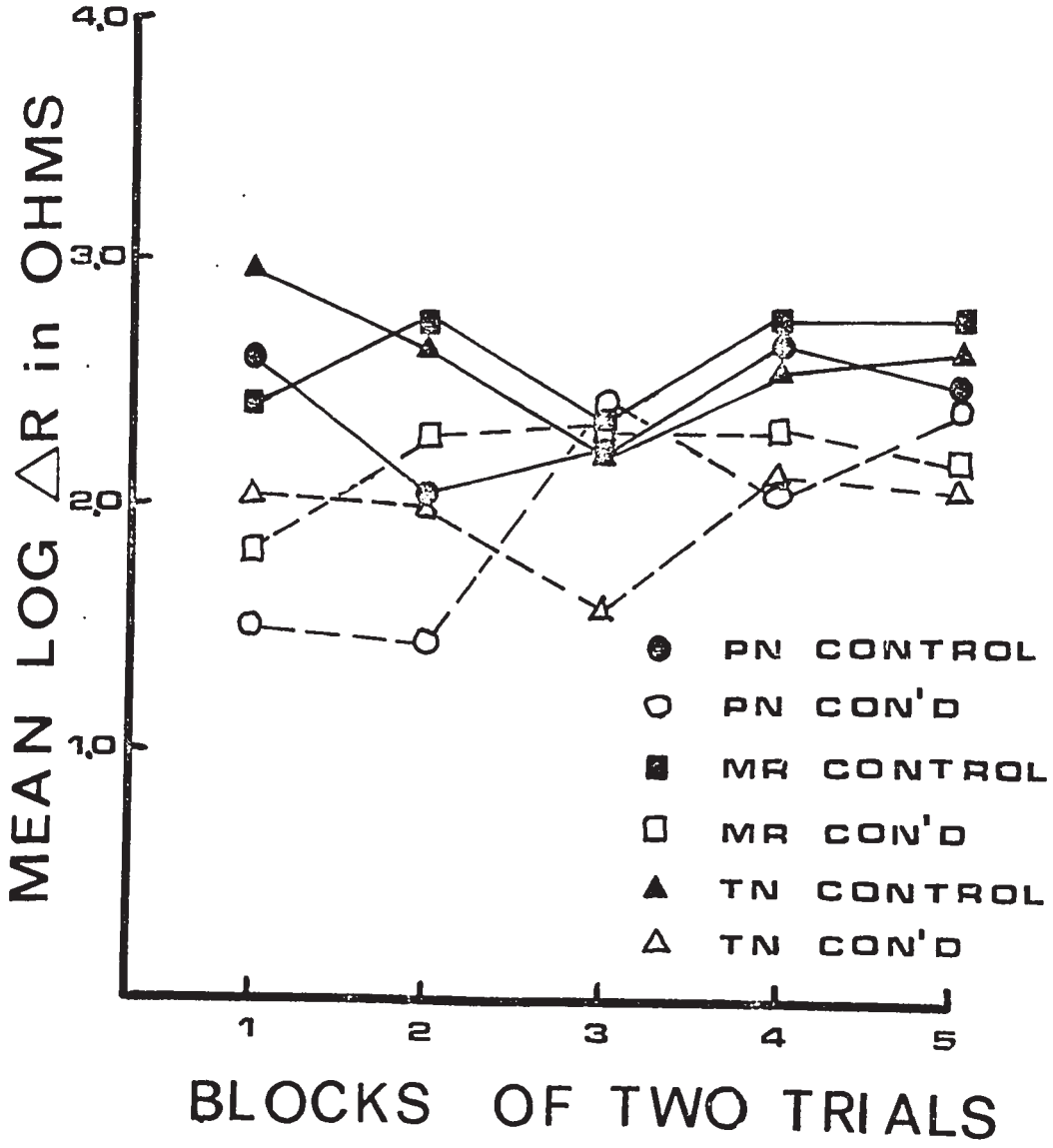

Figure 5. Reacquisition: Mean magnitude GSR as a function of Treatment group (PN/con'd, MR/con'd, TN/con'd, PN/control, MR/control, or TN/control) and Blocks of two trials.

attained roughly the same level of CS^- responding, at about the same rate, as did either group of corresponding nonretardates.

Additional analyses were performed on the inhibition test data of the retarded subjects in order to determine if etiological category was a significant variable affecting the measured strength of conditioned inhibition. Two one-way analyses of variance with Etiological Class as the only factor were computed on both the summation data (mean GSR magnitude over the five summation trials) and the reacquisition data (mean GSR magnitude over the ten CS^- alone trials). These analyses failed to show any significant effects.

DISCUSSION

The summation data clearly conformed to theoretical expectation. The finding that the CS^-/CS^+ combination elicited responses which were significantly smaller than those elicited by the CS^+ alone in both groups of nonretarded subjects which had previously received differential conditioning training, but not in the retardates who had also received such training, is consistent with the inhibition deficit hypothesis. The summation data of corresponding nonretardate controls further indicate that this suppressive effect was not merely due to a stimulus generalization decrement, since in these subjects, CS^-/CS^+ presentations produced responses of a significantly greater magnitude that did CS^+ presentations.

In terms of the summation data, it thus appears that while the former CS^- did become an effective conditioned inhibitor of the responding of nonretardates, it acquired no such inhibitory control over the responding of the mentally retarded subjects. As pointed out previously, however, the summation test alone cannot completely confirm a conditioned inhibition effect, especially in situations where attentional processes may be assumed to operate. A stimulus which does in fact lead to the selective attention of a subject, however, should not produce conditioned inhibitory effects in both the summation and reacquisition tests.

As there was no significant difference between retarded and nonretarded subjects, in terms of the rate of acquisition of CRs to the former CS^-, the reacquisition data do not directly support the inhibition deficit hypothesis. The data, however, are consistent with the hypothesis that in both retarded and nonretarded subjects the former CS^- gained some degree of inhibitory control. This is indicated by the fact that reacquisition responses of those subjects who had previously been differentially conditioned were of a significantly lesser magnitude than those of corresponding controls. If, as the summation data suggest is the case, retarded individuals suffer from a conditioned inhibition deficit, the finding that the rate of reacquisition of CRs was retarded to roughly the same extent in both retardates and nonretardates would not be predicted. One possible explanation of this discrepancy is that while both the reacquisition and summation tests are capable of detecting conditioned inhibitory effects, the reacquisition test may not be as sensitive to differences in the strength of conditioned inhibitors. The most direct method of empirically evaluating this suggestion would be to compare summation and reacquisition responding following different amounts of differential conditioning training and/or varying degrees of response differentiation in samples of nonretarded subjects. Such comparisons would provide a measure of the sensitivity of both the summation and reacquisition techniques, in terms of their measurement of suppressive effects of conditioned inhibitors of varying strength, as well as indicating the degree of correspondence between such measures based on the two techniques.

It may be suggested that the discrepancy between the summation

and reacquisition data could be accounted for in terms of hypothesized 'attentional' differences between retardates and nonretardates (see Zeaman & House, 1963). Assuming that rather than an inhibition deficit, retardates suffer some type of 'attentional deficit' such that they would pay less attention to the CS^- than would nonretardates, the CS^- would be expected to have little suppressive effect on the CS^+ responses of retardates in the summation test as compared to nonretardates. Furthermore, such inattention on the part of retardates would be expected to result in greater retardation of the development of CRs to the former CS^- in the reacquisition test as compared to nonretardates. Although the findings of the present experiment are somewhat consistent with the former prediction, the fact that the performance of retardates and nonretardates did not differ significantly during reacquisition testing does not support the latter. Moreover, the performance of both the retarded and nonretarded control subjects does not reflect any such attentional differences since their patterns of responding did not qualitatively differ in either the summation or reacquisition test. It thus appears that presumed attentional differences between retarded and nonretarded individuals cannot adequately account for the observed results.

