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The Disruption of State-Dependency By An Emotionally Important Conditioned Stimulus Paired With Food Reward And Food Seeking Responses

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THE DISRUPTION OF STATE-DEPENDENCY BY AN EMOTIONALLY IMPORTANT CONDITIONED STIMULUS PAIRED WITH FOOD REWARD AND FOOD SEEKING RESPONSES

A Thesis Submitted to the Graduate Division in Partial Fulfillment of the Requirements for the Degree of Master of Science

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Abstract

The purpose was to determine if a 1 Khz tone could disrupt state-dependency in a T-maze when the tone was paired with feeding and food seeking responses on training trials and later presented on testing trials while subjects were in the drug (D) state opposite of the training D state. The 64, 80 day old, male hooded rats were trained to an 18/20 criterion while in either: the D state, produced by 45 mg/kg intraperitoneal injections of chlordiazepoxide hydrochloride (Librium); or, the nondrug (ND) state, produced by sterile water. Beginning with the opposite of the training D state, D and ND states were alternated daily across 5 consecutive days of testing in the four experimental groups. The testing and training D states were always the same for the four control groups. Two no-tone experimental groups (one D trained; one ND trained) demonstrated state-dependent effects. The ability of the tone to disrupt state-dependency in the two transfer (tone) experimental groups (one D trained; one ND trained) could not be determined because of the possible influence of confounding variables. Results are discussed with regard to other disruption experiments.

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CHAPTER I

INTRODUCTION

Conditions upon which the performance of learned responses is contingent may vary with alterations between nondrug and drug states or with alterations among various drug states. As a result of change from nondrug state to drug state, from drug state to nondrug state, or from one drug state to another, conditioned responses easily elicited from an animal in the state in which training occurred may no longer be elicitable. After the animal is again in the state in which it was trained, responses may again be elicitable. This phenomenon is known as "state-dependent" or "dissociated" learning.

History of State-Dependent Learning

Lashley (1917) may have been the first investigator to suggest that learning which occurred while under the influence of a drug might not transfer to a nondrug state. Lashley administered strychnine or caffeine to rats prior to maze training and found that those rats required more time to relearn the maze during subsequent training without the drug than did the undrugged control group.

Attempting to determine the importance of peripheral, skeletal motor responding in learning, Harlow and Stagner (1933) used curare to produce complete flaccid paralysis in dogs and cats. Harlow and Stagner attempted to condition skeletal motor responding after their animals were paralyzed. They conditioned a smooth muscle response, pupillary dilation to pain, as a control activity because they thought it would not be influenced by the drug since the dosage of curare used would not paralyze smooth muscles. They reported that conditioned responses requiring move-

ment of striate muscles could not be established in their paralyzed animal subjects. They attempted to extinguish responses trained in the nondrug state by placing animals in the drug state and giving them the conditioned stimulus (CS) without the unconditioned stimulus (UCS). They found that when animals were returned to the nondrug state the conditioned skeletal motor responses were still elicitable. Harlow and Stagner reported that the pupillary response conditioned in the nondrug state was extinguished while their animals were in the drug state; however, it appears that no attempt was made to elicit the pupillary response after animals had returned to a nondrug state.

Harlow and Stagner observed a marked fall in blood pressure after the administration of curare. They thought that the fall in blood pressure might have affected the central nervous system of some of their animals and that it might have resulted in the failure of some animals to develop the pupillary conditioned response. Harlow and Stagner may have thought that the pupillary response would be among the first of any conditioned responses to be affected by changes in the central nervous system resulting from drug administration. Such thinking might account for their not mentioning the possibility that the fall in blood pressure might also affect the central nervous system in such a way as to interfere with the conditioning of skeletal motor responses.

Light and Gantt (1936) paralyzed the right hind legs of dogs by crushing nerve roots. Before regeneration of damaged motor nerves had taken place they applied shock to the foot of the paralyzed limb. The foot shock was accompanied by an auditory stimulus throughout a training period during which the damaged limbs remained paralyzed. After regeneration of the damaged nerves was complete Light and Gantt tested their

animals and found that leg withdrawal had been conditioned to the auditory stimulus although the animals were unable to make the response during training. Their subjects were not drugged at any time during the experiment. Their findings, regarding a response theory of learning, were in direct contradiction to those of Harlow and Stagner (1933) who, using curare to paralyze their animals, determined that movement was essential to the learning of conditioned skeletal motor responses.

Light and Gantt (1936) suggested that "cerebral anemia" (P. 22) might have affected the conditioning of skeletal motor responding in the curarized animals of the Harlow and Stagner (1933) experiment. "Cerebral anemia" referred to a condition caused by a fall in blood pressure following the administration of curare (Light and Gantt, 1936).

Girden and Culler (1937) attempted to account for the findings of Harlow and Stagner (1933) and Light and Gantt (1936). Girden and Culler (1937) reported that conditioned responses, established in animal subjects under the influence of curare, were not elicitable after the subjects had returned to a nondrug state, but were elicitable when animals were again in the curare drug state. Conversely, they reported that conditioned responses established in a nondrug state were not elicitable when the animals were placed in a drug state, but were again elicitable when animals returned to a nondrug state. Girden (1942) called this phenomenon "dissociation" (p. 219). Girden and Culler (1937) stated that previous failures to elicit conditioned responses trained under the influence of curare were a result of testing for conditioning after a return to the nondrug state.

Harlow (1940) experimented with cats to determine the effects of incomplete curare paralysis upon the conditioning and elicitation of

conditioned responses. He reported eliciting escape and pupillary conditioned responses while animals were under partial curare paralysis and eliciting those same responses with greater frequency after the animals had recovered from the drug state. Harlow also reported that some subjects "had not responded positively to the CS in the drugged state but did respond positively to the CS in the normal state" (p. 275). Harlow believed that conditioned escape responses, conditioned pupillary responses, and conditioned semitendinosus muscle responses were all components of a "conditioned emergency flight response" (p. 278). Because pupillary responses had been conditioned under curare paralysis "during a stage of cerebral depression that makes impossible the formation of conditioned escape responses involving gross responses of the skeletal musculature" (p. 279), Harlow believed that curare influenced various components of a total conditioned response pattern to varying degrees.

Girden (1942b) conditioned a pupillary response in dogs and cats while the animals were under the influence of curare or erythroidine and then tested for transfer of the response to the nondrug state. To prevent the smypathetic component of the autonomic nervous system from maintaining active dilation, sympathetic fibers innervating the subjects' pupils were severed. Girden found no transfer to the nondrug state. On the basis of these findings he rejected the contention of Harlow (1940) that curare could influence to varying degrees different components of a total conditioned response pattern. Girden (1942b) attributed the transfer of the conditioned pupillary response across drug states, reported by Harlow and Stagner (1933) and Harlow (1940), to observations of unconditioned rather than conditioned responses. Girden

(1942b) stated that "A pupillary conditioned response (CR) established under the drug has never been detected in the normal state" (P. 331).

In other experiments, Girden further elaborated the phenomenon of state-dependent learning. Girden (1940) conditioned dogs to an auditory CS in both drug and nondrug states after bilateral extirpation of the auditory cortex. The results showed that conditioned responses were transferred from the nondrug to the drug state, and from the drug to the nondrug state. Girden conjectured that conditioned responding in the normal undrugged animal is mediated by cortical pathways whereas conditioned responding in the drugged animal is mediated by subcortical systems. According to Girden, the subcortical systems of the undrugged animal are inhibited by an active cortex. In the drugged animal the cortex is suppressed by the drug and the subcortical systems are disinhibited. Consequently, learning was thought to be mediated by the systems active at the time of training, thus switching the active systems by changing drug states determines whether or not conditioned responses are elicited in the intact animal. Girden reasoned that the decorticate animal has only active subcortical systems whether drugged or undrugged and therefore will give conditioned responses in either drug or nondrug states regardless of which state the animal was trained in.

Girden (1942a) reported that autonomic responses conditioned in the drug state were absent after a return to the nondrug state. He stated that the suppression of a conditioned blood pressure response after a return to the nondrug state was a safe inference since other components of the conditioned response were suppressed after a return to the nondrug state.

Kellogg, Scott, Davis and Wolf (1940) reported that the conditioned leg flexion response elicited by Light and Gantt (1936) did not demonstrate conclusively that learning could occur without movement. Kellogg et al. (1940) recorded the activity of all limbs during a repetition of the Light and Gantt (1936) experiment. They found that the conditioned leg flexion response was accompanied by other movements and was part of a generalized pattern of which leg flexion was only one component.

Girden (1943) pointed out that cessation of all movement, of which a specific response could be a component, was not possible without death of the subject. He attempted to test the response theory relative to a state in which the restriction of all efferent responding was as near to complete as practicality would allow. Girden found that autonomic responses conditioned in a deep drug state, produced by curare or erythroidine, could be elicited in the mild drug state. His findings indicated that the central mechanism functioned in mediating the acquisition of conditioned responses during a period of total muscular paralysis caused by the drug and that the drug state from deep to mild intensities was continuous.

Girden (1947) demonstrated state-dependent learning in rhesus monkeys using erythroidine to produce the drug state. The results of that experiment confirmed data obtained in other experiments and demonstrated the phenomenon in an animal form previously untested.

Research on state-dependent learning waned through the late 1940's and most of the 1950's. During the 1960's there was a revival of interest in the area.

Heistad and Torres (1959) trained rats to press a lever for an aperiodic water reward. After a stable rate of responding was established,

they trained the same rats to give a conditioned emotional response (CER) consisting of crouching and cessation of lever pressing, following a clicker noise which signaled foot shock.

Heistad and Torres reported that the CER was lost when rats trained under saline were administered 5 mg/kg thioridazine hydrochloride. A CER learned under thioridazine was lost when the tranquilizer was withdrawn and saline substituted. These investigators interpreted their results to be evidence supporting a hypothesis that important aspects of internal stimulus conditions which affected the CER were altered by the presence and absence of the drug.

Stewart (1962) trained three groups of rats to perform differential escape responses dependent on differences between states produced by 4 mg/kg and 3 mg/kg chlorpromazine and 20 mg/kg imipramine vs. saline. After escape responses were established drugs other than those used in training were substituted for training drugs and tests for transfer were carried out. Stewart reported that chlorpromazine responses were given by animals in the states produced by acepromazine, perphenazine, and prothipendyl. She reported that no transfer to prochlorperazine or to imipramine was found in chlorpromazine trained animals. Responses learned while under the influence of imipramine did not transfer under chlorpromazine or acepromazine test conditions.

Overton (1964) trained rats to escape shock in a T-maze under drug and nondrug conditions. Some rats were trained to turn in one direction while in either a drug state produced by pentobarbital or a nondrug state produced by saline, and received no training in the opposite state. Other rats were trained to respond differentially by turning in one direction while in the drug state and in the opposite direction while

in the nondrug state. Overton tested rats trained in only one state, either drug or nondrug, in the state opposite of the training state and found complete dissociation with a heavy dosage of pentobarbital (25 mg/kg).

Overton found that rats trained to discriminate the nondrug state from the drug states produced by 0, 10, 15, 20, and 25 mg/kg pentobarbital showed transfer in amounts inversely related to the dosage used during training. He reported that complete state-dependence was an extreme form of the phenomenon. Overton compared the effectiveness of various extroceptive and interoceptive stimuli and drive states with that of a drug state in gaining differential response control. He found that a pentobarbital induced drug state gained response control significantly faster than any of the other conditions tested. He concluded that performance decrements resulting from drug state changes were apparently not a result of changes in sensory cues.

Overton (1966) reported on tests of the dissociative abilities of several depressant and atropine-like drugs. Dissociation was shown in the conditioned differential responding of rats in the T-maze. Partial dissociation was found between the nondrug state and states produced by some depressant drugs. Five drugs tested, alcohol, urethane, meprobamate, phenobarbital, and pentobarbital were found to be approximately equivalent in dissociative actions and were interchangeable. At selected dosages each of these five drugs when substituted for the others would elicit predominately drug responses from animals trained to discriminate drug and nondrug states. Learning which occurred under the influence of atropine was partially dissociated from learning which occurred under depressant drugs and learning which occurred in the nondrug state. Depressant drug effects which allowed dissociation from atropine learning

were not antagonized or mimicked by atropine effects. Animals trained under a depressant did not show a decrease in drug responses when the depressant and atropine were administered to the same animals during one test session. According to Overton, the results indicated that a single unidimensional process could not account for all dissociation resulting from drug state change.

Overton proposed two general theoretical models of the mechanisms underlying dissociation. In the first model drug states control responding by the same learning process which allow stimuli to control responding. When a drug state provides a control stimulus and the drug state is then changed the control stimulus is no longer present and responses conditioned to that control stimulus no longer occur. Reoccurrence of the conditioned response then becomes contingent upon a return to the training drug state or a drug state which will allow generalization to different but similar stimulus conditions. The degree of conditioned responding may then be dependent upon the degree of stimulus generalization. Overton suggests that drugs may act on a brain system which is not directly involved in learning but which projects to those systems that are. Drug action on such a system could alter the afferent neural impulses received by systems directly involved in learning thus resulting in variations of neural input similar to variations resulting from change in external stimulus conditions. Overton states that direct structural connections between drug affected systems and other learning systems may allow drug state changes to have a more powerful influence on responding than can be exercised by manipulation of external stimuli.

Overton's second model proposes that diffuse drug actions alter cell characteristics in brain regions that undergo structural change during

learning. He suggests that conditioned responding may be contingent upon a pattern of neural activity formed during training which consists of subtle changes in the transfer function of cells. The transfer functions of many neurons in such a system could be different in one drug state than in another. A change in drug state might alter or disrupt to varying degrees the interaction of cells in such a system resulting in partial or total loss of the conditioned response. When returned to the training drug state the cells would again have the same transfer function required to produce the pattern of neural activity established during training. A different but similar drug state might result in cellular activity capable of allowing a degree of conditioned responding depending upon how near the transfer function of cells is to that during training.

Overton (1967) conditioned rats to run to separate goal boxes in a three alley maze to escape shock. These rats discriminated correct choices according to three drug states produced by atropine, phenobarbital, and saline. Overton reported that unless low dosages of the drugs were used responding was disorganized and that a great deal of training under low dosage was required to reach criterion on the three choice task.

Overton (1968) trained rats to avoid foot shock in a T-maze by correctly responding to states produced by either no drug or pentobarbital. Rats in the first experiment were trained to discriminate between states produced by either 15 mg/kg pentobarbital or no drug and were given 1.6 mA shock in both states for approximately 50 training sessions. Overton then tested the subjects in both drug and nondrug states while varying shock intensities. He reported that testing with wide variations in shock intensity did not result in errors suggesting

that response control by the drug was not mediated through drug produced variations in shock-induced pain.

In a second experiment Overton (1968) tested various shock intensities to determine if those could govern responding by acting as discriminative stimuli during training. Rats were required to turn one direction when given 0.8 mA shock and the other direction when given 2.5 mA shock. Controls were trained unsuccessfully to alternate turns on successive days. No drugs were used. Results showed that differential responding was conditioned at a significantly lower rate than with conditions produced by 15 mg/kg pentobarbital vs. no drug.

Overton (1968), in a third experiment, tested the possibility that response control exercised by pentobarbital is mediated through sensory alterations in several modalities during training. To determine the importance of any drug produced analgesic effects influencing response control he devised three discriminations and trained a different group of rats on each discrimination. One group was trained to discriminate conditions pentobarbital (15 mg/kg) plus 0.8 mA shock vs. no drug plus 2.5 mA shock. A second group was trained to discriminate conditions pentobarbital plus 1.6 mA shock vs. no drug plus 1.6 mA shock. A third group was trained to discriminate conditions pentobarbital plus 2.5 mA shock vs. no drug plus 0.8 mA shock. Overton reasoned that if drug produced analgesic effects were important in response control then conditions pentobarbital plus 0.8 mA shock vs. no drug plus 2.5 mA shock would be the easiest to learn of the three discriminations. Conditions pentobarbital plus 2.5 mA shock vs. no drug plus 0.8 mA shock would be the most difficult discrimination to learn. Overton found that the rate of acquisition did not differ significantly among the three groups on the

discriminations tested. Thus, he concluded that drug produced analgesic effects alone were insufficient to regulate response control.

Overton (1968) carried out two experiments to test for differential response control of pentobarbital by mediation through alterations in visual cues. In the first experiment he trained sighted rats in the T-maze to differentiate pentobarbital 15 mg/kg vs. no drug during 30 training sessions. Those rats were then blinded. During continued training, after blinding, subjects showed only a small disruption of response control.

Overton (1968), in another experiment, blinded rats before training to determine whether or not subjects were aided in acquisition of response control by visual cues resulting from changes in drug states. Differential responding was conditioned in the T-maze. Overton reported that there were no significant differences between acquisition of blinded rats and sighted control rats.

Overton (1968) trained three groups of rats in the T-maze to differentiate various dosages of pentobarbital from either no drug or a different dosage of pentobarbital. One group was trained to differentiate conditions 10 mg/kg pentobarbital vs. no drug, another group to differentiate conditions 20 mg/kg pentobarbital vs. no drug, and a third group to differentiate 20 mg/kg pentobarbital vs. 10 mg/kg pentobarbital. Overton reported that rats trained to differentiate conditions 20 mg/kg pentobarbital vs. 10 mg/kg pentobarbital learned at about the same rate as rats trained to differentiate conditions 10 mg/kg pentobarbital vs. no drug. He found that rats trained to differentiate conditions 20 mg/kg pentobarbital vs. no drug gained response control faster than the other two groups. A test for tolerance was carried out by allowing a 20 day lapse without training or drug administration after differential responding was well established.

Those rats did not confuse 10 mg/kg pentobarbital with 20 mg/kg pentobarbital after returning to training, indicating little tolerance to the dissociative effects of pentobarbital during the previous training period.

Overton (1968) attempted to train rats to make a right turn while in a state produced by 10 mg/kg pentobarbital and a left turn while in a state produced by either saline or 20 mg/kg pentobarbital. The task provided rats with a very difficult discrimination since differential responding to three different states, rather than two, was required of each animal in the experimental group. Overton found that rats did not acquire differential responding easily or at a rate comparable to that of rats required to differentiate between only two drug states.

Otis (1964) conditioned rats injected with either 1.25 mg/kg chlorpromazine or saline to make an avoidance pole-jumping response when a flashing light signaled shock. Rats tested for retention in the state opposite the training state showed greater dissociation of the conditioned avoidance response (CAR) than did controls. Otis allowed a forty day rest period without drug administration or training following testing. He then retrained rats without injections, to give the CAR. Otis found that rats originally trained in a chlorpromazine state required more time to relearn the CAR than did rats originally trained in a saline state.

Barry, Etheredge, and Miller (1965) trained rats to press a lever for food reward and then punished the lever pressing response with shock until nearly all lever pressing had ceased. They next injected animals with either 10, 20, or 30 mg/kg of amobarbital sodium or saline of equal volumes and found that amobarbital resulted in resumed lever pressing. However, lever pressing resumed under the influence of amobarbital did not transfer to the nondrug state and apparently neither did any drug

induced reduction of fear. The data suggested an inverse relationship between drug dosage and performance in the nondrug state.

Sachs, Weingarten, and Klein (1966) experimented with chlorpromazine and chlordiazepoxide to determine the dossociative effects and the effects on acquisition of these two drugs. They trained rats to perform a hurdle jump CAR while in a state produced by injections of either chlorpromazine varying from 0.25 mg/kg to 2 mg/kg, 15 mg/kg chlordiazepoxide, or saline. Later they tested rats for transfer of the CAR following injections of either the substance administered during training or a dissimilar substance. Their results showed that in acquisition rats given doses of chlorpromazine as low as 0.25 mg/kg could not reach a criterion of 20/20 correct responses. Chlordiazepoxide trained rats reached criterion significantly faster than saline controls. They found that chlordiazepoxide trained rats did not transfer the CAR to the nondrug state. Rats trained in a nondrug state and tested under chlordiazepoxide trained rats did not transfer the CAR to the nondrug state. Rats trained in a nondrug state and tested under chlordiazepoxide showed a significant loss of the CAR. Chlordiazepoxide trained rats tested under amphetamine and chlorpromazine showed a much more severe loss of the CAR than did nondrug controls tested under the same conditions; however, only two animals trained under chlordiazepoxide were available for that test.

Iwahara, Iwasaki, and Nasegawa (1968) tested the effects of 2 mg/kg injections of chlorpromazine, homofenazine, and saline on a passive avoidance response (PAR). They trained undrugged rats by confining them to the shock compartment of a two compartment apparatus for a period of five minutes during which the rats received inescapable shock. They gave one group of rats a total of 10 shocks at the rate of two shocks per

minute and the other group a total of five shocks at the rate of one shock per minute. A day later they divided each of the two groups and unshocked controls into three subgroups to which they administered either chlorpromazine, homofenazine, or saline. They placed the rats in the unshocked compartments and recorded the time spent in each compartment during three minutes of unrestricted exploration. Two other tests without injections were given 48 and 96 hours after the first test.

Results for rats shocked five times per five minute period showed a PAR for saline tested rats but not for chlorpromazine and homofenazine tested rats. Results for rats shocked 10 times per five minute period showed a PAR for saline, chlorpromazine, and homofenazine tested rats. Dissociation was not confirmed in the two nondrug, nonshock tests which followed testing under drug.

Kubena and Barry (1969) tested the generalization of the stimulus characteristics of alcohol and atropine to other drugs. They trained rats in a conflict procedure by giving food reward for lever pressing on a fixed ratio (FR) schedule in states produced by injections of either 1200 mg/kg ethyl alcohol, 10 mg/kg atropine sulfate, or saline. They then gave the rats shock for lever pressing on the same FR schedule while in the opposite drug state.

Kubena and Barry trained other rats in a choice procedure to press one of two levers for food reward. The correct lever could be discriminated only on the basis of drug conditions; one lever was correct under drug, the other under saline. They used the same drugs and dosages which were used in the conflict procedure. Following training in both procedures Kubena and Barry tested their animals in states produced by drugs other than those used in training.

Their results for both procedures indicated generalization of the alcohol response to pentobarbital, chloridazepoxide, and chloral hydrate, all general depressants. The alcohol response did not generalize to chlorpromazine. Atropine responses generalized to scopolamine and did not generalize to atropine methyl bromide. Kubena and Barry point out the commonality of a central anticholinergic effect shared by atropine sulfate and scopolamine and state that atropine methyl bromide has anticholinergic effects which are peripheral.

Iwahara and Matsushita (1971) trained rats to avoid shock by discriminating black and white stimulus doors in a three section apparatus consisting of a start box, choice chamber, and goal box. They trained rats to a criterion of 18/20 correct responses in original discrimination learning while in states produced by injections of either 20mg/kg chlordiazepoxide or saline. They then divided the drug and saline groups so that four groups were formed. Two of these groups were retrained on the same discrimination while in the same drug state. The other two groups were retrained on the same discrimination originally learned but in the drug state opposite that in which original learning occurred. Iwahara and Matsushita again divided each of the groups so that eight groups were formed. They again repeated the original discrimination learning for all groups with the exception of the state in which learning occurred. During the second relearning period the training state was the same as the training for the first relearning period for four of eight groups. The other four groups were in the state opposite that in which the first relearning period occurred during the second relearning period. The criterion of 18/20 correct responses was used in the first and second relearning periods for all groups.

