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Simple and complex learning and problem solving situations were employed with mentally retarded children and adults. In the Rotation-Discrimination Complex and the Size-Discrimination Simple experiments, upper and lower test achievement subgroups were subjected to two basic types of visual discrimination at different levels of task complexity. Significant improvement in differentiation by the posterior intrinsic system was achieved by training retardates with a mental age of at least 5 1/2 years. Both groups solved a simple problem but with no improvement in differentiation. The function of intention was inefficient on both problems for both groups. On the Distance-Discrimination Complex boys did significantly worse than girls at the more difficult position. Distance perception was maturational and related to mental rather than chronological age. Incorrect responses were made with reference to a rigid, orderly space of parallels and perpendiculars. On the Hypothesis-Discrimination Complex the subjects were confronted with experimental situations in which they could choose either of two equally valid hypotheses, and when a choice was made, conditions were changed to bring about a switch to the other. Difficulty in shifting was associated with older chronological age. (Author/SN)

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FINAL REPORT
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February 1968

U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE

Office of Education
Bureau of Research

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The roster of the research team immediately involved follows:

Charlotte Henriette Anton
Aldon J. Bruce
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Charlotte Henriette Anton, Nora D. Hubbard, and Ronald Ragsdale participated in the preliminary planning and search of the literature for pertinent contributions. Victor Lind, Leslie D. McLean, Johnine M. Simpson, Mary Snelling, and Aldon J. Bruce shared in the actual experimentation with various groups of subjects. Johnine M. Simpson, Victor Lind, Aldon J. Bruce, Leslie D. McLean, and Mary Snelling also helped in building needed apparatus and equipment. Aldon J. Bruce, in addition to his other contributions, helped greatly with the preparation of the final report.

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FACTORS INFLUENCING LEARNING AND PROBLEM SOLVING BEHAVIOR IN THE MENTALLY RETARDED

I. General Introduction

In these studies of factors influencing learning and problem solving behavior in the mentally retarded several groups of retardates were placed in various situations where their learning or problem solving behavior could be examined.

In one group of experiments the neurally distinct functions of the posterior and frontal intrinsic systems involved in learning and problem solving were examined in connection with a relatively difficult sequence completion situation and a simpler block assembly problem. (Both differentiation and intention were involved.) In another set of studies educable mentally retarded were confronted with a linear distance matching task in which the positions of endpoints of the same interval were systematically varied.

Other studies involved situations in which severely retarded subjects were given an opportunity to solve problems by using either of two principles in a situation where two equally valid hypotheses were available and operative from the start. Then after the problem was solved, and special test procedures had revealed which principle had been used, new situations were presented where the previously successful hypothesis broke down and had to be discarded in favor of the originally non-chosen alternative, for success.

Learning and Problem Solving Behavior

Modification of behavior may be produced by altering singly or in any combination, genetic antecedents and components; sources of electromagnetic, mechanical, and chemical outputs involved in the stimulating situation; signals sent to receptors; the sensing or detecting devices themselves; afferent nerve pathways and other post-sensory transmitting media; the central nervous system; post-CNS transmitting mechanisms (mainly nerves and the blood stream);

effectors such as muscles and glands; or feedback and feedforward or anticipatory mechanisms.

Usually, however, only the modification of response tendencies which is mediated through reorganization of the central nervous system is considered learning. Behavior resulting when an organism is presented with a situation requiring an adjustment or a new series of actions for goal attainment, also brought about by (involving) neural changes is problem solving. Both learning and problem solving involve or require changes in the neural apparatus and differentiation in such terms is currently difficult, if not impossible. The required neural changes may take place relatively quickly or quite slowly and they may be of relatively short duration or approach permanence and irreversibility; but they must occur.

The crucial role of the central nervous system in learning and problem solving has been shown by extirpation techniques, electroencephalographic procedures, histological studies, chemical approaches, clinical data, split-brain preparations, microelectrode outcomes, electrically induced spreading depression phenomena, and computer simulation.

Mental Retardation

The official definition adopted in 1959 by the American Association on Mental Deficiency has a statistical or psychometric, rather than a neuropsychological, orientation and hence is largely service oriented. This official AAMD definition reads as follows:

> "Mental retardation refers to subaverage general intellectual functioning which originates during the developmental period and is associated with impairment in adaptive behavior" [87].

Such a psychometric concept of mental retardation, coupled with social incompetence as a basic criterion, has much practical utility for the professions providing services for the mentally retarded. Moreover this concept of mental retardation does not brand the condition as irreversible [209] and to the extent that it may, rather, lend itself to realistic hope for change in status within the reasonable pale of neurology and other remedial disciplines today, it has great value for the people concerned. To the extent that it may also lend itself to the early identification of those children needing specialized services and training in order to have reasonable success in developing their limited potentials, it has great value as well [131]. Nonetheless psychometric definitions lend themselves more to classification and treatment of symptoms than they do to research concerned with basic causes and ultimate understanding (prediction and control).

A neuropsychological approach to the concept of mental retardation, on the other hand, provides a logical framework for etiological research aimed at preventing, reversing, or treating therapeutically and educationally the patho-anatomic malformations, the destructive processes, and the genetic and biochemical disorders that are involved in mental retardation viewed as a great "constellation of syndromes" [208]. This approach does not exclude conditions having their genesis beyond the developmental period. Syndromes originating in disease processes, neurovascular trauma, metabolic disorders, physical injury, and senescence are probably not rooted in radically divergent mechanisms in brain physiology or pathology. Rather, the recent scientific assault upon brain mechanisms can be expected to provide the basic underlying principles for dealing with the whole constellation of syndromes. We are at present on the threshold of a great new phase of development of biochemical, genetic, neurophysiological, and neuropsychological studies of the brain [12, 24, 25, 67, 161, 220, 223, 233].

Whether the approach be psychometric or neuropsychological mental retardation is probably best thought of as being a matter of degree. From this point of view retardates as subjects are not regarded as qualitatively different from other subjects; they simply are individuals with a deficit of intellectual function resulting from varied determinants, but having as a common proximate cause a diminished efficiency of the central nervous system entailing slower and more limited intellectual development and poorer environmental adjustments. They are individuals who happen to be available for study in a given situation, subjects for whom research funds have been made available, or people who are intrinsically appealing or interesting to a given investigator. The mentally retarded are thus valid subjects, not only per se, but for general learning and problem solving research. Principles derived from learning and problem solving research anywhere apply, and causes may be sought in all domains regardless of psychometric or service classifications.

