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Gerald D. Pulvermacher
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THE EFFECT OF POSITIVE REINFORCEMENT
ON THE REACTION TIME OF YOUNG BRAIN-DAMAGED
AND NORMAL CHILDREN

by
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B.A., Sir George Williams University, 1968
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A Dissertation
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PREFACE

As in most major undertakings, the person who is generally credited with doing all of the work is in reality only a co-ordinator of the activity of many. It is in this spirit that I would like to express my gratitude to a host of people who have made major contributions to this study.

This research would certainly not have been possible without the direction and resourceful participation afforded to me by my advisor, Dr. B.P. Rourke, and the help of his staff, particularly Mrs. J. Orr. Equally deserving of mention are the other members of my committee, Dr. R. Orr, Dr. G. Holland, and Dr. A. McGhie, who were all instrumental in guiding the course of my work. I would also like to thank the Kent County Board of Education, the Windsor Hebrew Day School, and the I.O.D.E. Hospital, for giving me the opportunity to gather my data.

Finally, there are three people who are deserving of some special recognition. First, I extend my appreciation to Mrs. M. Hutchinson, who did such an admirable job in typing this manuscript. Secondly, there is little question in my mind that I would not have reached this point without the support, encouragement and understanding of my wife. And to Tracy, who frequently could not comprehend why her daddy was always working, I give all my love.
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CHAPTER I
INTRODUCTION

Purpose
The purpose of this study was to determine the effect which positive reinforcement would have on the performance of young brain-damaged (BD) and normal children on a reaction-time (RT) task. The independent variables under investigation were the following: neurological impairment and reinforcement versus non-reinforcement conditions. This introductory section deals with some theoretical formulations of attention, the input dysfunction model of psychopathology and its relation to schizophrenia and brain damage in adults and children, a behaviour-analytic analysis of attention, operant conditioning techniques, a statement of the problem, and the hypotheses under investigation.

Early Theories of Attention
Although Malebranche set forth the theory which identified "attention" with "clearness in consciousness" in the 18th century (Spearman, 1937), it was not until the 1950's, thirty years after McDougall's (1928) work appeared, that theoreticians and researchers again began to address themselves to a problem which has been almost as complex to define as that of "intelligence." A survey of early theories may help to explain the reasons why work in this area had generally been abandoned for such an extended period of time.
Among the first laboratories to address itself to the problem of "attention" was that of Wundt (Moray, 1969). "For Wundt it was attention which turned perception into apperception, and so played a central role in the account of sensation and perception (Moray, 1969, p.1)."

Titchner (1903) believed attention to be synonymous with "sensory clearness" and his elucidation of factors which might account for the role which attention played in perception left little to be added by modern day researchers. Amongst those factors he included intensity, extension (in space), duration, "certain qualitative aspects" (e.g., emotionally toned stimuli), repetition, suddenness, movement, novelty, association with ideas already present, accommodation of sense organs and the cessation of a stimulus. Berlyne (1951) later observed that one could infer "sensory clearness," or the intensity of attention, from behaviours such as the ability to recall a stimulus, the amplitude, latency and efficiency of the response, and the resistance of the response to extinction, satiation, and fatigue.

There were others who defined "attention" in terms of a definite concrete process. James (1890), and other members of the Functionalist School, maintained that attention is an active process whereby the subject actively attends to particular stimuli in the environment. While Oppenheimer (Spearman, 1937) felt that attention occurred as an early stage in a process, "Volkmann concluded that attention was a "holding steady." Lloyd Morgan, on the other hand, combined both stages: "We may describe attention as the bringing of something to the focus of consciousness and the holding it there."
With the appearance of Behaviourism, interest in "attention" began to decline. The cause of this regression is to be found in the swing in psychological methodology from an emphasis upon the phenomenal aspects of experience to the behavioural aspects, and the consequent tendency to reject terminology that had acquired a mentalistic tinge. Daishell's (1937) statement was characteristic of the objective position on attention:

"When a person takes up an attitude that will facilitate his response to some particular stimulus or stimuli, that attitude goes by the name of attending or attention (p.285)."

Thus, when Broadbent introduced his theory of attention in 1958, he noted that "attention," as a legitimate area of study for psychologists, had fallen "into bad odour because of the inability of introspective psychologists to agree with one another, or to provide objective evidence to back their assertions (p.109)." Moray (1969) attributed the renaissance of interest in the area of attention to three developments: (1) the use of operational definitions to describe the process of attention; (2) the advent of the Second World War and its demand on human operators to handle a high flow of information; and (3) the appearance of sophisticated laboratory equipment which made the study of attention more precise and meaningful.

In his discussion of attention, Moray (1969) noted that "attention" can denote several different processes. "Vigilance," for example, refers to signal detection, or the ability to detect some event as soon as it occurs, against a background of nothing happening. "Activation," en-
sentially means to get ready to deal with whatever happens next. Moray defined "set" as "a preparation to respond in a certain way, either cognitively (as in perceptual defense) or by overt motor responses (as in motor preparatory set)." Finally, "selective attention" refers to the ability to select and respond to one message when we are in fact faced by several messages; in other words, how do we ignore irrelevancies and attend to relevant information in the environment.

**Broadbent's Theory of Selective Attention**

Although numerous models of attention have been expounded in an attempt to explain the selective processes of attention (Berlyne, 1951; Deutsch & Deutsch, 1963; Hebb, 1949; Kristofferson, 1965) only Broadbent's (1958) model has been systematically applied to describe the cognitive and performance deficiencies in psychopathology. Broadbent (1958) advanced a theory of selective attention in which he hypothesized that all communication is channelled through the nervous system. To account for the observation that the organism is capable of responding selectively even when faced with a host of stimuli, despite its limited capacity for processing information, Broadbent proposed a "filter" theory of attention.

Broadbent (1958) regards the whole nervous system as a single channel, having a limit to the rate at which it can transmit information (channel capacity). The meaning that he ascribed to this postulate was that an organism will be unable to cope with more than a certain number of signals within a fixed period of time. This does not mean that one cannot do more than one thing at a time. This depends on the novelty of the stimuli. For example, a man can drive down a familiar street and talk
about his daily activities with his wife at the same time, doing both efficiently. He cannot, however, both drive down a new road and talk to his new boss at the same time without impairing his efficiency at one or both.

Broadbent further postulated that the limited capacity portion of the nervous system is preceded and protected by a selective device or filter, which passes only some of the incoming information. More recently, Broadbent (1971) has hypothesized that there may be additional mechanisms to help in the filtering process.

This selection process is not random and certain stimulus events have a greater probability of being selected due to the following: physical intensity, time since the last bits of information from that class of events entered the limited capacity channel, high frequency of sounds as opposed to low, and sounds as opposed to visual stimuli. Stimuli which the organism is more likely to attend to are also a function of the hierarchy of drives. Thus, if the organism is hungry, it will attend to stimuli which connote food. That information which is sensed and not selected into the channel is stored for short periods of time (seconds) in a short-term memory bank. Information from this store will be used if stimuli which are related to it become relevant. This must occur in a relatively short period of time, otherwise it will be lost.

Broadbent's "Filter Theory" and its Relationship to McGhie's Theory of Psychopathology of Selective Attention

A review of the literature revealed that, although the concept of attention has played a crucial role in various areas of applied psychology,
only one of the models of selective attention discussed earlier has been systematically applied to promote our understanding of psycho-pathology. McGhie (1969) proposed a theory of schizophrenic develop-
ment in which he regards the organism as an information-processing system which must selectively respond to relevant aspects of the en-
vironment and inhibit responses to irrelevant stimuli. This theory developed from Broadbent's (1958) model and Shakow's (1962) demonstra-
tion that schizophrenic subjects have greater difficulty than normals in maintaining a state of response readiness on psychomotor tasks where RT is the crucial variable. With practice, the normal individual no longer needs to attend to old stimuli, and focuses his attention on new ones. The schizophrenic, however, is not only deficient in his ability to focus on relevant stimuli but he is also influenced by peripheral stimuli. The schizophrenic reacts to old stimuli as if they were new ones, and he "perseverates," or reacts to new situations as if they were old. This breakdown in information processing is assumed to underlie all other symptomatology, including bizarre language, feelings of change and depersonalization, difficulty in reality testing and, especially, inefficiency in performance on cognitive tasks.

McGhie and Chapman (1961) speculated as to how a normal adult de-
velops an efficient information-processing system and correspondingly suggested a hypothesis for the breakdown of this system in schizophrenics. An outline of their model is as follows. Early in the child's develop-
ment, he is conceived of as being a passive receptor of all sensory infor-
mation, both from internal and external sources. At some point in time,
the child must learn to control and organize the incoming flow of sensory stimuli, and this involves an integration of present experience with past events to provide a degree of perceptual constancy. "By such processes we reduce, organize and interpret the otherwise chaotic flow of information reaching consciousness to a limited number of differentiated, stable and meaningful percepts from which our reality is constructed (McGhie & Chapman, 1961, p.112)." In the case of the schizophrenic, the faulty development of the "filter" mechanism leads to a breakdown in the individual's attention functions, hence exposing him to a flood of sensory impressions, not only from internal and external sources, but also from associations to these stimuli and past experiences; which have been stored in the memory bank. In other words, the schizophrenic again becomes a passive receptor of sensory information as in early childhood.