Iwahara and Matsushita found that chlordiazepoxide retarded learning of correct responses and lengthened latencies. They found that changing drug states resulted in a decrease of correct responding that was about equal for responses given during the first relearning period and responses overlearned in the second relearning period. They found a dissociative effect which occurred during the first relearning period and in the second relearning period for animals that had experienced only one state change. They did not find dissociation during the second relearning period for rats which had previously experienced training in both drug states. Latencies were not state-dependent.

Pusakulich and Nielson (1972) tested the hypothesis that "state-dependent learning is mediated by changes in brain excitability levels" (p. 33). They trained four cats to give a foreleg flexion response to avoid shock. The cats received electrical stimulation of either the left caudate nucleus or the left misencephalic reticular formation as a conditioned stimulus. The cats were initially trained in the nondrug state. Pusakulich and Nielson found response thresholds by gradually reducing CS intensity until CR's were no longer elicitable. They then determined response thresholds for dosages of sodium pentobarbital 5, 10, 12.5, and 15 mg/kg. Thresholds were checked at 1, 2, 4, 8, and 24 hours following injections. They reconditioned subjects in a state produced by 15 mg/kg pentobarbital. Another group of four cats was trained in the opposite sequence; first in the drug state and then in the nondrug state.

Pusakulich and Nielson reported that shifts in the level of brain excitability could not account for state-dependency and that the effect of the drug upon the CR threshold was dependent upon the subject's prior training in the drug state. Their results indicated that drug and nondrug

states were distinct prior to training in both states but not afterward. Following training in both states, threshold shifts that occurred as a result of varying drug conditions after training in only one state were no longer observable.

Gruber, Reed, and Block (1968) paired the sound of a door buzzer (CS) with stroking of the plantar surface of the feet (UCS) of hospital patients. The patients were in a light post-operative anesthesized state during conditioning of a galvanic skin response (GSR). A control group was given the CS and UCS unpaired and in random order during training in the drug state. A day later after recovery from anesthesia the patients were questioned to determine whether or not they were aware of the conditioning procedure on the previous day. Those subjects who were determined to be unaware were told that they would be given a "test of reflexes" (p. 151). The investigators gave the experimental and control groups extinction trials consisting of the CS alternating with a bell until neither elicited the GSR. After extinction trials were completed the patients were given reinforced trials with a non reinforced test trial given on every fourth trial until five test trials and 15 reinforced trials had been given. None of the 20 patients used in the experimental group had any recollection of conditioning under anesthesia after completion of all trials.

The investigators found that patients in the experimental group performed significantly better than controls on relearning of the conditioned GSR in the nondrug state. Transfer from the drug state to the nondrug state occurred without the patients' awareness of training after recovery from the drug state. Transfer was more evident on test trials during relearning than on extinction trials preceding relearning. The experimental

group was superior to the control group on extinction trials, but not significantly so.

Storm and Caird (1967) tested the dissociation hypothesis with 40 chronic male alcoholics ranging in age from 40 to 50 years. They randomly divided subjects into four groups in a 2 x 2 factorial design. One group was given alcohol during learning and relearning. A second group was given alcohol during learning but not during relearning. A third group was given no alcohol during learning but was given alcohol during relearning. A fourth group was not given alcohol during learning or relearning. A list of 12 two-syllable nouns was presented on a memory drum at a rate of 3 seconds per word. Subjects learned the list to a criterion of one perfect repetition. To minimize rehearsal between learning and relearning, Storm and Caird asked their subjects to return 48 hours later for a test of motor skills. When subjects returned relearning was carried out on the same word list and to the same criterion used in initial learning.

Storm and Caird found that when drug conditions existing during initial learning were the same during relearning fewer trials were required to reach criterion than when drug conditions existing during initial learning and relearning were different. They found that alcohol resulted in a significant decrement in final performance when given during learning but not when given during relearning.

Effects of Chlordiazepoxide

Training with chlordiazepoxide has resulted in dissociated learning in several experiments (Connelly, Connelly, & Epps, 1973; Connelly, Connelly, & Phifer, 1975; Iwahara & Matsushita, 1971; Overton, 1966; Sacks, Weingarten, & Klein, 1966).

According to a review by Randall and Schallek (1967), the administration of chlordiazepoxide, whether oral or by injection, results in increased food intake and weight gain in rats. When chlordiazepoxide was placed in rats' food, lower dosages were required for male than for female rats to cause an increase in food intake.

Hoogland, Miya, and Bousquet (1966) gave some of their rats intraperitoneal injections of either 100 mg/kg chlordiazepoxide or saline once a day for five successive days. On the sixth day, 24 hours after injections on the fifth day, they injected both the drug and saline groups with 100 mg/kg chlordiazepoxide and timed the duration of the drug induced paralysis which followed. They found that paralysis lasted for 65.0 minutes in the drug group and 113.3 minutes in the group that had previously been given saline; thus, tolerance to the muscle relaxant effects of the drug was easily developed. Hoogland et al. analyzed tissue samples, fecus, and urine and found that the rate of drug excretion was greater in the chlordiazepoxide group than in the group given the drug on the sixth day only; thus, repeated administration of chlordiazepoxide over the five day period resulted in tolerance to the biological effectiveness of the drug.

Sachs et al. (1966) found that rats trained under 15 mg/kg chlordiazepoxide learned a hurdle jumping CAR significantly faster than rats trained under saline. However, most of the difference between the chlordiazepoxide and saline trained groups appeared during the first two training sessions and differences were not significant on the five sessions following. Sachs et al. suggested that some responses to novel stimuli (e.g., orienting and freezing) may compete with the CAR during acquisition in the undrugged rat and then disappear as novel stimuli become habituated.

They cite evidence which indicates that hippocampal theta rhythm accompanies the emission of these responses to novel stimuli and that theta rhythm decreases as these stimuli become habituated. They state that drugs which abolish hippocampal theta rhythm are also reported to disrupt performance. They suggest that chlordiazepoxide may facilitate acquisition of the CAR via an action on the central nervous system which abolishes hippocampal theta rhythm and, as a result, the responses to novelty which accompany it. Sachs et al. observed a marked relaxation of muscles, occasional ataxia, and less tendency to freeze in rats drugged with chlordiazepoxide as compared to saline controls.

Hughes (1972) studied the effects of chlordiazepoxide upon exploration in rats and found an inverted U relationship between dosage strength and locomotion. He found that locomotion did not differ significantly when produced by injections of either saline or a 5.0 mg/kg of chlordiazepoxide, but that 2.5 and 3.75 mg/kg injections of the drug produced significantly more locomotion than saline or the 5.0 mg/kg dosage; 2.5 mg/kg produced slightly more locomotion than 3.75 mg/kg. Hughes found that 2.5 and 3.75 mg/kg chlordiazepoxide had little influence on rats' preferences for the novel half of the test apparatus, but that 5.0 mg/kg produced a marked preference for the familiar half.

Iwahara and Matsushita (1971) found that rats trained to avoid shock by discriminating black and white stimulus doors, learned somewhat faster under 20 mg/kg chlordiazepoxide than under saline, but the difference in acquisition was not significant. They found that the distribution range of running times was greater for drugged than undrugged rats throughout the experiment. The median running time for rats given only chlordiazepoxide was longer than that for rats given only saline.

Connelly et al. (1975) trained rats to escape shock in a modified T-maze after injections of either 40 mg/kg chlordiazepoxide or saline. During early training trials they observed less correct turns and longer running times in drugged than in undrugged rats.

Considering these cumulative findings, it would seem that the effects of chlordiazepoxide upon performance may be contingent upon a number of variables. Some variables which would seem to be probable determinants of the drug's effects on performance are: drug dosage and number of administrations within a given period; food intake and weight control; nature of the task involved.

Disruption of State-dependent Learning

Connelly et al. (1973) used emotionally important conditioned stimuli to disrupt dissociative learning in rats trained on a discrimination task. They trained four experimental groups, two dissociation and two transfer groups, to escape foot shock in a modified T-maze.

The dissociation group was trained to turn in one direction following an injection of 15 mg/kg chlordiazepoxide and in the opposite direction following an injection of sterile water (i.e., in the nondrug state). They trained the transfer group on a more complex discrimination which required discriminating between drug and nondrug states and between high and low frequency tones paired with foot shock. Their transfer groups learned to escape shock by running in one direction when presented with the high frequency tone while in the drug state, and by running in the opposite direction when presented with the low frequency tone while in the nondrug state. Both the tone and the drug state signaled the same direction of escape on any given trial during training.

Without using foot shock, Connelly et al. tested the experimental groups and controls for transfer across drug states following training. During testing they reversed the drug state-tone associations for the transfer groups so that rats which had previously been trained in the drug state with the high frequency tone were tested in the drug state but with the low frequency tone. The same rats which were also previously trained in the nondrug state with the low frequency tone were tested in the nondrug state but with the high frequency tone. Thus, the tone signaled escape in one direction while the drug state signaled escape in the opposite direction.

They found that rats in the dissociation group made correct turns in response to the drug state induced during testing. For those rats learning in the drug state was dissociated from learning in the nondrug state. They found that rats in the transfer groups made correct turns in response to the tone given during testing even though the drug state signaled escape in the direction opposite to that signaled by the tone. In their experiment, tones eliciting fear of foot shock were able to completely disrupt dissociative learning.

Connelly et al. (1975) used an emotionally important conditioned stimulus (EICS) to disrupt dissociative learning in a state-dependent learning design. They trained rats in a modified T-maze to escape shock by making a correct turn following an injection of either 40 mg/kg chlordiazepoxide or sterile water. This experiment differed from the Connelly et al. (1973) experiment in that rats in this experiment were trained to turn in only one direction, either left or right, and in only one drug state, either drug or nondrug. During training, Connelly et al. gave a

tone paired with foot shock to two transfer groups; one trained in the drug state, the other in the nondrug state.

Following training they tested the two transfer groups and the state-dependent experimental groups without shock in both drug and nondrug states. They presented the tone to both transfer groups throughout testing.

Connelly et al. found that their state-dependent experimental groups gave responses learned during training when the test drug state was the same as the training drug state, and random responses when the test drug state was opposite the training drug state. However, they found that the presentation of the tone to the transfer groups resulted in predominately training state responses regardless of which drug state the rats were tested in. The tone became an emotional memory prompter (EMP) mediating transfer between drug states and exercising more response control during testing than either the nondrug or drug state.

According to Connelly et al. (1975),

> Fear was associated with only a very general stimulus (the entire apparatus) for the SDL groups, and apparently this stimulus was not usable as a memory prompter when Ss were in the opposite drug state during testing. However, Transfer groups associated fear with a specific stimulus (the tone) which could be used to retrieve D-state information when in the ND test state (or vice versa). Though fear did not dissociate in the Transfer groups, it is perhaps more meaningful to say that the tone (representing this fear) could be used to disrupt drug-state dissociation (p. 142).

In these two experiments (i.e., Connelly et al., 1973; Connelly et al., 1975) the shock delivering apparatus, and especially the visual stimuli it provided, may have been indiscriminably camouflaged in the rat's perception of a complex environment. The rat could not "see" shock while he was in the apparatus and the grid floor in the safe goal boxes was identical to the electrified floor of the start box and choice section of the maze. Thus, it may have been very difficult for a rat not given tone to associate shock with any particular feature of the apparatus during a short training period and while under a great deal of stress. The tone, on the other hand, was a unique readily discriminable stimulus presented in the absence of other auditory stimuli (except for noise made by the rat) through a single sensory modality. Unlike the apparatus, the tone was present only shortly before and during the onset of shock. Like shock, the tone could not be seen by the rat and therefore it may have been more readily associated with shock than visual stimuli could be.

Since the tone was turned on as the rat was being placed on the electrified grid floor, and was turned off as soon as the rat reached safety, the emission of the conditioned escape response and the presentation of tone were nearly simultaneous in the transfer groups. Therefore, conditioned escape responding was probably more closely associated with the tone in the transfer groups than it was with any single discriminitive stimulus in the state-dependent experimental groups (Connelly et al., 1975) and the dissociation groups (Connelly et al., 1973). Thus, not only could the tone transfer fear across drug states but also the conditioned escape response instrumental in removing the rat from the feared situation.

During testing in the Connelly et al. experiment (1975) the apparatus alone may have been an adequate stimulus for eliciting fear of shock acquired in the opposite drug state, but it was not adequate to elicit the conditioned escape response. It is possible that fear elicited by the apparatus may have been antagonistic to attending and recall in the no tone groups during testing. Thus, the recall of conditioned escape responses already obscured by a drug state barrier may have been made more difficult by the presence of fear evoked by the apparatus.

It would seem that the fear of foot shock during training was as intense in the dissociation groups (Connelly et al., 1973) and state-dependent experimental groups (Connelly et al., 1975) as in the transfer groups in these experiments. It appears, however, that in order to effectively disrupt dissociation with fear, this intense "emotional energy" must be channeled toward a specific stimulus which is present during training and testing and closely associated with a specific conditioned response to an aversive stimulus.

From the research of Connelly et al. (1973) and Connelly et al. (1975) an important question arises as to whether or not intense fear is the only emotion which will produce an EICS that will serve an an EMP capable of disrupting state-dependency. If a tone can disrupt state dependency because it elicits fear of a powerful aversive stimulus and channels that fear into a specific conditioned response, can the same tone also disrupt state dependency by eliciting pleasant emotions accompanying positive reinforcement and channeling that emotional energy into a specific conditioned response?

Statement of the Problem

In the present study the problem was to determine if an EICS (tone) could disrupt state-dependency when paired with feeding and a specific conditioned response instrumental in obtaining a food reward for rats trained in a T-maze.

Predicted Trends in State-Dependency and Its Disruption

The following are some predicted outcomes of the present study and my reasons for predicting them:

1. State-dependency will be disrupted by the EMP (tone) because emotional energy will be efficiently channeled into a specific conditioned response by associating pleasant emotions and the conditioned response with the EMP.

2. The nature of the task involved will result in less disruption of state-dependency by the EMP than was shown by the tone transfer groups in the Connelly et al. (1975) experiment. I predict this outcome because I subjectively gauge the emotional energy to be channeled into all responding associated with the EMP in the present study to be of less intensity than that associated with the EMP in the Connelly et al. (1975) experiment. Therefore, I expect the EMP in the present study to be less efficient than the EMP in the Connelly et al. (1975) experiment.

3. Less state-dependency will be demonstrated by the state-dependent experimental groups in the present study than was demonstrated by the state-dependent experimental groups in the Connelly et al. (1975) experiment. I believe that intense fear elicited by the apparatus, and other general stimuli in close temporal proximity to placement in the apparatus, is partly responsible for the extreme degrees of drug-related dissociation found in some escape learning tasks. I believe that this intense

unchanneled fear is an antagonistic response which competes with attending and recall when the animal is inside of or near to the experimental apparatus while being tested in the drug state opposite of the training state. I, therefore, expect the absence of fear in the present study to result in more recall when animals are in the state opposite the training state, and, hence, more training state responding than was shown by the state-dependent experimental groups of the Connelly et al. (1975) experiment.

Hypotheses

1. If rats are trained to make correct turning responses in a T-maze for a food reward while in either a drug or non-drug state (produced by either 45 mg/kg chlordiazepoxide or sterile water), and if two groups of those rats (one group trained in the drug state, the other in the nondrug state) are conditioned to respond to an EMP (tone paired with feeding and responses instrumental in obtaining food) during training and are subsequently tested with the EMP in the drug state opposite the training drug state, then those two groups will demonstrate significantly more disruption of state-dependency than two other groups (one trained in the drug state, the other in the nondrug state) given the same training and testing but without the EMP.

2. If the degree of disruption of state-dependency in groups given the EMP in the present study is compared to the degree of disruption in the tone transfer groups of the Connelly et al. (1975) experiment, then the degree of disruption shown by the EMP groups in the present study will be less than that shown by the tone transfer groups of the Connelly et al. (1975) experiment.

3. If the degree of state-dependency demonstrated by the state-dependent learning experimental groups in the present study is compared to the degree of state-dependency demonstrated by the state-dependent learning experimental groups in the Connelly et al. (1975) experiment, then less state-dependency will be found in the present study than was found in the Connelly et al. (1975) experiment.

Significance of the Present Study

The present study is designed to determine whether or not positive reinforcement can produce an EMP capable of disrupting state-dependency under given experimental conditions. If the EMP can disrupt state-dependency in rats, then it is possible that the same can be done with humans. Thus, one possible practical application of disruption of state-dependency may be in developing an effective therapy for alcohol and drug abuse cases. However, the disruption of state-dependency in human beings is not the final goal of this research.

The present study is a small part of a total research program intended to discover and demonstrate methods of developing, harnessing, and directing emotional energy in humans. Such methods, when they are developed, will make a tremendous contribution to the behavioral sciences. By finding ways of developing, harnessing, and directing emotional energy man may, for example, be able to more effectively treat behavioral disorders, emotional disturbances, physical illness, and mental retardation. The effective control of emotional energy may improve education in the classroom and therapy in the mental health clinic. It may increase mental potential, self actualization, and life span.

The possibilities which are speculated upon here will certainly not be accomplished as a direct result of the present study. However, the present experiment may provide some small, but important, bits of information which, when compiled with a great deal of other research findings, may contribute to the development of these prospects. I sincerely believe that these prospects, as far reaching as these may seem, are attainable. Therefore, I have optimistically presented them here not intending to over dramatize the importance or implications of the present experiment, but in order to meaningfully relate this animal research to the lives of people. At this time it is impossible to determine the extent to which research in this general area might benefit mankind but the possibilities are tremendous and the goals well worth pursuing.

CHAPTER II

METHOD

Subjects

The subjects were 64 male hooded rats, 80 days old on arrival, purchased from Maxfield Farms, Ohio. Rats were kept in individual cages in a lighted room during the experiment. Five squads were run in order to obtain 64 subjects able to complete the experiment. An attempt was made to run 16 subjects in each of the first four squads; however, 14 rats died and the identity of two others was lost prior to training. Replacements were immediately available for some of the rats that died early in training. Whenever possible, replacements were run in the squads of the animals that were being replaced. Some of the replacements also died before completing the experiment. The fifth squad consisted of replacements for eight subjects missing from Squads 2, 3, and 4. The number of subjects that completed the experiment in Squads 1, 2, 3, 4, and 5 was 16, 12, 14, 14, and 8 respectively. The number of rats that died before completing the experiment in Squads 1, 2, 3, 4, and 5 was 2, 6, 6, 2, and 1 respectively.

Apparatus

The apparatus was a grey, wooden modified T-maze consisting of a start box, a choice section, and two goal boxes; one to the left and one to the right of the choice section. Each section was 14 cm wide, 23 cm long, and 13 cm high, except for the start box which was different only in that the walls were 28 cm high. One wooden, manually operated guillo-

tine door separated the start box from the choice section. Two manually operated Plexiglass guillotine doors (goal box doors) separated the choice section from each of the goal boxes. The goal box doors were transparent so that lighting in the goal boxes remained unchanged when the doors were closed. A three piece, removable, grey, wooden floor, 1 cm thick and 13 cm wide, rested on grids and ran throughout the maze. Running times were recorded from a Lafayette 0.01 Second Timer connected to photocells in the goal boxes and to a mercury switch attached to the start box lid. Feeding was timed with a stop watch. A grey Reliance seed cup (Item No. 728/2), 3.5 cm wide, 5.5 cm long, and 3 cm high, was attached to the floor in each goal box next to the end wall opposite of the goal box door. The location and depth of the seed cups was such that a rat could not see food in either cup until the animal had entered a goal box.

A 1 kHz, 600 MW tone was delivered by a Precision Audio Generator (Model No. E-310) and Scientific Power Amplifier (Model No. 382B) through a 13 cm radio speaker. The speaker was not enclosed and was located 40 cm directly above the center of the floor of the maze's choice section. The onset and termination of the 1 kHz tone was controlled by a foot-operated telegraph key.

A grey, wooden feeding box was used during pre-training to simulate feeding inside of the maze goal boxes. The feeding box was 14 cm wide, 23 cm long, and 13 cm high. The box was equipped with a seed cup (identical to those in the goal boxes), a 6 W, 125 volt light bulb on each side wall, and a removable, grey, wooden floor resting on grids. The feeding box was located on a table 2.5 meters directly behind the maze start box and was placed so that a rat feeding inside faced the same direction as when being placed in the maze's start box.

Drugs

Chlordiazepoxide hydrochloride (Librium) in 100 mg ampuls was diluted with 2.2 cc of sterile water per ampul. The diluted drug was injected in volumes of 0.001 cc per gram of body weight so that a constant dosage level of 45 mg/kg was maintained. Injections of equal volumes of sterile water produced the nondrug state. All injections were intraperitoneal (i.p.).

Design

Each of the 64 rats was randomly assigned to one of eight groups. There were two state-dependent learning (SDL) experimental groups, two SDL control groups, two transfer experimental (tone) groups, and two tone control groups. Eight rats were assigned to each group.

Procedure

Upon arrival at the lab all rats in Squad 1 were allowed free access to food (free feeding) for 72 hours. All other squads were allowed free feeding for 48 hours following arrival and every animal in all squads was allowed free access to water throughout the experiment.

Food deprivation. For Squad 1 deprivation was as follows: Four rats (not included as subjects) were assigned to a weight control group, given no training and were kept on free feeding while all other rats were placed on food deprivation. The weight control animals were weighed daily and 30% of that group's daily mean weight-increment was added daily to 80% of the free feeding weight (weight on the last day of free feeding) of each deprived subject to yield the deprived subject's running weight. The deprived subject's actual weight was then regulated toward the running weight on each successive day by regulating

feeding. This deprivation schedule was intended to decrease weight and then allow a gradual weight gain as training progressed; however, because the schedule appeared to be ineffective, running weights were re-calculated by adding 70% of the weight control group's daily mean increment to 70% of the free feeding weight of the deprived subjects. Although the new schedule apparently resulted in improved performance for some rats it was not considered adequate. A third and fourth schedule, neither of which required a weight control group, were then tried. Using the third schedule the subject's running weights were again recalculated by scheduling an increase in equal units from 70 to 85% of the free feeding weight of each subject over a 20 day period beginning with the first day of deprivation. The fourth schedule by which running weights were recalculated allowed an increase in equal units from 75 to 80% of each rat's free feeding weight over a 25 day period beginning with the first day of deprivation. The combined effect of these various schedules was to decrease weight throughout training until each rat reached the training criterion. Schedule changes were made 3, 7, and 15 days after training began. Although some replacement animals began training at a later date than other rats in the same squad all of the rats in each squad began deprivation on the same day and no rat required more than 12 days of training.

For Squads 2, 3, 4, and 5 deprivation was as follows: Each squad began pre-training on the day that its mean weight was reduced to 81% of its mean free feeding weight. Running weights were calculated according to the fourth schedule used in Squad 1; increase was allowed in equal units from 75 to 80% of the individual's free feeding weight over a 25 day period beginning on the first day of deprivation. However,

if an animal was maintained below its running weight plus 10 grams for three consecutive days and did not make a total of at least six correct responses over that 3 day period then its daily running weights were lowered by 10%.