Although there was no significant difference between retardate and nonretardate conditioning groups in terms of reacquisition responding as had been predicted, there is some evidence to suggest that this nonretardate-retardate equality in itself may indicate that the reacquisition responding of the nonretarded subjects was in fact retarded as compared to the performance of the mental defectives. The evidence which supports this contention is the frequent observation that retardates exhibit

weaker or slower acquisition of CRs than do nonretardates in single-cue conditioning (e.g., Lobb, 1968; Lobb & Nugent, 1966; Ross, Koski, & Yaeger, 1964). Thus, if the former CS^- had acquired no conditioned inhibitory properties whatsoever in either the retarded or nonretarded subjects, the retardates would be expected to be inferior to the nonretardates in terms of the rate of reacquisition. On the other hand, if the CS^- became a much stronger conditioned inhibitor in the nonretarded subjects than in the retardates, the nonretardates reacquisition responding would be expected to be suppressed to a much greater extent, possibly to the extent that the two intelligence groups would exhibit approximately equivalent patterns of reacquisition. Although the latter in fact occurred, this interpretation receives little support from the corresponding control group data, since there was little, if any, difference in the reacquisition performance of retarded and nonretarded subjects. Furthermore, in order to accurately evaluate such an interpretation, further comparative investigation of the effects of appropriate independent variables such as UCS duration and intensity, ISI and ITI, on single-cue conditioning in both mental defectives and nonretardates is necessary. If, for example, values of such conditioning parameters are found at which retardates and nonretardates consistently exhibited comparable rates of acquisition of CRs to a single CS, then such parameters could be used in reacquisition procedures designed to measure the strength of a suspected conditioned inhibitor in both types of subjects.

The control procedure employed in the present experiment appears to have provided an appropriate means of control to evaluate the accumulation of conditioned inhibitory strength by the CS^-. Examination of both the summation and reacquisition data indicates that for all subject groups which had previously received the control procedure, the pattern of results is what would be expected if the CS^- was not a conditioned inhibitor. These data indicate that rather than having a suppressive effect, the former CS^- of the control sequence elicited responding similar to that which would occur with a neutral stimulus. In the case of the reacquisition test, for example, the development of CRs to the control CS^- was more rapid and at higher level than it was to the former CS^- of the differential conditioning sequence for all groups. Similarly, the summation data of the control groups indicate that responses of a greater magnitude were elicited on CS^-/CS^+ trials than on CS^+ alone trials. Thus, rather than having an inhibitory effect, the former CS^- of the control sequence resulted in a slight increment in CS^+ responding. This increment may have partly been the result of a weak novelty effect, but more likely was due to the slight excitatory property of the CS^- resulting from generalized excitation from the positive to negative cue during the control phase of training. It is also conceivable that the CS^- of the control sequence may have developed slight excitatory properties due to random contiguous pairings with the UCS. That such contiguity, even in the absence of any CS-UCS contingency, can lead to the development of excitation has recently been documented by Kremer and Kamin (1971).

An additional finding of this experiment which is of interest, is that neither in the differential conditioning phase of training, nor in either of the two tests of conditioned inhibition were there any significant differences between the two major groups of nonretarded subjects. Thus, the additional differential conditioning training which the TN group received in comparison to the PN subjects failed to significantly alter the degree of response differentiation attained. Moreover, extended differential conditioning training failed to increase the conditioned inhibitory strength of the former CS^-, at least as measured by both the summation and reacquisition techniques in nonretarded subjects. This finding suggests that there is an upper limit to the strength which a conditioned inhibitor may attain under specific parameters, and once this limit is reached, further training will not increase its strength.

As there were no etiological class differences displayed, it may be that both structural and functional retardates perform equally well (or poorly, as the case may be) in the types of tasks used in the present experiment. The fact that diagnosed organicity did not differentially affect autonomic nervous system responsiveness and was not found to be a significant variable in this experiment indicates that perhaps a gross type of CNS damage is not a relevant dimension along which to classify mentally defective persons in regard to inhibitory functions. This conclusion, however, relies heavily upon both the validity and reliability of medical diagnoses; neither of which are impressively high (e.g., Chambers, 1971), especially with reference to the functional category.

REFERENCES

Baumeister, A. A., Beedle, R., & Urquhart, D. GSR conditioning in normals and retardates. *American Journal of Mental Deficiency*, 1964, *69*, 114-120.

Berger, A. Inhibition of the eyelid reflex in three etiologic groups of mentally retarded boys as compared with normals. *Training School Bulletin*, 1954, *51*, 146-152.

Brown, P. L., & Jenkins, H. M. Conditioned inhibition and excitation in operant discrimination learning. *Journal of Experimental Psychology*, 1967, *75*, 255-266.

Bull, V. A. III, & Overmier, J. B. Additive and subtractive properties of excitation and inhibition. *Journal of Comparative and Physiological Psychology*, 1968, *65*, 356-358.

Carlton, P. L., & Vogel, J. R. Habituation and conditioning. *Journal of Comparative and Physiological Psychology*, 1967, *63*, 348-351.

Carruthers, B. C. Effects of CS duration on classical conditioning of concurrent eyeblink and GSR in mental defectives. Unpublished M.A. thesis, University of Western Ontario, 1969.

Chambers, D. A. Objectives of behavioral measurement. Paper presented at 'Century Two' Symposium, London Psychiatric Hospital, London, Canada, May, 1971.

Denny, R. Research in learning and performance. In H. A. Stevens & R. Heber (Eds.), *Mental Retardation*. Chicago: University of Chicago Press, 1964.

Diamond, S., Balvin, R. S., & Diamond, F. R. *Inhibition and choice*. New York: Harper and Row, 1963.

Eccles, J. C. *The physiology of synapses*. New York: Academic Press, 1964.

Ellis, N. R. A behavioral research strategy in mental retardation: Defense and critique. *American Journal of Mental Deficiency*, 1969, *73*, 557-560.

Estes, W. K. *Learning theory and mental development.* New York: Academic Press, 1970.

Estes, W. K., & Skinner, B. F. Some quantitative properties of anxiety. *Journal of Experimental Psychology,* 1941, *29,* 390-400.

Gormezano, I. Classical conditioning. In J. B. Sidowski (Ed.), *Experimental methods and instrumentation in psychology.* York: McGraw-Hill, 1966.

Grings, W. W., Lockhart, R. A., & Sameron, L. E. Conditioning autonomic responses of mentally subnormal individuals. *Psychological Monographs,* 1962, *76* (39, Whole No. 558).

Hammond, L. J. Retardation of fear acquisition when the CS has previously been inhibitory. *Journal of Comparative and Physiological Psychology,* 1968, *66,* 756-759.

Hammond, L. J. A traditional demonstration of the properties of Pavlovian inhibition using differential CER. *Psychonomic Science,* 1967, *5,* 337-338.

Heal, L. W. Research strategies and research goals in the scientific study of the mentally subnormal. *American Journal of Mental Deficiency,* 1970, *75,* 10-15.

Heal, L. W., & Johnson, J. T. Jr. Inhibition deficits in retardate learning and attention. In N. R. Ellis (Ed.), *International review of research in mental retardation.* Vol. IV. New York: Academic Press, 1970.

Heal, L. W., Ross, L. E., & Sanders, B. Reversal and partial reversal in mental defectives and normal children of a comparable age. *American Journal of Mental Deficiency,* 1966, *71,* 411-416.

Heber, R. (Ed.) A manual on terminology and classification in mental retardation. *Monograph Supplement, American Journal on Mental Deficiency,* 1958, *64,* No. 2.