The distinction often made (most recently by Zigler [234])between the organic and the familial, or cultural-familial, retardate has considerable value insofar as it calls attention to the important role of experiential, motivational, and personality factors in those cases where there is no demonstrable pathology, but it can be very misleading if it implies that the genetic antecedents and the central nervous systems of the familial are "normal" albeit low normal. Thus Zigler [234] writes on page 294, "Once one adopts the position that the familial mental retardate is not defective or pathological but is essentially a normal individual of low intelligence then the familial retardate no longer represents a mystery but, rather, is viewed as a particular manifestation of the general developmental process" and again, on page 298, "The familial retardate, on the other hand, is seen as a perfectly normal expression of the population gene pool, of slower and more limited intellectual development than the individual of average intellect." This position can be stultifying if it causes us to quit looking for neural and other causes once the subject is so labelled.

The so-called organic cases of mental retardation are directly comparable to the specimens produced by man through extirpation, chemical, mechanical, vascular, electrical and other techniques in his experimentation with central nervous systems and serve to help us better understand the functioning of the central nervous system and its ancillary mechanisms. In some of these organic cases the modifications in the structural components of behavior with its concomitant deficits in cognitive behavior have been traced to gene aberations, infections, glandular malfunctioning, chromosomal defects, toxic agents, and cerebral injury.

It does not follow, however, that where no demonstrable pathology is involved there is no organic basis for in a broader and very real sense all behavior whether it be superior, average, impaired, or defective has an organic basis involving among other things genetic components and the central nervous system. Certainly organic does not signify or require defectiveness or pathology. It can mean just what it says; that there is an organic or structural basis for the behavior. In this sense all retardation (or anything else) is organic. The advantage of this position is not in its exclusion of research concerned with the experiential, motivational, or personality factors in the mentally retarded; for it does not involve any such restriction or prohibition; but rather in its complete awareness of the need for research dealing with "organic" (CNS structure and function, sense organs, transmitters, feedback mechanisms etc.) as well.

"Cultural-familial" retardate behavior is certainly organically rooted in the sense that the central nervous system lies at its root though the precise nature of the involvement may be difficult to ascertain. If this faulty or inadequate central nervous system in turn is attributed to genetic antecedents and related mechanisms because of the lack of demonstrable pathology then the question remains as to the nature of this gene determined behavior, how it operates, and how it may be modified and controlled.

Studies concerned with the modification of responses through manipulation of the stimulating situation in and of themselves are inadequate for complete causal explanations. In learning and problem solving there must be some modification of the central nervous system and therefore its role must be understood if we are to have full knowledge. Indeed some modification in behavior may be relatively simple if we make necessary alterations in the central nervous system and virtually impossible if we do not. Getting rats [61] or blowflies [46] to gorge themselves literally to the bursting point is currently impossible without a lesion of the ventromedial area of the hypothalamus in the case of the rat and a cutting of the recurrent nerve or interruption of the ventral cord in the case of the blowfly.

Actually studies concerned exclusively with the modification of response through manipulation of the stimulating situation may or may not be dealing with learning and problem solving behavior. If the central nervous system is merely utilized and not altered then regardless of its degree of participation learning or problem solving behavior is not under study.

Just as studying overt behavior alone will not tell us how brains function and are metabolized, so chemical, electrical, or histological studies alone will not enable us to understand learning and problem solving. We certainly wouldn't know the composition of computer components, let alone be able to build such a machine, if all we knew were the inputs and outputs involved. On the other hand even if the composition and functions of the various components were thoroughly understood we could not accurately predict or control outcomes if we knew nothing of the inputs.

This difficult problem is perhaps best handled by close collaboration between physiologists, neurologists, psychologists, and geneticists, but in the absence of such teamwork psychologists should select behavior situations where they may be fairly certain that central nervous system alterations are necessary for successful completion of the tasks utilized leaving determination of the precise nature of the neural changes involved and how these may be controlled and facilitated, for the future. Physiologists, geneticists, and neurologists, for their part, must choose very carefully the pre- and post-behavioral tasks they employ in studying intervening variables.

The basic problem is to ascertain the essential characteristics of slowly developing mentality and the determination of precisely how such slow mentality evolves in the first place. It is easy to be beguiled by the measurement of the intellectual shortcomings of the mentally retarded and to forget to search for the underlying reasons for their shortcomings. We should not get lost in the behavioral implications of etiological differences nor become implicated in cross sectional investigations of how the mentally retarded differ from the normal in various areas of behavior [17]. As Morrison [156] has pointed out we should try to ascertain the laws that govern observed facts.

II. ROLE OF NEOCORTICAL MECHANISMS IN COGNITIVE BEHAVIOR

Today part of the actual physiological mechanism of memory appears to have been detected by cytochemical, microphotometric, and electron-microscopic techniques of investigation [13, 97, 158, 159, 160, 195, 207]. The study of the relationship between the base ratios in the ribosenucleotides in the neurones and the functions of genetic and psychic memory in the molecules is proceeding intensively in many laboratories. Genetic memory depends upon complimentary recognition of one nucleotide by another via highly specific hydrogen-bonding. It is specified by nucleotide sequences in the DNA and is relatively stable from one generation to another. The base sequence in DNA specifies the base sequence of a class of RNA termed template or messenger RNA (mRNA) which in turn specifies the sequence of amino acids in protein. Nirenberg [159] asserts there is no doubt that gene function can be altered temporarily by environmental factors in reproducible and specific ways, but the mechanisms which control such alteration have not been fully clarified.

Other physiological mechanisms of the brain may soon be revealed in the inherent biochemical, structural, and functional properties of the neurones and their electrical activities. Young [233] has noted the evidence that changes in RNA do occur during learning, but he also noted that it does not necessarily follow that the changes in the RNA specifically embody a coded representation of learning in any direct chemical transformation. The actual storage in memory takes place by changing the state of cells, i.e., by closing the unneeded channels. The occurrence of such changes in the cells and their channels depends in the first place upon the condition and synthesis of the ribosenucleotides, which may well be altered in turn with the changes. The underlying mechanism for closing the unneeded channels has been postulated to be the manufacture or release of a flood of inhibitor around the presynaptic junctions of the mnemonic units to be closed; the release of the flood being triggered by small specialized cells upon receipt of appropriate signals [233].

It has been proposed for many years that some of the small cells in the spinal cord have the function of producing an inhibitory chemical at the presynaptic junctions of certain spinal pathways [51]. It is now thought this same kind of process has been extended in the course of evolution into the higher cortical classifying cells that learn. Young believes the connection between this process of producing an inhibitory chemical and RNA metabolism may soon be found in the special mechanisms of synthesis of needed inhibitor enzymes.