Because of difficulties with weight control, drug effect on weight, hunger, and responding, the amount of food increase given once an animal reached its running weight was subjectively determined and frequently resulted in continuing weight loss although the rate of weight loss usually decreased once the running weight was reached.

In order to compare weight changes among squads a mean and standard deviation were derived, for each squad, for weight on the first day of pre-training, the lowest weight reached by each rat, and weight on the last day of testing. Each mean weight was then converted to a percent of the mean free feeding weight of the squad from which that mean was derived. These data are presented in Table 1.

TABLE 1

MEAN WEIGHTS, STANDARD DEVIATIONS, AND MEAN WEIGHTS AS PERCENTS OF MEAN FREE FEEDING WEIGHTS FOR THE FIRST DAY OF PRE-TRAINING, THE LOWEST WEIGHT REACHED, AND THE LAST DAY OF TESTING

	First Day of Pre-Training			Lowest Weight Reached			Last Day of Testing		
Squad No.	$\bar{x}$(gm)	σ(gm)	$\bar{x}$/ff wt.[a](%)	$\bar{x}$(gm)	σ(gm)	$\bar{x}$/ff wt.(%)	$\bar{x}$(gm)	σ(gm)	$\bar{x}$/ff wt.(%)
1	241.39	22.56	86	215.52	21.86	77	220.79	21.56	78
2	242.81	17.98	81	220.51	17.48	74	229.42	14.64	77
3	286.38	21.25	81	249.29	17.34	71	253.13	19.31	72
4	255.96	17.37	81	213.86	22.02	68	215.32	20.53	68
5	229.29	11.58	81	197.26	10.81	69	200.85	10.18	71
All Squads Combined	251.17	27.37	82	219.29	24.71	72	223.90	24.53	73

[a]ff wt. free feeding weight.

Pre-training. All subjects were individually given 5 minutes of free exploration in the maze on each of the 2 days preceding the first day of training. During these exploration periods the start box door and both goal box doors were open and no food was placed anywhere in the maze.

On the same 2 days, following the exploration period, each rat was placed in the feeding box and retained there until it had eaten four food pellets (Noyes, formula A, size 45 mg) from the seed cup, or until 5 minutes had passed, depending upon which occurred first. Following the time in the feeding box the rats were returned to the home cages until the following day. Four rats (no. 34, 35, 38, and 54) failed to eat in the feeding box on both days of pre-training. Those four rats were given no food in the home cages following the second day of pre-training. On the morning after the second day of pre-training the four were individually placed in the feeding box and retained there until each had eaten four food pellets. The longest time required for any of the four to eat in the feeding box was 13 minutes. All other subjects had eaten in the feeding box by the end of the second day of pre-training. The purpose of this pre-training was to facilitate acquisition during training.

Training states. All groups were trained while in either the drug (D) or the nondrug (ND) state. One SDL experimental group, one SDL control group, one SDL transfer experimental group, and one SDL tone control group were trained while in the D state. Conversely, one SDL experimental group, one SDL control group, one SDL transfer experimental group, and one SDL tone control group were trained while in the ND state. For each group trained while in the D state, one other group, identical in all other respects, was trained while in the ND state.

Training. All of the eight groups were trained to respond for a food reward in the T-maze. Each rat was trained to make a correct turn, which was in the direction opposite to its position preference, and to run to a correct goal box located in the same direction; both goal box doors were open on every training trial. Each rat was trained to turn in only one direction, either left or right, and in only one D state, either the D or the ND state. Each of 58 of the rats were given five massed training trials per day until reaching the training criterion of 18/20 responses correct over 4 consecutive days. Because the training criterion was not determined until after training of the first squad had begun three rats (no. 21, 24, and 31) were given one extra day of training after reaching the training criterion and three other rats (no. 27, 36, and 39) were given two extra days of training.

Exactly four food pellets were placed in the seed cup of the correct goal box before each training trial while the seed cup of the incorrect goal box was kept empty. On each training trial the rat was placed in the start box with the start box door and both goal box doors open and allowed 30 seconds to enter the correct goal box. After the rat moved into the choice section the start box door was gently closed behind the animal. If the rat entered the incorrect goal box, that goal box door remained open. Once the rat entered the correct goal box, the goal box door was gently closed. On training trials 1, 2, and 5 the rat was pushed into the correct goal box if the animal was anywhere in the maze except in the correct goal box 30 seconds after being placed in the start box. On training trials 3 and 4, the rat was pushed into the correct goal box after 30 seconds if the animal was in the incorrect goal box or the choice section. However, if the rat remained in the

start box for 30 seconds on training trials 3 and 4 the animal was gently pushed into the choice section, the start box door was closed, and an additional 20 seconds was allowed for running into the correct goal box. After those 20 seconds had passed the rat was pushed into the correct goal box if the animal had not entered. When a rat did not enter either goal box within 30 seconds, a "No choice" with a 30-second running time was recorded. When a rat entered the incorrect goal box and remained there until 30 seconds had elapsed, an incorrect response with a 30-second running time was recorded. When a rat entered the incorrect goal box and then entered the correct goal box before 30 seconds had elapsed, an incorrect response was recorded with a running time equal to the number of seconds required for reaching the correct goal box.

Once the rat had entered the correct goal box (i.e., the animals rear quarters passed the goal box door opening) 10 seconds was allowed for the animal to place its nose beneath and inside of the rim of the seed cup. If the rat did not place its nose in the cup within 10 seconds, the rat's body was held so that the rat's nose touched the food pellets in the cup for 3 seconds and then the animal was gently released. The rat was allowed to remain in the correct goal box for not longer than 15 seconds after its nose was seen beneath and inside of the rim of the seed cup regardless of the number of food pellets eaten. The rat was removed from the correct goal box in less than the allotted time if the animal had been in the correct goal box for at least 10 seconds and had eaten all four of the food pellets. After each trial the rat was removed from the correct goal box, placed in the carrying cage for 10 seconds, and then given another trial until five trials were completed. Occasionally a rat was found in the carrying cage between trials chewing on food pellets

carried back from the maze or on feces and was allowed to finish chewing before being given the next trial. Disinfectant deodorant (Zepyramic, E.P.A. Regulation No. 1270-166-AA) was sprayed into the choice section of the maze after each trial to prevent rats from tracing odor trails.

Tone in training. The crucial variable in this experiment was the pairing of a 1 Khz tone with a food reward and the conditioned response that was instrumental in getting that reward. Only the two transfer experimental groups and the two tone control groups were given the 1 Khz tone. The tone was presented to these groups on every training trial. The tone was turned on as rats were placed in the start box and turned off as rats were lifted out of the correct goal box.

Correct response. For both training and testing trials, a correct response was defined as a rat's running to the correct goal box in a running time of 30 seconds or less without taking one or more steps with each of the animal's four feet in the direction of the incorrect goal box after the front half of the animal's body enters the choice section.

Running times. During training a rat's running time began when the animal was placed in the start box and ended when the animal entered the correct goal box or when 30 seconds had elapsed before the animal reached the correct goal box. During testing the running time began when the animal was placed in the start box and ended when the rat entered either the correct or the incorrect goal box or when 30 seconds had elapsed before entry into either goal box was made.

Injections. Rats given a M injection were run 25 minutes after the injection and were never given more than a single injection on any one day. Rats given a D injection were run 25 minutes after the injection only if the animals' bodies appeared limp when lifted, and only if the animal

was mobile. If a rat did not appear limp 25 minutes after a single D injection, a second D injection, equal to 67% of the first dosage, was immediately given and the animal was run 25 minutes later provided that limpness appeared and provided that the animal was mobile. If limpness did not appear 25 minutes after the second D injection, a third D injection, equal to 67% of the first dosage, was given and the rat was run 25 minutes later if it was mobile. No rat required more than three D injections (i.e., one injection at full dosage followed by two injections at 67% of the first dosage level) to become limp on any one day. During training a total of 53 second D injections were given, eight of which were followed by third D injections. During testing 38 second D injections and no third D injections were given.

Mobility. Rats given D injections were placed on a table after being judged limp. If the rat remained immobile (did not walk or crawl) for 20 seconds, a 5 gm Purina Lab Chow food pellet was held approximately 1 cm from the rat's nose and moved away only as fast as was necessary to keep the pellet out of the rat's mouth. If the rat was unable to follow the food pellet, the animal was detained in the home cage and re-checked at 15 minute intervals. When the rat was able to crawl 20 cm toward the food pellet, trials were given. No rat was detained for more than 2 hours because of immobility without being removed from the experiment.

Testing. Three massed trials per day were given on 5 consecutive days of non-reward testing which began for each rat 24 hours after the completion of training. For the two SDL control groups and the two tone control groups, the testing D state was always the same as the training D state. For the two SDL experimental groups and the two transfer experimental (tone groups), the testing D state was the opposite of the training

D state on Testing Days 1, 3, and 5, but the same as the training D state on Testing Days 2 and 4. The 5-day testing sequence for those four groups was ND-D-ND-D-ND if rats were trained while in the D state, and D-ND-D-ND-D if rats were trained while in the ND state. Using this single alteration schedule the testing D state on the first day of testing was always the opposite of the training D state, thus predisposing the rat to make training state responses in the opposite D state. Throughout testing both goal box doors were open so that rats could enter either goal box and no food was placed anywhere in the maze. On each testing trial the rat was placed in the start box with the start box door open. The start box door was gently closed after the rat entered the choice section. After entering either goal box, the goal box door was gently closed behind the rat, the animal was detained in the goal box for 10 seconds, placed in the carrying cage for 10 seconds, and then given another trial until three trials were completed. If a rat remained in the start box or choice section for more than 30 seconds after placement in the start box, both goal box doors and the start box door were closed, the rat was removed from the maze ending that trial, and a "No choice" with a 30-second running time was recorded. Just as in training, body limpness was the criterion used to determine whether rats were to be run following a single D injection or given a second D injection equal to 67% of the first dosage. Mobility was demonstrated by the same criterion used in training before trials were given. Zepynamic Disinfectant Deodorant was sprayed into the choice section after each trial.

Tone in testing. The 1 Kls tone was given to the two transfer experimental groups and the two tone control groups on all testing trials regardless of the D state present. The tone was turned on as each rat

was placed in the start box and remained on until the rat had been detained in either goal box for 10 seconds or until the rat was removed from the start box or choice section in the event that no choice was made within 30 seconds.

Reminder trials. Every rat was given three reminder trials 15 minutes after the last test trial on both Testing Days 2 and 4. No reminder trials were ever given on Testing Days 1, 3, or 5. If a rat had not entered either goal box within 30 seconds on any reminder trial, the animal was gently pushed into the correct goal box. Reminder trials were identical to training trials in all other respects including E state, food reward, presence or absence of the tone, and free access to either goal box. Zoyvanic Disinfectant Deodorant was sprayed into the choice section after each trial.

CHAPTER III

RESULTS

Response measures. The response measures taken for each subject for each day of training and testing were:

1. The number of correct responses.

2. The proportion of correct responses (the resulting fraction, expressed as a percent, when the number of correct responses per day is the numerator and the total number of trials per day minus the number of "No choice" trials per day is the denominator).

3. The number of first trial correct responses (the number of responses made on the first trial of each day which are correct).

4. The median latency (the median running time of each day's trials; where the median latencies of a group are referred to as being longer or shorter than that of another group the reference is to the daily total of the median latencies of each group).

Criteria for a state-dependent effect. Because state-dependency occurs in varying degrees and is not an "all or none" phenomenon groups' performances were compared to the following criteria in order to determine the presence or absence of a state-dependent effect:

1. A group demonstrates a state-dependent effect is its performance is not significantly above the random (50%) level of responding on Testing Days 1, 3, and 5, and is significantly above the random level on Testing Days 2 and 4.

2. A group demonstrates a state-dependent effect if its performance is significantly below the control group's performance on Testing Days

1, 3, and 5, not significantly below the control group's performance on Testing Days 2 and 4, and above the random level of responding on Testing Days 2 and 4.

3. A group demonstrates a state-dependent effect if its median latencies are significantly longer than those of the control group on Testing Days 1, 3, and 5, and not significantly longer than those of the control group on Testing Days 2 and 4.

The performance of each of the four experimental groups was matched to each of the first two criteria on all response measures except median latencies. The performance of each of the four experimental groups was matched to the third criterion only, for the median latency measure. If a group met either of the first two criteria on any of the response measures other than median latencies, or met the third criterion in the case of median latencies, the group was considered to have demonstrated a state-dependent effect on the response measure in question. In accordance with a normal binomial distribution a group had to meet the requirements of at least one of the criteria on all of the 5 days of testing in order for the state-dependent effect to be significant at the .05 level.

Criteria for a transfer (disruption) effect attributable to the tone. In order for the disruption of a state-dependent effect to be attributed to the tone all of the following criteria had to be met except on the median latency measure. On the median latency measure only the first two criteria had to be met:

1. A transfer experimental group must perform significantly better on Testing Days 1, 3, and 5, than a SDL experimental group which was trained in the same D state as the transfer experimental group.

2. The SDL group, to which the transfer experimental group is compared, must demonstrate a state-dependent effect according to the established criteria.

3. The transfer experimental group must perform above the random level of responding on Days 1, 3, and 5 on the response measure in question with the exception of the median latency measure.

Statistical Treatment

An analysis of variance (ANOVA) for a two-way classification (Bruning & Kintz, 1968) was used to compare differences in the number of trials required to reach the training criterion (see Appendix F, Tables 2-46 for ANOVA summary tables; Tables 47-52 for levels of significance for ANOVAs). The two factors in this analysis were the D states (D vs. ND) and the tone conditions (tone vs. no tone).

An ANOVA for a three-factor experiment with repeated measures on one factor (Winer, 1971) was applied to data collected on each of the four response measures. Each of the four response measures was analyzed for training and testing separately. The two factors imposed during both training and testing were the D states and the tone conditions. The third factor was D state change vs. no change (ND-D-ND-D-ND and D-ND-D-ND-D vs. ND-ND-ND-ND-ND and D-D-D-D-D) over the 5 days of testing. Training data were analyzed for the first day and the last 4 days of training only. Four rats (no. 50, 59, 81, and 85) reached the training criterion in only 4 days. "Projected" scores and percents were assigned to the fifth day of training and are the same as those made on the fourth (last) day of training by each of those four rats.

Because sampling error was found between groups having identical treatments during training, and since there was no difference in the training of each experimental group and its control group, data from each experimental group were combined with data from the corresponding control group (e.g., transfer-D experimental data were combined with tone-D control data) so that the eight groups could be treated as four (trained D-no tone, ND-no tone, D-tone, and ND-tone; N=16 rats per group) for the purpose of analysis. The ANOVA (Winer, 1971) was applied to these combined data for training only in order to guage the degree of sampling error which contributed to significant differences shown.

Sub-ANOVAs (Winer, 1971) were used to make the following comparisons on each response measure for both training and testing: SDL experimentals vs. transfer experimentals, SDL controls vs. tone controls, SDL experimentals vs. SDL controls, transfer experimentals vs. tone controls.

The Mann-Whitney U test (U test) converted to a z score formula and corrected for tied ranks (Siegel, 1956) was used to make the following comparisons on each response measure for each day of training and testing: SDL-D experimentals vs. transfer-D experimentals, SDL-ND experimentals vs. transfer-ND experimentals, SDL-D controls vs. tone-D controls, SDL-ND controls vs. tone-ND controls, SDL-D experimentals vs. SDL-D controls, SDL-ND experimentals vs. SDL-ND controls, transfer-D experimentals vs. tone-D controls, transfer-ND experimentals vs. tone-ND controls (see Appendix B, Tables 53-60 for z scores and levels of significance for U tests). All U tests applied to training data were two-tailed. U tests applied to testing data for experimental vs. control and experimental vs. experimental comparisons were one-tailed; control vs. control comparisons were two-tailed. The U test was used for these com-

parisons because it is suitable for small independent samples, is more sensitive than the sign test (Van Dalen, 1962, p. 338), and does not require matched pairs.

The performance of each of the eight groups was compared to the 50% (random) level of responding on each day of testing. This comparison was made on three of the responses measures excluding median latencies. In order to make this comparison ǂ scores (one-tailed, Bruning & Kingz, 1968) were derived for the differences between: each group's daily total number of correct responses and 12 (a group's mean number of correct responses for 24 trials when 50% are correct) on the number correct measure; each group's daily total number of first trial correct responses and 4 (a group's mean number of correct responses for 8 trials when 50% are correct) on the number of first trial correct measure. On the proportion correct measure z scores (one-tailed) were derived for differences between each group's daily proportion of correct responses and 50% by using a formula for the comparison of two proportions (Downie, & Starry, 1977; see Tables 61-63 for z scores and levels of significance for comparisons to the random level of responding).

The t-test (Bruning & Kintz, 1968) was used to compare all D to ND trained rats for differences in weight decrements occurring between the first and second days of training. The U test (two-tailed; Siegel, 1956) was used to make the same comparison for each squad taken separately. The U test was also used to compare the two D trained experimental groups combined to the two ND trained experimental groups combined for differences in weight decrements occurring between the first and second days of testing.

Training

The ANOVA of the number of trials required to reach the training criterion shows that there were no significant differences caused by the D states, the tone conditions, or interaction (range = 20 - 60 trials).

Number of correct responses. The overall ANOVA of the number of correct responses shows a significant tone condition-D state interaction (for the 5 days combined), $F(3, 56) = 3.83$, $p < .025$, and a significant tone condition-D state-days (across days of training) interaction, $F(12, 224) = 2.75$, $p < .001$.

SDL-D experimentals performed significantly more correct responses than SDL-D controls on the first day of training, $z(8, 8) = 3.01$, $p < .005$, and SDL-ND controls performed significantly more correct responses than SDL-ND experimentals on the next to the last day of training, $z(8, 8) = 2.61$, $p < .01$ (see Figure 1, Appendix A). Since, during training, there were no differences in treatments between SDL-D experimentals and SDL-D controls or between SDL-ND experimentals and SDL-ND controls these differences shown by U tests are a result of sampling error.

The ANOVA between the four groups formed by combining experimental and control groups according to treatment during training (combined groups ANOVA) shows no significant interaction effects on the number correct measure. Therefore much or all of the tone condition-D state interaction and the tone condition-D state-days interaction shown in the overall ANOVA is due to sampling error. The same is true of each of the significant differences, except the days effect, shown in the four sub-ANOVAs.

The overall ANOVA, the combined groups ANOVA, and each of the four sub-ANOVAs (SDL experimentals vs. SDL controls; transfer experimentals

vs. tone controls; SDL experimentals vs. transfer experimentals; SDL controsl vs. tone controls) show a significant days effect which is a result of learning, F (4, 224) = 306.83, p<.001 for the overall ANOVA; F (4, 240) = 258.46, p<.001 for the combined groups ANOVA; F (4, 112) = 57.40, 66.66, 120.68, and 198.19 respectively for the sub-ANOVAs, p<.001 for each sub-ANOVA.

Proportion of correct responses. The overall ANOVA of the proportion of correct responses shows a significant tone condition-D state interaction, F (3, 56) = 3.41, p<.025, and a significant tone condition-D state-days interaction, F (12, 224) = 2.64, p<.005.

SDL-D experimentals performed a significantly higher proportion of correct responses than SDL-D controls on the first day of training, z (8, 8) = 2.56, p<.025, and SDL-ND controls performed a significantly higher proportion of correct responses than SDL-ND experimentals on the next to the last day of training z (8, 8) = 2.61, p<.01 (see Figure 2, Appendix A). Thus sampling error, between the same groups and on the same days as on the number correct measure, resulted in significant differences on the proportion correct measure. Since the combined groups ANOVA shows no significant interaction, the interaction shown in the overall ANOVA and all significant differences, except the days effect, shown in the four sub-ANOVAs, are largely, or totally due to sampling error.

The overall ANOVA, the combined groups ANOVA, and each of the four sub-ANOVAs (SDL experimentals vs. SDL controls; transfer experimentals vs. tone controls; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls) show a significant days effect which is a result of learning, F (4, 224) = 166.76, p<.001 for the overall ANOVA; F (4, 240) = 152.90, p<.001 for the combined groups ANOVA; F (4, 112) = 30.26, 36.38, 72.88, and 95.01 respectively for the sub-ANOVAs, p<.001 for each sub-ANOVA.

Number of first trial correct responses. The only significant differences shown in the overall ANOVA of the number of first trial correct responses and the combined groups ANOVA is the days effect, $F(4, 224) = 60.06$, $p<.001$; $F(4, 240) = 61.50$, $p<.001$ respectively, which is also significant in each of the four sub-ANOVAs (SDL experimentals vs. SDL controls; transfer experimentals vs. tone controls; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls), $F(4, 12) = 20.71$, 10.86, 23.43, and 38.69 respectively, $p<.001$ for each sub-ANOVA (see Figure 3, Appendix A). All significant differences, except the days effect, shown in the sub-ANOVAs are largely, or totally, due to sampling error since these differences are not shown in the overall ANOVA or the combined groups ANOVA.

Median latencies. The overall ANOVA of median latencies shows a significant tone condition-D state interaction, $F(3, 56) = 4.74$, $p<.01$, and a significant tone condition-D state-days interaction, $F(12, 224) = 2.49$, $p<.005$.

SDL-D experimentals had significantly lower median latencies than SDL-D controls on the first day of training, $z(8, 8) = 2.90$, $p<.005$, and transfer-ND experimentals had significantly lower median latencies than tone-ND controls on the next to the last day of training, $z(8, 8) = 2.37$, $p<.025$ (see Figure 4, Appendix A). Again these differences shown by U tests are due to sampling error since, during training, there are no differences in treatments between SDL-D experimentals and SDL-D controls or between transfer-ND experimentals and tone-ND controls.

Since the combined groups ANOVA shows no significant interaction on this response measure, the tone condition-D state interaction and the tone condition-D state-days interaction shown in the overall ANOVA and all

significant differences, except the days effect, shown in the four sub-ANOVAs, are greatly affected by sampling error.

The overall ANOVA shows that ND trained rats had significantly lower median latencies than D trained rats over the 5 days analyzed, $F(3, 56) = 3.75$, $p<.025$. The combined groups ANOVA also shows that this effect is significant, $F(1, 60) = 7.31$, $p<.01$. These differences are largely due to the depressing effect of Librium.

The overall ANOVA, the combined groups ANOVA, and each of the four sub-ANOVAs (SDL experimentals vs. SDL controls; transfer experimentals vs. tone controls; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls) show a significant days effect which is a result of learning, $F(4, 224) = 261.76$, $p<.001$ for the overall ANOVA; $F(4, 240) = 239.66$, $p<.001$ for the combined groups ANOVA; $F(4, 112) = 63.42$, 51.60, 95.23, and 181.20 respectively for the sub-ANOVAs, $p<.001$ for each sub-ANOVA.

Testing

The number of correct responses. The overall ANOVA of the number of correct responses shows that variations in D treatment combinations resulted in significant differences in performance over the 5 days of testing $F(3, 56), = 9.29$, $p<.001$, and that there was a significant D-treatment combination-days interaction, $F(12, 224) = 9.37$, $p<.001$.

The sub-ANOVA between SDL experimentals and SDL controls shows a significant experimentals vs. controls-days interaction, $F(4, 112) = 5.24$, $p<.001$, and a significant experimentals vs. controls-D treatment combination-days interaction, $F(4, 112) = 9.83$, $p<.001$ (see Figures 1 & 5, Appendix A).