Thus, when asked to perform a cognitive task involving either perception, association or conceptual thinking, the level of performance of the members of the schizophrenic group is invariably inferior to that of the normal controls. From the point of view of this input dysfunction model of schizophrenia, this deficit in performance can be attributed to the breakdown in the selective functions of the information-processing system, with the ensuing flood of stimulation interfering with performance.

In the area of perception, for example, psychomotor tasks, in which RT is the dependent variable, require that the subject maintain a state of response readiness, which Shackow (1962) called a "major set." The RT
task is structured so that there is a warning signal which indicates
the beginning of a trial, a preparatory interval (PI), which is that
period of time between the onset of the warning signal and the onset
of the RT stimulus, and finally the RT stimulus itself. In order to
respond appropriately and efficiently, the subject must disregard all
irrelevant stimuli, or "minor sets," during the PI. It is here that
the schizophrenic is expected to encounter some difficulty (McGhie,
1969).

The hypothesis that the performance of schizophrenics should be
poorer than that of normals on psychomotor tasks because of their in-
ability to inhibit attention to minor, irrelevant sets, leads to three
testable deductions (Bass, 1966). First, schizophrenics should have
slower RTs than do normals. Not only is there a good deal of evidence
to support this hypothesis (e.g., Shakow & McCormick, 1965), but it has
also been found (King, 1961) that the faster the RT, the better the
mental health rating among psychiatric patients. The second hypothesis
states that variations in stimuli should adversely affect schizophrenics
more than normals. Due to the schizophrenic's difficulty in maintain-
ing a major set, it is expected that varying the length of the PI will
adversely affect schizophrenics more than it will normal subjects. All
the evidence (e.g., Rodnick & Shakow, 1940) supports this prediction.
In addition, a study by Zahn, Rosenthal and Shakow (1963) demonstrated
that schizophrenics were especially hampered by a long PI on the preced-
ing trials. Apparently, they had the tendency to perseverate; that is,
if the previous trial was a long one, then they would "expect" the sub-
sequent interval to be long as well. Similarly, Chapman and McGhie (1962) and McGhie, Chapman and Lawson (1964, 1965) demonstrated that schizophrenics have more difficulty attending to relevant aspects of a stimulus situation if distracted by irrelevant aspects than do normals.

Finally, conditions that enhance maintaining the major set are expected to improve the performance of schizophrenics more than that of normals, and should eliminate the relative decrement in schizophrenic performance. The results of studies which have utilized aversive stimulation at the precise moment when the relevant stimulus is presented (e.g., Pascal & Swensen, 1953), have demonstrated that schizophrenic subjects improved their performance to a greater extent than did normals.

Similar results have been reported in studies in which performance is readily interfered with by the inability to inhibit bizarre associations. These bizarre associations have been observed to act as distractors and result in performance deficits (Chapman, 1958).

Cameron (1938, 1939) suggested that the performance of schizophrenics on various category sorting tasks was deleteriously affected by their inability to confine their conceptual thinking within the narrow range of concepts set by the sorting tasks (cited by McGhie, 1969). He found that this was due to their tendency to formulate concepts on the basis of irrelevant features of the objects or upon personal associations with the objects. He called this tendency "overinclusive ness." As an explanation, Cameron suggested that the difficulty which the schizophrenic encountered was due to a disturbance in his ability to attend to the
relevant aspects of the stimulus situation. Chapman (1956), based on
the findings of Payne and his colleagues (Payne, Caird & Laverty, 1964;
Payne & Hewlett, 1960; Payne, Mattusseck & George, 1959), was of the
opinion that overinclusiveness is another example of the breakdown in
the schizophrenic's "filter" mechanism.

The Input Dysfunction Model and Brain-Damage

McGhie (1969) has further speculated that the performance of brain-
damaged individuals can also be explained in terms of a breakdown in the
information filtering abilities of these persons. Although the be-
behavioural concomitants of brain-damage may vary considerably due to the
nature of the injury, the extent and severity of the damage, as well as
the personality of the individual, certain behavioural manifestations
are still considered to be quite characteristic of such patients. McGhie
(1969) notes that the key symptoms of the "brain-damage syndrome" include:

1) ...impairment of comprehension, interference
with elaboration of impressions, defects in
orientation and retention, difficulty in ac-
tivation of memories and marked fluctuation
of the level of attention.

2) Affective disorder in the forms of emotional
stability...

3) Character change in the form of conduct foreign
to the patient's natural disposition...

(Henderson & Gillespie, 1962)

Following Goldstein's (1939) observations, McGhie notes that one of
the most common difficulties which brain-injured people encounter, and characteristic of a deficit in attention, is an inability to ignore unessential details in their environment. Goldstein pointed out that not only could the disturbed patient not limit his attention to the interviewer's questions, but in addition, when placed in an experimental situation, his performance on a task requiring the identification of figures was deleteriously affected by the background in which the figure was embedded. An examination of the research, however, leads one to believe that not all the behavioural manifestations of brain-damage are the same for adults and children.

Experiments Employing Brain-Damaged Adults

Rosvold, Mirsky, Sarason, Bramsone, and Beck (1956) designed the Continuous Performance Test (CPT) to measure sustained attentiveness in experimental subjects. The design of the instrument was based on certain electroencephlographic (EEG) evidence which suggested that brain-damaged individuals would show inferior ability, as compared to non-brain-injured people, on tests requiring sustained attention. The waking EEGs of many brain-damaged patients show either random bursts of hypersynchronous (high amplitude) activity intruding upon normal activity of the brain or a general hypersynchrony. Hypersynchronous activity is also evident from the recording of the brain of a sleeping subject. If hypersynchrony is associated with reduced attention, as suggested by its presence during sleep, then the hypersynchrony of patients with cerebral dysfunction might also indicate reduced attention. Using the CPT technique, Rosvold and his colleagues were able to demonstrate defects of attention in brain-damaged individuals.
If, in fact, brain injury does cause a deficit in attention, it might be expected that brain-damaged patients would manifest poor performance on RT tasks. DeRenzi and Faglioni (1965) found a positive correlation between scores obtained on a visual RT test and degree of brain-damage. They concluded that latency on such tasks might prove to be a sensitive measure of the severity of brain-damage. Costa (1962) demonstrated that the RTs of brain-injured subjects were significantly longer than those of normal subjects under all PI conditions. Their findings essentially replicated the results of Benton and Blackburn (1957) and Benton and Joynt (1959). Independent studies by McDonald (1964), where the interstimulus interval was varied, and Benton, Sutton, Kennedy, and Brokaw (1962), who examined RT as a function of whether the stimulus was preceded by an identical stimulus, a different stimulus of the same modality, or a different modality stimulus, found that there was no significant difference between the performance of the brain-damaged group and the normal controls. Thus, the results with respect to attentional deficits in brain-damaged adults are somewhat equivocal.

Experiments Employing Brain-Damaged Children

Results of experiments which involved comparisons of brain-damaged and normal children on a number of cognitive tasks are much more conclusive than are the results of studies with adult subjects. Werner and Strauss (1939) investigated Goldstein's "figure-ground syndrome" using brain-damaged and normal children as their subjects. It was concluded that the inferior attempt of the brain-damaged children to identify a pattern from within a distracting background was caused by their greater
susceptibility to distraction from irrelevant stimuli in the ground. As the background became more distracting (an effect produced by varying the figure and ground to differing degrees) their performance in comparison to normals and non-brain-injured defective children became correspondingly worse (Werner & Strauss, 1941).

To determine whether this attentional deficit was limited to the visuomotor field, Werner and Strauss (1941) constructed an equivalent of the Marble-Board Test to assess the performance of brain-damaged children in the tactual modality. The subjects were to run their fingers over a board with raised rubber tacks and identify a figure which was embedded in other tacks having flat enamelled tops. The disrupting effect of the distracting stimuli was again recorded and the findings were replicated on tasks requiring auditory perception (Werner & Bowers, 1941), memory (Werner, 1946), and concept formation (Werner, 1949). "It would appear, therefore, that a deficit in attention constitutes a more general factor in organic impairment in children (McGhie, 1969, p.147)."

Spivack (1963) has criticized the methodology of the figure-ground experiments. His primary concern is that the ground in which the figure is embedded often occupies the dominant amount of space and thus the subject's poor performance may reflect his indecision regarding the correct test strategy; that is, the subject may report the ground instead of the figure. A further complicating factor (McGhie, 1969) is that the distracting stimuli which the subjects are required to ignore may themselves be meaningful, therefore requiring a response from the subject. "It follows, then, that a low score on the figure-ground task may reflect a
conflict between the experimenter and the subject as to what stimuli are relevant or irrelevant rather than a failure to inhibit responsiveness to known irrelevancies (McGhie, 1969, p.111)."