Earlier explanations of the memory process proposed the maintenance of nerve impulses in reverberating circuits, as well as plastic changes at synapses, involving swelling, outgrowth, or multiplication of presynaptic terminals and dendritic networks. But it is altogether possible that nature has evolved only a single process for information coding and storage in biology [139], namely, the so-called holy trinity of DNA, RNA, and protein [71]. The brain may depend for its learning and memory functions upon modification of neuronal RNA, but the mechanism for this modification is still to be determined. The DNA of the chromosomes contained within the nucleus of each neurone can replicate itself. The DNA also passes on to the RNA a code of information in the chemical form of a specific sequence of nucleotides making up the nucleic acid molecule. RNA then moves from the nucleus into the cytoplasm and there presides over the synthesis of protein molecules, in such a way that a specific sequence of nucleotides in the RNA molecule becomes translated into a specific sequence of amino acids in a polypeptide molecule. The polypeptides are combined into protein molecules of specific sorts. Perhaps some of these specific sorts of protein molecules synthesized under the RNA template will be found to be the neural transmitter substances or hormones responsible for consequent patterns of post-synaptic excitation and inhibition [71]. While it seems improbable that transmitter hormones, such as acetylcholine and noradrenaline, are directly involved in the memory coding process as such, the mechanism by which the nerve impulse activates the innervated structure for liberation of such potent transmitter hormones must be a function of RNA [198].

It has been postulated that fixation of patterns of neural information is produced by transduction of modulated frequency in flux inputs, i.e., ion flux, from impulses coming into centers over afferent pathways into specifically structured macromolecules, probably RNA molecules. Schmitt [198] raised the question as to whether the electrical energy in the ionic fluxes representing the informational input from sensory nerve fibres would suffice for the transduction to specific chemical synthesis of RNA molecules as hypothesized.

Nevertheless, Hyden [100] still affirms his working hypothesis that changes in the specific base composition of RNA could arise through ion flux associated with electrical patterns of sensory and motor activities and could persist for a long time. In some of his learning experiments Hyden [96, 97, 98, 99] discussed a conceptual model for intraneuronal storage of information, assuming cytoplasmic RNA with specific base composition to serve this purpose. The RNA changes found in his experiments show clearly that both the neurone and its glia collaborate in synthesizing specific RNA fractions during the learning of a complicated motor and sensory pattern. According to those experiments, the modulated frequency of nerve impulses generated in a neurone by a specific stimulation induces a new sequence of nucleotide residues along the backbone of the RNA molecule. When the RNA has thereby been specified, this leads to a specification of the protein being formed through the mediation of the messenger RNA. A combination of this specific protein with the complementary molecule activates the transmitter substance at the points of contact with the next neurone. This in turn allows the coded information to pass on to the next neurone in the network. The protein in this neurone responds to the same type of electrical pattern whenever it is repeated because it had once been specified through a modulated frequency ion flux of the same pattern. Hoagland [89] has pointed out that the specific RNA and protein are constantly being produced in the neurone in such quantities that, from a statistical point of view, the supply of molecules can be estimated to furnish the necessary permutation possibilities to store the memory experiences of a lifetime.

An outstanding characteristic of neurones is their capacity to produce, among other substances, great amounts of RNA and proteins [97]. In large mammalian neurones the amount of DNA is only about 8-10 uug per nucleus, but in sharp contrast the amount of cytoplasmic RNA, the main part of which is ribosomal RNA, reaches values up to 2, 000 uug [100]. It is not yet known whether the main part of all this ribosomal RNA of the neurone nucleus is an inert but necessary framework of the cell or whether the bulk of the ribosomal RNA comprises a specialized RNA memory storage serving the nervous function [188].

It is here postulated that the available store of memory RNA in the particular mnemonic networks involved in any given behavior operates by the process of inhibiting or exciting cell channels selectively to shape and integrate behavior. Moreover only certain portions of the fund of memory functionally involved can appear in conscious memory at a given moment under the regulation of the mechanism of attention, which itself is an expression of the integration taking place. The delayed appearance of conscious mind and rational will in infancy may be a function of the slow development of a necessary and sufficient fund of memory, in addition to the necessary neural maturation and development of myelinization and dendrites [29]. Flechsig's myelinogenetic law of neural function which holds that certain types of neurones do not start to function until myelinization is complete has been widely accepted [38, 216]. However, even with neural development completed, there is an apparent slowness of learning in childhood indicating that considerable time is required for the accumulation of a sufficient number of appropriately interconnected trained memory cells, called mnemons, to integrate certain behaviors [232, 233]. The classifying cells at many levels are presumably able to make relatively permanent cumulative mnemonic records, subject to interference and forgetting, of those responses to each type of circumstance which are likely to be good for the organism during the long developmental period of infancy and early childhood. The onset of consciousness of self and of abstract language skills shape and accelerate this process along unique lines for each personality. The so-called classifying cells [233] apparently correspond to the cortical "analyzers" discussed by other scientists [137, 166].

It is further suggested that the necessary and sufficient fund of memory, referred to hereafter as the mnemonic network capability (MNC), which enables the central nervous system to integrate any given behavior, be conceived as the insight threshold. The increasingly complex mnemonic network capability requisite for adaptive behavior on an ascending scale of phylogenetic behavioral elaboration (PBE) may similarly be conceived as the insight threshold gradient (ITG). These concepts are illustrated in the following model: see Figure 1. It seems evident that an organism very low on the MNC scale will be below the insight threshold for behavior along considerable upper portions of the PBE scale. This accounts theoretically for the trial-and-error behavior of severely retarded subjects, approaching pure randomness unaffected by relevant cues [10, 148].

In accordance with this theoretical approach, Young [233] postulated that learning consists in the limitation of choice between alternatives and the storage of this new pattern of behavior in the appropriate mnemonic network. To learn how to train such a learning mechanism more efficiently, searching experiments must be devised to study the electrical and chemical patterns of coding and classifying