SDL-ND experimentals performed significantly fewer correct responses than SDL-ND controls on Day 1, z (8, 8) = 3.28, $p<.001$, Day 3, z (8, 8) = 2.58, $p<.005$, and Day 5, z (8, 8) = 2.72, $p<.005$, and more correct responses than controls on Days 2 and 4. SDL-D experimentals performed significantly fewer correct responses than SDL-D controls on Day 1, z (8, 8) = 2.92, $p<.005$, Day 3, z (8, 8) = 2.48, $p<.025$, and Day 5, z (8, 8) = 2.24, $p<.025$, not significantly fewer correct responses than controls on Day 2, and more correct responses than controls on Day 4. Neither SDL-ND nor SDL-D experimentals performed significantly above the random level on Days 1, 3, or 5. Both SDL-ND and SDL-D experimentals performed significantly above the random level on Day 2, $z = 3.06$, $p<.005$; $z = 2.24$, $p<.025$ respectively, and Day 4, $z = 3.88$, $p<.001$; $z = 3.06$, $p<.005$ respectively (see Figure 5, Appendix A).

These results indicate that both SDL-ND and SDL-D experimentals were state-dependent on this measure; however, a comparison of the number of correct responses to the proportion of correct responses (compare Figures 5 & 6, Appendix A) indicated that Librium may have had a strong depressing effect on responding in the SDL-ND experimental group on Days 1, 3, and 5. This effect may be more clearly seen by comparing the number of correct responses to the number of turns (The number of turns equal the number of correct plus incorrect responses minus the number of "No choice" trials; compare Figures 5 & 8, Appendix A).

The sub-ANOVA between transfer experimentals and tone controls shows a significant experimentals vs. controls-D treatment combination-days interaction, F (4, 112) = 8.23, $p<.001$.

Transfer-ND experimentals performed significantly fewer correct responses than tone-ND controls on Day 1, z (8, 8) = 3.19, $p<.001$, not

significantly fewer correct responses than controls on Days 3 and 5, and more correct responses than controls on Days 2 and 4 (see Figure 5, Appendix A). Transfer ND experimentals were significantly above the random level of responding on Day 3, $z = 1.84$, $p < .05$, but not on Days 1 and 5. Therefore it can be stated that there was transfer across D-states in the transfer-ND experimental group on Day 3 provided that SDL-ND experimentals were state-dependent on this response measure.

Transfer-D experimentals performed significantly fewer correct responses than tone-D controls on Day 1, $z\ (8, 8) = 2.18$, $p < .025$, exactly the same number of correct responses as controls on Day 3, not significantly fewer correct responses than controls on Day 5, and more correct responses than controls on Days 2 and 4 (see Figure 5, Appendix A). Transfer-D experimentals did not, however, perform significantly above the random level on Days 1, 3, or 5 and did perform significantly above the random level on Day 2, $z = 3.06$, $p < .005$, and Day 4, $z = 3.06$, $p < .005$. Therefore transfer-D experimentals were state-dependent on this response measure throughout testing, even though the group did not perform significantly fewer correct responses than controls on Days 3 and 5.

The proportion of correct responses. The overall ANOVA of the proportion of correct responses shows a significant D treatment combinations effect over the 5 days of testing, $F\ (3, 56) = 5.49$, $p < .005$, and a significant D-treatment combination-days interaction, $F\ (12, 224) = 4.52$, $p < .001$.

The sub-ANOVA between SDL experimentals and SDL controls shows a significant experimentals vs. controls-D treatment combination-days interaction, $F\ (4, 112) = 5.60$, $p < .001$ (see Figures 2 & 6, Appendix A).

SDL-ND experimentals performed a higher proportion of correct responses than SDL-ND controls on Days 1, 2, and 4, a significantly lower proportion than controls on Day 3, z (8, 8) = 1.86, $p<.05$, and a not significantly lower proportion than controls on Day 5. However, SDL-ND experimentals performed significantly above the random level on Day 1, $z = 2.15$, $p<.025$, and Day 5, $z = 1.78$, $p<.05$. Therefore SDL-ND experimentals were not state-dependent on this response measure.

SDL-D experimentals performed a significantly lower proportion of correct responses than SDL-D controls on Day 1, z (8, 8) = 2.60, $p<.005$, and Day 3, z (8, 8) = 3.19, $p<.001$, a not significantly lower proportion than controls on Days 2 and 5, and a higher proportion than controls on Day 4 (see Figure 6, Appendix A). SDL-D experimentals performed significantly above the random level on Day 2, $z = 2.45$, $p<.01$, and Day 4, $z = 3.85$, $p<.001$, but not on Days 1, 3, or 5. Therefore SDL-D experimental were state-dependent on this response measure.

The sub-ANOVA between transfer experimentals and tone controls shows a significant experimentals vs. controls-D treatment combination-days interaction, F (4, 112) = 4.99, $p<.005$.

Transfer ND experimentals performed a significantly lower proportion of correct responses than tone-ND controls on Day 1, z (8, 8) = 2.26, $p<.025$, a not significantly lower proportion than controls on Day 5, and a higher proportion than controls on Days 2, 3, and 4 (see Figure 6, Appendix A). Transfer-ND experimentals performed above the random level on Day 2, $z = 3.72$, $p<.001$, Day 3, $z = 3.40$, $p<.001$, Day 4, $z = 4.12$, $p<.001$, and Day 5, $z = 1.79$, $p<.05$, but not on Day 1. Since, on Day 3, transfer-Nd experimentals performed a significantly higher proportion of correct responses than SDL-ND experimentals, z (8, 8) = 1.87, $p<.05$, the

tone may have improved performance on that day. However, state dependency was not disrupted by the tone on this response measure since SDL-ND experimentals were not state-dependent on this measure.

Transfer-D experimentals performed a significantly lower proportion of correct responses than tone-D controls on Day 1, z (8, 8) = 2.71, $p<.005$, a not significantly lower proportion than controls on Days 3 and 5, and a higher proportion than controls on Days 2 and 4 (see Figure 6, Appendix A). Transfer-D experimentals performed significantly above the random level on Day 2, $z = 3.23$, $p<.001$, and Day 4, $z = 3.23$, $p<.001$, but not on Days 1, 3, or 5. Therefore transfer-D experimentals were state-dependent on this response measure.

The number of first trial correct responses. The overall ANOVA of the number first trial correct responses shows a significant D-treatment combinations effect over the 5 days of testing, F (3, 56) = 8.08, $p<.001$, and a significant D-treatment combination-days interaction, F (12, 224) = 2.56, $p<.005$.

The sub-ANOVA between SDL experimentals and SDL controls shows a significant experimentals vs. controls-D treatment combination-days interaction, F (4, 112) = 5.31, $p<.001$ (see Figures 3 & 7, Appendix A).

SDL-ND experimentals performed significantly fewer first trial correct responses than SDL-ND controls on Day 1, z (8, 8) = 2.24, Day 3, z (8, 8) = 1.86, $p<.05$, and Day 5, z (8, 8) = 1.86, $p<.05$ (see Figure 7, Appendix A) and more first trial correct than controls on Days 2 and 4. SDL-ND experimentals performed significantly above the random level on Day 2, $z = 2.48$, $p<.01$, and Day 4, $z = 2.48$, $p<.01$, but not on Days 1, 3, or 5. These results indicate that SDL-ND experimentals were state-dependent, however, this group was not state-dependent on the proportion

correct measure and may have met the criteria for state-dependency on this measure only because of the depressing effect of Librium.

SDL-D experimentals performed significantly fewer first trial correct responses than controls on Day 3, $z\ (8, 8) = 3.0$, $p<.005$, and Day 5, $z\ (8, 8) = 2.0$, $p<.025$, not significantly fewer first trial correct than controls on Day 1, more first trial correct than controls on Day 2, and the same number of first trial correct as controls on Day 4 (see Figure 7, Appendix A). SDL-D experimentals performed significantly above the random level on Day 2, $z = 1.77$, $p<.05$, and Day 4, $z = 1.77$, $p<.05$, but not on Days 1, 3, or 5. Therefore SDL-D experimentals were state-dependent on this response measure even though the experimentals did not perform significantly below controls on Day 1.

The sub-ANOVA between transfer experimentals and tone controls shows no significant interaction. However, some significant differences shown by U tests are not detected by the sub-ANOVA of the four groups.

Transfer-ND experimentals performed significantly fewer first trial correct responses than tone-ND controls on Day 1, $z\ (8, 8) = 2.24$, $p<.025$, and Day 3, $z\ (8, 8) = 1.86$, $p<.05$, not significantly fewer first trial correct than controls on Days 2 and 5, and the same number of first trial correct as controls on Day 4 (see Figure 7, Appendix A). Transfer-ND experimentals performed significantly above random level on Day 2, $z = 1.77$, $p<.05$, and Day 4, $z = 1.77$, $p<.05$, but not on Days 1, 3, or 5. Therefore transfer-ND experimentals met a criterion for a state-dependent effect on this response measure even though experimentals did not perform significantly fewer first trial correct responses than controls on Day 5.

Transfer-D experimentals performed not significantly fewer first trial correct responses than tone-ND controls on Days 1, 2, and 5, the same num-

ber of first trial correct as controls on Day 3, and more first trial correct than controls on Day 4 (see Figure 7, Appendix A). However, transfer-D experimentals performed significantly above the random level on Day 4 only, $z = 1.06$, $p < .01$. Therefore transfer-D experimentals did not show a state-dependent effect on this response measure; nor did the group show transfer attributable to the tone.

Median Latencies. The overall ANOVA of median latencies shows a significant D-treatment combinations effect over the 5 days of testing, $F(3, 56) = 11.44$, $p < .001$, and a significant D-treatment combination-days interation, $F(12, 224) = 8.53$, $p < .001$.

The sub-ANOVA between SDL-experimentals and SDL controls shows a significant experimentals vs. controls-days interaction, $F(4, 112) = 5.33$, $p < .001$, and a significant experimentals vs. controls-D treatment combination-days interaction, $F(4, 112) = 3.77$, $p < .01$ (see Figures 4 & 9, Appendix A).

SDL-ND experimentals had significantly longer median latencies than SDL-ND controls on Day 1, $z(8, 8) = 3.51$, $p < .001$, Day 3, $z(8, 8) = 2.52$, $p < .01$, and Day 5, $z(8, 8) = 2.92$, $p < .005$, and shorter median latencies than controls on Days 2 and 4 (see Figure 9, Appendix A). Therefore SDL-ND experimentals met the criterion for a state-dependent effect on this response measure; however, the state-dependent effect shown may be partially, or totally, due to the depressing effect of Librium.

SDL-D experimentals had significantly longer median latencies than SDL-D controls on Day 5, $z(8, 8) = 1.79$, $p < .05$, not significantly longer median latencies than controls on Days 1 and 4, shorter median latencies than controls on Day 2, and the same length of median latencies as controls on Day 3 (see Figure 9, Appendix A). Therefore SDL-D experimentals

were not state-dependent on this response measure; the significant experimentals vs. controls-days interaction shown in the sub-ANOVA is mostly a result of relatively large differences between SDL-ND experimentals and SDL-ND controls on Days 1 and 5.

The sub-ANOVA between transfer experimentals and tone controls shows a significant experimentals vs. controls-D treatment combination-days interaction, $F(4, 112) = 3.99$, $p<.005$.

Transfer-ND experimentals had significantly longer median latencies than tone-ND controls on Day 1, $z(8, 8) = 3.26$, $p<.001$, Day 3, $z(8, 8) = 3.05$, $p<.005$, and Day 5, $z(8, 8) = 3.26$, $p<.001$, and shorter median latencies than controls on Days 2 and 4 (see Figure 9, Appendix A). Therefore transfer-ND experimentals met the criterion for a state-dependent effect on this response measure. Transfer-ND experimentals had significantly shorter median latencies than SDL-ND experimentals on Day 1, $z(8, 8) = 2.08$, $p<.025$. Therefore there may have been some degree of transfer in the transfer-ND experimental group on Day 1 provided that there was some degree of state-dependency in the SDL-ND experimental group, rather than only the depressing effect of Librium.

SDL-D experimentals had not significantly longer latencies than controls on Day 1 and shorter latencies than controls on Days 2, 3, 4, and 5. Therefore SDL-D experimentals did not demonstrate a state-dependent effect on this response measure; nor did the group show transfer attributable to the tone since SDL-D experimentals did not demonstrate a state-dependent effect on this measure either.

State-dependent effects compared to those found in a foot-shock experiment. The degree of state-dependency found in the present experiment was compared to that found in the Connelly et al. (1975) experiment on the

number correct measure only. The number of correct responses made on Testing Days 1, 3, and 5 was totaled for each SDL experimental group in each of the two experiments. The total score of each of the four groups was then converted to a percent of the number of correct responses possible for the three days combined. These percents were smaller for both ND and D trained rats in the present experiment (38.89% and 43.06% respectively) than for the corresponding groups in the Connelly et al. (1975) experiment (56.67% and 57.50% respectively). When the percent correct was derived by dividing each experimental group's number of correct responses by the number of correct responses performed by its control group the direction of the differences was the same; the percents were smaller for both ND and D trained rats in the present experiment (46.90% and 54.39% respectively) than for the corresponding groups in the Connelly et al. (1975) experiment (56.67% and 58.47% respectively). Therefore, in contradiction to one hypothesis, the state-dependent effect shown in the present experiment was not less than that shown in the Connelly et al. (1975) experiment. However, the reader is reminded that the state-dependent effect, shown in the SDL-ND experimental group of the present study, may be partially, or totally, a result of the depressing effect of Librium on responding.

Drug effect on weight. The t-test shows that Librium injections resulted in no significant differences in weight decrement between the day of the first injection (i.e., the first day of training) and the day following. However, U tests show that weight decrements between the two days were significantly greater for ND trained rats than for D trained rats in Squad 3, $U\ (7, 7) = 3.50$, $p < .005$, and Squad 4, $U\ (7, 7) = 3.00$, $p < .005$. The U test shows that the two D trained experimental groups

combined had significant larger weight decrements than the two ND trained experimental groups combined between the first testing day (first day in the D state opposite the training D state) and the second, U (16, 16) = 40, $p < .002$.

CHAPTER IV

DISCUSSION

The results support only one of the three hpyotheses. The first hypothesis predicted that the transfer experimental groups would demonstrate disruption of state-dependency attributable to the tone. The second hypothesis predicted that the transfer experimental groups would demonstrate less disruption than the transfer experimental groups of the Connelly et al. (1975) experiment. The first hypothesis is not supported for the same reason that the second hypothesis is confirmed; that is, no transfer across D-states is demonstrated in this experiment which could be attributed to the tone. The third hypothesis predicted that the degree of state-dependency demonstrated by the SDL experimental groups would be less than that demonstrated by the SDL experimental groups of the Connelly et al. (1975) experiment. The third hypothesis is not supported by the results for either SDL-ND or SDL-D experimental groups.

Absence of a transfer effect. When the tone acts as an EP its power to disrupt state-dependency may be in direct proportion to the intensity of the emotion it is paired with. The tone may have failed to result in a transfer effect because the feeding of deprived rats does not generate emotion of sufficient intensity to overcome the D state barrier; even when that emotion is channeled into behavior by the tone. The intensity of emotion paired with the tone might have been low if the quantity

of food received in the home cages caused rats to perceive feeding in the maze as a relatively unimportant event.

The tone may not have caused a transfer effect because of a weak association with feeding resulting from long irregular CS-UCS intervals. Early in training, while attending to novel stimuli may have been maximal, the interval between the on set of tone and feeding often exceeded 30 seconds. In an escape learning task, such as that used in the Connelly et al. (1975) experiment, the tone and foot shock can be paired in close temporal contiguity consistently throughout training.

The tone may be somewhat aversive to rats prior to any training. If this is true then rats may more readily associate the tone with a powerful aversive UCS, such as foot-shock, than with feeding.

The administration of large doses of Librium may have resulted in a diminished ability to hear the tone. If this is true then D trained rats may have been unable to associate the tone with feeding during training. Consequently the tone would not prompt memory for those rats during testing. ND trained rats that made an association between the tone and feeding would not be able to hear the tone, or hear it as well, while in the D state on Testing Days 1, 3, and 5. Consequently the tone would not act as an EMP for those rats either.

There is no apparent diminishing effect on hearing caused by Librium in the Connelly et al. (1975) experiment; nor is there any weakening effect due to food deprivation in that experiment since deprivation was not used. A poor physical condition may have resulted from food deprivation in the present experiment and may have decreased hearing in drugged rats. However, if a poor physical condition did not contribute to a hearing loss in drugged rats, then attributing the absence of a transfer effect to

D effects on hearing may be just another way of saying that emotion is not as intense in the present experiment as in the Connelly et al. (1975) experiment.

Some degree of extinction may have occurred in the transfer experimental groups due to non reward testing trials. Therefore the tone may have prompted memory across D states even though no disruption effect, attributable to the tone, is shown on any of the response measures according to the established criteria. Note that in order for disruption to be attributed to the tone all of the criteria listed had to be met except on the median latencies measure; on that measure both of the first two criteria had to be met.

State-dependency. The SDL experimental groups of the present study did not demonstrate less state-dependency than the SDL experimental groups of the Connelly et al. (1975) experiment. Therefore no support is given to my contention that extreme fear contributes to a more pronounced state-dependent effect in escape learning tasks with foot shock (see predicted trends, introduction section). The important implication in regard to the mechanism of the tone as an EMP is this; the EMP which disrupts state-dependency in a foot shock experiment is disrupting a physiological state resulting from D effects rather than a predominantly "emotional" state.

It is important to remember that the state-dependent effect shown by the SDL-ND experimental group may have been partially, or totally, a result of the depressing effects of Librium on responding. SDL-ND experimentals were not state-dependent on the proportion correct measure. It may be that the group was not state-dependent and performed correct responses to the extent that D depression would allow, or it may be that these ND trained rats were even more state-dependent than SDL-D experimentals and

consequently had more "No choice" trials. If there was state-dependency in the ND trained group, the rats may have not only forgotten which direction to run for food reward, but also that there was food reward. All rats given D injections were mobile, according to the criterion described in the method section, before trials were given.

Sampling error. Sampling error was found in training on as late as the next to the last training day. Since there was sampling error between groups which were not significantly different in performance until the next to the last training day, previous equality in performance does not assure the absence of sampling error. Therefore it is possible that unknown factors, which resulted in sampling error during training, may have also resulted in differences during testing; even though there was no sampling error shown by U tests on the last training day. There are no means of detecting sampling error in testing which are comparable to those used for training since no two groups experienced identical treatments during testing.

Drug effect on weight, deprivation, and responding. During the training and testing of the first squad it appeared that Librium caused decreases in the weight decrements of deprived rats. Some D trained rats experience large (up to 24 gm) overnight weight decrements following a sterile-water injection on the first day of testing. Some ND trained rats showed unexpected overnight weight gains (up to 18 gm) after a single Librium injection on the first day of testing following several consecutive days of weight decrement. I considered these observations to be an indication that Librium administration resulted in fluid retention which added to rats' weight. The significant difference in weight decrements shown between all ND and all D trained experimental animals support this

contention. Since there seemed to be no readily available control for this factor feeding was regulated subjectively. The problems posed were: keeping rats at or near the running weight; keeping hunger as constant as possible across groups; keeping rats alive and healthy; avoiding over feeding and a resulting decline in correct responding. In general the quantity of food consumed in the home cages was increased during training and continued to increase throughout testing. The large proportion of deaths occurring in the D trained groups may have been a result of administering Librium to rats that were equally as weak from food deprivation as those rats in the ND trained groups; or, the death rate may have been higher in the D trained groups because Librium injections resulted in rats being too severely deprived as a result of a "false weight".

Extinction and reinforcement effects. During testing both SDL-ND and tone-ND controls showed a decrease in performance following Day 1, an increase following Day 2, a decrease following Day 3, and another increase following Day 4. This trend occurred on both the number correct and proportion correct measures (see Figures 5 & 6, Appendix A). This trend may have been caused by an extinction effect following non reward trials on Days 1 and 3 and reinforcement as a result of reminder trials on Days 2 and 4.

A similar trend can be seen in the SDL-D control group on the proportion correct measure over the first 4 days of testing, but not on Day 5. The number of turns made by the SDL-D control group decreased following Day 2, increased following Day 3, and increased again following Day 4. If over feeding (relative to previous feedings) in the home cages resulted in more turns in the direction of the position preference, and if a poor physical condition resulted in a smaller number of turns made,

then the SDL-D control group's performance might indicate an extinction-reinforcement trend combined with the effects of: a weakened physical condition on Day 3; a better physical condition on Day 4 (possibly resulting from increased food intake); over feeding, appearing on Day 5 (compare Figures 6 & 8, Appendix A).

The tone-D control group may not show a trend similar to ND controls due to an over feeding effect occurring on Day 3. The number of turns made by this group was only one less on Days 3 and 4 than on Days 1 and 2; yet there is a slump in the number of correct responses performed on Day 3 (compare Figures 5 & 8, Appendix A). If the group's responding on the proportion correct and number correct measured had been elevated on Day 3, the group would show the same trend as the ND control groups.

Groups demonstrating a state-dependent effect would not be expected to show an extinction-reinforcement trend like the ND controls for two reasons; non rewarded correct responses would not be remembered in the opposite D state if rats were state-dependent; less extinction would result, if there was some degree of transfer, due to a smaller number of non rewarded correct responses. The latter would also be true of ND trained rats that were not state-dependent but were suppressed by the D on Testing Days 1, 3, and 5.

Transfer-ND and transfer-D experimental groups show a trend on the number correct measure which does not indicate an extinction effect following non reward trials on Testing Days 1 and 3. Both of these groups show a decrease in correct responding following reminder trials on Days 2 and 4 (see Figure 5, Appendix A). These trends are like those of the SDL experimental groups. A similar trend is shown by the transfer-D experimental group on the proportion correct measure. The transfer-ND experimental group

does not show a trend similar to the SDL experimentals on the proportion correct measure due to a relatively high level of responding on Day 3 (see Figure 6, Appendix A). Here it is important to remember that SDL-ND experimentals did not demonstrate a state-dependent effect on the proportion correct measure.

I stated earlier that the transfer experimental groups may have failed to demonstrate a transfer effect because of extinction on Days 1, 3, and 5. However, it may be that these groups experienced little, or no, extinction as result of non reward test trials because the memory of non rewarded trials was state-dependent. Note that on the number correct measure and the proportion correct measure the performances of the transfer-D experimental groups were not lower on Day 4 than on Day 2. The same is true of the transfer-ND experimental group (see Figures 5 & 6, Appendix A). Since performance was not lower on Day 4 than on Day 2 in either of these two groups a cumulative extinction effect, resulting from nine out of twelve trials not being reinforced prior to Day 4, is not indicated. The relatively high levels of performance demonstrated on Day 4 may be the result of the memory of reinforced reminder trials on Day 2 and the absence of the memory of non rewarded trials on Days 3 and 4.

The trends and causes discussed here are speculation only, but do offer some explanations of differences between group's performances. Because of the possible influence of the confounding variables discussed the ability of the tone to act as an EMP in this experiment cannot be determined.