Hilgard, E. R., & Bower, G. H. *Theories of Learning.* New York: Appleton-Century-Crofts, 1966.

Hull, C. L. *A behavior system: An introduction to behavior theory concerning the individual organism.* New Haven: Yale University Press, 1952.

Jenkins, H. M. Generalization gradients and the concept of inhibition. In D. I. Mostofsky (Ed.), *Stimulus generalization.* Stanford: Stanford University Press, 1965.

Johnson, J. T., Jr. Developmental aspects of inhibition and the orienting reflex. Unpublished doctoral dissertation. George Peabody College for Teachers, Nashville, Tennessee. 1968.

Johnson, J. T., Jr., & Heal, L. W. CS-UCS interval in differential eyelid conditioning with retardates. *Psychonomic Science,* 1967, *9,* 327-328.

Kimble, D. P. Hippocampus and internal inhibition. *Psychological Bulletin,* 1968, *70,* 285-295.

Kimmel, H. D., & Pennypacker, H. S. Differential GSR conditioning as a function of the CS-UCS interval. *Journal of Experimental Psychology,* 1963, *65,* 559-563.

Konorski, J. *Conditioned reflexes and neuron organization.* Cambridge: Cambridge University Press, 1948.

Kremer, E., & Kamin, L. J. The truly random control procedure: associative or nonassociative effects in rats. *Journal of Comparative and Physiological Psychology,* 1971, *74,* 203-210.

Lipman, R. S. Learning: Verbal, perceptual-motor, and classical conditioning. In N. R. Ellis (Ed.), *Handbook of mental deficiency: Psychological theory and research.* New York: McGraw-Hill, 1963.

Lobb, H. Trace GSR conditioning with benzedrine in mentally defective and normal adults. *American Journal of Mental Deficiency,* 1968, *73,* 239-246.

Lobb, H., & Kaplun, J. Protection of GSR conditioning by Dextroamphetamine. *Canadian Journal of Psychology,* 1970, *24* (1).

Lobb, H., & Nugent, C. M. Interaction between intelligence level and interstimulus trace interval in electrodermal conditioning. *American Journal of Mental Deficiency,* 1966, *70,* 548-555.

Lockhart, R. A., & Grings, W. W. Interstimulus interval effects in GSR discrimination conditioning. *Journal of Experimental Psychology,* 1964, *67,* 209-214.

Lubow, R. E. Latent inhibition: effects of frequency of nonreinforced pre-exposure of the CS. *Journal of Comparative and Physiological Psychology,* 1965, *60,* 454-457.

Marinesco, G., & Kreindler, A. Des réflexes conditionnels. *Journal Psychologie,* 1933, *30,* 855-886.

Montagu, J. D., & Coles, E. M. Mechanism of measurement of the galvanic skin response. *Psychological Bulletin,* 1965, *65,* 261-279.

Ohlrich, E. S. The effect of CS-UCS interval on the single-cue differential eyelid conditioning of retarded children. Unpublished doctoral dissertation, University of Wisconsin, 1963.

Ohlrich, E. S., & Ross, L. E. Acquisition and differential conditioning of the eyelid response in normal and retarded children. *Journal of Experimental Child Psychology*, 1968, *6*, 181-193.

Pavlov, I. P. *Conditioned reflexes*. London: Oxford University Press, 1927.

Rescorla, R. A. Pavlovian conditioned inhibition. *Psychological Bulletin*, 1969, *72*, 77-94.

Rescorla, R. A. Pavlovian conditioning and its proper control procedures. *Psychological Review*, 1967, *74*, 71-80.

Rescorla, R. A., & Lolordo, V. M. Inhibition of avoidance behavior. *Journal of Comparative and Physiological Psychology*, 1965, *59*, 406-412.

Rodnick, E. H. Does the interval of delay of conditioned responses possess inhibitory properties? *Journal of Experimental Psychology*, 1937, *20*, 507-527.

Ross, L. E. Classical conditioning and discrimination learning: Research with the mentally retarded. In N. R. Ellis (Ed.), *International review of research in mental retardation*, Vol. I, New York: Academic Press, 1966.

Ross, L. E., & Wilcox, S. M. Conditioned stimulus parameters and the interstimulus interval: the processing of CS information in differential conditioning. Mimeographed copy of paper prepared for classical conditioning symposium, University of Wisconsin, Madison, Wisconsin, 1969.

Ross, L. E., Koski, C. H., & Yaeger, J. Classical eyelid conditioning of the severely retarded: partial reinforcement effects. *Psychonomic Science*, 1964, *I*, 253-254.

Schnur, P., & Ksir, C. J. Latent inhibition in human eyelid conditioning. *Journal of Experimental Psychology*, 1969, *80*, 388-389.

Scott, K. G. Intelligence and learning. In H. C. Haywood (Ed.), *Psychometric intelligence*. New York: Appleton-Century-Crofts, 1970.

Sechenov, I. *Selected Works*. Moscow: State Publishing House, 1935.

Sergeev, B. F. On the localization of inhibition in the extinction of conditioned reflexes to chain stimuli. *Pavlov Journal of Higher Nervous Activity*, 1961, *11*, 695-700.

Sherrington, C. S. *The integrative action of the nervous system.* New Haven: Yale University Press, 1906.

Sokolov, E. N. Neuronal models and the orienting reflex. In M. A. Brazier (Ed.), *The central nervous system and behavior.* New York: Josiah Macy Foundation, 1960.

Spence, K. W. *Behavior theory and conditioning.* New Haven: University Press, 1956.

Spence, K. W. *Behavior theory and learning. Selected papers.* Englewood Cliffs: Prentice-Hall, 1960.

Spence, K. W., & Platt, J. R. Effects of partial reinforcement on acquisition and extinction of the conditioned eyeblink in a masking situation. *Journal of Experimental Psychology,* 1967, *74*, 259-263.

Szwejkowska, G. The effect of a primary inhibitory stimulus upon the positive salivary conditioned reflex. *Bulletin De L'Academic Polonaise Des Sciences,* 1957, *5*, 393-396.

Szwejkowska, G., & Konorski, J. The influence of the primary inhibitory stimulus upon the salivary effect of excitatory conditioned stimulus. *Acta Biological Experimentalis,* 1959, *19,* 162-174.

Winer, B. J. *Statistical principles in experimental design, 2nd Edition.* New York: McGraw-Hill, 1971.

Zeaman, D., & House, B. J. The role of attention in retardate discrimination learning. In N. R. Ellis (Ed.), *Handbook of mental deficiency: Psychological theory and research.* New York: McGraw-Hill, 1963.

APPENDIX A

Sequences of stimuli and schematic of procedures used in the differential conditioning phase of training

TABLE 1

Sequence of CS^+, CS^-, and paired CS^+-UCS (P) trials used in the differential conditioning phase of training for both the conditioning and control procedures.