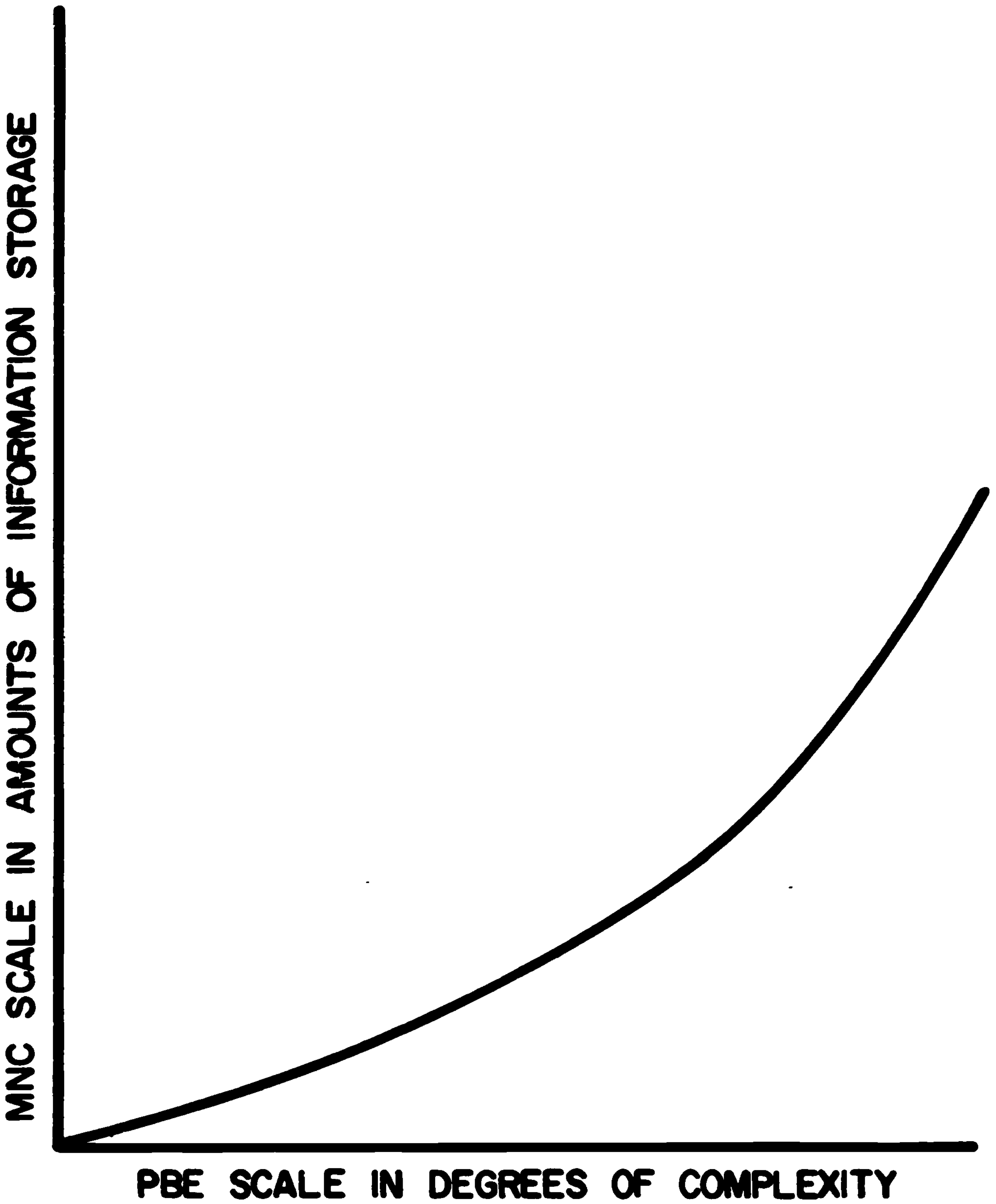

Fig. 1 MODEL OF INSIGHT THRESHOLD GRADIENT

signals as brain mechanisms use them to limit choice in behavior. Educational theory and methodology can only proceed empirically, with no real scientific basis, until this coding and classifying system is understood. The physiologist will approach the problem by devising experiments to determine the biochemical and bioelectrical changes in the classifying cells at a single occasion of learning. The neuropsychologist, on the other hand, will approach the problem operationally by analyzing behavior on repeated trials on diagnostically-chosen standard laboratory discrimination tasks to isolate the characteristics of mnemonic network capability on either side of the insight threshold gradient for such basic standard tasks. Several discrimination tasks are gaining an established record of particular, circumscribed, diagnostic function, especially in ablation studies [150], and in electrical stimulation studies of brain function [205]. It is proposed here that object and spatial discrimination tasks, discrimination reversal tasks, learning-set discrimination tasks, and tactile, visual, and aural models of these discrimination tasks, on the one hand, or delayed reaction tasks and double or single alternation tasks, on the other, be selected diagnostically in order to isolate the differential characteristics of mnemonic function in the contrasting infra- and supra-threshold categories of performance. The importance of the frontal lobe and the function of inhibition in delayed reaction tasks and in discrimination reversal tasks should focus the attention of experimenters on the exceptional significance of these particular tasks for this kind of analytic approach [28, 116, 117, 118, 119, 125, 199, 206].

It is recognized, however, that the physiologist is not going to find all the answers to the coding system for memory in the reductionist approach with the electron microscope [112]. Somehow it will be necessary to understand a macromolecular coding system for memory by deciphering an input pattern of electrical activity involving a complex array of neuronal networks and an equally complex output pattern of electrical activity which integrates the behavior of the organism. The significance of these patterns of activity of the neurones has come under investigation in recent years by means of the microelectrode, the amplifier, the oscilloscope, and the computer. These studies indicate that the pattern code may lie in the modulated frequency of the nerve impulses in the mnemonic network. Hence, the neurophysiologist is concerned not only with the "molecular" question of synthesis of RNA in the neurones, for example, but also with the "molar" question of the decoding of electrical activities of the whole mnemonic system [86]. At this juncture the techniques of neurophysiology and the techniques of neuropsychology merge into an interdisciplinary investigation of the mechanisms of learning and memory [81]. The combination, however, is not likely to overwhelm the obstacles to operationism, for in dealing with the central nervous system via its behavioral output all scientists of whatever specialty will encounter that puzzling but obvious deviation of behavior from control by the environmental stimulation [86]. Even more puzzling is the unmistakable deviation of behavior of the distractible mentally retarded subject despite controlled laboratory stimulation. It is a common feature of attempts to organize experiments with mentally retarded subjects, especially at the more severe levels of retardation where research is most needed, to find that the behavioral pattern cannot be readily or meaningfully controlled by the experimental stimuli. Whatever activity may be ongoing in the so-called autonomous central processes apparently completely precludes the integration of relevant experimental stimulation or focus of attention upon relevant cues in the experimental problem-solving situations [105].

If experimental stimulation is to result in a modification of function at the appropriate classifying cells, the excitation produced by the stimulus must not only be conducted to those cells but must also dominate a large cortical region of divergent conduction where the mass ratio of so-called "association" to "sensory" cortex is large. Conduction from the sensory surface to the cortical projection areas, of course, is in parallel and straightforward seriatim. This presents no great difficulty to the experimenter. The same population of units can be reliably excited, time after time. This follows because the units which begin together and are simultaneously excited also end together and reinforce one another's action at the next synaptic station in serial order along the afferent pathway toward the cortical projection area [85, 86]. Upon arrival at the cortex, however, a relatively small sensory projection area cannot possibly dominate a large region of divergent conduction without some other mechanism to augment the excitation. The same relative difficulty in domination is true of the large thalamocortical sectors which comprise the so-called "association" areas as well as the other rhinencephalic and basal gangliar areas that may be involved in higher mental processes [86, 193]. The augmenting or sensitizing mechanism that enables a small sensory projection area to interact with such a large cortical region has been described by Lorente de Nó [134]. He described the histological structure of these cortical regions of divergent conduction as systems of closed pathways capable of self-maintained activity. Hebb [84] has called these systems "cell assemblies," and Lashley [127] called them "trace systems." The relationship of these systems to the classifying cells or the small specialized signalling cells described by Young [233] is not clear. However, in general functional context the so-called "cell assemblies" and "trace systems" seem to incorporate the classifying cells and the small specialized cells as a whole functional system.