The fact remains, however, that numerous reports exist in the literature which indicate that the performance of brain-injured children is inferior to normals on most experimental tasks. Stevens, Boystun, Dykman, Peters, and Sinton (1967) concluded from their work that a deficiency in attentional processes was one of the primary defects among minimally brain-damaged children. Luria (1961), noting that his experimental group of brain-damaged children performed at a level significantly inferior to the normal controls on a task which required the subject to respond to only one of two stimuli, suggested that the cardinal feature of children with cerebral dysfunction is an inability to inhibit responses to irrelevant stimuli. Campanelli (1968) found that brain-damaged children between the ages of 8 and 12 made significantly more errors on the CPT than did the normal controls. Furthermore, he found that those brain-injured children with non-focal lesions were more severely disturbed than were children with focal lesions.

It seems apparent, therefore, that children with brain-damage are indeed inferior on tasks requiring sustained attention. A number of studies (e.g., Czudner & Marshall, 1967; Czudner & Bourke, 1972; Bourke & Czudner, 1972) have addressed themselves to the problem of identifying a brief assessment procedure which would reliably differentiate between brain-damaged and normal children on the basis of this demonstrated inequality in attentional abilities.
Proposition 1. The performance of brain-damaged children on a task which requires the ability to maintain a state of response readiness, while filtering out irrelevant cues, should be inferior to that of normal children.

Reaction Time as a Dependent Variable

Rodnick and Shakow (1940) demonstrated that RT on psychomotor tasks is a highly reliable dependent variable which consistently differentiates between normal and schizophrenic adults. Czudner and Marshall (1967) replicated the findings of Rodnick and Shakow, using retarded, normal and schizophrenic children as the experimental subjects. They found that, under both regular and irregular preparatory interval conditions, the RT of the schizophrenic group was significantly inferior to those of the other two groups. Noting Goldfarb's (1961) observations (i.e., that schizophrenic children might also be afflicted with neurological deficits) Czudner and Marshall re-evaluated the performance of their schizophrenic subjects and found that those who were suspected of having neurological impairment were, in fact, inferior on tasks requiring them to maintain a state of response readiness.

A subsequent study (Czudner & Rourke, 1970) confirmed the hypothesis that children with cerebral dysfunction perform more poorly than do normal subjects on a visual RT task. An additional finding, which is especially relevant to the study about to be proposed, was that although brain-damaged subjects were more adversely affected than were normals when the length of the FI was varied in a random fashion, it was also found that these differences were prominent at younger age levels (8-10),
whereas they tended to disappear at older age levels (11-14). Similar results (Czudner & Rourke, 1972; Rourke & Czudner, 1972) were found when RT was evaluated in the auditory and visual modalities. Czudner (1971a) attempted to explain this developmental difference on the basis of reactive inhibition ($I_R$). He hypothesized that younger subjects with BD (6-9) are more influenced by (pay more attention to) the length of the PI on previous trial, since they apparently have a greater deficit in attention. Consequently, they build up a greater amount of $I_R$ which then takes longer to dissipate. This being the case, they are not totally prepared for the subsequent trials. More recently, Czudner (1971b) has interpreted this difference in performance as being the consequence of motivational inequalities. That is, young subjects with neurological impairment appeared to be less motivated to sustain attention and perform to capacity than did the older brain-damaged subjects.

The view that performance of subjects with brain-damage on an RT task is adversely affected due to lack of motivation is not new. Goldstein (1942) was one of the first to suggest that poor RT performance was due to a lack of motivation. Although his subjects consisted mainly of adults who had sustained brain injuries during the war, he reported that using simple and choice RT as dependent variables, the patients showed RTs well within normal limits, at least during the first ten trials. On the ensuing trials, however, these patients demonstrated both a lengthening in RT and an increase in variability, whereas normal subjects did not manifest these changes within the limits investigated (30 trials).

The work of subsequent researchers (Benton & Blackburn, 1957; Black-
burn, 1958; Farber & Spence, 1956) reinforced Goldstein's hypothesis. Blackburn (1958) introduced, for example, two types of motivational cues into the experimental situation. One group of subjects was reinforced verbally for RT performance, either positively ("good" or "fine") or negatively ("poor" or "too slow"). A second group was given relaxing or reassuring instructions ("you've been doing fine so far but just relaxing seems to help everybody"). In comparing the performance of his brain-damaged group with that of his normal controls, Blackburn found that following the presentation of his standard instructions (which did not include any phrases designed to increase motivation), the brain-damaged group showed a decrement in performance while the controls showed improvement. Both groups manifested performance improvements under relaxation instructions, although the increment for the brain-injured group was greater than that for the controls (the difference, however, did not reach a statistically significant level). Within groups it was observed that verbal urging and censure produced the greatest gains, especially within the brain-damaged groups. Shankweiler (1959) replicated this study and found similar results.

A more contemporary approach to the study of the motivational effects on the performance of subjects with brain-damage is being directed towards the systematic manipulation of external contingencies following desired performance. The technique which has been used to promote attending behaviour is operant conditioning.
Principles of Operant Conditioning

Operant conditioning procedures have been used to modify various deviant, disruptive, or maladaptive human behaviours (Yates, 1971). An "operator" may be defined as any behaviour which can be controlled by its environmental consequences (Bijou & Baer, 1961). One may conceptualize the situation as follows: a behaviour is usually emitted at a certain frequency and this frequency level may be altered by the consequences which follow that behaviour. There are at least two general ways in which a response can have stimulus consequences. Stimuli which strengthen the immediately preceding response are termed "positive reinforcers." Some stimuli, when subtracted from the environment, also strengthen the immediately preceding response, and these are called "negative reinforcers."

To adequately discuss all possible response contingencies one must include those stimuli which, when added to the environment, weaken the immediately preceding response. Thus, the introduction of "punishing stimuli" following a response will reduce the probability that the response will again be emitted. Similarly, there are stimuli which, when subtracted from the environment, will also weaken the immediately preceding response. For example, taking a desired toy away from a child is a form of punishment.

One other condition which may follow a response is the discontinuance of the contingency. This pattern is termed "extinction" and its results are that the response returns to the level of strength characterizing it before it was altered by the reinforcement or punishment contingency. This level has been referred to as the "operator level."
Of extreme importance to the technology of operant conditioning is the principle of "discrimination." This principle describes the frequent observation that the effects of reinforcement, punishment and extinction are restricted to the stimuli settings in which they take place. This is frequently observed in attempts to modify disruptive classroom behaviour. For example, in one setting (say, the school) the response may be extinguished, but at home it may be positively reinforced. Where there are stimuli correlated with the typical consequences which a response will meet, should it occur, those stimuli usually acquire a controlling function of the response. They are termed "discriminative stimuli." When discrimination fails, the result is usually called "stimulus generalization."

**A Behaviour Analytic Interpretation of Attending**

Some authors (e.g., Cromwell, et al., 1963; Strauss & Kephart, 1955) maintain that short attention span, distractibility and hyperactivity refer to different phenomena. Despite this, differences among the behavioural referents of these concepts are often difficult to specify. Cromwell states: "According to their (Strauss' and his associates') conception of the brain-injured child, an environment of overstimulation should exaggerate the symptoms of distractible behaviour, short attention span, and superactivity (1963, p.64)." In short, it appears as though the above researchers have dealt with the behaviours of short attention span, distractibility and hyperactivity as distinct phenomena.

An operant analysis presents a paradigm of "attention span" which refers to a series of specific behavioural events. Thus attending
behaviour is considered to be synonymous with task perseverance which is under the influence of positive reinforcement, whereas other behaviour which is incompatible with attention goes unreinforced. On the other hand, a short attention span is observed when reinforcement is contingent upon behaviour that is incompatible with attending to the task of interest, and attending behaviour goes unreinforced.

The Use of Positive Reinforcement to Modify Attending and Related Behaviours in Children

The most commonly used principle of operant behaviour modification has been positive reinforcement. Positive reinforcement has been applied to the problems of increasing behaviours currently occurring at low rates, reinstating behaviours once present but no longer exhibited, substituting appropriate responses for avoidance behaviour, and building completely new behavioural repertoires (Yates, 1971).

Positive reinforcement has been used to develop increased social interaction with other children (Allen, Hart, Buell, Harris & Wolf, 1964; Baer & Wolf, 1968), increase climbing and other forms of active play (Johnston, Kelley, Harris & Wolf, 1966) and increase non-crawling behaviour (Harris, Johnston, Kelley & Wolf, 1964). Birnbrauer, Bijou, Wolf and Kidder (1965) have described a procedure in which "token" reinforcers, exchangeable for food, toys, and school supplies, have been used in conjunction with programmed instruction procedures to develop and maintain academic study behaviour in retarded children. Kerr, Meyerson and Michael (1965) established vocal responses in a previously
mute retarded girl by reinforcing any vocalization which the child emitted. Giles and Wolf (1966) established appropriate defecation and urination in several retarded males by reinforcing approaches to, then sitting on, and then successful elimination in the toilet. Conditioning techniques employing positive reinforcement have also been applied to the control of operant crying (Hart, Allen, Buell, Harris & Wolf, 1964) and the elimination of tantrum behaviour (Williams, 1959).