Suggestions for a food reward experiment. The intensity of emotion associated with the tone might be increased in a food reward experiment if rats were forced to rely entirely on a food supply made available only in the presence of the tone. Decreasing the CS-UCS interval might result in

a stronger association between the tone and feeding. Turning the tone off and then immediately removing the animal from an abundant food supply might make the tone more important since termination of the tone would signal the end of feeding. More reminder trials might prevent extinction.

A D dosage of less than 45 mg/kg might be sufficient to result in state-dependency in a food reward experiment and would probably result in a decrease of the D effect on weight. Rats could be trained in the ND state and at the same time be made tolerant of the depressing effects of the D by daily D injections following training trials.

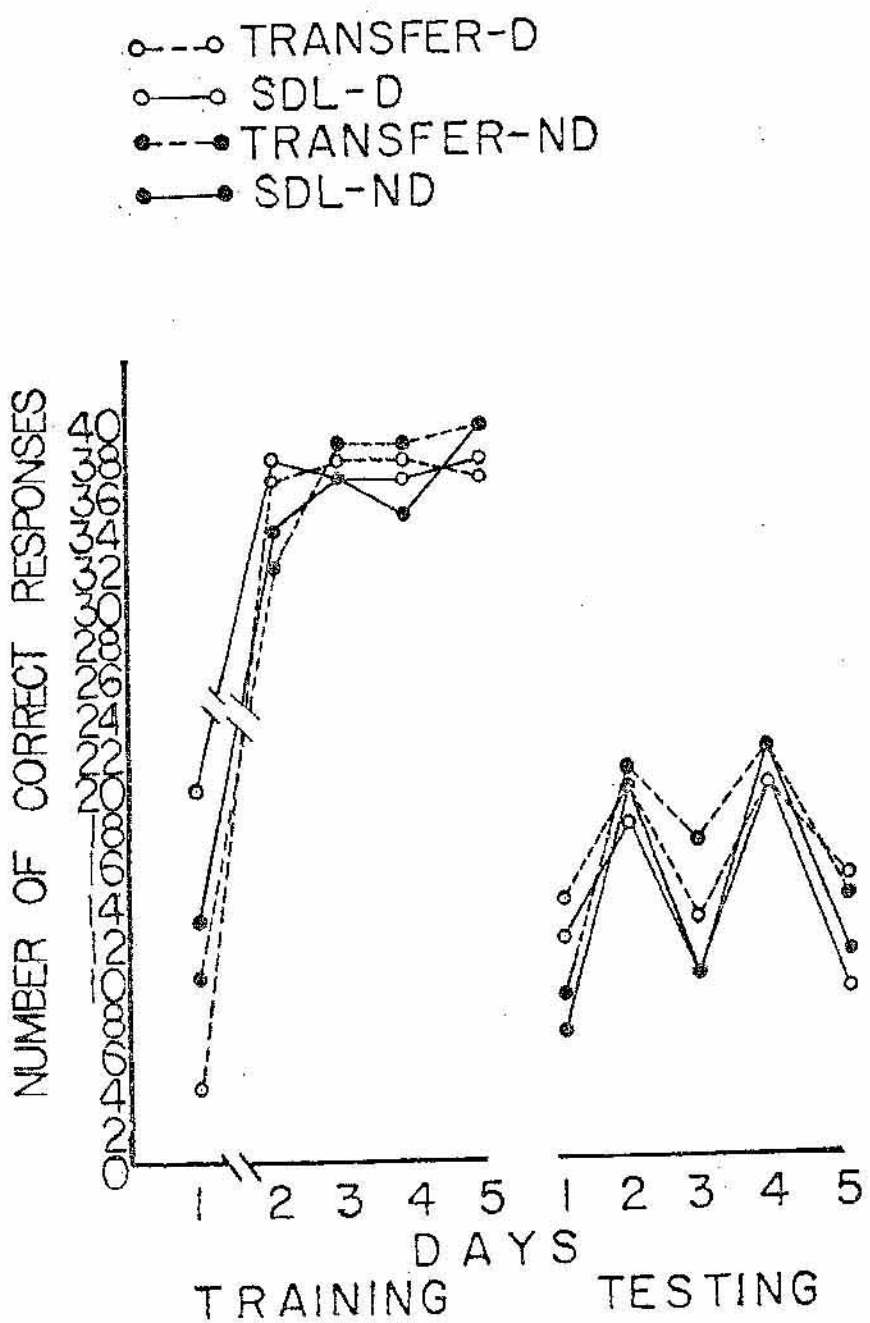
In the present experiment squads were lowered to 80% of the free feeding weight within 5 to 7 days. Extending the period between the beginning of deprivation and the time the rat reaches the running weight might result in a better physical condition of the animal. Extending the period might also prevent a decrease in motivation resulting from over feeding in an attempt to keep the animal from dropping below the running weight. Scheduling the daily quantity of food to be given to rats as a percent of the free feeding weight would prevent any D effect on weight from influencing feeding; however, the percentages required might be different for D than for ND trained rats and would need to be determined in a pilot study.

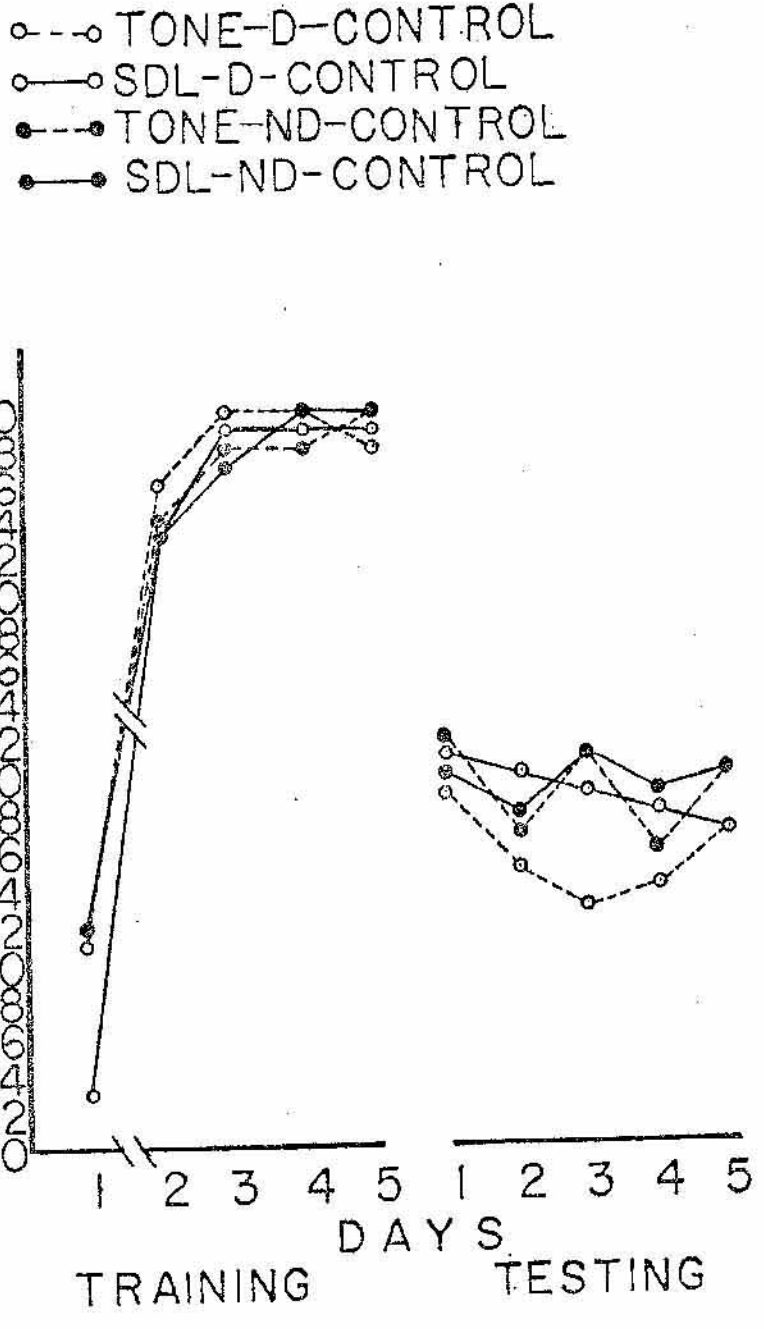
Further attempts to disrupt state-dependency, with positively reinforced conditioned stimuli, might add a new dimension to the effective use and control of "emotional energy" by conditioned stimuli; at the same time some new light might be shed on the physiology of emotion. Therefore an attempt to modify the present design might be justified and further investigation of the variables manipulated here are warranted.

APPENDICES

APPENDIX A

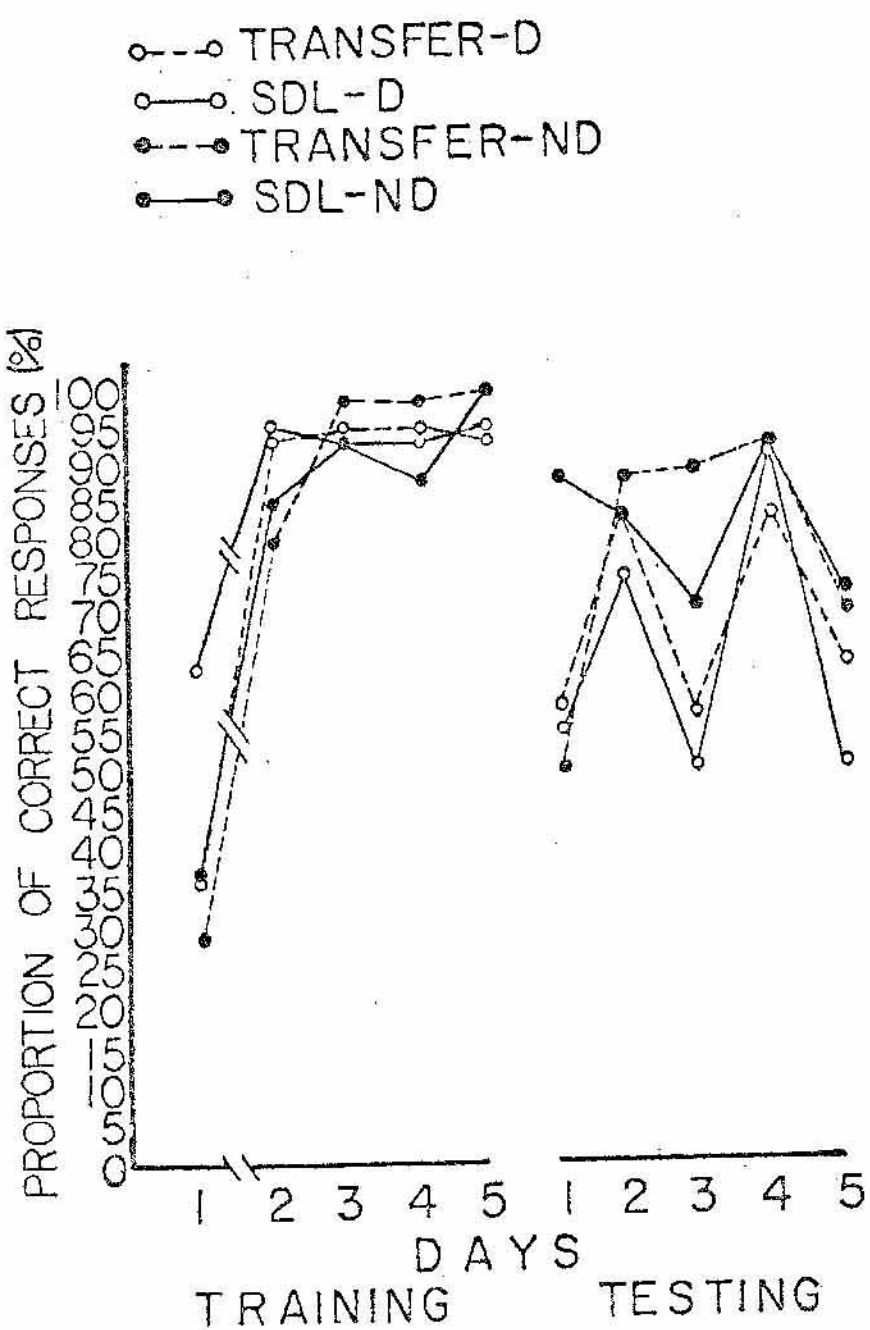
Figure 1 - The number of correct responses for training and testing; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls.

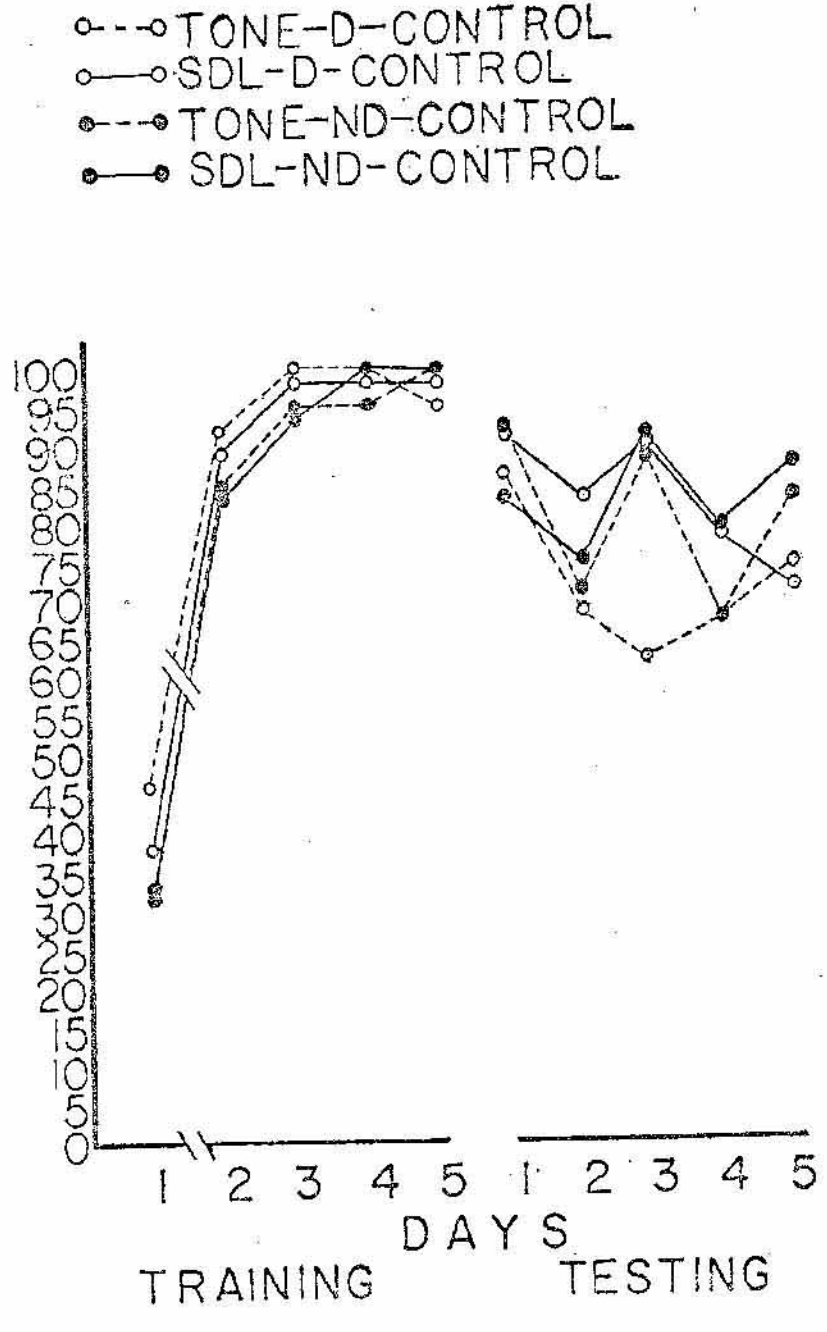

TRANSFER-D
SDL-D
TRANSFER-ND
SDL-ND
NUMBER OF CORRECT RESPONSES
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING


TONE-D-CONTROL
SDL-D-CONTROL
TONE-ND-CONTROL
SDL-ND-CONTROL
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING

APPENDIX A

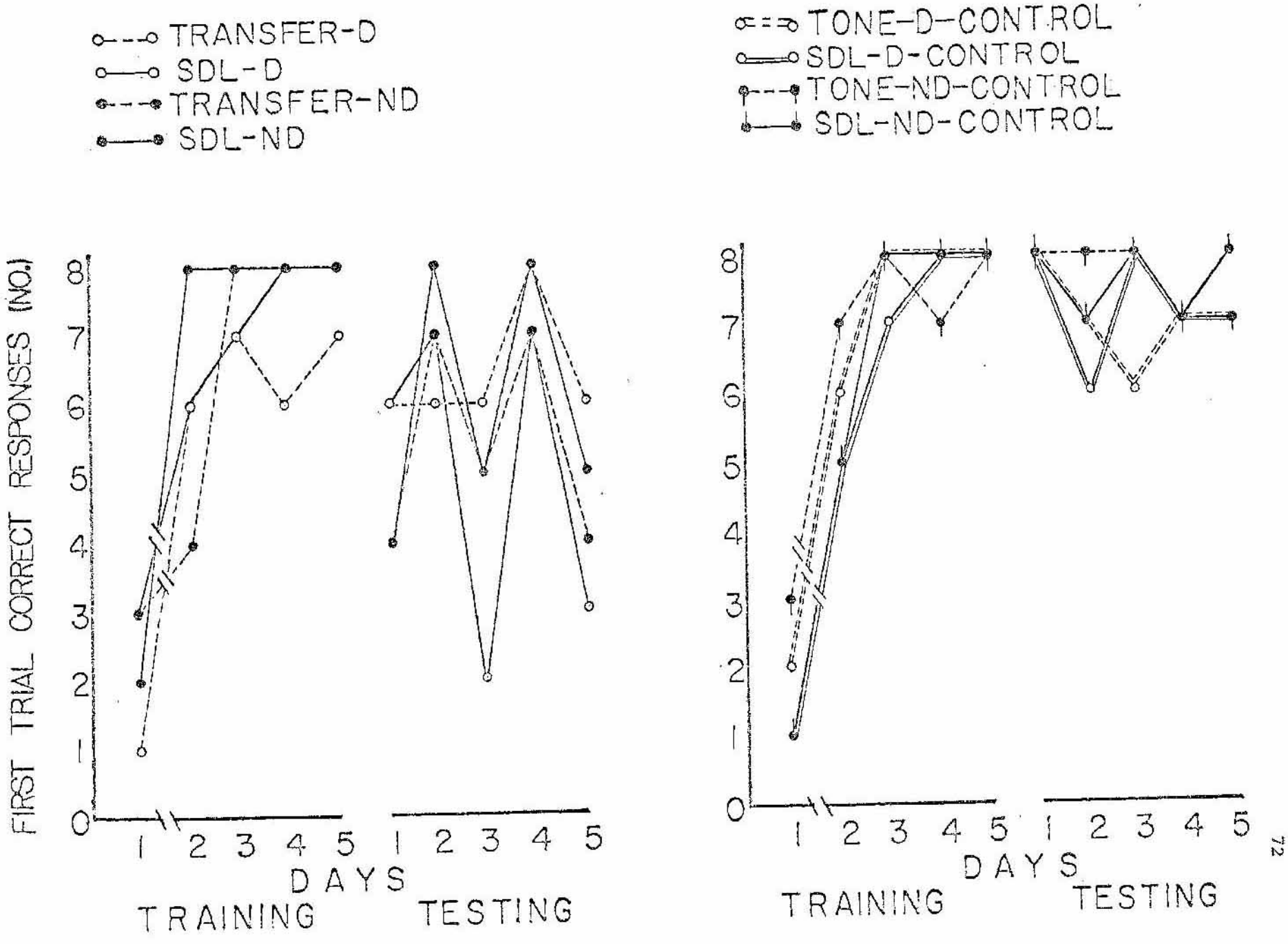
Figure 2 - The proportion of correct responses for training and testing; SDL experimentals vs. tranfer experimentals; SDL controls vs. tone controls.


TRANSFER-D
SDL-D
TRANSFER-ND
SDL-ND
PROPORTION OF CORRECT RESPONSES (%)
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING


TONE-D-CONTROL
SDL-D-CONTROL
TONE-ND-CONTROL
SDL-ND-CONTROL
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING

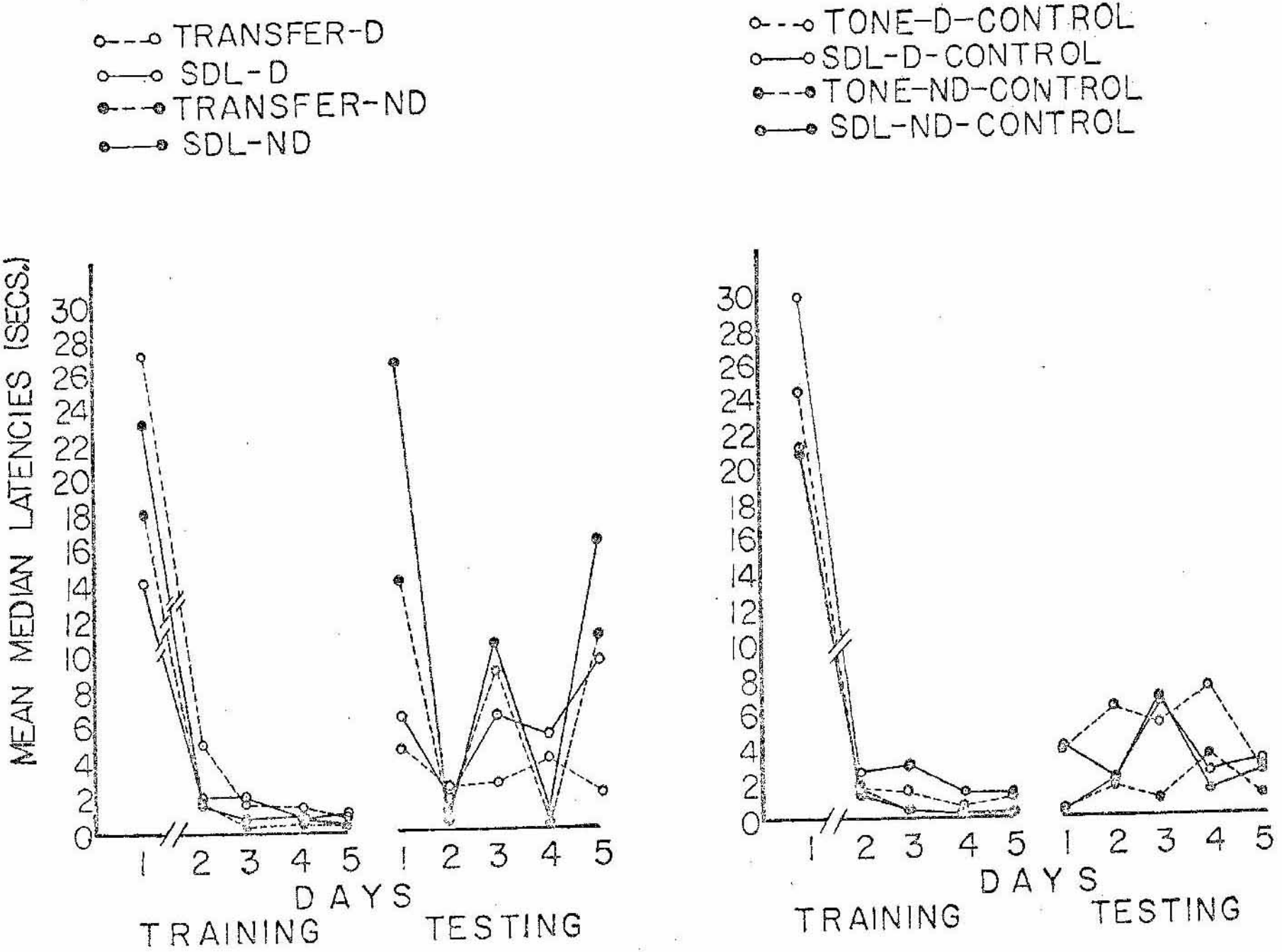
APPENDIX A

Figure 3 - The number of first trial correct responses for training and testing; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls.