Some seventy years ago the model of the brain proposed by Flechsig [59] described the brain as being composed of receiving areas (the sensory cortex) which functioned primarily to transmit receptor events to adjacent areas of so-called "association" cortex. In the "association" cortex, these sensory neural events were supposedly elaborated and integrated with other neural events before being transmitted to the motor areas of the cortex serving as the principal effector mechanism for all cerebral activity. Recent data obtained by the use of electronic amplifying devices to study neural events [68] make it necessary to modify this old model considerably. Some of the more recent anatomical and histological information about cognitive structures of the brain, actually suggest a new model of the brain and its mechanism.

Not all of the cerebralmantle has been related to cognitive behavior. The medial and basal limbic structures, which have been related to behavior other than cognitive, do not pass through a six-layered embryonic stage and have been designated as "allocortex" or "juxtallocortex." The dorsolateral portions of the cerebral cortex that are related to cognitive behavior do pass through a six-layered embryonic stage and have been designated the "isocortex" [179].

Pribram [173, 174] divides the isocortex into two sectors on the basis of thalamo-cortical relationships determined by the neurohistological technique of retrograde degeneration of neurones in the thalamus following cortical resection [30, 35, 177]. Rose and Woolsey [190] provided the key to the two isocortical sectors by dividing the thalamic nuclei into (1) extrinsic nuclei receiving large tracts of extra-thalamic efferents and (2) intrinsic nuclei receiving the major portions of their direct afferents from within the thalamus.

The portions of the cortex labeled as "extrinsic sectors" in the above system of classification correspond generally to those usually designated as primary projection areas. Those portions labeled "intrinsic" correspond generally to those usually designated as so-called "association" areas. But Pribram prefers to adhere to the currently less loaded terms, "extrinsic" and "intrinsic." He offers two anatomical techniques which permit approximately the same subdivision of the isocortex: (1) one derived from cell body stains; and (2) the second derived from nerve fibre stains [171]. He also offers further support for this basic classification of the isocortex from electrophysiological data [120, 185]. This proposed classification of isocortex into extrinsic and intrinsic sectors does not solve all of the anatomical problems of differential mapping and understanding the functions of these areas [32]. The evidence is, however, sufficient to warrant the division of the cerebral isocortex according to whether its major input derives, via the thalamus, directly from the periphery or whether that input is largely intracerebral.

Bell and Magendie's law, which defines sensory in terms of afferents in the dorsal-spinal and motor in terms of efferents in the ventral-spinal roots, should not be over-generalized to the isocortex. Input from the extracerebral structures reaches the portions of the cortex usually referred to as "motor" as well as to those known as "sensory" areas. Somatic afferents are distributed to both sides of the central fissure, reaching the precentral "motor" areas as well as the postcentral "sensory" areas. This marked overlap of input-output is not limited to the somatic extrinsic system [173, 174]. A similar overlap is evident in the neural mechanisms related to the special senses as well [14, 108, 221]. Because of this afferent-efferent overlap in the extrinsic system, Pribram has suggested that the intrinsic areas need not be considered as association centers upon which pathways from the extrinsic sensory sectors converge to integrate neural events for motor activation. The extrinsic sectors must be further investigated before the differences in detail of the organization and function of the overlapping input to and output from each neurally distinct extrinsic sector will be understood.

It is well established that neurally distinct portions of the extrinsic isocortex are organized to serve distinct classes of behavior. Kluver [114] has shown that the monkey deprived of its occipital lobes reacts visually only to total luminous flux and is deprived of its differential reactions to more complex visual events. Lashley [123] found that a monkey deprived of the precentral cortex reacts only with grossly organized movements and loses more discretely organized patterns of movement. Pribram [173, 174] concludes that invasion of the extrinsic cortical sectors results in impairment that may be characterized as a limit placed on the complexity of possible behavior.

It is apparent that cognitive processes are being inferred operationally from discriminative and problem-solving behavior in neuropsychological experiments. There is a large group of neuropsychological experiments made on the monkey during the past two decades to analyze brain mechanisms [150, 154, 172, 178, 180, 182]. Pribram pointed out how these experiments helped to delineate the organization of the intrinsic cortex in choice behavior. The intrinsic cortex has been found to have two anatomically distinct intrinsic sectors; (1) a posterior or parieto-temporo-preoccipital, and (2) an anterior or frontal. The posterior sector itself may be subdivided into areas, each of which serves a separate sense modality. When the infero-temporal area of the posterior intrinsic system is invaded by resection, the animal is unable to make a great variety of visual choices even though it had learned to make them preoperatively. Occipito-parietal resections interfere with performance of the somesthetic choice problems but not with performance of the visual choice problems. Converse findings have followed infero-temporal resections. Bagshaw and Pribram [14] found evidence that the anterior temporal region functions selectively in taste and olfaction.

The evidence seems conclusive that specific portions of the posterior intrinsic sector of the isocortex are related to discriminative behavior in one or another of the special senses or in somesthesis. It may be concluded, therefore, that cognitive processes are served not only by modality-specific extrinsic mechanisms but by modality-specific intrinsic mechanisms as well [173, 174].

Completely separate behavior factors have been shown to be related to the posterior and to the frontal intrinsic sectors. With reference to the frontal intrinsic sector, Jacobsen [103] has shown an invariant relationship between lesions of the frontal intrinsic system of monkey and decrements in performance of delayed reaction problems. Resections of the frontal intrinsic sector have no effect on the many other types of choice behavior [173, 174]. Pribram concludes that any notion of the frontal lobes as a general "higher" integrative mechanism is quite untenable.

The effects on choice behavior of frontal intrinsic sector lesions have been found to extend to a variety of tasks of the delayed reaction type. Resection of the frontal intrinsic sector is known to impair performance on spatial alternation as well as on non-spatial, visual object alternation [104, 183, 184]. There is also impairment on alternation of two patterns of response rate contingent on two schedules of reinforcement, the deficiency appearing when the alternation is made dependent on the animal's own prior behavior rather than on environmental cues. It seems therefore that resections of the frontal intrinsic sector will impair performance on delayed reaction tasks whenever a correct choice depends exclusively on transient events not present at the time the choice is made irrespective of sensory modality. These experiments highlight the diagnostic value of double alternation tasks and delayed reaction tasks.

The experimental basis for stating that different cortical areas serve different functions applies equally to the intrinsic and the extrinsic sectors. Any effect of resection of the intrinsic system may be duplicated by extensive resection of the extrinsic systems with additional field effects from the latter. For example, resection of the intrinsic visual system limits the complexity of the organism's possible visual behavior but not so much as resection of the extrinsic visual system or that of the receptor sense organ [173, 174]. Thus, there is a hierarchical organization of neural systems in vision, and there are very probably many other hierarchical organizations of isocortical systems serving other categories of behavior. Apparently functionally separable parts of the isocortex do not necessarily serve different functions, but rather may serve the same function in hierarchical fashion.