A series of recent studies have been concerned with the problem of increasing the attention span of children who are typically described as being either hyperactive, brain-damaged, or both (e.g., Allen, Henke, Harris, Baer & Reynolds, 1967; Martin & Powers, 1967; Patterson, Jones, Whittier & Wright, 1965; Walker & Buckley, 1968). In almost all cases, the children in these studies were reported to be verbally and physically provoking other children, not completing tasks, talking out of turn, and being easily distracted from a given task by ordinary classroom stimuli such as minor noises, movements of others and even changes in lighting conditions.

Experimenters have employed a variety of positive reinforcement models in an attempt to modify attending behaviour. In the Walker and Buckley (1968) study,

...the subject was told that when a given interval of time had elapsed, in which no distractions had occurred, a click would sound and the experimenter would enter a single check mark in a cumulative re-
cording form which would indicate that the subject had earned a point. The subject was told that attending to the click represented a distraction and would result in loss of the point for that interval. The subject was allowed to exchange his points for a model of his choice at the conclusion of the treatment period. The number of points necessary for the model (160) was specified to the child when treatment began. ...Attending behaviours for the subject involved looking at the assigned page, working problems, and recording responses. Non-attending behaviours were defined as those which were incompatible with task-oriented (attending) behaviour (p.246).

Allen, et al. (1967) used social reinforcers, including talking to the subject from a distance of three feet or less, touching, and supplying him with additional materials for his activity. Techniques of withholding these reinforcers consisted of turning away from the subject, not looking or smiling at him, not speaking to him, and attending to other children in the classroom. Patterson, et al. (1965) used a combination of candy and social reinforcement to improve the attending behaviour of two young boys, aged 10 and 10½ years, both of whom had incurred injuries to the brain.

Kerr (1962) worked with two brain-injured girls who supposedly had short attention spans and were irresponsible and hyperactive in the classroom (based on hospital staff reports). He devised the task of folding 2x5 cards along a dotted line and placing each folded card in a container. During the first session, the traditional technique of urging the child to continue was used whenever
a lag in productivity occurred, i.e., the experimenter would say such things as, "come on, you can do better than that," and approximately 10 minutes of productive behavior was obtained. During the next two sessions, experimenter attention was made contingent upon task performance and a full hour of work was obtained each time. Two more sessions of the traditional technique and two more reinforcement sessions replicated these efforts.

In every case described above, the attending behavior of the children improved substantially when compared to their operant level. Systematic variation of the treatment conditions, that is, making reinforcement non-contingent upon the production of the appropriate responses, or the introduction of extinction conditions, resulted almost immediately in a drastic reduction in the desired behavior. When the response-contingent reinforcement conditions were re-introduced, the frequency of the desired behavior improved concurrently. It may be hypothesized, therefore, that the judicious dispensing of positive reinforcement can result in the modification and control of attending behavior.

**Proposition 2.** Under conditions of positive reinforcement, there should be less difference in RT performance between young BD and normal children than under no reinforcement conditions.
Problem

The independent variables under investigation included the presence or absence of neurological dysfunction and reinforcement versus non-reinforcement conditions. The dependent variables were RT to a visual stimulus, number of blocks of trials completed, and number of reinforcers earned.

The purpose of this study was to answer the following questions. Would the application of a positive reinforcement procedure enable the young brain-damaged child to improve his performance on a task in which the primary ability called for was that of maintaining a state of response readiness? Furthermore, would the introduction of this contingency affect the young brain-damaged child's performance in such a way that it would be no different from that of the young normal child's? If so, the ramifications of these findings for the treatment and training of young children with cerebral dysfunction would seem important.

Hypotheses

(1) Under conditions of positive reinforcement, there should be less difference in RT performance between young brain-damaged and normal children than under no reinforcement conditions.

(2) There should be no difference in performance between those normal children receiving PR and those under NR conditions.
CHAPTER II

METHODOLOGY AND PROCEDURE

Subjects

Two groups of subjects were employed: (1) a "brain-damaged" or experimental group, and (2) a normal or control group. These two groups, consisting of 14 subjects (Ss) in the brain-damaged group and 16 in the normal group, were further subdivided into brain-damaged and normal groups receiving positive reinforcement (PR) and brain-damaged and normal groups receiving no reinforcement (NR).

Group 1 consisted of 14 children aged 6 years 8 months to 8 years 3 months, selected from the files of the Neuropsychology Unit at I.O.D.E. Hospital in Windsor. In actuality, there were 6 additional Ss tested. All six had to be excluded, however, for reasons related to the criteria for selection. The brain-damaged Ss were selected on the basis of neuropsychological and electroencephlographic (EEG) evidence of cerebral dysfunction (Appendix A describes the neuropsychological and EEG history of each S). The "neuropsychological evidence" of cerebral dysfunction "means that a "blind" interpretation of the results of a battery of neuropsychological tests....judged these
results to be consistent with the presence of cerebral dysfunction (Rourke & Czudner, 1972)." Furthermore, in reviewing the electroencephlographic indications of brain damage, it is probably true to say that, in general, these Ss were suffering from what would best be described as relatively mild, chronic cerebral dysfunction.

The children in the control group were selected from the Kent County Public School system. The children tested under PR conditions came from one school, and those under NR were from another. Attempts were made to match these Ss as closely as possible with their counterparts in the brain-damaged group on the variables of age, sex, and I.O. This proved to be a difficult task for the later two variables. It has been previously found (Jones, 1937), however, that differences in sex should not contribute to RT performance, and that there is no correlation between I.O. and RT, other than when the two extreme groups (below 80 and above 115) are compared (Scott, 1940). The Pearson Product-Moment Correlation Co-efficient obtained in the present study for I.O. and RT yielded a co-efficient of -.14, which proved not to be statistically significant. No S with any detectable sensory or motor impairment, based on teachers' reports of classroom and academic behaviour, were included in the control group. A short form of the WISC was administered to the potential Ss to assure that their Full Scale I.O. score fell within the limits achieved by the experimental group. The overall average of the estimated Full Scale I.O. was 91-109 with a mean of 101.
TABLE 1
DESCRIPTION OF SUBJECTS

<table>
<thead>
<tr>
<th></th>
<th>Normal Subjects</th>
<th>Brain-Damaged Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR</td>
<td>NR</td>
</tr>
<tr>
<td>A. Age:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (months)</td>
<td>92</td>
<td>88</td>
</tr>
<tr>
<td>Range (months)</td>
<td>89-95</td>
<td>84-94</td>
</tr>
<tr>
<td>Standard Deviation (months)</td>
<td>1.884</td>
<td>3.354</td>
</tr>
<tr>
<td>B. I.O.:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>102.62</td>
<td>101.25</td>
</tr>
<tr>
<td>Range</td>
<td>96-109</td>
<td>91-108</td>
</tr>
<tr>
<td>Standard Deviation</td>
<td>4.808</td>
<td>7.516</td>
</tr>
<tr>
<td>C. Sex:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Female</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>
Glasser and Zimmerman (1969) reviewed approximately twenty abbreviated forms of the WISC. Addressing themselves to the reliability of this form of intellectual assessment, they noted that there should be at least a correlation of .90 between the brief form and the Full Scale score. A correlation of this magnitude resulted in a variance of no more than nine I.O. points above or below the "true" I.O. in two-thirds of the cases. They further reported that, as the number of subtest combinations were increased, the reliability tended to increase correspondingly.

Based on the Glasser and Zimmerman (1969) review of the different abbreviated forms of the WISC, the following combination of WISC subtests were used to assess the intellectual functioning of the normal sample: Comprehension, Arithmetic, Vocabulary, Picture Arrangement and Object Assembly. At age levels 7½, 10½ and 13½, this combination correlated with the Full Scale score .94, .96 and .96 respectively.

Apparatus and Procedure

The apparatus consisted of two Hunter Decade Interval Timers (Model 100C and 111C), one Hunter Klockcounter (Model 1520) with an accuracy of 0.001 seconds and a Hunter Timer (Model 1512). The RT stand consisted of a telegraph key, and two 7.5-W light bulbs, one serving as a warning signal (white light) and the other as the RT stimulus (red light). In addition, a dispenser was mounted on the RT stand which automatically dropped a token (shiny penny) into a plastic box, in full view of the S. At the conclusion of the session, the tokens were redeemed for a small toy (see Appendix B). These toys,
each having a predetermined value, were displayed in the experimental room (the values were displayed along with the corresponding item). The timers were arranged so that the experimenter (E) was able to manipulate the length of the PI. An assistant recorded the RT, the number of reinforcers earned and reset the timer following each presentation. The Klockcounter was connected to the telegraph key so that the S had to press the key to set the warning signal. Releasing the key before the appearance of the red light stopped the apparatus and the trial was repeated (this occurred less than 2 percent of the time).

To initiate the session, each S was given three practice trials at each of the four levels of PI, that is 2-, 4-, 6- and 8-seconds. In this phase, the PI levels were presented under the regular condition (Czudner, 1971a), and the S was given instructions to that effect, as well as being told to respond as quickly as possible (see Appendix C - Instructions). Each S, therefore, had a total of twelve practice trials.