TRANSFER-D
SDL-D
TRANSFER-ND
SDL-ND
TONE-D-CONTROL
SDL-D-CONTROL
TONE-ND-CONTROL
SDL-ND-CONTROL
FIRST TRIAL CORRECT RESPONSES (NO.)
0
1
2
3
4
5
6
7
8
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING

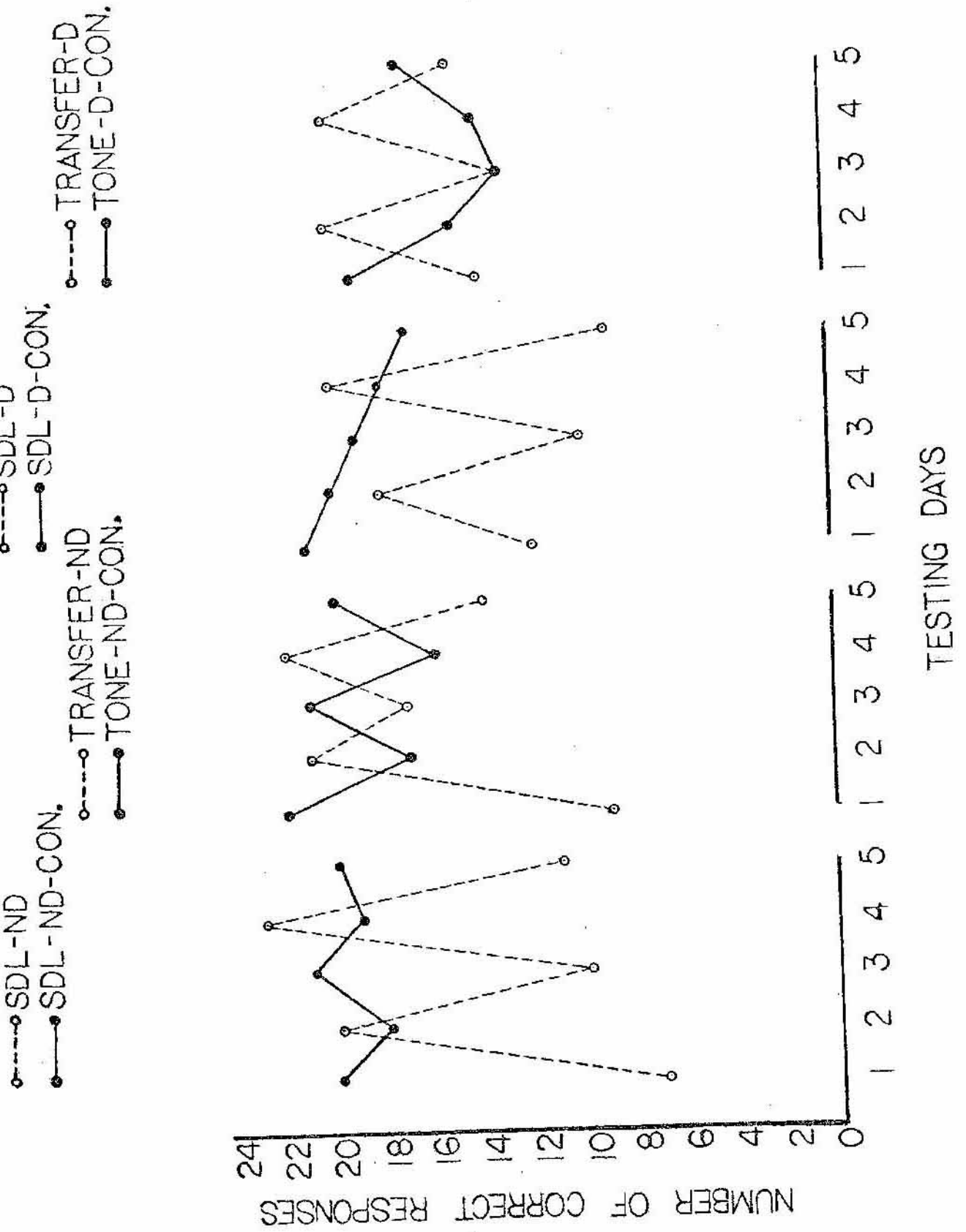
APPENDIX A

Figure 4 - The mean median latencies for training and testing; SDL experimentals vs. transfer experimentals; SDL controls vs. tone controls.


TRANSFER-D
SDL-D
TRANSFER-ND
SDL-ND
TONE-D-CONTROL
SDL-D-CONTROL
TONE-ND-CONTROL
SDL-ND-CONTROL
MEAN MEDIAN LATENCIES (SECS.)
0
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
1 2 3 4 5 1 2 3 4 5
DAYS
TRAINING
TESTING

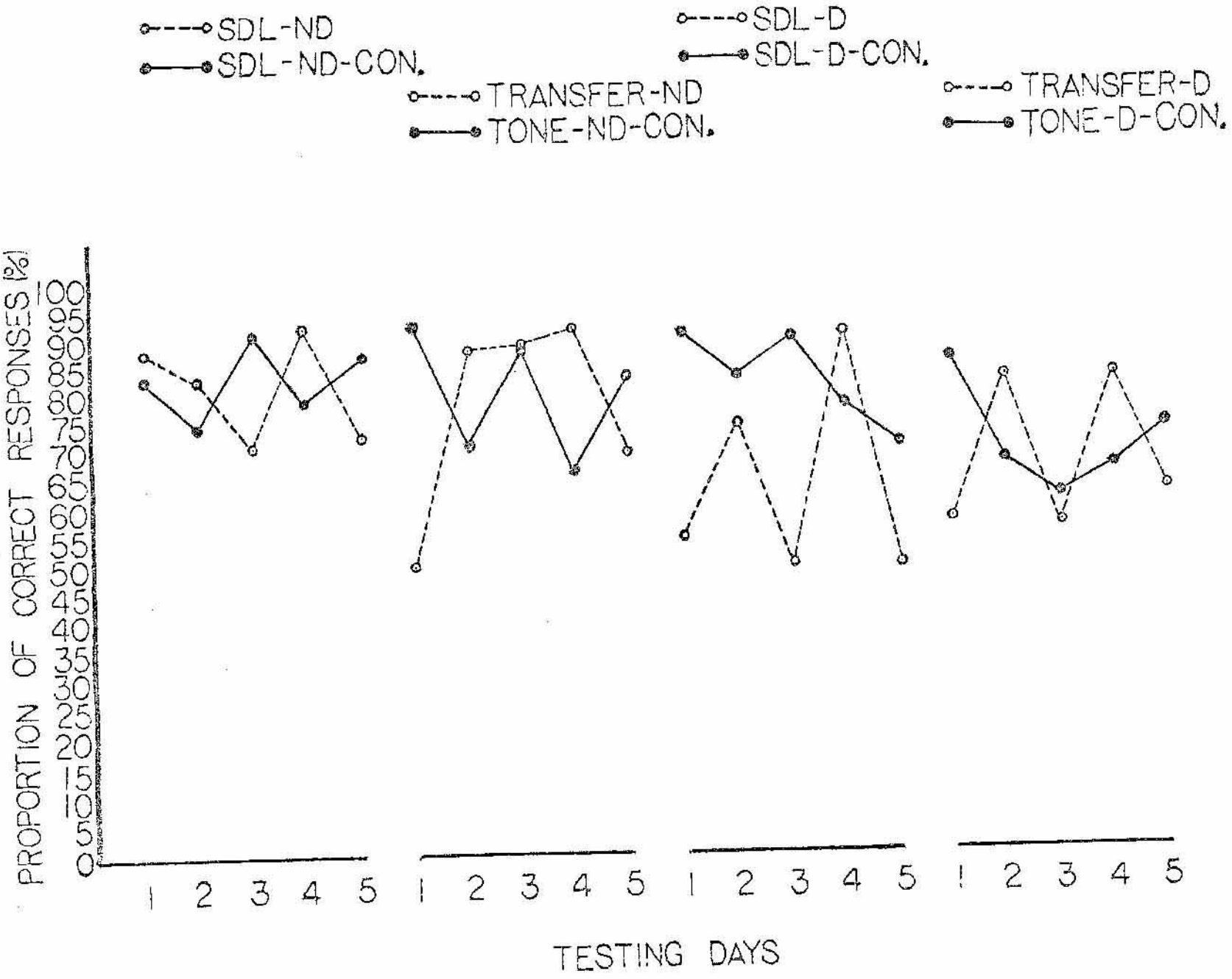
APPENDIX A

Figure 5 - The number of correct responses for testing; experimentals vs. controls.


SDL-ND
SDL-ND-CON.
SDL-D
SDL-D-CON.
TRANSFER-ND
TONE-ND-CON.
TRANSFER-D
TONE-D-CON.
NUMBER OF CORRECT RESPONSES
0
2
4
6
8
10
12
14
16
18
20
22
24
1 2 3 4 5
TESTING DAYS

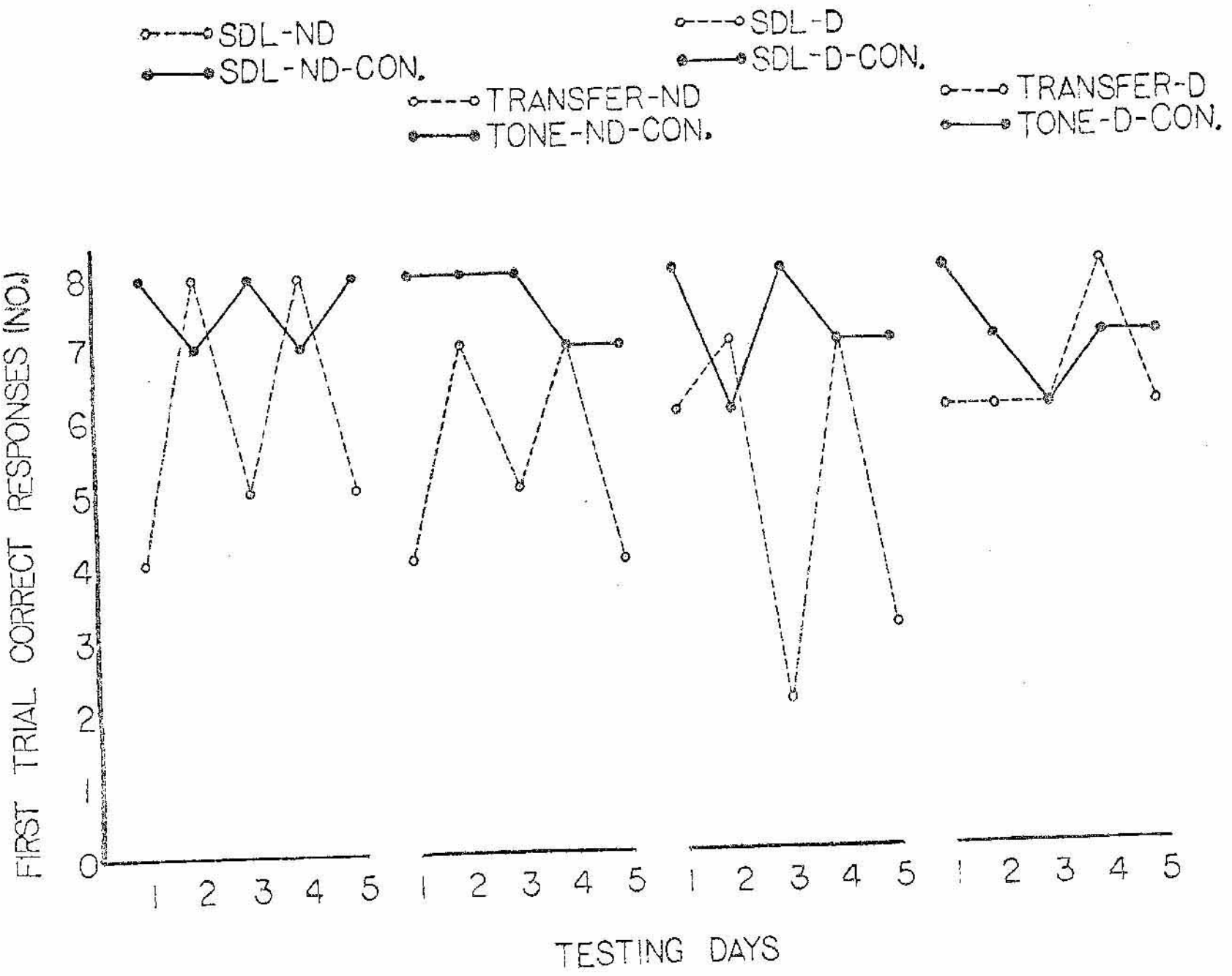
APPENDIX A

Figure 6 - The proportion of correct responses for testing; experimentals vs. controls.


SDL-ND
SDL-ND-CON.
TRANSFER-ND
TONE-ND-CON.
SDL-D
SDL-D-CON.
TRANSFER-D
TONE-D-CON.
PROPORTION OF CORRECT RESPONSES (%)
0
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
95
100
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TESTING DAYS

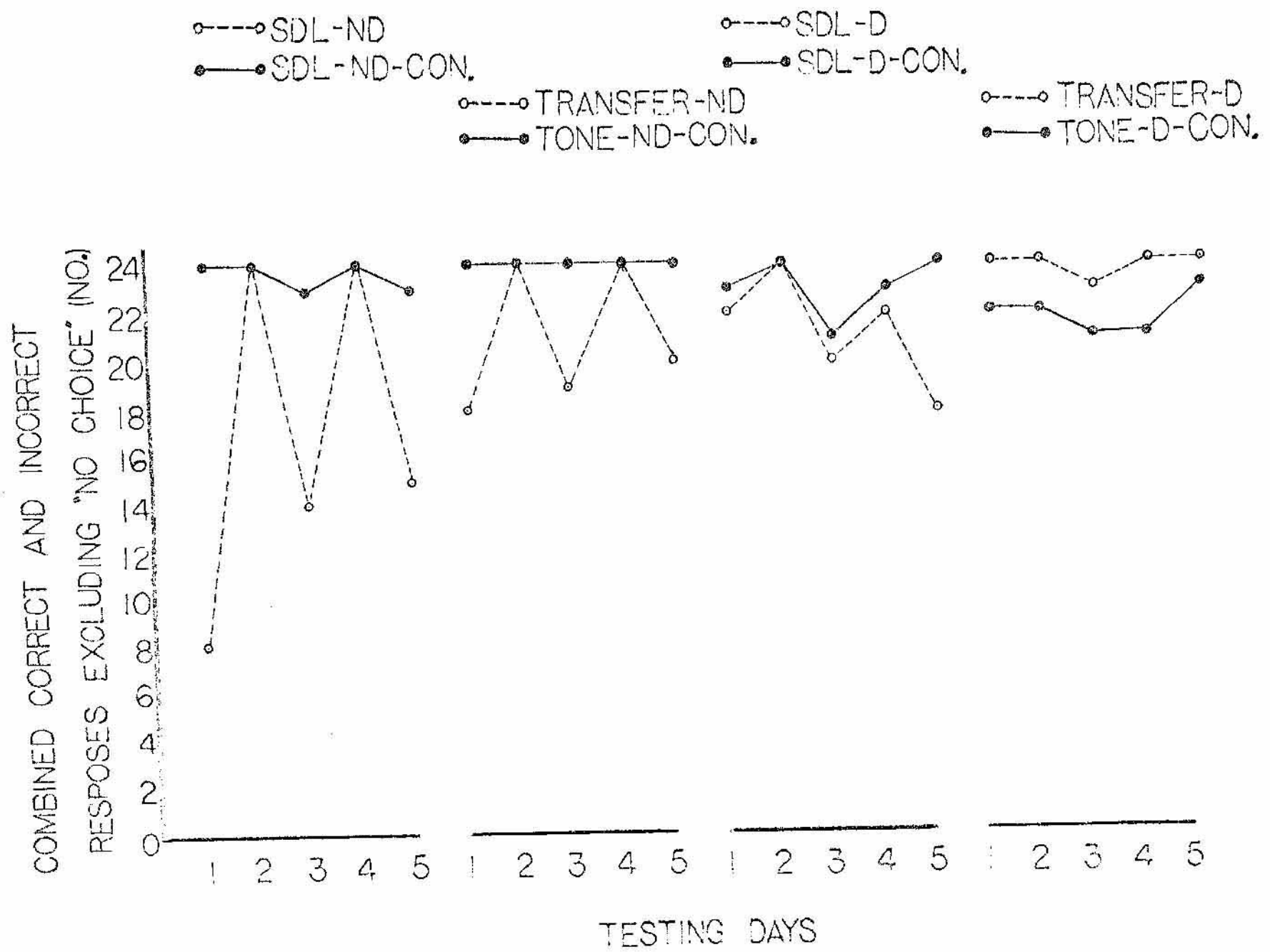
APPENDIX A

Figure 7 - The number of first trial correct responses for testing; experimentals vs. controls.


SDL-ND
SDL-ND-CON.
TRANSFER-ND
TONE-ND-CON.
SDL-D
SDL-D-CON.
TRANSFER-D
TONE-D-CON.
FIRST TRIAL CORRECT RESPONSES (NO.)
0
2
4
6
8
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TESTING DAYS

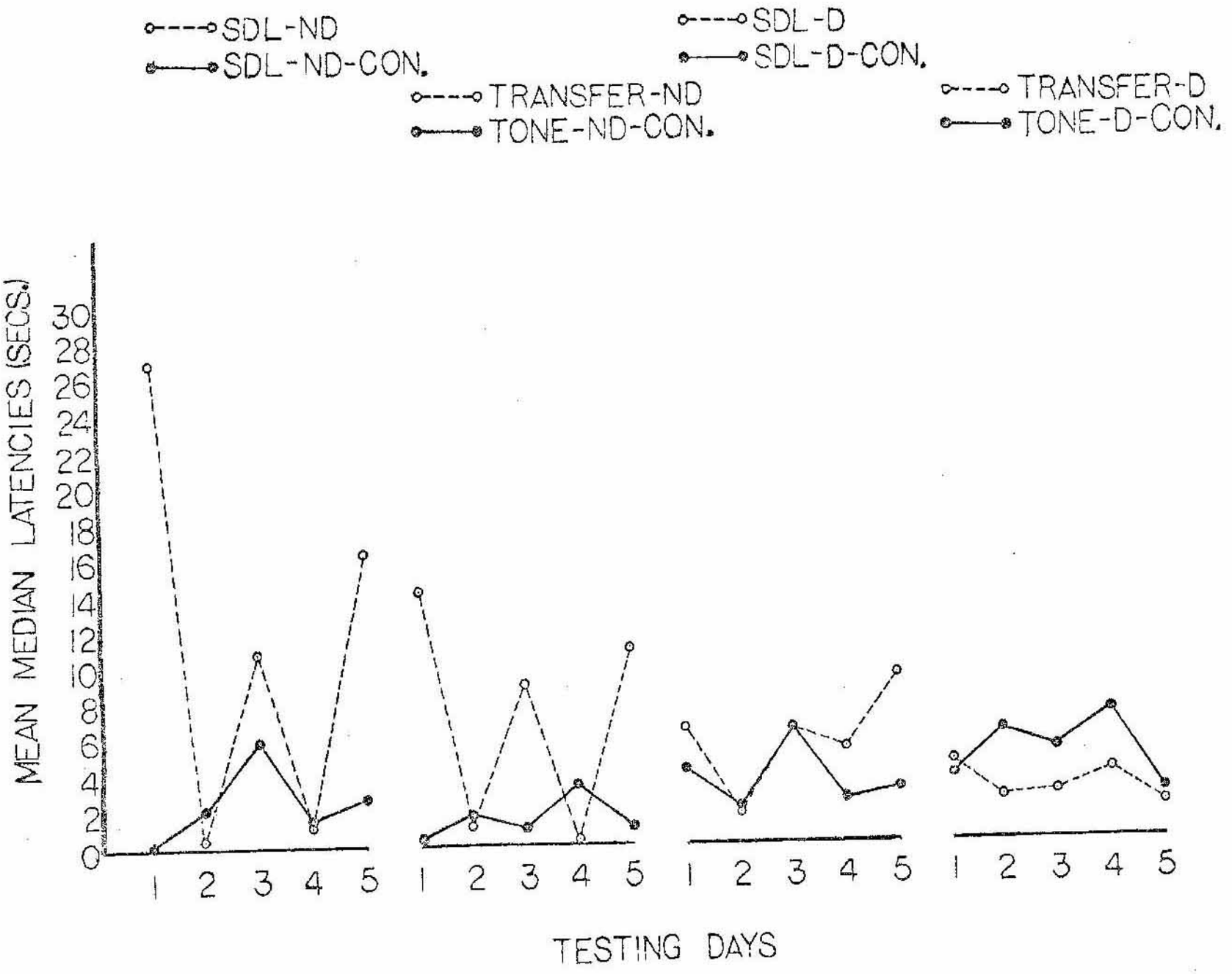
APPENDIX A

Figure 8 - The number of turns for testing; experimentals vs. controls.


SDL-ND
SDL-ND-CON.
SDL-D
SDL-D-CON.
TRANSFER-ND
TONE-ND-CON.
TRANSFER-D
TONE-D-CON.
COMBINED CORRECT AND INCORRECT
RESPOSES EXCLUDING "NO CHOICE" (NO.)
0
2
4
6
8
10
12
14
16
18
20
22
24
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
2 3 4 5
TESTING DAYS

APPENDIX A

Figure 9 - The mean median latencies for testing; experimentals vs. controls.