The old model of cerebral organization in cognitive processes formulated with the "reflex" as prototype, the so-called transcortical reflex model, stating that input is organized in the extrinsic "sensory," elaborated in the intrinsic "associative," and relayed to the extrinsic "motor" sectors, has fallen into further doubt with each new experiment [31, 125, 181, 219]. It is therefore concluded that the hierarchical organization of extrinsic and intrinsic systems is a better model. In this new model, the hierarchical relationship between intrinsic and extrinsic systems can be attributed to convergence of efferents of the two systems at subcortical loci, such as the midbrain colliculi, posterior and anteromedial hypothalamus, and the reticular system of the mesencephalon [155, 175, 176]. Whitlock and Nauta [227] have shown by silver straining techniques that both the intrinsic and the extrinsic sectors implicated in vision by neurophysiological experiments are efferently connected with the superior colliculus. The output of the intrinsic systems, in this manner, influences the extrinsic systems and by regulation of the peripheral sensory mechanism in a centrifugal action influences the input of the extrinsic systems as well.

Pribram has recently concentrated his investigations on the intrinsic systems of the forebrain (telencephalon), because neuropsychological experiments [79, 172] have shown these intrinsic sectors to be especially related to problem-solving processes. He has described in some detail an intrinsic nuclear group and its projections on the forebrain for each of the major thalamic subdivisions, i.e., (1) the posterior intrinsic system, related to the external portion of the thalamus and the dorso-latero-posterior cerebral convexity, and (2) the frontal intrinsic system, related to the internal core of the thalamus and the fronto-medio-basal areas of the cerebral hemispheres.

Experiments aimed at a neuropsychological analysis of the posterior intrinsic system have shown that monkeys with posterior intrinsic sector lesions have deficiencies in differentiation in problem-solving. During the searching portion of the experiment, when it is necessary for the animal to move on successive trials each of the stimulus objects until the positive cue is identified, such behavior is impaired in animals with posterior intrinsic sector ablations. These deficiencies in differentiative behavior in problem solving differ in some respects from those produced by lesions of the extrinsic systems [175, 176, 182]. For example, (1) drastic bilateral removal of an extrinsic sector severely limits differentiative behavior in the modality, and only in the modality, served by that sector. (2) Under some conditions of simple response requirements, differentiative behavior seems unimpaired after drastic posterior intrinsic sector resection. (3) Under other conditions, having in common the requirement that two or more separate responses be systematically related to the differences between the environmental relationships that determine the stimuli, differentiative behavior is severely impaired after posterior intrinsic sector ablations. This kind of impairment has been found in the visual mode in problems requiring brightness, color, form, pattern, size, and flicker discriminations [149, 151, 152]; in problems requiring successive and simultaneous discriminations [182]; in problems requiring successions of learning-set discriminations [32, 189]; and in problems requiring oddity discriminations and matching from sample [78].

Pribram attributes the relationship between intrinsic and extrinsic systems to convergence of efferents from the two systems at a subcortical locus as noted earlier. This hierarchical convergence model bears considerable resemblance to the theory of signals from small specialized cells converging on mnemonic classifying cells to close unneeded channels and integrate behavioral output as proposed by Young [233]. The similarity increases as Pribram develops the model. The specific effects in behavior of the intrinsic systems are explained on the basis of efferents to a subcortically located neural mechanism that has specific functions. These efferents partition (which could be accomplished by the technique of closing unneeded channels) the afferent activity that results in the neural events in the extrinsic sectors, events initiated by and corresponding to the input variables.

Pribram [175, 176] draws upon the theory of games [218] and mathematical learning theory [56] to develop this process of partitioning afferent activity. Partitioning determines the extent of the range of possibilities to which an element or a set of elements can be assigned. Partitioning therefore results in patterns of information, i.e., information given by the elements of the subsets resulting from the partition. The posterior intrinsic sector mechanism provides both the referent and units (perhaps stored in the mnemonic networks), though not the elements to be specified. Continued intrinsic sector activity results in a sequence of patterns of partitions of increasing complexity, which in turn allow more and more precise specification of particular elements in the subsets of events occurring in the extrinsic systems. Thus, through continued posterior intrinsic sector activity, more and more information can be conveyed by any given input. As a result, the organism's differentiative behavior remains invariant under a progressively narrower range of systems of transformation of the input.

Mental retardation, throughout most of its range and constellations of syndromes, seems to be characterized by impairment of precisely the above type of posterior intrinsic sector activity, as well as by other impairments. Because of this type of impairment, the partitioning program of the posterior intrinsic sector fails to operate on the neural events that are initiated by the input and fails to transform them into other neural events which can lead to ever increasingly finer differentiative behavior [70].

The programing of the activities of the posterior intrinsic sectors remains in question at this time, but the concept of changing the state of the cells, closing the unneeded channels, and storing these changes in the base ratios of the ribosenucleotides in the cell structure, provides a working hypothesis. Interaction between posterior and frontal intrinsic sector activities integrates the ongoing program in the posterior sectors with the simultaneously ongoing program of intention in the frontal sectors to produce better adaptation of behavior to its consequences.

The classifying cells at many levels (a hierarchy of extrinsic sector, posterior intrinsic sector, frontal intrinsic sector, and subcortical structure) are presumably able to make mnemonic records of which responses to each type of circumstance are likely to be good for the organism [233]. Pribram holds that in his model the engrams consist of encoded programs. Such a model is consonant with a mnemonic network composed of the complex hierarchy of systems already outlined, but it is emphasized that the posterior intrinsic sectors are not conceived as the loci of storage of an ever increasing number of minutely specific engrams of partitioned subsets. Pribram [175, 176] accepts the evidence as overwhelmingly against the presence in the nervous system of such minutely specific engrams [126]. The evidence of potential storage of minute engrams in RNS molecules [100] or in the vast number of dendritic channels and their synaptic intersections in the neuropil [163], however, would seem to permit the storage of minutely specific engrams of a lifetime with no risk of overload. Further study will be necessary to specify the nature of the engrams. Pribram's model requires the posterior intrinsic sector to be the loci of storage of its own engrams of encoded programing mechanisms, whether specific or general. This concept of storage can be readily translated into our model of closing of unneeded channels and an RNA memory system.

The kind of defect in differentiative behavior that results from lesions (possibly from various kinds of pathology in mental retardation) of the extrinsic and posterior intrinsic sectors of the forebrain can be characterized by stating the variety of transformations of the input under which behavior remains invariant [56, 175, 176]. (1) Following extensive bilateral resections of the extrinsic sectors, behavior will remain invariant under a great variety of transformations of the input. This is very similar to the concept of rigidity in mental retardation [109]. (2) Following lesions of the posterior intrinsic sector, differentiative behavior, but not very simple actions, will remain invariant under the more complex transformations of input. Unique responses requiring absolute differentiations will be precluded, of course, in the extreme case.