Conditioning				Control**			
Trial*#	Stimulus	Trial #	Stimulus	Trial #	Stimulus	Trial #	Stimulus
11	P	43	CS^-	11	P	43	CS^-
12	CS^-	44	CS^-	12		44	CS^-
13	CS^+	45	P	13	CS^+	45	P
14	P	46	CS^+	14	P	46	CS^+
15	CS^-	47	P	15		47	P
16	CS^+	48	CS^+	16	CS^-	48	
17	P	49	P	17	P	49	P
18	CS^-	50	CS^-	18		50	
19	CS^-	51	P	19	CS^+	51	P
20	P	52	CS^-	20	P	52	CS^-
21	CS^+	53	P	21	CS^-	53	P
22	CS^-	54	CS^+	22		54	CS^+
23	P	55	CS^-	23	P	55	
24	P	56	CS^+	24	P	56	
25	CS^+	57	P	25		57	P
26	CS^-	58	CS^-	26	CS^-	58	
27	CS^+	59	CS^+	27		59	CS^+
28	P	60	P	28	P	60	P
29	CS^-	61	CS^-	29		61	
30	CS^-	62	CS^-	30	CS^-	62	CS^-
31	P	63	P	31	P	63	P
32	CS^+	64	P	32	CS^-	64	P
33	CS^-	65	CS^+	33	CS^+	65	
34	P	66	CS^-	34	P	66	CS^+
35	P	67	CS^-	35	P	67	
36	P	68	CS^-	36	P	68	CS^+
37	CS^-	69	P	37	CS^-	69	P
38	CS^+	70	CS^+	38	CS^+	70	CS^-
39	CS^-	71	CS^-	39	CS^-	71	
40	CS^-	72	P	40	CS^-	72	P
41	P	73	P	41	P	73	P
42	CS^+	74	CS^+	42	CS^+	74	CS^-

*Consecutive 20 sec. periods of possible stimulus occurrence (see following schematic - Figure 1).

**Only those CS^+ and CS^- presentations on which it was possible to measure the GSR (i.e., more than 3 sec. removed from any adjacent stimulus) are presented.

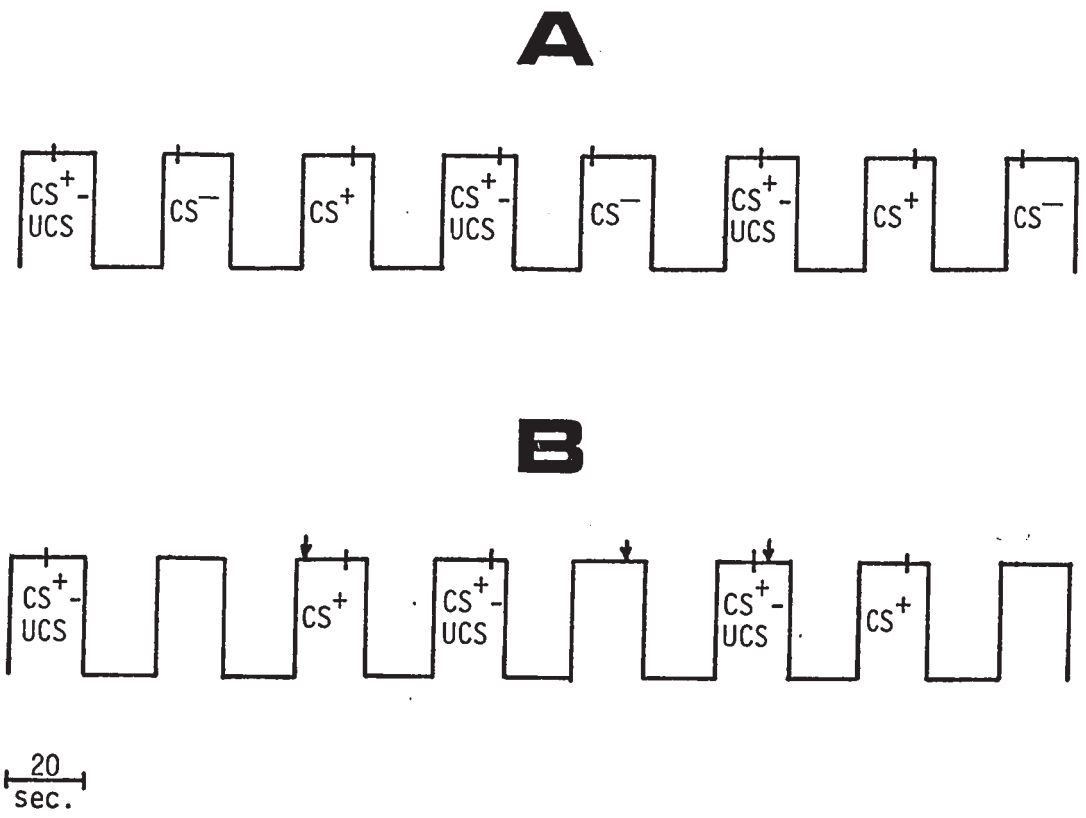

Figure 1. Schematic comparison of conditioning and control procedures used in the differential conditioning phase of training. (A. A typical eight-trial block of conditioning trials consisting of 3 CS^+-UCS, 3 CS^-, and 2 CS^+ alone presentations whose onsets are indicated by the vertical dash in each 20 sec. period of possible stimulus occurrence. B. A corresponding block of control trials in which the distribution and onsets of CS^+-UCS and CS^+ presentations remain the same, but CS^- presentations (↓) occur randomly. CS^- trials occur randomly at any point during any 3 of the 8 periods of possible stimulus occurrence, with only one CS^- occurring once in any such interval.)

APPENDIX B

Summaries of statistical analyses and

corresponding tables of means

TABLE 1

Summary of the correlation analysis performed on five habituation trials, including mean GSR magnitudes and resistance levels.

Trial	$\overline{X}$ log ΔR (Ω)	$\overline{X}$ log R (Ω)	r
1st Habituation	2.857	5.032	-0.13
3rd "	2.252	5.019	0.07
4th "	2.047	4.767	0.19
7th "	1.928	4.830	-0.21
9th "	1.827	4.621	0.11

TABLE 2

Summary of analysis of variance of GSR magnitude during the habituation phase of training as a function of Groups (PN, MR, or TN), Treatment, (con'd/sum., con'd/reacq., control/sum., control/reacq.), Stimulus Type (tone or light diminution), and Trials.

Source	*df*	MS	*F*
Between Ss			
A (Groups)	2	5.736	0.84
D (Treatment)	3	1.119	0.17
AD	6	8.115	1.19
Ss w. groups	84	6.796	
Within Ss			
B (Trials)	4	64.877	46.64***
AB	8	2.050	1.47
BD	12	0.859	0.62
ABD	24	1.074	0.77
BxSs w. groups	336	1.391	
C (Stimulus Type)	1	9.205	6.99**
AC	2	0.967	0.73
CD	3	0.380	0.29
ACD	6	1.447	1.10
CxSs w. groups	84	1.317	
BC	4	2.885	2.45*
ABC	8	0.354	0.30
BCD	12	1.735	1.47
ABCD	24	1.247	1.06
BCxSs w. groups	336	1.179	

* $p<.05$
** $p<.025$
*** $p<.001$

TABLE 3

Mean GSR magnitude during habituation as a function of Groups, Treatment, Stimulus Type, and Trials.

		TRIALS (ORDINAL PRESENTATIONS)									
		1		2		3		4		5	
		L	T	L	T	L	T	L	T	L	T
CON'D/ REACQ.	PN	2.91	2.91	1.70	1.12	0.75	1.15	1.91	0.98	1.35	1.07
	MR	2.73	3.35	2.96	2.00	2.09	1.80	2.96	2.17	2.13	1.82
	TN	3.00	3.48	2.78	2.20	1.85	1.55	2.23	1.88	1.29	0.96
CON'D/ SUM.	PN	3.37	3.31	2.75	1.70	2.35	1.86	1.56	2.09	2.34	0.86
	MR	2.56	3.45	1.92	1.96	0.81	1.72	1.95	1.71	1.56	1.74
	TN	3.50	3.32	2.15	2.31	2.68	1.94	2.26	2.37	2.52	1.38
CONTROL/ REACQ.	PN	3.35	3.29	1.66	1.72	1.26	1.69	2.25	1.34	0.98	0.38
	MR	3.26	3.35	2.67	2.35	2.27	1.79	1.87	2.38	2.87	1.81
	TN	2.81	2.79	2.49	2.15	2.31	2.17	2.07	2.29	1.91	1.01
CONTROL/ SUM.	PN	3.28	3.26	2.69	2.35	2.87	1.36	1.63	2.31	1.73	1.62
	MR	2.93	2.92	1.65	2.02	2.28	1.69	2.04	2.20	1.88	1.09
	TN	2.58	2.53	1.64	2.69	2.24	2.09	1.87	1.42	1.38	1.70

TABLE 4

Summary of analysis of variance of the magnitude of the habituation responses of retarded subjects as a function of Sex, Trials, Stimulus Type (tone or light diminution), and Etiological Class (structural or functional).