The scores achieved on the first block following the practice procedure were considered as being baseline trials. This block consisted of eight trials, two at each PI level, but under irregular (Czudner, 1971a) PI conditions. At this stage, neither the NR or PR group were treated differently. They were again instructed to respond as quickly as possible.

During the experimental phase, all the Ss were exposed to the irregular procedure only, that is, the PIs were presented in a random
order for at least five blocks of trials (see Appendix D for order of presentation). The order of presentation for each successive S was counterbalanced. That is, half the Ss were administered the PI in the forward direction, the other half in the reverse direction. Again, all Ss were told to respond as quickly as possible and to always try to improve on their previous effort. The PR group received the additional instruction that each time that their RT latency was faster than a criterion set by the E, a token would automatically be dispensed. It was explained how these pennies would be used to buy toys. They were then asked to inspect the toys in the room which were displayed in such a way that they would not distract them during their performance.

Each S in the PR condition was exposed to at least five blocks of trials. Thus, at least 40 trials beyond the practice (12 trials) and baseline (8 trials) blocks were completed by each S. If, however, S had earned at least two reinforcers on the fifth experimental block, he was then exposed to additional blocks of trials until such time as he had received either one or zero reinforcers in any one block. At this point, the experimental session was terminated.

Following a session with a S who had been included in the PR group, the next S was then included in the NR group and his trial blocks were yoked to that of the immediately preceding S who was exposed to the PR condition. For example, if the first S to be tested was assigned to the PR condition and he had successfully completed seven blocks beyond the practice and baseline trials, then the next S in the NR group also had seven blocks of trials beyond the
the practice and baseline blocks. The Ss in the NR groups, of course, were not given the opportunity to earn tokens for their performance. They were simply instructed to perform as quickly as possible and to try to improve their performance on each succeeding trial.

As was mentioned previously, the criteria which the Ss in the PR group had to meet in order to earn a reinforcer were set by the E. This criterion (.800 seconds) was initially set at a level where all Ss in the PR condition, be they normal or brain-damaged children, were expected to succeed in earning the required number of reinforcers. This criterion (which was based on the slowest RT score achieved by a brain-damaged subject under irregular PI conditions in the Cudner and Rourke (1972) study) was subsequently decreased by 0.050 seconds at the beginning of each new block of trials.
CHAPTER III
PRESENTATION AND ANALYSIS OF RESULTS

Experimental Design

The data were analyzed by the application of three analyses of variance and two t-tests. A 2x2x2x4 analysis with repeated measures on the last two factors was carried out to determine whether a statistically significant difference existed between the RT of the brain-damaged and normal subjects prior to the introduction of treatment, that is, the "baseline" period. There were two groups (i.e., brain-damaged vs. normals), two types of treatment (i.e., PR vs. NR), two trials at each level of PI, and four levels of PI (2-, 4-, 6- and 8-seconds).

A 2x2x4x8 analysis, with repeated measures on the last two factors, was employed to determine whether PR had a beneficial effect on the performance of brain-damaged and normal subjects. There were two groups (i.e., brain-damaged vs. normals), two forms of treatment (i.e., PR vs. NR), four levels of PI (i.e., 2-, 4-, 6- and 8-seconds), and eight blocks of trials. The first eight blocks were selected for the analysis because this was the least number of blocks achieved by the poorest S in any group.

A 2x2x3 analysis, with repeated measures on the last factor, was used to evaluate the effect on performance of all Ss at the 2-sec interval, depending on whether the 2-sec interval was preceded by a 4-, 6- or 8-sec interval. In this analysis, there were two groups.
(i.e., brain-damaged vs. normals), two treatment conditions (i.e., PR vs. NR), and the three levels of PI which preceded the 2-sec interval (i.e., 4-, 6- and 8-secs).

One t test was carried out to compare the difference in the number of blocks attained by the BD and normal groups. The other was to test the difference in the number of reinforcements earned by each of the groups.

Results

Table 2 contains the means for RT under both trials of the baseline condition for the BD and normal Ss. The overall means (i.e., both trials combined) are plotted in Figure 1. An analysis of this data is presented in Table 3.

It is evident (Table 3) that the group main effect was statistically significant ($p < 0.05$). In other words, a separation was obtained between the RT performance of the Ss in the brain-damaged group and that of the normals (N) prior to the introduction of the treatment conditions. Inspection of Figure 1 would seem to indicate that this difference is particularly clear at the 2-sec PI, and to a lesser degree at the 4-sec, 8-sec, and 6-sec PIs, in that order. Furthermore, there was a statistically significant ($p < 0.01$) PI main effect; that is, the level of the PI had a statistically significant effect on performance. Additionally, a statistically significant ($p < 0.05$) PI x Group interaction was obtained. An analysis of this interaction, employing the Newman-Keuls comparison between means, yielded statistically significant ($p < 0.05$) differences between the brain-damaged and normal groups, but
<table>
<thead>
<tr>
<th>Interval (Seconds)</th>
<th>Brain-Damaged</th>
<th></th>
<th>Normal</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR</td>
<td>NR</td>
<td>PR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td>Tr(1)</td>
<td>Tr(2)</td>
<td>Tr(1)</td>
<td>Tr(2)</td>
</tr>
<tr>
<td>2</td>
<td>.450</td>
<td>.674</td>
<td>.611</td>
<td>.529</td>
</tr>
<tr>
<td>4</td>
<td>.415</td>
<td>.384</td>
<td>.499</td>
<td>.438</td>
</tr>
<tr>
<td>6</td>
<td>.376</td>
<td>.370</td>
<td>.457</td>
<td>.354</td>
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<tr>
<td>8</td>
<td>.419</td>
<td>.375</td>
<td>.452</td>
<td>.454</td>
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</tbody>
</table>

*Table 2: Mean reaction time (in seconds) of brain-damaged and normal subjects at the 4 levels of PI during the baseline procedure.*
FIG. 1. Mean RT of brain-damaged and normal subjects at the four levels of PI during the baseline-procedure.
TABLE 3
ANALYSIS OF VARIANCE OF BRAIN-DAMAGED AND NORMAL SUBJECTS' PERFORMANCE DURING THE BASELINE CONDITION

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
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<td>0.2354</td>
<td>4.880</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Treatment</td>
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<td>0.0350</td>
<td>0.724</td>
<td></td>
</tr>
<tr>
<td>Group x Treatment</td>
<td>1</td>
<td>0.0154</td>
<td>0.319</td>
<td></td>
</tr>
<tr>
<td>Ss within Group Error</td>
<td>26</td>
<td>0.0482</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>3</td>
<td>0.1419</td>
<td>8.673</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>PI x Group</td>
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<td>0.0552</td>
<td>3.375</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>PI x Treatment</td>
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<td></td>
</tr>
<tr>
<td>PI x Group x Treatment</td>
<td>3</td>
<td>0.0146</td>
<td>0.895</td>
<td></td>
</tr>
<tr>
<td>PI x Ss within error</td>
<td>78</td>
<td>0.0163</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trials</td>
<td>1</td>
<td>0.0113</td>
<td>0.908</td>
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<tr>
<td>Trials x Group</td>
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<td>0.0002</td>
<td>0.020</td>
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<td>Trials x Ss within error</td>
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<tr>
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</tr>
<tr>
<td>Treatment x PI x Trials</td>
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<td>1.557</td>
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</tr>
<tr>
<td>Group x Treatment x PI x Trials</td>
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<td>0.0191</td>
<td>1.161</td>
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<tr>
<td>PI x Trials x Ss within error</td>
<td>78</td>
<td>0.0164</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
no such differences existed within groups. Finally, a statistically significant ($p < .05$) Trial$\times$Group$\times$Treatment interaction was obtained. A review of the data contained in Table 2 indicates that, during baseline, except for the $S$s in the N NR group, the $S$s in the other 3 groups tended to improve on their RT performance from trial one to trial two.