The neuropsychological analysis of behavior affected by the frontal intrinsic system has demonstrated the mechanism by which the frontal intrinsic sector influences intentional behavior. Pribram

[175, 176] demonstrated that in multiple object choice behavior the intentions of animals with lesions in the frontal intrinsic sector are guided less than those of controls by the behaviorally relevant consequences of their prior actions. Intentions are determined by two classes of variables, namely, (1) the dispositions of the organisms influenced by cyclical physiological needs, and (2) an estimate about the actions of other parts of the system in the behavioral situation. Animals with lesions appeared deficient in evaluating the second class of variables, because the performance of this group reflected the number of alternatives in the situation with increasing handicap. The results of a separate experiment involving constant (fixed) interval operant conditioning under conditions of food deprivation showed that deprivation influences total rate of response but that frontal intrinsic sector lesions affect the distribution of rate of performance across the intervals indicating little evaluation of the schedule involved. These findings support the thesis that resection of the frontal intrinsic sector impairs intentional behavior by interfering with ability to make an estimate about the actions of other parts of the system in terms of consequences.

A clear-cut impairment in intentional behavior has also been demonstrated by means of delayed reaction and alternation problems using animals with lesions in the frontal intrinsic sector [27, 130]. These two kinds of problems have significant diagnostic value for the effects of frontal intrinsic sector lesions. Although these problems are usually classified with those used primarily to study differentiative behavior, analyses of results of these problems emphasize their recurrent regularities and place them in a larger class of problems that can be distinguished from problems that require differentiative behavior only [65, 175, 176]. The recurrence, at the time response is permitted, of some of the events present in the predelay situation constitutes the essential recurring aspect of the delayed reaction problem [153, 154, 183]. The important determinant of response in these problems is the outcome of the animal's previous reactions, i.e., either (1) the outcome of the reaction in the predelay situation of the delay problem, or (2) the outcome of the previous alternation in the other problem. Lesions of the frontal intrinsic sector characteristically impair such guidance of behavior by the relevant outcomes of prior actions.

Pribram also proposes a model of the frontal intrinsic and the mediobasal forebrain mechanisms in terms of the variety of transformations of descriptions of the outcome under which behavior remains invariant. (1) Following extensive bilateral resections of the mediobasal systems, behavior remains invariant over a wide variety of transformations of outcome. Even gross changes in the amount of food deprivation minimally alter the rate of response to food [47, 63, 225]. This type of deficit is seen in severe mental retardation as well. (2) Frontal intrinsic sector lesions, on the other hand, affect intentional behavior that remains invariant only under the more restricted ranges of transformations of outcome. This impairment is probably involved in the low efficiency of performance of the mentally retarded on problems they can solve.

As in the case of the posterior intrinsic mechanisms, Pribram conceives that the frontal intrinsic mechanism partitions the events in the mediobasal forebrain systems, i.e., the dispositional events aroused by cyclical variations in the physiological needs of the organism. Partitioning results in distributions of intentions that are determined by the elements of the subsets resulting from the partition. Thus, the frontal intrinsic mechanism provides both referent and units, but not the elements that specify the intentional behavior of any given situation. The mechanism in operation results in an increasingly complex sequence of distributions of intentions which in turn allow more and more precise specifications of intent that can be conveyed for any given outcome. Where there is no lesion or impairment of the frontal intrinsic sector, then, the organism's intentional behavior remains invariant under a progressively narrower range of systems of transformations of outcomes. Storage of encoded programs is required, not storage of an ever-increasing number of minutely specific preferences. Presently, it is better to recognize that this postulate is quite plausible, and may very well be confirmed, but is not necessarily required by the nature of neural mechanisms [100, 163]. This model is, however, in full accord with the postulated hierarchical convergence model of mnemonic network structure previously discussed. The frontal intrinsic and mediobasal forebrain structures and mechanisms constitute interacting networks in a hierarchy that sends out converging efferents upon allocortical and subcortical loci, such as the hippocampal gyrus, uncus, dentate convolution, pyriform cortex, entorhinal cortex, cingular gyrus, amygdaloid nucleus, and the tegmental region of the midbrain. Analyses of the functional relations of these neural structures to learning by means of ablation, resection, drug, microelectrode, impedance, and computer studies are in progress [3, 4, 5, 6, 7].

It has occurred to some that the neural mechanism of signal analysis in the brain might involve comparison of wave processes on a statistical basis [8, 23, 58]. Differentiations might be made on the statistical probability that the afferent patterns in the brain are significantly different from intrinsic, mnemonic patterns which the past experiences of the brain have set its mnemonic networks to "expect." The complexities of coding in patterns of neural discharge [136] are compounded by sequential transformations in serial synaptic stations and by great variations in the degree of convergence and divergence of different neuronal aggregates [23]. The findings of Whitfield [226] in the otic system indicate that the rate of firing becomes progressively less as the impulses proceed through serial synaptic stations on their way to the cortical projection area and that the rate of unit firing becomes less dependent upon the strength of the stimulus at each successive synapse. This means that the intensity of the stimulus is no longer being signalled simply by frequency of discharge and that the coding has been changed somehow, perhaps by the distribution of excitation and inhibition among the fibres of the pathways by centrifugal control from central processes [23].

A recent approach [136] to the study of this electrical activity in isolated ganglia provided a systematic analysis of the different kinds of responses available after electrical stimulation by various frequencies. The relationships between electrical activity in the afferent and efferent fibres of isolated ganglia and the processes of plasticity, rudimentary learning, memory, facilitation, inhibition, post-tetanic potentiation, post-tetanic effect on spontaneous activity, and habituation were examined. An in vitro preparation of the sixth abdominal and the metathoracic ganglion of the Blatta orientalis was utilized.

Six types of response of this preparation to electrical stimulation were recorded and analyzed in regard to the possibility of a plasticity of response dependent on the past experience of the ganglia, i.e., the immediate past experimental stimulation. (1) A servile response was recorded, in which the ganglia gave a constant response until they became fatigued. This response could be modified in most species by immediate past stimulation that resulted in post-tetanic potentiation, the simplest form of memory in the cells lasting for an appreciable interval. (2) A labile response was observed, in which there was a characteristic cessation of response to stimuli at a certain frequency range but capability of renewed response by increasing the frequency markedly. Thus this response type could be modified by a long-lasting habituation as a result of the immediate past stimulation of the ganglia and was subject to prompt dishabituation by a sharp change in the stimulation. (3) An intratetanic response was noted, in which the discharge was evoked some time after the tetanus was applied and after some summation of stimulation. This response evidenced some plasticity by summation of stimulation before discharge. (4) An afterdischarge response, in which the bursts appeared promptly but only after an irregular few of a series of stimuli, such as twenty tetani of one-second duration given at 9-second intervals. This represented an unpredictable plasticity in behavior of the ganglia and occurred with or without the classical delayed response activity. (5) A delayed response, in which the discharge activity appeared after considerable delay and summation of stimulation including summation with previous delayed responses to reach increasing response levels. This type of response represented great plasticity in reaction of the ganglia to stimulation and sometimes resulted in activity in the responding unit lasting for several minutes. (6) A natural response, in which the activity started only after summation of several stimuli of high frequency and took a course of increasing discharge frequency until it reached a maximum and then declined. This kind of natural behavior of the ganglia represented a complete pattern of spontaneous response discharge once the tetani reached a certain threshold and denoted the maximum electrical plasticity of the cell in an all-or-none style of function [136].