SDL-ND
SDL-ND-CON.
SDL-D
SDL-D-CON.
TRANSFER-ND
TONE-ND-CON.
TRANSFER-D
TONE-D-CON.
MEAN MEDIAN LATENCIES (SECS.)
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
TESTING DAYS

APPENDIX B

TABLE 2

ANOVA of the Number of Trials Required to Reach the Training Criterion, ALL Groups

Source	SS	df	MS	F	
Between groups	160.54	3			
Rows	112.89	1	112.89	1.04	NS
Columns	47.26	1	47.26		
Interaction	0.39	1	0.39		
Within	6498.44	60	108.31		
Total	6658.99				

Note: Rows - Drug States

Columns - Tone-No Tone Conditions

TABLE 3

ANOVA of the

Number of Correct Responses,

Training, All Groups

Source	SS	df	MS	F	
Between subjects	29.72	63			
A	0.03	1	0.03		
B	0.38	3	0.13		
AB	4.98	3	1.66	3.83	($p < .025$)
sub w. groups [error (between)]	24.32	56	0.43		
Within Subjects	725.20	256			
C	589.28	4	147.32	306.83	($p < .001$)
AC	2.58	4	0.65	1.34	NS
BC	9.95	12	0.83	1.73	($p < .10$)
ABC	15.84	12	1.32	2.75	($p < .001$)
C x Subgroups [error (within)]	107.55	224	0.48		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 4

ANOVA of the Number of Correct Responses, Training, D-No Tone vs. ND-No Tone vs. D-Tone vs. ND-Tone

Source	SS	df	MS	F	
Between subjects	29.72	63	[illegible]		
A	0.03	1	0.03		
B	0	1	0		
AB	0.08	1	0.08		
Sub w. groups [error (between)]	29.61	60	0.49		
Within Subjects	734.20	256			
C	589.28	4	147.32	258.46	($p < .001$)
AC	2.58	4	0.65	1.14	NS
BC	4.36	4	1.09	1.91	NS
ABC	0.53	4	0.13		
Cx Subgroups [error (within)]	137.45	240	0.57		

Note: A - Tone - No Tone Conditions

B - Drug States

C - Days

TABLE 5

ANOVA of the

Number of Correct Responses, Training,

SDE Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	13.50	31			
A	0.40	1	0.40		
B	1.23	1	1.23	2.94	($p < .10$)
AB	0.22	1	0.22		
sub w. groups /error (between)/	11.65	28	0.42		
Within Subjects	332.40	128			
C	103.21	4	25.80	57.40	($p < .001$)
AC	158.41	4	39.60	88.09	($p < .001$)
BC	15.21	4	3.80	8.46	($p < .001$)
ABC	5.21	4	1.30	2.90	($p < .05$)
Cs Subgroups /error (within)/	50.35	112	0.45		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 6

ANOVA of the

Number of Correct Responses, Training,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	15.79	31			
A	2.26	1	2.26	5.28	($p < .05$)
B	1.06	1	1.06	2.47	NS
AB	0.51	1	0.51	1.18	NS
Sub w. groups /error (between)/	11.97	28	0.43		
Within Subjects	393.20	128			
C	137.84	4	34.46	66.66	($p < .001$)
AC	194.71	4	48.68	94.16	($p < .001$)
BC	0.91	4	0.23		
ABC	1.84	4	0.46		
Cs Subgroups /error (within)/	57.90	112	0.52		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 7

ANOVA of the Number of

Correct Responses, Training

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	18.14	31			
A	1.41	1	1.41	2.66	NS
B	0.16	1	0.16		
AB	1.81	1	1.81	3.42	($p < .10$)
sub w. groups [error (between)]	14.78	28	0.53		
Within Subjects	342.80	128			
C	263.35	4	65.84	120.68	($p < .001$)
AC	11.25	4	2.81	5.16	($p < .001$)
BC	3.25	4	0.81	1.49	NS
ABC	3.85	4	0.96	1.76	NS
Cx Subgroups [error (within)]	61.10	112	0.55		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 8

ANOVA of the Number of
Correct Responses, Training,
SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	11.57	31			
A	0.90	1	0.90	2.64	NS
B	0.23	1	0.23		
AB	0.90	1	0.90	2.64	NS
sub w. groups /error (between)/	9.55	28	0.34		
Within Subjects	382.40	128			
C	328.79	4	82.20	198.19	($p < .001$)
AC	1.79	4	0.45	1.08	NS
BC	3.84	4	0.96	2.31	($p < .10$)
ABC	1.54	4	0.38		
Cx Subgroups /error (within)/	46.45	112	0.41		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 9

ANOVA of the Proportion of Correct Responses, Training, All Groups

Source	SS	df	MS	F	
Between subjects		63			
A	51.00	1	51.00		
B	725.00	3	241.67		
AB	2480.00	3	826.67	3.41	($p < .025$)
sub w. groups [error (between)]		56	242.68		
Within Subjects		256			
C		4		166.76	($p < .001$)
AC	1234.00	4	308.50	1.10	
BC	3733.00	12	311.08	1.11	
ABC	8917.00	12	743.08	2.64	($p < .005$)
Cx Subgroups [error (within)]		224	281.08		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 10

ANOVA of the Proportion of

Correct Responses, Training,

D-No Tone vs. ND-No Tone vs. D-Tone vs. ND-Tone

Source	SS	df	MS	F	
Between subjects	16845.39	63			
A	51.20	1	51.20		
B	632.82	1	632.82	2.36	NS
AB	45.0	1	45.0		
sub w. groups [error (between)]	16116.37	60	268.61		
Within Subjects	264333.60	256			
C	187487.52	4	46871.88	152.90	($p < .001$)
AC	1233.71	4	308.43	1.01	NS
BC	1930.46	4	482.62	1.57	NS
ABC	108.91	4	27.23		
Cx Subgroups [error (within)]	73573.0	240	306.55		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 11

ANOVA of the Proportion of

Correct Responses, Training,

SDI Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	7306.00	31			
A	4.00	1	4.00		
B	432.00	1	432.00	1.85	
AB	316.00	1	316.00	1.35	
sub w. groups [error (between)]	6554.00	28	234.07		
Within Subjects		128			
C		4	7466.50	30.26	($p < .001$)
AC		4		52.78	($p < .001$)
BC	5845.00	4	1461.25	5.92	($p < .001$)
ABC	3637.00	4	909.25	3.68	($p < .01$)
Sx subgroups [error (within)]		112	246.78		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 12

ANOVA of the Proportion of

Correct Responses, Training,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	9417.00	31			
A	238.00	1	238.00		
B	744.00	1	744.00	2.52	
AB	262.00	1	262.00		
sub w. groups [error (between)]	8273.00	28	295.46		
Within Subjects		128			
C		4		36.38	($p < .001$)
AC		4		53.34	($p < .001$)
BC	823.00	4	205.75		
ABC	1079.00	4	269.75		
Cx Subgroups [error (within)]		112	304.35		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 13

ANOVA of the Proportion of

Correct Responses, Training,

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	8760.00	31			
A	846.00	1	846.00	3.67	($p < .10$)
B	562.00	1	562.00	2.44	NS
AB	903.00	1	903.00	3.92	($p < .10$)
sub w. groups /error (between)/	6449.00	28	230.32		
Within Subjects		128			
C		4		72.88	($p < .001$)
AC	5976.00	4	1494.00	5.22	($p < .001$)
BC	2300.00	4	575.00	2.01	NS
ABC	2060.00	4	515.00	1.80	NS
Cx Subgroups /error (within)/		112	286.02		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 14

ANOVA of the Proportion of
Correct Responses, Training,
SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	8065.00	31			
A	360.00	1	360.00	1.41	NS
B	141.00	1	141.00		
AB	422.00	1	422.00	1.65	NS
Sub w. groups /error (between)/	7142.00	28	255.07		
Within Subjects		128			
C		4		95.01	($p < .001$)
AC	966.00	4	241.50		
BC	598.00	4	149.50		
ABC	1149.00	4	287.25	1.04	NS
Cx Subgroups /error (within)/		112	276.13		

Note: A = Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 15

ANOVA of the Number of

First Trial Correct Responses,

Training, All Groups

Source	SS	df	MS	F	
Between subjects	6.20	63			
A	0.01	1	0.01		
B	0.25	3	0.08		
AB	0.64	3	0.21	2.25	($p < .10$)
sub w. groups [error (between)]	5.30	56	0.09		
Within Subjects	49.60	256			
C	24.61	4	6.15	60.06	($p < .001$)
AC	0.24	4	0.06		
BC	0.31	12	0.03		
ABC	1.49	12	0.12	1.21	
Cx Subgroups [error (within)]	22.95	224	0.10		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 16

ANOVA of the Number of

First Trial Correct Responses, Training,

D-No Tone vs. MD-No Tone vs. D-Tone vs. MD-Tone

Source	SS	df	MS	F	
Between subjects	6.20	63			
A	0.01	1	0.01		
B	0.20	1	0.20	2.0	NS
AB	0.02	1	0.02		
sub w. groups [error (between)]	5.97	60	0.10		
Within Subjects	49.60	256			
C	24.61	4	6.15	61.50	($p < .01$)
AC	0.24	4	0.06		
BC	0.05	4	0.01		
ABC	0.42	4	0.11	1.10	NS
Cx Subgroups [error (within)]	24.28	240	0.10		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 17

ANOVA of the Number of

First Trial Correct Responses, Training,

SDL Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	2.94	31			
A	0.01	1	0.01		
B	0.31	1	0.31	3.33	($p < .10$)
AB	0.06	1	0.06		
sub w. groups /error (between)/	2.57	28	0.09		
Within Subjects	24.40	128			
C	7.06	4	1.77	20.71	($p < .001$)
AC	7.21	4	1.80	21.15	($p < .001$)
BC	0.29	4	0.07		
ABC	0.29	4	0.07		
Cx Subgroups /error (within)/	9.55	112	0.09		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 18

ANOVA of the Number of
First Trial Correct Responses, Training,
Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	2.84	31			
A	0.16	1	0.16	1.88	
B	0.31	1	0.31	3.69	($p < .10$)
AB	0.06	1	0.06		
sub w. Groups /error (between)/	2.32	28	0.08		
Within Subjects	25.60	128			
C	5.35	4	1.34	10.86	($p < .001$)
AC	5.75	4	1.44	11.67	($p < .001$)
BC	0.35	4	0.09		
ABC	0.35	4	0.09		
Cx Subgroups /error (within)/	13.80	112	0.12		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 19

ANOVA of the Number of

First Trial Correct Responses, Training,

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	3.10	31			
A	0.40	1	0.40	4.57	($p < .05$)
B	0.23	1	0.23	2.57	NS
AB	0.02	1	0.02		
sub w. groups /error (between)/	2.45	28	0.09		
Within Subjects	24.80	128			
C	10.71	4	2.68	23.43	($p < .001$)
AC	0.29	4	0.07		
BC	0.09	4	0.02		
ABC	0.91	4	0.23	2.00	($p < .10$)
Cx Subgroups /error (within)/	12.80	112	0.11		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 20

ANOVA of the Number of

First Trail Correct Responses, Training,

SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	3.10	31			
A	0.23	1	0.23	2.21	NS
B	0.03	1	0.03		
AB	-0.00	1	-0.00		
sub w. groups [error (between)]	2.85	28	0.10		
Within Subjects	24.80	128			
C	14.03	4	3.51	38.69	($p < .001$)
AC	0.40	4	0.10	1.10	NS
BC	0.10	4	0.02		
ABC	0.13	4	0.03		
Cx Subgroups [error (within)]	10.15	112	0.09		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 21

ANOVA of the

Median Latencies, Training,

All Groups

Source	SS	df	MS	F	
Between subjects	1713.44	63			
A	0.77	1	0.77		
B	236.37	3	78.79	3.75	($p < .025$)
AB	298.91	3	99.64	4.74	($p < .01$)
sub w. groups [error (between)]	1177.39	56	21.02		
Within Subjects		256			
C		4	5682.29	261.76	($p < .001$)
AC	19.21	4	4.80		
BC	371.34	12	30.95	1.43	
ABC	647.31	12	53.98	2.49	($p < .005$)
Cx Subgroups [error (within)]	4862.67	224	21.71		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 22

ANOVA of the

Median Latencies, Training,

D-No Tone vs. ND-No Tone vs. D-Tone vs. ND-Tone

Source	SS	df	MS	F	
Between subjects	1713.42	63	27.20		
A	0.79	1	0.79		
B	182.58	1	182.58	7.31	($p < .01$)
AB	31.18	1	31.18	1.25	NS
sub w. groups [error (between)]	1498.87	60	24.98		
Within Subjects	28629.41	256	111.83		
C	22729.16	4	5682.29	239.66	($p < .001$)
AC	19.16	4	4.79		
BC	56.30	4	14.08		
ABC	133.54	4	33.39	1.41	NS
Cx Subgroups [error (within)]	5691.25	240	23.71		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 23

ANOVA of the

Median Latencies, Training,

SDL Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	625.70	31			
A	2.90	1	2.90		
B	86.34	1	88.34	5.96	($p < .025$)
AB	119.09	1	119.09	8.03	($p < .01$)
sub w. groups [error (between)]	415.37	28	14.83		
Within Subjects		128			
C	4550.99	4	1137.75	63.42	($p < .001$)
AC	6533.75	4	1633.44	91.05	($p < .001$)
BC	472.62	4	118.15	6.59	($p < .001$)
ABC	361.55	4	90.39	5.04	($p < .001$)
Cx Subgroups [error (within)]	2009.19	112	17.94		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 24

ANOVA of the

Median Latencies, Training,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	766.37	31			
A	58.51	1	58.51	2.35	
B	7.89	1	7.89		
AB	1.51	1	1.51		
sub w. groups [error (between)]	698.46	28	24.94		
Within Subjects		128			
C	5375.83	4	1343.96	51.60	($p<.001$)
AC	6629.95	4	1657.49	63.64	($p<.001$)
BC	87.50	4	21.88		
ABC	12.36	4	3.09		
CxSubgroups [error (within)]	2917.11	112	26.05		

Note: A - Experimentals vs. Controls

B - Drug States

C - Days

TABLE 25

ANOVA of the

Median Latencies, Training

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	802.57	31			
A	45.29	1	45.29	2.36	NS
B	30.84	1	30.84	1.61	NS
AB	189.83	1	189.83	9.91	($p < .005$)
sub w. groups /error (between)/	536.61	28	19.16		
Within Subjects		128			
C	9471.18	4	2367.79	95.23	($p < .001$)
AC	110.44	4	27.61	1.11	NS
BC	15.03	4	3.76		
ABC	479.00	4	119.75	4.82	($p < .005$)
Cx Subgroups /error (within)/	2784.71	112	24.86		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 26

ANOVA of the

Median Latencies, Training,

SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between Subjects	889.15	31			
A	29.98	1	29.98	1.31	NS
B	183.82	1	183.82	8.03	($p < .01$)
AB	34.58	1	34.58	1.51	NS
sub w. groups /error (between)/	640.78	28	22.88		
Within Subjects		128			
C		4	3361.78	181.20	($p < .001$)
AC	44.96	4	11.24		
BC	167.16	4	41.79	2.25	($p < .10$)
ABC	32.63	4	8.16		
Cx Subgroups /error (within)/	2077.96	112	18.55		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 27

ANOVA of The Number

of Correct Responses, Testing,

All Groups

Source	SS	df	MS	F	
Between subjects	53.60	63			
A	0.15	1	0.15		
B	15.18	3	5.06	9.29	($p < .001$)
AB	7.73	3	2.58	4.73	($p < .01$)
sub w. groups /error (between)/	30.52	56	0.55		
Within Subjects	148.40	256			
C	13.04	4	3.26	8.43	($p < .001$)
AC	2.52	4	0.63	1.63	NS
BC	43.46	12	3.62	9.37	($p < .001$)
ABC	2.78	12	0.23		
Cx Subgroups /error (within)/	86.60	224	0.39		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 28

ANOVA of the Number of
Correct Responses, Testing,
SDL Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	35.50	31			
A	0.02	1	0.02		
B	18.23	1	18.23	31.21	($p < .001$)
AB	0.90	1	0.90	1.54	NS
sub w. groups [error (between)]	16.35	28	0.58		
Within Subjects	77.60	128			
C	7.48	4	1.87	4.91	($p < .005$)
AC	7.98	4	1.99	5.24	($p < .001$)
BC	4.52	4	1.13	2.97	($p < .025$)
ABC	14.98	4	3.74	9.83	($p < .001$)
Cx Subgroups [error (within)]	42.65	112	0.38		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 29

ANOVA of the Number of

Correct Responses, Testing,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	15.54	31			
A	0.16	1	0.16		
B	0.51	1	0.51	1.05	NS
AB	1.41	1	1.41	2.92	($p < .10$)
sub w. groups [error (between)]	13.47	28	0.48		
Within Subjects	73.20	128			
C	5.28	4	1.32	3.31	($p < .025$)
AC	2.50	4	0.63	1.57	NS
BC	7.65	4	1.91	4.80	($p < .005$)
ABC	13.13	4	3.28	8.23	($p < .001$)
Cx Subgroups [error (within)]	44.65	112	0.40		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 30

ANOVA of the Number of

Correct Responses, Testing,

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	15.20	31			
A	4.23	1	4.23	10.80	($p < .005$)
B	0.03	1	0.03		
AB	-0.00	1	-0.00		
sub w. groups /error (between)/	10.95	28	0.39		
Within Subjects	107.20	128			
C	46.21	4	11.55	24.16	($p < .001$)
AC	2.21	4	0.55	1.16	NS
BC	4.41	4	1.10	2.31	($p < .10$)
ABC	0.81	4	0.20		
Cx Subgroups /error (within)/	53.55	112	0.48		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 31

ANOVA of the Number of

Correct Responses, Testing,

SHL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	25.99	31			
A	2.26	1	2.26	3.23	($p < .10$)
B	2.76	1	2.76	3.94	($p < .10$)
AB	1.41	1	1.41	2.01	NS
Sub w. groups [error (between)]	19.57	28	0.70		
Within Subjects	41.20	128			
C	3.98	4	0.99	3.37	($p < .025$)
AC	1.52	4	0.38	1.29	NS
BC	1.90	4	0.47	1.61	NS
ABC	0.75	4	0.19		
Wx Subgroups [error (within)]	33.05	112	0.30		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 32

ANOVA of the Proportion of

Correct Responses, Testing,

All Groups

Source	SS	df	MS	F	
Between subjects		63			
A	440.00	1	440.00		
B	9425.00	3	3141.67	5.49	($p < .005$)
AB	4377.00	3	1459.00	2.55	($p < .10$)
sub w. groups [error (between)]		56	572.41		
Within Subjects		256			
C	4253.00	4	1063.25	1.99	($p < .10$)
AC	1711.00	4	427.75		
BC		12	2423.00	4.52	($p < .001$)
ABC		12	903.50	1.68	($p < .10$)
Cx Subgroups [error (within)]		224	535.55		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 33

ANOVA of the Proportion of
Correct Responses, Testing,
SDL Experimental vs. SDL Controls

Source	SS	df	MS	F	
Between subjects		31			
A	1134.19	1	1134.19	2.01	
B	7102.19	1	7102.19	12.60	($p < .005$)
AB	163.81	1	163.81		
sub w. groups [error (between)]		28	563.71		
Within Subjects		128			
C	1924.31	4	481.08		
AC	4242.06	4	1060.52	1.72	
BC	1405.44	4	351.36		
ABC		4	3449.95	5.60	($p < .001$)
Cx Subgroups [error (within)]		112	616.41		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 34

ANOVA of the Proportion of

Correct Responses, Testing,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects		31			
A	1221.00	1	1221.00	2.37	
B	16.75	1	16.75		
AB	144.25	1	144.25		
sub w. groups /error (between)/		28	514.80		
Within Subjects		128			
C	8787.50	4	2196.88	4.66	(p<.005)
AC	1532.25	4	383.06		
BC	8798.25	4	2199.56	4.67	(p<.005)
ABC	9410.50	4	2352.63	4.99	(p<.005)
C x Subgroups /error (within)/		112	471.26		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 35

ANOVA of the Proportion of

Correct Responses, Testing,

EDI Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects		31			
A	635.88	1	635.88		
B	4357.69	1	4357.69	6.58	($p < .025$)
AB	1.94	1	1.94		
sub w. groups [error (between)]		29	662.18		
Within Subjects		123			
C		4	5729.50	8.18	($p < .001$)
AC	5433.69	4	1358.47	1.94	NS
BC	2229.63	4	557.41		
ABC	2185.06	4	546.27		
C x Subgroups [error (within)]		112	699.94		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 36

ANOVA of the Proportion of
Correct Responses, Testing,
SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects		31			
A	3018.88	1	3018.88	6.26	($p < .025$)
B	1160.88	1	1160.88	2.41	NS
AB	1161.13	1	1161.13	2.41	NS
sub w. groups [error (between)]		28	482.62		
Within Subjects		128			
C	5619.63	4	1404.91	3.79	($p < .01$)
AC	3178.31	4	794.58	2.14	($p < .10$)
BC	2569.56	4	642.39	1.73	NS
ABC	1751.88	4	437.97	1.18	NS
Cx Subgroups [error (within)]		112	371.15		

Note: A - Tone- No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 37

ANOVA of the Number of

First Trial Correct Responses, Testing,

All Groups

Source	SS	df	MS	F	
Between subjects	12.52	63			
A	0.03	1	0.03		
B	3.56	3	1.19	8.08	($p < .001$)
AB	0.71	3	0.24	1.61	
sub w. groups [error (between)]	8.23	56	0.15		
Within Subjects	35.60	256			
C	1.45	4	0.36	2.83	($p < .025$)
AC	0.05	4	0.01		
BC	3.92	12	0.33	2.56	($p < .005$)
ABC	1.53	12	0.13		
Cx Subgroups [error (within)]	28.65	224	0.13		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 38

ANOVA of the Number of

First Trial Correct Responses, Testing,

SDL Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	6.99	31			
A	0.01	1	0.01		
B	2.26	1	2.26	13.37	($p < .005$)
AB	0.01	1	0.01		
sub w. groups /error (between)/	4.73	28	0.17		
Within Subjects	18.00	128			
C	0.90	4	0.23	1.99	
AC	0.90	4	0.22	1.99	
BC	1.15	4	0.29	2.55	($p < .05$)
ABC	2.40	4	0.60	5.31	($p < .001$)
Cx Subgroups /error (within)/	12.65	112	0.11		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 39

ANOVA of the Number of

First Trial Correct Responses, Testing,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	5.90	31			
A	0.10	1	0.10		
B	1.23	1	1.23	7.89	($p < .01$)
AB	0.22	1	0.22	1.45	
sub w. groups [error (between)]	4.35	28	0.16		
Within Subjects	17.20	128			
C	0.23	4	0.06		
AC	0.65	4	0.16	1.20	
BC	0.65	4	0.16	1.20	
ABC	0.53	4	0.13		
Cx Subgroups [error (within)]	15.15	112	0.14		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 40

ANOVA of the Number of

First Trial Correct Responses, Testing,

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	5.98	31			
A	0.10	1	0.10		
B	0.0	1	0.0		
AB	0.63	1	0.63	3.33	($p < .10$)
sub w. groups [error (between)]	5.25	28	0.19		
Within Subjects	26.80	128			
C	4.15	4	1.04	5.60	($p < .001$)
AC	0.65	4	0.16		
BC	0.75	4	0.19	1.01	NS
ABC	0.50	4	0.13		
Cx Subgroups [error (within)]	20.75	112	0.19		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 41

ANOVA Of the Number of

First Trial Correct Responses, Testing,

SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	3.14	31			
A	0.01	1	0.01		
B	0.16	1	0.16	1.47	NS
AB	0.01	1	0.01		
sub w. groups [error (between)]	2.98	28	0.11		
Within Subjects	8.80	128			
C	0.35	4	0.09	1.24	NS
AC	0.27	4	0.07		
BC	0.13	4	0.03		
ABC	0.15	4	0.04		
Cx Subgroups [error (within)]	7.90	112	0.07		