The means obtained by the 4 groups during the treatment procedure are described in Table 4 and plotted in Figure 2. The statistical analysis of this data is presented in Table 5. The statistically significant ($p < .05$) group main effect means that the overall performance of both normal groups combined was superior to that of both brain-damaged groups combined. Again, as in the baseline condition, a statistically significant ($p < .01$) PI main effect and PI $\times$ group ($p < .05$) interaction were obtained. In other words, the level of PI had a significant effect on the performance of all groups and this effect was particularly clear (see Figure 2) at the 2-sec interval for both brain-damaged groups. Furthermore, the blocks main effect proved to be statistically significant. An inspection of the raw data revealed that the RT performance of $S$s in all groups tended to deteriorate over blocks. The $F$ ratio for the Group $\times$ Treatment interaction did not attain statistical significance. Consequently, the null hypothesis that there is no difference between the performance of the B PR and both normal groups, has to be accepted. A Newman-Keuls comparison between the means obtained by the 4 groups confirmed the null hypothesis.
<table>
<thead>
<tr>
<th>Interval (Seconds)</th>
<th>Brain-Damaged</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR</td>
<td>NR</td>
</tr>
<tr>
<td>2</td>
<td>.628</td>
<td>.795</td>
</tr>
<tr>
<td>4</td>
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</tr>
<tr>
<td>8</td>
<td>.399</td>
<td>.601</td>
</tr>
</tbody>
</table>
FIG. 2. Mean RT at the four levels of PI for each group during treatment condition.
TABLE 5
ANALYSIS OF VARIANCE OF BRAIN-DAMAGED AND NORMAL SUBJECTS' PERFORMANCE DURING TREATMENT CONDITIONS

<table>
<thead>
<tr>
<th>Source</th>
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<td>1</td>
<td>2.483</td>
<td>4.0015</td>
<td></td>
</tr>
<tr>
<td>Group x Treatment</td>
<td>1</td>
<td>1.688</td>
<td>2.7192</td>
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</tr>
<tr>
<td>Ss within Group Error</td>
<td>26</td>
<td>0.621</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>3</td>
<td>1.395</td>
<td>38.9457</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>PI x Group</td>
<td>3</td>
<td>0.140</td>
<td>3.9127</td>
<td>&lt; .05</td>
</tr>
<tr>
<td>PI x Treatment</td>
<td>3</td>
<td>3.746</td>
<td>2.4413</td>
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</tr>
<tr>
<td>PI x Group x Treatment</td>
<td>3</td>
<td>0.026</td>
<td>0.7402</td>
<td></td>
</tr>
<tr>
<td>PI x Ss within error</td>
<td>78</td>
<td>0.036</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blocks</td>
<td>7</td>
<td>0.326</td>
<td>4.9742</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>Blocks x Group</td>
<td>7</td>
<td>0.039</td>
<td>0.5928</td>
<td></td>
</tr>
<tr>
<td>Blocks x Treatment</td>
<td>7</td>
<td>0.104</td>
<td>1.5818</td>
<td></td>
</tr>
<tr>
<td>Blocks x Group x Treatment</td>
<td>7</td>
<td>0.086</td>
<td>1.3138</td>
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<tr>
<td>Blocks x Ss within error</td>
<td>182</td>
<td>0.065</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI x Blocks</td>
<td>21</td>
<td>0.048</td>
<td>0.9653</td>
<td></td>
</tr>
<tr>
<td>PI x Group x Blocks</td>
<td>21</td>
<td>0.019</td>
<td>0.3841</td>
<td></td>
</tr>
<tr>
<td>PI x Treatment x Blocks</td>
<td>21</td>
<td>0.042</td>
<td>0.8272</td>
<td></td>
</tr>
<tr>
<td>PI x Group x Treatment x Blocks</td>
<td>21</td>
<td>0.058</td>
<td>1.1153</td>
<td></td>
</tr>
<tr>
<td>PI x Blocks x Ss within error</td>
<td>546</td>
<td>0.050</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6 contains the overall means for the baseline and treatment conditions in the four experimental groups. Although there appears to be a relatively large discrepancy (approximately 50 percent) between the baseline and treatment conditions for the BD NR group, with the performance of the other 3 groups remaining at approximately the same level, none of these differences attained statistical significance.

A sequence analysis was undertaken to determine whether performance at the 2-sec PI was affected by the length of the interval which preceded it. Table 7 describes the means at the 2-sec PI as a function of whether they were preceded by the 8-, 6-, or 4-sec intervals. From the slopes of the curves in Figure 3, which describes the data presented in Table 7 pictorially, it can be observed that there seems to be a direct relationship between performance at the 2-sec interval and the length of the preceding interval. Table 8 contains a summary of the results of the analysis of this data and indicates that there was, in fact, a statistically significant sequential interval main effect (p < .01). Furthermore, a test for trends indicates that this relationship, for both brain-damaged groups, is linear in nature and statistically significant (p < .05). In other words, as the length of the preceding PI increases, there is a corresponding decrement in performance at the 2-sec interval for both brain-damage groups at least from the 8 to 6 sec. PI. The length of the preceding PI does not appear to affect the performance of the normal groups at the 2-sec interval in any differential manner. In addition, a statistically significant
# TABLE 6

OVERALL MEANS FOR THE BASELINE AND TREATMENT CONDITIONS

IN THE FOUR EXPERIMENTAL GROUPS

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>BD PR</td>
<td>.433</td>
<td>.468</td>
</tr>
<tr>
<td>BD NR</td>
<td>.474</td>
<td>.655</td>
</tr>
<tr>
<td>N PR</td>
<td>.422</td>
<td>.386</td>
</tr>
<tr>
<td>N NR</td>
<td>.440</td>
<td>.394</td>
</tr>
<tr>
<td>GROUP</td>
<td>PRECEDING INTERVAL</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>--------------------</td>
<td></td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>BD PR</td>
<td>.752</td>
<td>.552</td>
</tr>
<tr>
<td>BD NR</td>
<td>.869</td>
<td>.793</td>
</tr>
<tr>
<td>N PR</td>
<td>.496</td>
<td>.454</td>
</tr>
<tr>
<td>N NR</td>
<td>.520</td>
<td>.495</td>
</tr>
</tbody>
</table>
FIG. 3. Means for the two-second interval when preceded by the eight-second, six-second and four-second intervals.
TABLE 8
ANALYSIS OF VARIANCE AT THE 2-SECOND PI BY THE BRAIN-DAMAGED
AND NORMAL SUBJECTS AS A FUNCTION OF THE PRECEDING PI

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>1</td>
<td>1.288</td>
<td>11.3706</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>Treatment</td>
<td>&gt;1</td>
<td>0.221</td>
<td>1.9518</td>
<td></td>
</tr>
<tr>
<td>Group x Treatment</td>
<td>1</td>
<td>0.122</td>
<td>1.0740</td>
<td></td>
</tr>
<tr>
<td>Ss within Group Error</td>
<td>26</td>
<td>0.113</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PI</td>
<td>2</td>
<td>0.100</td>
<td>5.1337</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>PI x Group</td>
<td>2</td>
<td>0.022</td>
<td>1.1391</td>
<td></td>
</tr>
<tr>
<td>PI x Treatment</td>
<td>2</td>
<td>0.010</td>
<td>0.4932</td>
<td></td>
</tr>
<tr>
<td>PI x Group x Treatment</td>
<td>2</td>
<td>0.005</td>
<td>0.2686</td>
<td></td>
</tr>
<tr>
<td>PI x Ss within error</td>
<td>52</td>
<td>0.020</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
group main effect was obtained (p < .01). In other words, the performance of all brain-damaged Ss at the 2-sec interval was generally inferior to that of the normal Ss. A Newman-Keuls analysis between means revealed that the performance of the BD NR group at the 2-sec interval was significantly different from that of all other groups, regardless of the level of the preceding PI (p < .05). Both normal groups did not differ from each other, while the performance of the BD PR group did not differ from the normal groups, except when the level of the preceding PI was 8 sec.

The final two analyses involved a comparison of the number of reinforcements earned and the number of blocks of trials completed by the BD PR and N PR groups. Table 9 contains the means obtained by the BD PR and N PR groups. In both cases, the differences did not approach statistical significance.
### Table 9

Mean number of reinforcements earned and blocks of trials completed by the brain-damage and normal groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Number of Reinforcements Earned</th>
<th>Mean Number of Blocks Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td>BD PR</td>
<td>60.67</td>
<td>10.3</td>
</tr>
<tr>
<td>N PR</td>
<td>66.38</td>
<td>10.2</td>
</tr>
</tbody>
</table>
CHAPTER IV

DISCUSSION

There were three dependent variables which related to the first major hypothesis of this study, that is, under conditions of positive reinforcement, there should be less difference in RT performance between young brain-damaged and normal children than under no reinfor- cement conditions. These dependent variables were RT, number of blocks of trials completed, and number of reinforcements earned. An analysis of the RT data revealed that the differences in performance which were obtained prior to the introduction of positive reinforcement conditions, were maintained following the institution of treatment.

That is to say, employing this dependent variable, positive reinforcement does not appear to have a significant effect on the RT performance of young brain-damaged subjects. This finding contradicts those of a number of previous studies (Blackburn, 1958; Goldstein, 1942; Kerk, 1962; Martin & Powers, 1967) which have demonstrated the beneficial effects of introducing incentives on the performance of Ss who have difficulty maintaining a state of response readiness. Furthermore, the present finding tends to confirm McGhee's (1969) hypothesis that the inability of brain-damaged children to maintain a state of response readiness can be attributed to a structural deficiency. This deficiency is described as being due to a breakdown in the individual's ability to

-48-
selectively attend to relevant aspects of the environment. Consequently, such persons are highly distractible and this is supposedly reflected in their poor RT performance.

The fact that the brain-damaged and normal Ss did not differ in the number of blocks of trials which they completed or the number of reinforcements which they earned offers somewhat conflicting evidence and suggests that the introduction of positive contingencies for desirable behaviour will, in fact, produce some change in performance. Furthermore, it might be noted at this point that, in the course of testing Ss in the BD NR group, following their completion of the required number of trials, the E instructed 4 of these Ss that the experiment would continue and that now a penny would fall into the box if they were able to match or "beat" a time set by the E. In other words, the E introduced the PR condition with some of the Ss in the BD NR group. This procedure is referred to in the behavioural literature as an A-B-A design (Ullman & Krasner, 1969). Unfortunately, this design was not entertained early enough in the experiment to collect a sufficient quantity of data to produce meaningful statistical results; however, over the number of trials that this procedure was instituted, it was observed that there was a distinct change for the better in the performance manifested by the Ss in this group. This reversal in performance occurred despite the fact that the Ss in this group were probably fatigued and quite likely bored with the task (two of these Ss had already been exposed to 123 trials prior to
the modification in procedure).