Source	*df*	MS	*F*
Between Ss			
A (Sex)	1	9.672	1.38
D (Etiological Class)	1	1.659	0.24
AD	1	9.884	1.41
Ss w. groups	28	7.029	
Within Ss			
B (Trials)	4	16.429	13.10**
AB	4	4.373	3.49*
BD	4	1.825	1.46
ABD	4	0.572	0.46
BxSs w. groups	112	1.254	
C (Stimulus Type)	1	0.844	0.39
AC	1	0.720	0.34
CD	1	0.012	0.01
ACD	1	0.975	0.46
CxSs w. groups	28	2.141	
BC	4	1.671	1.45
ABC	4	1.563	1.35
BCD	4	1.630	1.41
ABCD	4	1.580	1.37
BCxSs w. groups	112	1.154	

* $p<.01$
** $p<.001$

TABLE 5

Mean GSR magnitude of the habituation responses of retardates as a function of Sex, Trials, Stimulus Type, and Etiological Class.

		TRIALS (ORDINAL PRESENTATIONS)									
		1		2		3		4		5	
		L	T	L	T	L	T	L	T	L	T
STRUCTURAL	FEMALE	3.24	3.09	2.37	2.81	1.97	2.31	2.31	2.03	1.36	1.46
	MALE	2.38	3.15	2.59	2.30	1.75	1.80	2.57	1.75	2.95	1.64
FUNCTIONAL	FEMALE	2.98	3.49	1.90	0.79	1.84	1.19	1.15	2.18	1.38	1.07
	MALE	2.89	3.36	2.34	2.43	1.89	1.71	2.79	2.51	2.76	2.29

APPENDIX B (Con't)

TABLE 6

Summary of analysis of variance of GSR magnitude on the last two eight-trial blocks of the differential conditioning phase of training, as a function of Groups (PN, MR or TN), Treatment (conditioning or control), Trial Type (CS^- or CS^+), and Blocks of trials.

Source	*df*	MS	*F*
Between Ss			
A (Groups)	2	1.618	0.88
D (Treatment)	1	8.803	4.78*
AD	2	0.994	0.54
Ss w. groups	90	1.843	
Within Ss			
B (Blocks)	1	0.012	0.01
AB	2	1.705	1.86
BD	1	2.408	2.63
ABD	2	0.891	0.97
BxSs w. groups	90	0.917	
C (Trial Type)	1	34.453	48.06***
AC	2	0.747	1.04
CD	1	6.642	9.26**
ACD	2	1.602	2.24
CxSs w. groups	90	0.717	
BC	1	4.067	8.65**
ABC	2	0.604	1.28
BCD	1	0.791	1.68
ABCD	2	1.122	2.39
BCxSs w. groups	90	0.470	

* $p<.05$
** $p<.01$
*** $p<.001$

APPENDIX B (Cont'd)

TABLE 7

Mean GSR magnitudes on the last two eight-trial blocks of the differential conditioning phase of training as a function of Groups, Treatment, and Trial Type.

			BLOCKS OF EIGHT TRIALS			
			1		2	
			CS^+	CS^-	CS^+	CS^-
CONDITIONING	PN	PN	3.25	2.47	3.31	2.16
	MR	MR	2.97	2.68	3.12	1.84
	TN	TN	2.87	2.24	3.10	2.06
CONTROL	PN	PN	2.02	2.38	3.01	2.52
	MR	MR	2.61	1.88	2.51	1.72
	TN	TN	2.56	2.27	2.52	2.45

TABLE 8

Summary of analysis of variance of GSR magnitude on the summation test as a function of Groups (PN, MR or TN), Trial Type (CS^-/CS^+ or CS^+), Treatment (conditioning or control), and Trials.

Source	*df*	MS	*F*
Between Ss			
A (Groups)	2	24.910	3.02*
D (Treatment)	1	13.995	1.70
AD	2	0.022	0.01
Ss w. groups	42	8.252	
Within Ss			
B (Trials)	4	1.443	0.99
AB	8	0.617	0.42
BD	4	2.370	1.62
ABD	8	1.795	1.23
BxSs w. groups	168	1.461	
C (Trial Type)	1	1.858	1.49
AC	2	5.723	4.60*
CD	1	33.412	26.85**
ACD	2	6.197	4.98*
CxSs w. groups	42	1.245	
BC	4	1.080	0.75
ABC	8	1.551	1.08
BCD	4	2.654	1.85
ABCD	8	1.129	0.79
BCxSs w. groups	168	1.432	

* $p<.05$
** $p<.001$

APPENDIX B (Con't)

TABLE 9

Mean GSR magnitude during summation testing as a function of Groups, Trial Type, Treatment, and Trials.

		BLOCKS OF TWO TRIALS									
		1		2		3		4		5	
		SUM	CS^+	SUM	CS^+	SUM	CS^+	SUM	CS^+	SUM	CS^+
CON'D	PN	2.48	2.14	1.86	2.31	1.64	2.96	2.75	3.20	1.86	2.69
	MR	2.41	1.84	1.83	0.72	1.28	1.83	1.87	1.00	1.55	1.48
	TN	1.55	2.29	2.15	2.68	1.17	2.23	1.22	2.66	0.79	2.47
CONTROL	PN	2.29	1.82	2.68	1.96	2.56	2.00	2.36	1.21	2.23	1.17
	MR	1.34	1.00	2.37	0.96	1.87	1.30	1.20	0.92	1.02	0.69
	TN	1.89	1.05	1.46	1.65	2.28	1.41	1.19	1.70	2.51	0.61

APPENDIX B (Con't)

TABLE 10

Summary of analysis of variance of GSR magnitude on the reacquisition test as a function of Groups (PN, MR, or TN), Blocks of trials, and Treatment (conditioning or control).

Source	*df*	MS	*F*
Between Ss			
A (Groups)	2	1.009	0.34
C (Treatment)	1	14.547	4.95*
AC	2	0.198	0.07
Ss w. groups	42	2.940	
Within Ss			
B (Blocks)	4	0.554	0.69
AB	8	0.861	1.06
BC	4	1.000	1.24
ABC	8	0.259	0.32
BxSs w. groups	168	0.809	

* $p<.05$

APPENDIX C

Individual GSR data

TABLE 1

GSRs (ΔR in mm.) elicited by both the visual (light diminution) and auditory stimulus (tone) during habituation for the PN subjects.