Note: A - Tone-No Tone Conditions

B - Drug States

C - Days

TABLE 42

ANOVA of the

Median Latencies, Testing,

All Groups

Source	SS	df	MS	F	
Between subjects	6819.60	63			
A	140.54	1	140.54	2.01	
B	2397.90	3	799.30	11.44	($p<.001$)
AB	366.91	3	122.30	1.75	
sub w. groups [error (between)]	3914.25	56	60.90		
Within Subjects		256			
C	1181.81	4	295.45	8.21	($p<.001$)
AC	377.92	4	94.48	2.63	($p<.05$)
BC	3679.99	12	306.67	8.53	($p<.001$)
ABC	379.21	12	31.60		
Cx Subgroups [error (within)]	8056.98	224	35.97		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 43

ANOVA of the

Median Latencies, Testing,

SDL Experimentals vs. SDL Controls

Source	SS	df	MS	F	
Between subjects	4440.78	31			
A	310.14	1	310.14	4.64	($p < .05$)
B	1420.40	1	1420.40	21.24	($p < .001$)
AB	838.05	1	838.05	12.53	($p < .005$)
Sub w. groups /error (between)/	1872.19	28	66.86		
Within Subjects	8640.97	128			
C	871.24	4	217.81	4.98	($p < .005$)
AC	931.75	4	232.94	5.33	($p < .001$)
BC	1284.19	4	321.05	7.35	($p < .001$)
ABC	659.49	4	164.87	3.77	($p < .01$)
Cx Subgroups /error (within)/	4894.30	112	43.70		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 44

ANOVA of the

Median Latencies, Testing,

Transfer Experimentals vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	2037.86	31			
A	330.10	1	330.10	7.71	($p < .01$)
B	138.87	1	138.87	3.24	($p < .10$)
AB	370.34	1	370.34	8.65	($p < .01$)
sub w. groups /error (between)/	1198.55	28	42.81		
Within Subjects	5235.37	128			
C	59.96	4	14.99		
AC	116.59	4	29.15		
BC	481.60	4	120.40	3.37	($p < .025$)
ABC	571.01	4	142.75	3.99	($p < .005$)
Cs Subgroups /error (within)/	4006.20	112	35.77		

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 45

ANOVA of the

Median Latencies, Testing,

SDL Experimentals vs. Transfer Experimentals

Source	SS	df	MS	F	
Between subjects	3070.13	31			
A	455.19	1	455.19	7.21	($p < .025$)
B	830.90	1	830.90	13.16	($p < .005$)
AB	16.36	1	16.36		
sub w. groups [error (between)]	1767.74	28	63.13		
Within Subjects		128			
C	2940.90	4	735.23	13.19	($p < .001$)
AC	368.89	4	92.22	1.65	NS
BC	1800.31	4	450.08	8.07	($p < .001$)
ABC	236.48	4	59.12	1.06	NS
Cx Subgroups [error (within)]	6243.13	112	55.74		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 46

ANOVA of the

Median Latencies, Testing,

SDL Controls vs. Tone Controls

Source	SS	df	MS	F	
Between subjects	2525.74	31			
A	20.88	1	20.88		
B	343.26	1	343.26	4.48	($p < .05$)
AB	15.03	1	15.03		
sub w. groups [error (between)]	2146.57	28	76.66		
Within Subjects	2086.22	128			
C	50.47	4	12.62		
AC	101.96	4	25.49	1.57	NS
BC	70.15	4	17.54	1.08	NS
ABC	49.80	4	12.45		
Cx Subgroups [error (within)]	1813.84	112	16.20		

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 47

Levels of Significance

for ANOVAs, All Groups

	Training				Testing			
	The No. of Correct Responses	Proportion of Correct	The No. of First Trial Correct Responses	Median Latencies	The No. of Correct Responses	Proportion of Correct	The No. of First Trial Correct Responses	Median Latencies
Between Subjects								
A	*NS	NS	NS	NS	NS	NS	NS	NS
B	NS	NS	NS	.025	.001	.005	.001	.001
AB	.025	.025	.10	.01	.01	.10	NS	NS
Sub Gr. Groups /error (between)/								
Within Subjects								
C	.001	.001	.001	.001	.001	.10	.025	.001
AC	NS	NS	NS	NS	NS	NS	NS	.05
BC	.10	NS	NS	NS	.001	.001	.005	.001
ABC	.001	.005	NS	.005	NS	.10	NS	NS
Cx Subgroups /error (within)/								

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

*NS. not significant

TABLE 48

Levels of Significance

for ANOVAs, Combined Groups, Training

	The No. of Correct Responses	Proportion of Correct	The No. of First Trial Correct Responses	Median Latencies
Between Subjects				
A	NS	NS	NS	NS
B	NS	NS	NS	.01
AB	NS	NS	NS	NS
Sub W. Groups [error (between)]				
Within Subjects				
C	.001	.001	.001	.001
AC	NS	NS	NS	NS
BC	NS	NS	NS	NS
ABC	NS	NS	NS	NS
Cx Subgroups [error (within)]				

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 49

Levels of Significance for Sub-ANOVAs,

SDL Experimentals vs. SDL Controls

	Training				Testing			
	The No. of Correct Responses	Proportion of Correct Responses	The No. of First Trial Correct Responses	Median Latencies	The No. of Correct Responses	Pro. of Correct Responses	The No. of First Trial Correct Responses	Median Latencies
Between Subjects								
A	NS	NS	NS	NS	NS	NS	NS	.05
B	.10	NS	.10	.025	.001	.005	.005	.001
AB	NS	NS	NS	.01	NS	NS	NS	.005
Sub W. Groups /error (between)/								
Within Subjects								
C	.001	.001	.001	.001	.005	NS	NS	.005
AC	.001	.001	.001	.001	.001	NS	NS	.001
BC	.001	.001	NS	.001	.025	NS	.05	.001
ABC	.05	.01	NS	.001	.001	.001	.001	.01
Cx Subgroups /error (within)/								

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 50

Levels of Significance for Sub-ANOVAs,

Transfer Experimentals vs. Tone Controls

	Training				Testing			
	The No. of Correct Responses	Proportion of Correct	The No. of First Trial Correct Responses	Median Latencies	The No. of Correct Responses	Proportion of Correct	The No. of First Trial Correct Responses	Median Latencies
Between Subjects								
A	.05	NS	NS	NS	NS	NS	NS	.01
B	NS	NS	.10	NS	NS	NS	.01	.10
AB	NS	NS	NS	NS	.10	NS	NS	.01
Sub N. Groups /error (between)/								
Within Subjects								
C	.001	.001	.001	.001	.025	.005	NS	NS
AC	.001	.001	.001	.001	NS	NS	NS	NS
BC	NS	NS	NS	NS	.005	.005	NS	.025
ABC	NS	NS	NS	NS	.001	.005	NS	.005
Cx Subgroups /error (within)/								

Note: A - Experimentals vs. Controls

B - Drug Treatment Combinations

C - Days

TABLE 51

Levels of Significance for Sub-ANOVAs.

SDL Experimentals vs. Transfer Experimentals

	Training				Testing			
	The No. of Correct Responses	Proportion of Correct Responses	The No. of First Trial Correct Responses	Median Latencies	The No. of Correct Responses	Proportion of Correct Responses	The No. of First Trial Correct Responses	Median Latencies
Between Subjects								
A	NS	.10	.05	NS	.005	NS	NS	.025
B	NS	NS	NS	NS	NS	.025	NS	.005
AB	.10	.10	NS	.005	NS	NS	.10	NS
Sub W. Groups /error (between)/								
Within Subjects								
C	.001	.001	.001	.001	.001	.001	.001	.001
AC	.001	.001	NS	NS	NS	NS	NS	NS
BC	NS	NS	NS	NS	.10	NS	NS	.001
ABC	NS	NS	.10	.005	NS	NS	NS	NS
Cx Subgroups /error (within)/								

Note: A - Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 52

Levels of Significance for Sub-ANOVAs,

SDL Controls vs. Tone Controls

	Training				Testing			
	The No. of Correct Responses	Proportion of Correct Responses	The No. of First Trial Correct Responses	Median Latencies	The No. of Correct Responses	Proportion of Correct Responses	The No. of First Trial Correct Responses	Median Latencies
Between Subjects								
A	NS	NS	NS	NS	.10	.025	NS	NS
B	NS	NS	NS	.01	.10	NS	NS	.05
AB	NS	NS	NS	NS	NS	NS	NS	NS
Sub W. Groups /error (between)/								
Within Subjects								
C	.001	.001	.001	.001	.025	.01	NS	NS
AC	NS	NS	NS	NS	NS	.10	NS	NS
BC	.10	NS	NS	.10	NS	NS	NS	NS
ABC	NS	NS	NS	NS	NS	NS	NS	NS
Cx Subgroups /error (within)/								

Note: A = Tone-No Tone Conditions

B - Drug Treatment Combinations

C - Days

TABLE 53

Z Scores for Mann-Whitney U Tests

and Levels of Significance for Training

The Number of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-ND Exp. vs. SDL-ND Cont.	0.22 NS	0.39 NS	.0 NS	2.61 < .01	0 NS
SDL-D Exp. vs. SDL-D Cont.	3.01 < .005	1.65 < .10	0.69 NS	1.12 NS	0.62 NS
Tran.-ND Exp. vs. Tone-ND Cont.	0.96 NS	0.73 NS	0.62 NS	0.62 NS	0 NS
Tran.-D Exp. vs. Tone-D Cont.	1.47 NS	0.49 NS	1.46 NS	1.46 NS	0.52 NS
SDL-ND Exp. vs. Tran.-ND Exp.	0.76 NS	0.73 NS	1.12 NS	2.0 < .05	0 NS
SDL-D Exp. vs. Tran.-D Exp.	2.41 < .025	0.52 NS	0.14 NS	0.52 NS	0.52 NS
SDL-ND Cont. vs. Tone-ND Cont.	0.28 NS	0.39 NS	0.52 NS	1.46 NS	0 NS
SDL-D Cont. vs. Tone-D Cont.	1.57 NS	0.92 NS	1.0 NS	1.0 NS	0.62 NS

TABLE 8

z Scores for Mann-Whitney U Tests

and Levels of Significance for Training

The Proportion of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-ND Exp. vs. SDL-ND Cont.	0.37 NS	0.39 NS	0 NS	2.61 < .01	0 NS
SDL-D Exp. vs. SDL-D Cont.	2.56 < .025	0.77 NS	0.69 NS	1.12 NS	0.62 NS
Tran.-ND Exp. vs. Tone-ND Cont.	1.08 NS	0.73 NS	0.62 NS	0.62 NS	0 NS
Tran.-D Exp. vs. Tone-D Cont.	1.28 NS	0 NS	1.46 NS	1.46 NS	0.43 NS
SDL-ND Exp. vs. Tran.-ND Exp.	0.76 NS	0.73 NS	1.12 NS	2.0 < .05	0 NS
SDL-D Exp. vs. Tran.-D Exp.	2.41 < .025	0.52 NS	0.14 NS	0.52 NS	0.52 NS
SDL-ND Cont. vs. Tone-ND Cont.	0.16 NS	0.39 NS	0.52 NS	1.46 NS	0 NS
SDL-D Cont. vs. Tone-D Cont.	1.34 NS	0.37 NS	1.0 NS	1.0 NS	0 NS

TABLE 55

Z Scores for Mann-Whitney U Tests

and Levels of Significance for Training

The Number of First Trial Correct Responses

	Day 1		Day 2		Day 3		Day 4		Day 5	
SDL-MD Exp. vs. SDL-MD Cont.	0.62	NS	1.36 < .10		0	NS	0	NS	0	NS
SDL-D Exp. vs. SDL-D Cont.	1.12	NS	0.52	NS	0	NS	0	NS	0	NS
Tran.-MD Exp. vs. Tone-MD Cont.	0	NS	1.57	NS	0	NS	1.0	NS	0	NS
Tran.-D Exp. vs. Tone-D Cont.	0.62	NS	0	NS	1.0	NS	1.46	NS	1.0	NS
SDL-MD Exp. vs. Tran.-MD Exp.	0.52	NS	2.24 < .025		0	NS	0	NS	0	NS
SDL-D Exp. vs. Tran.-D Exp.	1.12	NS	0	NS	0	NS	1.46	NS	1.0	NS
SDL-MD Cont. vs. Tone-MD Cont.	1.12	NS	1.12	NS	0	NS	1.0	NS	0	NS
SDL-D Cont. vs. Tone-D Cont.	0.62	NS	0.52	NS	1.0	NS	0	NS	0	NS

TABLE 56

Z Scores for Mann-Whitney U Tests

and Levels of Significance for Training

Median Latencies

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-MD Exp. vs. SDL-MD Cont.	0.73 NS	0.52 NS	0.79 NS	1.68 < .10	0.84 NS
SDL-D Exp. vs. SDL-D Cont.	2.90 < .005	1.26 NS	0.74 NS	1.89 < .10	0.84 NS
Tran.-M Exp. vs. Tone-MD Cont.	0.44 NS	0.53 NS	0.84 NS	2.37 < .025	0.53 NS
Tran.-D Exp. vs. Tone-D Cont.	0.62 NS	1.26 NS	0.63 NS	1.79 < .10	0.95 NS
SDL-MD Exp. vs. Tran.-MD Exp.	0.95 NS	0.74 NS	1.31 NS	1.16 NS	0.32 NS
SDL-D Exp. vs. Tran.-D Exp.	2.31 < .025	1.15 NS	0.32 NS	1.58 NS	0.63 NS
SDL-MD Cont. vs. Tone-MD Cont.	0.11 NS	0.11 NS	0.15 NS	0.47 NS	0.05 NS
SDL-D Cont. vs. Tone-D Cont.	1.46 NS	1.16 NS	1.34 < .10	1.89 < .10	1.05 NS

TABLE 57

Z-Scores for Mann-Whitney U Tests

and Levels of Significance for Testing

The Number of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-ND Exp. vs. SDL-ND Cont.	3.28 < .001	1.0 NS	2.58 < .005	1.46 < .10	2.72 < .005
SDL-D Exp. vs. SDL-D Cont.	2.92 < .005	0.81 NS	2.46 < .01	1.06 NS	2.24 < .025
Tran.-ND Exp. vs. Tone-ND Cont.	3.19 < .001	1.59 < .10	1.27 NS	2.03 < .025	1.34 < .10
Tran.-D Exp. vs. Tone-D Cont.	2.13 < .025	1.89 < .05	0.32 NS	2.32 < .025	0.97 NS
SDL-ND Exp. vs. Trans-ND Exp.	0.68 NS	0.49 NS	1.69 < .05	0 NS	0.66 NS
SDL-D Exp. vs. Tran.-D Exp.	1.0 NS	0.71 NS	1.12 NS	0.24 NS	2.45 < .01
SDL-ND Cont. vs. Tone-ND Cont.	1.0 NS	0.39 NS	0 NS	1.06 NS	0 NS
SDL-D Cont. vs. Tone-D Cont.	0.97 NS	1.76 < .10	1.93 < .10	1.70 < .10	0.06 NS

TABLE 58

Z Scores for Mann-Whitney U Tests

and Levels of Significance for Testing

The Proportion of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-ND Exp. vs. SDL-ND Cont.	0.49 NS	1.0 > NS	1.86 < .05	1.46 < .10	1.27 NS
SDL-D Exp. vs. SDL-D Cont.	2.60 < .005	0.81 NS	3.19 < .001	1.46 < .10	1.27 NS
Tran.-ND Exp. vs. Tone-ND Cont.	2.26 < .025	2.0 < .025	0.90 NS	2.08 < .025	0.68 NS
Tran.-D Exp. vs. Tone-D Cont.	2.71 < .005	1.20 NS	0.26 NS	2.32 < .025	1.35 < .10
SDL-ND Exp. vs. Tran.-ND Exp.	1.36 < .10	1.9 NS	1.87 < .05	0 NS	0.06 NS
SDL-D Exp. vs. Tran.-D Exp.	0.12 NS	0.71 NS	1.14 NS	1.0 NS	0.96 NS
SDL-ND Cont. vs. Tone-ND Cont.	1.0 NS	0.39 NS	0.52 NS	1.06 NS	0.49 NS
SDL-D Cont. vs. Tone-D Cont.	0.52 NS	1.21 NS	3.01 < .005	2.01 < .05	0.28 NS

TABLE 59

z Scores for Mann-Whitney U Tests

and Levels of Significance for Testing

The Number of First Trial Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-HD Exp. vs. SDL-HD Cont.	2.24 < .025	1.0 NS	1.86 < .05	1.0 NS	1.86 < .05
SDL-D Exp. vs. SDL-D Cont.	1.46 < .10	0.62 NS	3.0 < .005	0 NS	2.0 < .025
Tran.-HD Exp. vs. Tone-HD Cont.	2.24 < .025	1.0 NS	1.86 < .05	0 NS	1.57 < .10
Tran.-D Exp. vs. Tone-D Cont.	1.46 < .10	0.62 NS	0 NS	1.0 NS	0.62 NS
SDL-HD Exp. vs. Tran.-HD Exp.	0 NS	1.0 NS	0 NS	1.0 NS	0.89 NS
SDL-D Exp. vs. Tran.-D Exp.	0 NS	0.62 NS	1.94 < .05	1.0 NS	1.46 < .10
SDL-HD Cont. vs. Tone-HD Cont.	0 NS	1.0 NS	0 NS	0 NS	1.0 NS
SDL-D Cont. vs. Tone-D Cont.	0 NS	0.62 NS	1.46 NS	0 NS	0 NS

TABLE 60

Z Scores for Mann-Whitney U Tests

and Levels of Significance for Testing

Median Latencies

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-D Exp. vs. SDL-D Cont.	3.51 < .001	0.89 NS	2.52 < .01	0.21 NS	2.95 < .005
SDL-T Exp. vs. SDL-T Cont.	1.37 < .10	0 NS	0.32 NS	0.53 NS	1.79 < .05
Tran.-D Exp. vs. Tone-D Cont.	3.26 < .001	1.0 NS	3.05 < .005	2.20 < .025	3.26 < .001
Tran.-T Exp. vs. Tone-T Cont.	0.74 NS	0.21 NS	0.42 NS	1.26 NS	0.21 NS
SDL-D Exp. vs. Tran.-D Exp.	2.08 < .025	0.11 NS	0.32 NS	2.0 < .025	1.01 NS
SDL-T Exp. vs. Tran.-T Exp.	0.21 NS	0.79 NS	0.53 NS	0.84 NS	1.94 < .05
SDL-D Cont. vs. Tone-D Cont.	0.11 NS	0.26 NS	1.42 NS	0.42 NS	1.10 NS
SDL-T Cont. vs. Tone-T Cont.	0.11 NS	0.63 NS	0.89 NS	0.95 NS	0.53 NS

TABLE 61

z Scores For Comparisons to the
50% (Random) Level of
Responding for Testing
The Number of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-ID Exp.	[a]2.24<.025	3.06<.005	[a]1.02 NS	3.38<.001	[a]0.61 NS
SDL-D Exp.	[a]0.20 NS	2.24<.025	[a]1.02 NS	3.06<.005	[a]1.43<.10
SDL-ID Cont.	3.06<.005	2.24<.025	3.47<.001	2.65<.005	3.06<.005
SDL-D Cont.	3.47<.001	3.06<.005	2.65<.005	2.24<.025	1.84<.05
Trans-ID Exp.	[a]1.43<.10	3.47<.001	1.84<.05	3.38<.001	0.61 NS
Trans.-D Exp.	0.61 NS	3.06<.005	0.20 NS	3.06<.005	1.02 NS
Tone-ID Cont.	3.38<.001	1.84<.05	3.47<.001	.143<.10	3.06<.005
Tone-D Cont.	2.65<.005	1.02 NS	0.20 NS	0.61 NS	1.84<.05

[a]z scores represent differences between the 50% level of responding and performance below that level.

TABLE 62

z Scores for Comparisons to the

50% (Random) Level of

Responding for Testing

The Proportion of Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDI-ID Exp.	2.15<.025	3.23 < .001	1.57 < .10	4.12 < .001	1.72 < .05
SDI-D Exp.	0.47 NS	2.45 < .01	0 NS	3.85 < .001	0 NS
SDI-ID Cont.	3.23<.001	2.45 < .01	3.93 < .001	2.84 < .005	3.55 < .001
SDI-D Cont.	3.93<.001	3.23 < .001	3.67 < .001	2.69 < .005	2.06 < .025
Trans.-ID Exp.	0 NS	3.72 < .001	3.40 < .001	4.12 < .001	1.79 < .05
Trans.-D Exp.	0.73 NS	3.23 < .001	0.67 NS	3.23 < .001	1.27 NS
Tone-I Cont.	4.12<.001	2.06 < .025	3.72 < .001	1.57 < .05	3.29< .001
Tone-T Cont.	3.38<.001	1.69 < .05	1.10 NS	1.56 < .10	2.30 < .025

TABLE 63

z Scores for Comparisons to the

50% (Median) Level of

Responding for Testing

The Number of First Trial Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-D Exp.	[a]0.35 NS	2.48 < .01	0.35 NS	2.48 < .01	0.35 NS
SDL-D Exp.	1.06 NS	1.77 < .05	[a]1.77 < .05	1.77 < .05	[a]1.06 NS
SDL-D Cont.	2.48 < .01	1.77 < .05	2.48 < .01	1.77 < .05	2.48 < .01
SDL-D Cont.	2.48 < .01	1.06 NS	2.48 < .01	1.77 < .05	1.77 < .05
Trans.-D Exp.	[a]0.35 NS	1.77 < .05	0.35 NS	1.77 < .05	[a]0.35 NS
Trans.-D Exp.	1.06 NS	1.06 NS	1.06 NS	2.48 < .01	1.06 NS
Cons-D Cont.	2.48 < .01	2.48 < .01	2.48 < .01	1.77 < .05	1.77 < .05
Cons-D Cont.	2.48 < .01	1.77 < .05	1.06 NS	1.77 < .05	1.77 < .05

[a]z scores represent differences between the 50% level of responding and performance below that level.

TABLE 63

a Scores for Comparisons to the
50% (Random) Level of
Responding for Testing
The Number of First Trial Correct Responses

	Day 1	Day 2	Day 3	Day 4	Day 5
SDL-M Exp.	[a]0.35 NS	2.48 < .01	0.35 NS	2.48 < .01	0.35 NS
SDL-F Exp.	1.06 NS	1.77 < .05	[a]1.77 < .05	1.77 < .05	[a]1.06 NS
SDL-M Cont.	2.48 < .01	1.77 < .05	2.48 < .01	1.77 < .05	2.48 < .01
SDL-F Cont.	2.48 < .01	1.06 NS	2.48 < .01	1.77 < .05	1.77 < .05
Tran.-M Exp.	[a]0.35 NS	1.77 < .05	0.35 NS	1.77 < .05	[a]0.35 NS
Tran.-F Exp.	1.06 NS	1.06 NS	1.06 NS	2.48 < .01	1.06 NS
Tone-M Cont.	2.48 < .01	2.48 < .01	2.48 < .01	1.77 < .05	1.77 < .05
Tone-F Cont.	2.48 < .01	1.77 < .05	1.06 NS	1.77 < .05	1.77 < .05

[a]a scores represent differences between the 50% level of responding and performance below that level.

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