These observations led the Ex to examine the RT performance and evidence of pathology of the brain-damaged Ss more closely. It was noted that, during the baseline sessions, the RT of Ss in both the BD/PR and BD NR groups ranged from .319 sec to .556 sec. During treatment, however, the RT of Ss in the BD PR group ranged from .302 to .623 sec, while the RT of Ss in the BD NR group ranged from .389 to 1.031 sec. With this in mind, the EEG opinions of the Ss in the BD NR group were re-examined. It was found that the two Ss in this group with the superior RTs (See Appendix A: Ss #1 and #7) had less conclusive evidence of brain dysfunction in their EEGs, while those with the poorest RTs (Ss #8, #13 and #14) had much more conclusive EEG evidence of brain dysfunction. As a result, it may be concluded that the lack of support obtained for the main hypothesis (that the RT performance of young brain-damaged Ss can be modified by rewarding desirable performance) is, at least, partially a function of the fact that some of the Ss in the BD NR group did not have conclusive evidence of brain-damage. This eventuated in their performance more closely approximating that of normal rather than brain-damaged Ss. Furthermore, this suggests that, in future studies, additional weight should be placed on the EEG opinion in the selection of young brain-damaged Ss. Given, however, that the results pertaining to 2 of the 3 dependent variables were in the predicted direction, and that PR reversed the effect of NR in brain-damaged Ss, it is concluded that hypothesis #1 is supported.
As was the case in both Czudner and Rourke studies (1970, 1972), the level of the PI had a significant effect on the RT performance of all the Ss. This was true both during the baseline and treatment conditions. In addition, however, a PI x Group interaction was also obtained under each condition. That is, although the performances of Ss in all groups were affected by the level of the PI, the differential effect on the brain-damaged groups was greater than on the normal group. The relationship obtained between PI and performance was such that, as the level of PI decreased, RT increased. Consequently, performance was poorest at the 2-sec interval. A sequential analysis was effected and it was found that, for the brain-damaged groups only, as the level of the preceding interval increased, performance at the 2-sec PI deteriorated. Performance of the normal Ss at the 2-sec PI seemed equally affected by all other levels of PI. This phenomenon was also obtained by Czudner and Rourke (1972), and lends support to the hypothesis that young brain-damaged children are affected to a greater extent than normal Ss by the length of the preceding PI.

Czudner and Rourke (1970) attempted to account for this finding by postulating that the longer the level of the preceding PI, the greater the buildup of reactive inhibition. The poorer performance on the subsequent interval, particularly if it happens to be of short duration, would be due to the insufficient time available for the reactive inhibition to dissipate. If one accepts this hypothesis, then the improvement in performance of the brain-damaged Ss in the PR group (Figure 3) can be explained in terms of the attenuating function of
positive reinforcement on the effects of reactive inhibition.

The second major hypothesis, that positive reinforcement will not alter the RT performance of young normal children, was supported. As was expected, normal children already find the experimental interaction sufficiently reinforcing in the form of attention to maintain desirable behaviour. Consequently, they are already functioning close to their physiological limit, and concrete rewards, such as the toys in the present study, do not appreciably alter their performance.

The results of the present study, therefore, suggest that positive reinforcement can have beneficial effects on the ability of young brain-damaged children to maintain a state of response readiness. It is clear, however, that future studies will have to take careful precautions to insure that their brain-damaged sample consists of Ss who have conclusive evidence of cerebral dysfunction. This point was highlighted in the present research when one potential S, who had three EEG examinations prior to the study, had to be excluded because a fourth examination revealed that the EEG patterns which he manifested were within normal limits. In addition, it is advisable that more than one block of trials be obtained as a baseline measure. In fact, RT performance at baseline might prove to be a helpful criterion for assessing the presence of brain-damage (De Renzi & Faglioni, 1965; Rourke & Czudner, 1972).
CHAPTER V

SUMMARY AND CONCLUSIONS

The primary purpose of this study was to determine whether positive reinforcement could have a beneficial effect on the performance of young brain-damaged children on a RT task, which requires that the S maintain a state of response readiness (attention). A series of previous investigations, none of which included external incentives, had determined that young, brain-damaged Ss performed considerably poorer than normals on such tasks.

The hypotheses under investigation were as follows:

**Hypothesis 1.** Under conditions of positive reinforcement, there should be less difference in RT performance between young brain-damaged and normal children than under no reinforcement conditions.

This hypothesis was supported. No significant difference was found between the numbers of blocks of trials completed and number of reinforcements earned between the BD PR and N PR groups. It was also observed, however, although there was a tendency for PR to produce mild changes in RT performance in the predicted direction, these differences were not large enough to eventuate in statistically significant results.

It was suggested that one possible reason for the lack of support for this hypothesis was that some of the Ss in the BD NR group had only borderline evidence of brain-damage. Consequently their performance
was consistent with what would be expected of normal Ss. It was noted that future studies should take extensive precautions to insure that their Ss are in fact brain-damaged.

**Hypothesis 2.** There should be no difference in performance between those normal children receiving PR and those under NR conditions. This hypothesis was supported. It was hypothesized that these Ss are sufficiently motivated to perform close to their physiological limit simply because of the social reinforcement afforded by the experimental situation. PR, therefore, in the form of concrete rewards, was not expected to, and did not, result in drastic modifications to that performance.
APPENDIX A

E.E.G. OPINIONS AND NEUROPSYCHOLOGICAL IMPRESSIONS FOR EACH OF THE BRAIN-DAMAGED CHILDREN

S - 1 (7-11 years, female)

E.E.G. Opinion

Tracing considered to be borderline, it is suggestive of epileptoid activity in the temporal regions of the brain.

Neuropsychological Impression

An hypothesis of mild, chronic cerebral dysfunction would appear to be warranted in this case. The area of maximal involvement would appear to be the posterior temporo-parietal regions of both cerebral hemispheres. The left cerebral hemisphere would appear to be more involved than is the right.
S - 2 (7-10 years, male)

**E.E.G. Opinion**

This E.E.G. is considered to be abnormal because of an increased amount of theta activity. This E.E.G. is consistent with mild brain dysfunction.

**Neuropsychological Impression**

This particular pattern of abilities and deficits is consistent with what we have come to expect from children suffering the effects of relatively mild cerebral dysfunction maximally involving the temporal regions. In this particular case the right hemisphere would appear to be somewhat more involved than is the left.

S - 3 (7-7 years, male)

**E.E.G. Opinion**

Borderline, compatible with disturbed function in the right temporal region of the brain, with possible epileptic activity.

**Neuropsychological Impression**

These deficits, it should be stressed, are only of a mild nature and should not be seriously debilitating. If cerebral dysfunction be present, it is likely of a mild chronic nature and not interfering greatly with adaptive abilities.

S - 4 (7-9 years, male)

**E.E.G. Opinion**

This E.E.G. is abnormal and is compatible with seizure disorder or epileptic disorder. Epileptogenic focus is thought to be in the deep midline structures of the brain.
Neuropsychological Impression

This pattern of abilities and deficits is suggestive of mild, chronic cerebral dysfunction maximally involving the temporal-parietal region of the right cerebral hemisphere.

S - 5 (7-9 years, male)

E.E.G. Opinion

The background of this E.E.G. is essentially the same as a previous E.E.G. It again recorded a spike activity in the right posterior temporal region. It is indicative of dysfunction of this area of the brain. The sleep activation recording again records the same spike pattern in the right temporal-occipital region, however with a phase reversal pattern in the right posterior temporal region, it is indicative of, by clinical history, an epileptogenic lesion in this area.

Neuropsychological Impression

Although many difficulties may be accountable, to a great extent, to difficulty in attending, the possibility of mild, chronic cerebral dysfunction is still raised. The temporo-parietal regions of both cerebral hemispheres would appear to be maximally involved, especially this region within the left hemisphere.

S - 6 (7-10 years, male)

E.E.G. Opinion

This E.E.G. is abnormal and compatible with epileptic disorders. Epileptogenic activity is probably located in the deep midline structures of the brain. The possibility of right temporal lobe epilepsy also exists.
Neuropsychological Impression

The possibility of mild dysfunction at a sub-cortical level might be raised to account for the steadiness problem with the left hand.

S - 7 (5-10 years, male)

E.E.G. Opinion

E.E.G. is borderline for age group suggestive of bi-temporal disturbance of function and possible epileptic activity.

Neuropsychological Impression

This particular pattern of abilities and deficits is consistent with what we have come to expect from children who are currently suffering the effects of mild, chronic cerebral dysfunction. In this particular case, this hypothesized dysfunction would appear to involve maximally the temporal regions bi-laterally and the adjacent right parietal region.
S - 8 (8-3 years, male)

**E.E.G. Opinion**

Diagnostic of dysfunction, right temporo-parietal region of the brain.