	Light					Tone				
	1	2	3	4	5	1	2	3	4	5
	2.5	0.0	0.0	0.0	0.0	3.5	0.0	0.0	0.0	0.0
	3.0	1.0	0.0	0.0	1.0	5.0	0.0	0.0	0.0	0.0
	7.0	2.0	1.0	2.5	1.5	4.5	1.5	1.0	0.5	0.5
	6.5	1.0	0.0	1.0	0.0	9.0	2.0	4.0	0.0	0.0
	5.0	2.0	6.0	12.0	3.0	5.5	4.5	6.5	1.0	10.5
	8.0	0.0	0.0	0.0	0.0	8.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	1.5	0.5	0.0	0.0	0.0	0.0	0.0
Con'd.	8.5	1.0	0.0	4.5	0.0	53.5	0.0	0.0	2.0	1.0
	4.0	9.5	4.0	0.0	12.5	16.0	11.0	0.5	1.5	0.0
	4.5	4.0	4.5	6.5	16.5	9.5	6.5	0.5	6.0	5.0
	19.5	0.0	1.0	1.0	0.5	4.5	0.0	0.0	0.0	0.0
	7.0	6.0	8.5	0.0	4.0	8.0	6.0	0.0	1.5	0.0
	18.5	3.5	2.0	0.0	0.0	3.5	0.0	20.0	1.0	0.0
	5.0	2.0	0.0	0.0	0.0	2.5	0.0	0.0	1.0	0.0
	1.5	4.0	0.0	1.5	1.5	2.0	0.0	2.5	1.0	0.0
	4.0	1.0	5.0	13.0	2.0	4.5	3.5	5.5	0.0	9.5
	4.0	0.5	0.5	2.5	0.0	5.5	1.5	0.0	2.0	0.0
	12.0	4.0	0.5	2.0	2.0	5.5	2.0	4.0	0.0	0.0
	5.0	0.0	0.5	12.0	0.0	6.0	2.0	0.0	0.0	0.0
	5.0	0.5	3.5	1.0	1.0	6.0	1.0	3.0	0.5	0.0
	5.5	0.0	0.0	0.0	0.0	2.0	1.0	0.5	0.0	0.0
	5.0	3.5	0.0	2.0	0.5	5.0	0.0	1.0	1.0	0.0
	8.0	0.0	0.0	0.0	0.0	8.0	0.0	0.0	0.0	0.0
	3.5	0.5	0.0	2.0	0.0	3.5	0.0	0.5	2.0	2.5
Control	9.0	1.5	0.5	0.0	0.0	6.5	0.5	0.0	0.0	10.0
	3.0	1.5	1.0	0.0	0.5	1.0	0.5	1.0	0.0	0.0
	6.5	1.0	2.5	2.0	0.5	5.5	1.0	1.5	3.5	0.0
	1.0	0.0	0.5	0.0	1.0	3.0	2.0	0.0	1.5	0.0
	3.5	3.0	2.5	3.5	0.0	4.0	2.0	0.0	2.5	0.0
	4.5	8.0	5.0	0.0	7.0	4.0	2.0	1.0	2.5	3.0
	17.0	9.0	9.5	14.0	0.0	14.0	0.0	0.0	6.0	2.0
	5.5	4.5	2.0	4.0	4.0	7.0	2.0	2.0	3.5	6.0

TABLE 2

GSRs (ΔR in mm.) elicited by both the visual (light diminution) and auditory stimulus (tone) during habituation for the MR subjects.

	Light					Tone				
	1	2	3	4	5	1	2	3	4	5
Con'd.	4.0	3.0	0.0	4.0	0.0	14.0	2.5	0.0	0.5	0.0
	0.0	2.5	3.5	7.5	0.0	11.0	0.0	0.0	7.0	2.0
	4.0	1.0	0.0	0.5	0.5	0.5	1.5	0.5	1.0	1.0
	14.0	10.0	12.0	10.0	3.5	9.0	12.0	6.0	9.5	4.0
	2.0	1.0	0.5	1.0	1.5	8.0	5.5	2.5	3.5	3.0
	2.0	6.0	0.5	1.0	1.0	6.5	4.0	3.5	0.5	0.0
	1.0	3.0	2.5	2.5	4.0	2.0	0.0	1.0	0.0	0.0
	5.0	0.5	0.5	2.0	2.5	14.0	0.0	0.0	0.0	1.5
	2.0	0.0	0.0	0.5	0.0	7.5	3.5	5.0	0.0	1.5
	27.0	1.0	0.0	0.0	0.0	22.0	0.0	0.0	0.0	0.0
	0.0	3.0	0.0	2.0	2.0	2.5	0.0	0.0	0.0	1.0
	0.0	0.0	0.0	0.0	2.5	2.5	1.0	0.0	8.0	0.0
	13.0	9.0	13.0	9.0	0.0	8.0	14.0	6.5	19.0	4.5
	9.0	0.0	0.0	0.0	0.0	8.0	0.0	5.0	2.0	1.5
	5.0	1.0	1.5	3.5	2.0	9.5	1.0	0.0	0.0	0.0
	2.5	8.0	0.0	12.0	12.0	10.0	9.0	13.0	6.5	8.5
Control	13.0	6.0	1.5	0.0	0.5	14.0	5.0	1.0	0.5	0.0
	5.5	4.0	4.0	0.0	2.0	5.5	0.0	0.0	3.0	0.0
	5.5	2.0	0.5	2.0	1.0	2.0	2.0	1.0	0.5	3.0
	4.5	4.0	4.5	6.5	10.0	9.5	6.5	4.5	6.0	5.0
	4.5	1.5	3.5	2.0	2.5	4.0	2.0	1.5	0.5	1.0
	6.0	2.5	7.5	3.5	1.0	8.0	3.5	3.0	6.0	2.0
	2.0	2.0	0.0	0.0	4.0	10.0	3.5	0.0	0.5	0.0
	2.0	0.0	0.0	1.0	1.5	2.5	0.0	0.0	0.0	1.0
	19.0	9.0	17.0	19.0	1.5	8.0	2.5	5.5	0.5	0.0
	5.0	0.0	0.0	0.0	0.0	8.5	0.0	0.0	0.0	0.0
	13.0	9.0	11.0	9.0	3.5	9.0	11.0	6.0	8.0	4.0
	10.0	5.5	3.0	2.0	0.0	6.5	4.0	5.0	4.0	0.0
	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	5.5	0.0	1.0	0.0	2.0	2.5	0.0	0.0	0.5	0.0
	0.0	0.0	0.5	1.5	5.0	6.0	5.5	8.5	5.0	1.0
	5.0	1.5	1.5	4.0	2.0	2.5	2.5	0.0	2.5	2.0

TABLE 3

GSRs (ΔR in mm.) elicited by both the visual (light diminution) and auditory stimulus (tone) during habituation for the TN subjects.

	Light					Tone				
	1	2	3	4	5	1	2	3	4	5
Con'd.	12.0	11.0	0.0	6.0	0.0	12.0	0.0	0.0	0.0	0.0
	19.5	6.5	8.5	4.5	0.0	15.5	4.0	0.5	3.0	0.0
	7.5	8.0	5.0	4.5	3.0	7.5	5.0	2.0	6.0	3.0
	6.0	1.0	0.0	1.5	0.5	3.5	0.5	0.0	0.0	0.0
	6.5	2.0	0.5	0.0	0.0	6.5	2.0	0.5	3.0	0.5
	17.5	6.0	2.0	1.0	0.0	15.0	3.5	0.5	2.0	0.5
	0.5	1.5	1.5	1.0	1.0	16.0	0.0	0.0	0.0	0.0
	32.0	0.0	13.0	0.0	0.0	10.0	1.0	4.0	0.0	0.0
	6.5	0.0	1.0	1.0	0.5	4.5	0.0	0.0	0.0	0.0
	3.0	0.0	2.0	0.0	4.0	7.0	0.0	2.0	4.5	2.0
	2.5	4.0	0.0	1.5	1.5	2.0	2.0	1.0	1.0	0.5
	17.0	24.0	10.0	13.0	8.0	16.0	7.0	10.0	9.0	0.0
	8.0	4.0	3.0	5.0	4.0	4.5	3.5	2.5	2.0	4.0
	6.0	3.0	2.0	0.5	1.0	8.0	2.5	0.0	2.0	1.0
	11.0	13.0	1.0	6.0	1.0	1.5	6.0	0.0	14.0	0.0
Control	3.0	1.0	0.0	0.5	0.0	7.0	3.0	2.5	1.5	0.0
	1.5	1.5	1.0	0.0	0.5	1.5	0.0	0.5	0.5	0.0
	10.5	4.0	2.5	2.0	2.0	5.0	1.5	4.0	1.0	1.0
	8.0	3.0	2.0	2.5	1.0	4.5	3.5	2.5	3.5	3.5
	0.0	0.0	0.5	0.0	0.5	0.0	0.0	0.0	0.0	0.0
	5.0	3.5	3.0	1.0	1.0	6.0	1.5	3.0	1.0	0.5
	4.0	1.5	0.5	2.5	0.0	4.5	1.5	0.0	1.0	0.0
	2.5	0.5	0.5	1.5	1.0	2.0	1.0	1.5	0.5	0.0
	5.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	11.5	6.5	8.5	4.5	2.0	15.0	8.0	3.5	3.0	2.0
	5.0	0.0	1.5	2.0	0.0	0.0	0.5	1.5	1.5	1.0
	0.0	0.0	1.5	0.0	0.0	6.0	4.5	0.0	0.0	0.0
	8.0	4.0	3.0	5.0	4.0	4.5	3.5	2.5	2.0	4.0
	20.0	4.5	7.0	0.0	0.0	7.0	6.0	1.0	0.0	0.0
	2.5	4.0	0.5	2.0	1.0	2.5	2.0	1.0	1.0	0.5
	0.0	0.0	0.0	1.0	0.5	5.5	2.5	1.0	0.0	1.0

TABLE 4

Mean GSRs (ΔR in mm.) elicited by the CS^+ and CS^- on each of the last two eight-trial blocks of the differential conditioning phase of training for the PN subjects.

Treatment	S	CS^+		CS^-		# of blocks to criterion
		#1	#2	#1	#2	
Conditioning/ Reacquisition	1	7.0	11.0	1.5	0.0	3
	2	8.5	7.8	5.7	2.7	3
	3	1.3	2.0	0.8	0.3	4
	4	4.8	4.8	0.0	0.3	2
	5	16.3	14.8	12.0	3.2	4
	6	2.0	1.5	0.0	0.0	4
	7	2.8	4.3	1.0	1.5	3
	8	3.0	6.5	3.5	0.3	3
Conditioning/ Summation	21	7.3	16.5	3.3	6.8	4
	22	7.3	6.5	3.5	1.2	3
	23	1.5	1.0	0.8	0.2	4
	24	11.8	12.5	1.5	2.3	3
	25	19.3	6.5	8.0	1.7	3
	26	0.3	0.8	0.0	0.0	3
	27	2.8	3.5	2.2	1.0	4
	28	12.3	16.5	7.8	2.8	4
Control/ Reacquisition	11	1.0	3.0	0.8	1.8	
	12	3.0	1.5	2.0	1.0	
	13	0.0	5.5	1.0	3.7	
	14	8.0	13.0	11.5	9.3	
	15	1.0	1.0	0.5	0.7	
	16	1.0	18.5	5.0	8.7	
	17	14.0	3.0	5.0	10.0	
	18	0.0	8.0	0.0	0.3	
Control/ Summation	31	0.0	3.5	1.0	4.2	
	32	1.0	1.0	0.8	0.5	
	33	1.5	5.3	2.0	6.8	
	34	5.0	4.0	2.0	3.0	
	35	4.0	4.5	2.8	0.0	
	36	0.0	0.0	0.0	0.3	
	37	0.0	4.5	0.0	0.5	
	38	0.5	7.5	8.5	0.0	

APPENDIX C (Con't)

TABLE 5

Mean GSRs (ΔR in mm.) elicited by the CS^+ and CS^- on each of the last two eight-trial blocks of the differential conditioning phase of training for the MR subjects.

Treatment	S	CS^+		CS^-		# of blocks to criterion
		#1	#2	#1	#2	
Conditioning/ Reacquisition	1	0.3	0.8	0.3	0.0	7
	2	2.0	2.3	0.5	0.0	6
	3	1.2	2.3	0.7	0.3	7
	4	4.3	6.5	0.8	0.0	4
	5	2.0	2.3	2.0	0.8	5
	6	9.5	9.3	5.3	4.0	6
	7	1.3	2.0	0.5	0.5	6
	8	2.0	1.5	1.5	0.0	6
Conditioning/ Summation	21	6.5	4.5	1.7	0.0	4
	22	3.0	2.8	1.7	0.2	5
	23	0.5	1.8	1.2	0.3	6
	24	6.8	5.0	1.5	0.8	7
	25	1.8	2.8	0.3	0.5	6
	26	3.8	8.0	2.5	1.0	4
	27	1.8	5.5	1.8	1.0	7
	28	6.5	9.0	3.7	4.5	5
Control/ Reacquisition	11	4.5	4.3	3.0	2.5	
	12	1.5	4.5	3.0	2.5	
	13	0.3	1.8	0.0	0.0	
	14	3.0	1.8	1.3	1.2	
	15	1.5	1.0	0.5	1.3	
	16	0.8	0.5	0.5	0.0	
	17	5.0	0.0	2.5	0.0	
	18	1.5	8.0	0.8	0.0	
Control/ Summation	31	0.5	9.3	0.0	0.2	
	32	1.5	1.5	0.0	1.0	
	33	1.3	1.5	0.8	0.5	
	34	0.5	1.3	0.0	0.5	
	35	7.3	0.0	4.0	1.0	
	36	2.0	0.3	0.8	0.0	
	37	0.0	1.8	0.0	0.2	
	38	1.5	1.5	1.9	2.3	

APPENDIX C (Con't)

TABLE 6

Mean GSRs (ΔR in mm.) elicited by the CS^+ and CS^- on each of the last two eight-trial blocks of the differential conditioning phase of training for the TN subjects.

Treatment	S	CS^+		CS^-		# of blocks
		#1	#2	#1	#2	
Conditioning/ Reacquisition	1	11.5	13.0	4.2	4.0	7
	2	0.5	1.3	0.0	0.0	6
	3	5.3	4.0	1.3	1.0	7
	4	2.5	2.5	1.2	0.3	4
	5	1.0	1.5	0.0	0.0	5
	6	3.3	1.3	1.0	0.2	6
	7	4.5	6.8	1.8	1.3	6
	8	5.0	2.3	2.5	2.3	6
Conditioning/ Summation	21	0.0	2.0	0.8	0.3	4
	22	1.5	1.8	0.5	0.2	5
	23	13.0	7.5	6.2	2.2	6
	24	2.3	2.0	0.5	0.5	7
	25	5.8	3.5	0.5	1.7	4
	26	0.3	3.5	0.5	1.7	4
	27	3.5	4.5	3.3	0.0	7
	28	5.5	6.8	1.7	2.2	5
Control/ Reacquisition	11	2.0	1.0	2.0	0.7	
	12	0.0	0.0	0.0	6.0	
	13	3.5	4.0	2.0	4.0	
	14	6.5	7.8	5.8	5.8	
	15	1.3	0.8	1.5	0.8	
	16	1.8	2.5	1.8	2.0	
	17	1.5	2.5	0.8	1.0	
	18	1.0	1.5	1.3	0.0	
Control/ Summation	31	1.5	0.5	0.0	0.0	
	32	2.3	1.5	1.7	2.0	
	33	1.5	0.0	0.8	1.0	
	34	0.0	1.3	0.0	0.5	
	35	3.8	3.0	3.5	1.0	
	36	2.0	3.5	0.8	3.7	
	37	2.0	1.0	1.0	0.5	
	38	2.3	2.8	1.3	1.8	

TABLE 7

GSRs (ΔR in mm.) elicited by the compound CS^+/CS^- stimulus and CS^+ during the summation test for the PN, MR, and TN subjects who had previously received conditioning training.