**Neuropsychological Impression**

This neuropsychological profile is suggestive of mild, chronic cerebral dysfunction maximally involving the temporal-parietal regions bi-laterally. There is some indication that the right cerebral hemisphere may be more involved than is the left.

S - 9 (7-0 years, male)

**E.E.G. Opinion**

This E.E.G. is abnormal and consistent with focal epilepsy in the right temporal area.

**Neuropsychological Impression**

On the basis of the limited amount of information available to us, it would appear that this boy is experiencing cerebral dysfunction which may involve the middle and anterior regions of the brain somewhat more so than the posterior regions. Additionally, it was felt that the extremely bizarre behaviour exhibited by the child is somewhat suggestive of psychomotor epilepsy.

S - 10 (6-9 years, male)

**E.E.G. Opinion**

This is a borderline E.E.G. because of some mild disorganization of the background and mild bi-posterior disturbance. These changes are non-specific as to clinical corelation.
Neuropsychological Impression

This particular pattern of abilities and deficits is consistent with what we have come to expect from children who are currently suffering the effects of lesions below the level of the cerebral cortex. In this particular case, this hypothesized dysfunction would appear to involve structures adjacent to the tentorium.

S - 11 (7-11 years, male)

E.E.G. Opinion

Compatible with dysfunction most marked in the parietal region of the brain on the right more than the left.

Neuropsychological Impression

There was considerable evidence to suggest an impairment in this boy's adaptive abilities as a consequence of cerebral dysfunction. The area of maximal involvement would appear to be the temporal and temporoparietal regions of the right cerebral hemisphere. These regions within the left cerebral hemisphere would also appear to be involved.

S - 12 (7-11 years, male)

E.E.G. Opinion

This E.E.G. which consists mostly of drowsiness and sleep does not show any strong focal or paroxysmal features. However, the background is somewhat disorganized suggesting mild disturbance of cerebral function.
Neuropsychological Impression

This pattern of abilities and deficits is similar to that which we have come to expect from children who are currently suffering the effects of mild cerebral dysfunction.

E.E.G. Opinion

S - 13 (7-10 years, male)

E.E.G. is mildly abnormal because of slight generalized slowing of the background and some disorganization. The E.E.G. changes suggest a mild, diffuse depression of cerebral function.

Neuropsychological Impression

This neuropsychological profile is not indicative of any serious impairment in his adaptive abilities as a consequence of cerebral dysfunction. At most, the possibility of mild cerebral dysfunction involving primarily the temporo-parietal regions of the cerebral hemispheres might be raised.

S - 14 (7-1 years, male)

E.E.G. Opinion

Abnormal sleep recording diagnostic of rather severe epileptic activity in the temporal areas of the brain, right more than left.

Neuropsychological Impression

This pattern of abilities and deficits is similar to that which we have come to expect from children who are currently suffering the debilitating effects of mild chronic cerebral dysfunction. In this particular case, this hypothesized dysfunction would appear to involve maximally the temporal and adjacent cortical regions of both cerebral hemispheres, more so on the right side.
APPENDIX B

LIST OF REINFORCERS AND VALUES

<table>
<thead>
<tr>
<th>Item</th>
<th>Normal Group</th>
<th>Brain-Damaged Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>B = Boys</td>
<td></td>
<td></td>
</tr>
<tr>
<td>G = Girls</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Badminton Set</td>
<td>85(B)/80(G)</td>
<td>75(G)/80(B)</td>
</tr>
<tr>
<td>Frisbee</td>
<td>70(B&amp;G)</td>
<td>65(B&amp;G)</td>
</tr>
<tr>
<td>Bubbles</td>
<td>35(B)/40(G)</td>
<td>30(B)/35(G)</td>
</tr>
<tr>
<td>Archery Set</td>
<td>80(B)</td>
<td>75(B)</td>
</tr>
<tr>
<td>Marbles</td>
<td>40(B)/35(G)</td>
<td>35(B)/30(G)</td>
</tr>
<tr>
<td>Parachuter</td>
<td>58(B)</td>
<td>53(B)</td>
</tr>
<tr>
<td>Whistle</td>
<td>30(B&amp;G)</td>
<td>25(B&amp;G)</td>
</tr>
<tr>
<td>Football</td>
<td>75(B)</td>
<td>70(B)</td>
</tr>
<tr>
<td>Cap Bomb</td>
<td>52(B)</td>
<td>47(B)</td>
</tr>
<tr>
<td>Black Bat</td>
<td>38(B&amp;G)</td>
<td>33(B&amp;G)</td>
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<tr>
<td>Bolo-Bat</td>
<td>47(B&amp;G)</td>
<td>42(B&amp;G)</td>
</tr>
<tr>
<td>Swim Ring</td>
<td>66(B)/58(G)</td>
<td>61(B)/53(G)</td>
</tr>
<tr>
<td>Purse with Umbrella</td>
<td>85(G)</td>
<td>80(G)</td>
</tr>
<tr>
<td>Skipping Rope</td>
<td>66(G)</td>
<td>61(G)</td>
</tr>
<tr>
<td>Doll Cut-Outs</td>
<td>75(G)</td>
<td>70(G)</td>
</tr>
<tr>
<td>Skip-It</td>
<td>52(G)</td>
<td>47(G)</td>
</tr>
</tbody>
</table>

Substitutes

Raggedy Ann Miniature Doll 30(G)
Doctor Kit 75(B)
David Cassidy Colouring Book 30(B&G)
<table>
<thead>
<tr>
<th>Activity</th>
<th>Normal Group</th>
<th>Brain-Damaged Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Invisible Ink Game</td>
<td></td>
<td>55(B&amp;G)</td>
</tr>
<tr>
<td>Hot Rod Book</td>
<td></td>
<td>50(B)</td>
</tr>
<tr>
<td>Finger Paints</td>
<td></td>
<td>60(B&amp;G)</td>
</tr>
<tr>
<td>Chess and Checkers Game</td>
<td></td>
<td>80(B&amp;G)</td>
</tr>
<tr>
<td>Battle-Battle Game</td>
<td></td>
<td>80(B&amp;G)</td>
</tr>
</tbody>
</table>
APPENDIX C
INSTRUCTIONS TO SUBJECTS

Practice Session: (Both groups-Regular Procedure-3 trials at each PI)

We are going to play a game which should prove to be a lot of fun. You see in front of you a board on which there is a white and a red light, as well as a telegraph key. When you press down on the key the white light will come on (demonstrate). When this red light appears, you are to take your finger off the key as quickly as possible (demonstrate). You are not to take your finger off the key before the red light comes on, otherwise the machine will stop and we will have to start that over again.

Now here is where the fun comes in. We will play this game a number of times and each time I would like you to try and raise your finger from the key as fast as you can. Do you understand? O.K. Let's begin.

1 MINUTE REST AFTER PRACTICE SESSION

Baseline Session: (Both Groups-Irregular Procedure-2 trials at each PI)

Now that you have had some practice we are going to do this some more. This time I would like to see you again take your finger off the key as fast as you can. O.K. Let's begin.

1 MINUTE REST AFTER BASELINE SESSION
Experimental Session:

(a) No Reinforcement Group:

Now let's see if you can do it even faster.

(b) Positive Reinforcement Group:

Now let's see if you can do it even faster. This time, though, you will know that you have gone faster when a shiny penny falls into that box (point out). At the end of the session, depending on how many pennies you have won, you will be able to buy one of these toys (point out). For example, if you have won 30 pennies you can buy the parachuter which costs 30 pennies, or the bubbles and the bat which cost 14 and 16 pennies. Do you understand? O.K. Begin.
APPENDIX D

ORDER OF PRESENTATION OF PREPARATORY INTERVALS

Baseline

2 8 6 4
6 2 8 4

Criterion Set to .800 Seconds

Block 1

8 2 6 4
6 8 4 2

Criterion Set to .750 Seconds

Block 2

4 6 8 2
6 2 4 8

Criterion Set to .700 Seconds

Block 3

6 8 4 2
4 2 8 6

Criterion Set to .650 Seconds

Block 4

6 4 8 2
2 8 4 6

Criterion Set to .600 Seconds

Block 5

8 4 2 6
6 4 8 2

Criterion Set to .550 Seconds

Block 6

4 8 2 6
6 8 2 4

Criterion Set to .500 Seconds

Block 7

6 8 4 2
4 2 8 6
<table>
<thead>
<tr>
<th>Block 8</th>
<th>4 6 8 2</th>
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<tbody>
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<td>8 4 6 2</td>
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Criterion Set to .450 Seconds

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Criterion Set to .400 Seconds

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Criterion Set to .350 Seconds

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Criterion Set to .300 Seconds

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Criterion Set to .250 Seconds

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<tr>
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Criterion Set to .200 Seconds
REFERENCES


Cameron, N. Reasoning, regression and communication in schizophrenics. *Psychological Monographs*, 1938, 50, 1-34.


Czudner, G. Personal communication, March, 1971 (b).


Scott, W.S. Reaction time of young intellectual deviates. Archives of Psychology, 1940, No. 256.


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