	Sum. trials					CS^+ trials				
	1	2	3	4	5	1	2	3	4	5
PN	8.0	0.0	18.0	11.0	8.0	6.0	7.5	3.0	31.0	17.0
	1.5	1.5	8.0	3.0	0.0	3.5	0.5	2.0	2.0	7.0
	0.0	0.0	0.0	0.0	0.0	0.0	1.0	3.0	1.0	0.5
	3.5	0.0	0.0	7.5	0.5	0.0	0.0	13.5	4.0	1.0
	7.5	3.0	2.5	5.0	2.0	8.0	6.0	4.0	4.0	2.0
	0.0	0.5	0.0	0.5	0.0	0.0	0.0	0.0	2.0	0.0
	4.0	4.0	1.5	2.5	1.5	3.0	3.5	4.0	2.0	1.0
	15.0	9.0	0.0	4.5	6.0	27.0	9.0	8.5	15.0	19.0
MR	0.0	1.5	0.0	1.0	0.0	0.0	0.0	0.0	0.0	5.0
	1.0	0.0	0.0	0.0	6.0	5.0	0.0	1.0	0.0	0.0
	1.0	1.0	1.0	2.0	0.0	2.0	0.0	1.0	0.0	1.0
	1.0	1.0	1.0	3.5	0.0	1.0	0.0	5.0	0.0	0.0
	3.5	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0
	1.0	3.5	0.0	2.5	0.0	0.0	1.0	0.0	0.5	1.0
	1.5	0.0	1.5	0.0	3.0	1.0	0.0	1.5	2.0	0.0
	2.5	9.0	0.5	5.5	5.5	5.0	3.5	5.5	1.5	5.5
TN	3.0	0.5	0.0	0.0	0.0	2.5	0.0	0.0	1.0	1.5
	0.0	0.0	0.0	0.0	0.0	0.5	1.0	3.5	1.5	1.0
	3.0	9.5	8.5	6.5	7.0	7.5	9.5	8.0	13.0	12.0
	0.0	1.5	0.0	0.0	0.0	2.0	2.0	1.5	0.5	1.0
	0.0	1.0	0.0	4.0	0.0	3.0	1.0	0.0	1.0	1.0
	2.0	0.0	0.0	0.0	0.0	0.0	4.0	0.5	0.0	1.0
	0.0	1.0	1.0	0.0	2.0	0.0	6.0	2.5	11.5	0.0
	6.0	6.0	4.5	3.5	0.0	9.0	4.0	3.5	11.0	2.0

TABLE 8

GSRs (ΔR in mm.) elicited by the compound CS^+/CS^- stimulus and CS^+ during the summation test for the PN, MR, and TN subjects who had previously received the control procedure.

	Sum. trials					CS^+ trials				
	1	2	3	4	5	1	2	3	4	5
PN	1.5	9.5	10.0	1.0	0.0	0.0	0.0	5.5	2.0	0.0
	1.0	1.5	3.0	2.5	0.5	1.0	0.5	0.0	0.0	0.0
	3.0	4.0	14.0	8.5	12.0	4.5	5.5	3.5	8.5	13.0
	3.0	3.0	4.0	9.5	4.5	2.5	3.5	2.5	0.0	2.0
	5.0	2.5	7.5	5.0	6.5	3.0	0.0	2.0	4.5	1.5
	8.0	1.0	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0
	0.0	0.0	0.0	2.0	1.0	0.0	9.0	0.0	0.0	0.0
	0.0	4.0	6.0	0.0	0.0	0.0	6.0	11.0	0.0	0.0
MR	0.5	3.0	0.0	6.0	0.0	0.0	0.0	0.5	0.0	0.0
	0.0	0.5	9.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	2.0	1.0	2.0	0.0	1.5	1.5	1.5	1.0	0.5	1.0
	0.0	0.5	0.5	6.5	0.0	0.0	0.0	0.0	0.0	0.0
	0.0	0.0	4.0	0.0	0.0	1.0	0.0	0.0	0.0	4.0
	0.0	6.0	0.0	0.0	1.5	0.0	0.0	0.0	0.0	0.0
	4.5	2.5	2.5	1.5	1.0	1.0	1.0	2.0	1.5	0.5
TN	0.0	0.0	1.5	0.0	4.5	0.0	0.0	0.0	0.0	0.0
	6.0	3.5	8.5	5.0	6.0	4.0	1.0	3.0	4.0	0.5
	0.0	0.0	2.0	0.0	0.0	0.0	2.0	0.0	0.0	0.0
	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	7.0	1.0	0.0	0.0	1.0	1.0	0.0	0.5	1.0	0.0
	1.0	0.0	2.0	0.0	3.5	0.0	1.0	5.0	1.0	0.0
	1.5	2.0	3.5	3.0	1.0	1.0	0.5	0.0	0.5	0.0
	2.0	2.5	2.5	3.5	1.5	0.0	1.5	1.0	2.0	1.0

TABLE 9

Mean GSRs (ΔR in mm.) elicited by the $\overline{CS}$ on each of the five two-trial blocks of the reacquisition test for the PN, MR, and TN subjects.

	Conditioning					Control				
	1	2	3	4	5	1	2	3	4	5
PN	0.0	0.0	0.0	0.5	0.3	2.0	1.3	0.5	2.5	0.3
	0.8	3.0	1.0	2.8	2.8	3.0	1.3	2.3	2.8	3.0
	0.3	0.3	1.0	1.3	0.8	1.5	0.0	0.5	3.5	1.5
	0.0	0.0	0.5	0.0	0.0	3.8	1.5	0.8	1.5	1.5
	0.8	5.5	5.8	6.8	6.8	0.5	0.3	0.5	1.0	0.8
	0.0	0.0	0.5	0.3	1.0	7.3	5.5	2.8	7.0	10.3
	0.3	2.5	0.8	0.5	2.5	4.8	1.5	0.3	0.0	0.8
	0.8	0.0	2.3	0.0	0.8	0.0	0.0	0.0	0.8	0.0
MR	0.0	0.3	0.0	0.3	0.0	1.8	0.8	0.5	0.3	1.5
	3.0	0.0	1.8	2.8	3.3	1.0	0.5	0.8	2.8	0.5
	1.3	1.5	1.8	1.0	1.5	1.8	4.5	0.5	0.5	0.3
	0.0	0.3	0.8	0.0	0.0	3.5	1.8	2.8	3.3	3.5
	1.5	2.3	2.8	1.8	2.0	0.3	1.3	0.3	1.5	0.8
	1.0	2.3	1.8	1.8	2.0	2.8	0.8	1.5	1.0	0.8
	0.0	1.5	0.3	1.3	1.8	1.8	1.5	0.0	1.0	4.0
	1.8	0.8	0.5	1.0	0.5	0.0	2.5	8.5	2.8	4.3
TN	5.0	11.5	0.0	3.3	9.0	1.5	1.0	0.8	0.8	1.0
	0.0	0.3	0.3	1.0	0.0	2.5	0.3	1.3	1.0	2.3
	1.0	0.8	3.5	1.5	1.5	6.3	4.5	2.5	6.0	8.3
	0.3	0.0	0.8	1.3	0.8	3.8	2.8	6.8	2.3	4.5
	1.3	0.0	0.0	0.0	1.5	0.3	0.3	0.0	0.0	0.0
	0.0	0.3	0.3	0.8	1.3	4.8	2.5	1.5	2.0	2.5
	1.3	4.8	5.5	3.5	0.0	2.8	1.8	2.5	2.8	2.0
	1.0	0.8	0.0	0.0	0.5	1.0	0.5	0.0	1.8	0.8