The electrical activity of this kind of preparation recorded outside of or beyond the period of stimulation should perhaps properly be designated as spontaneous behavior induced by, but proceeding independently of, the threshold stimulation. Considered as spontaneous activity, the frequency of this activity probably has a certain characteristic statistical value for each type of neural aggregation, which value it attains in an all-or-none pattern of discharges after threshold tetanic stimulation. Hence, Luco [136] holds that all six of these types of electrical response of nerve fibres fall into three general stages: (1) The pattern follows a certain law of servility before the spontaneous activity appears. (2) The pattern of discharge becomes very unpredictable because of the continuously changing frequency and type of spontaneous discharges to certain kinds of stimulation. (3) The pattern of discharge develops an expected and predictable pattern as a consequence of stabilization in the frequency characteristic of its spontaneous activity.

While these characteristics of ganglial function may provide a neurophysiological mechanism for a short-lasting memory, this mechanism cannot account for memory that persists after total and long-lasting cessation of electrical activity. Luco, therefore, postulates that the past experience of stimulation of the ganglial fibres leaves a trace in the autogenic generator that modifies the frequency of the spontaneous activity triggered by the stimulation or causes a lowering of the threshold for the release of this spontaneous activity. Delayed responses and natural responses are therefore postulated to be the characteristic expressions of an autogenic spontaneous activity triggered by stimulation.

The isolated preparation is not the only means of study of the electrical activity of the central nervous system. Stimulation and recording by way of implanted microelectrodes in the intact and unrestrained functional animal and in some cases in the human brain being treated for neurological disorders are available techniques [22, 24, 25]. Recent approaches of this type [3, 24, 25] have focused experimental attention on the hippocampal systems in analyses of the electrical activities of the brain and the relationship of these activities to the laying down of a memory trace. The great antiquity of the hippocampal systems in the evolution of the brain and the essential stability of its basic structure in the face of immense evolutionary changes in the remainder of the cerebral mantle make it highly probable that these systems may constitute the classifying cells that have outputs that lead directly to behavioral output [233]. Experimental evidence indicates the apparent relationship of hippocampal integrity to aspects of recent memory and directed attention involving discriminative choice [3]. The memory trace may actually be laid down outside the hippocampal system [167], perhaps in the exterior and posterior-frontal intrinsic systems of the forebrain in discriminative behavior, but integrity of its interrelations with these seemingly unrelated cortical and subcortical regions of the hippocampal systems may be vital to the appropriate recall of previously learned discriminative habits [9].

This new approach seeks to determine changes in patterns of electrophysiological activity closely correlated with the acquisition of the learned discriminative task by taking measures of electrical activity in the hippocampal system and related structures during the course of learning. It also seeks to determine whether such changes in electrical patterns would reveal anything about the fundamental nature of the processes by which information is stored in cerebral tissue [3]. Some changes of this nature have already been detected by the application of a series of increasingly refined computing techniques [4, 7, 8]. Because of the nature of these results, it has been postulated that the electrical brain wave process is of basic importance in the handling of information in cerebral systems. It has also been postulated that the neuron in the cerebral system functions as a phase comparator of the patterns of waves which sweep across the neuronal surface in complex spatio-temporal patterns that arise both intrinsically and extrinsically [3].

The recording of wave patterns and the computer analysis of these patterns of electrical activity of the hippocampal systems of the temporal lobe, together with the external and posterior-frontal intrinsic systems of the forebrain, during the course of acquisition of a discriminative habit may yet develop the code that unlocks the transactional mechanisms in cerebral tissue in the learning process and storage in memory. A technique has been developed for measuring impedance in small volumes of hippocampal neuronal and glial tissue with implanted electrodes. This technique has revealed substantial but transient impedance shifts that are confined to the period of the discriminative performance after learning has risen appreciably above chance levels [5, 6]. It was found that the "evoked" impedance change persisted in undiminished degree with considerable overtraining and that these impedance changes were abolished by extinction of the learned habit and redeveloped with retraining. It has also been found that these impedance changes do not appear simultaneously at the same level of learning and to the same degree in different hippocampal structures, i.e., the symmetric left and right hippocampi. During the course of learning, the concomitant changes in averaged hippocampal impedance records were found to occur more slowly in one side than in an essentially symmetrical hippocampal placement in the opposite dominant hemisphere until performance reached the one hundred per cent level, at which time the records became similar from both left and right hippocampi [3].

A simple averaging technique for computer analysis of hippocampal wave trains with the onset of the averaged epoch time-locked to the moment of presentation of the test situation and a reinforcing tone has been developed and applied with significant results by Adey and Walter [7]. This technique has shown that the hippocampal electrical activity in the pre-approach epoch behaved as a random phenomenon and progressively diminished as such after several discriminations, but at the same time the rhythmic averages during the discriminative epoch grew progressively larger. The regularity of the rhythmic averages in the hippocampal system were found to persist steadily into overtraining [3], which indicates the inherent continuing relationship between this hippocampal system and performance of learned behavior. It has also been discovered that the regularity of the rhythmic averages in the midbrain and subthalamic records tends to decline once the learned performance has been substantially overtrained. It is altogether possible that the hippocampal systems are functionally identical with the classifying cells postulated by Young [233]. The classifying cells stand at the electrical gateway to the appropriate channels for behavioral output and function by the postulated mechanism of closing unneeded channels. Such a mechanism must therefore be activated on every occasion of performance of the learned discriminative behavior and presumably must be activated by the same characteristic pattern of rhythmic averages in the electrical wave processes. Only in such an essential classifying cell mechanism, however, would this activation always be required in overtraining or in subsequent retrieval of stored mnemonic experiences of discriminative learning. Hence, the gradual decline of these rhythmic averages in other brain structures after learning and during overtraining would occur because they were needed for laying down of the memory trace in all the systems involved in the integration, but not for subsequent performance. The classifying cells of the hippocampal systems may therefore be postulated to be preset by coding in the base ratios of their ribosenucleotides, or their small signalling cells, or both, for the transaction of identical and similar discriminative performances in the future. The autogenic electrotonic activity of the dendritic trees of the small cells will then be coded or modulated by the RNA storage via glia cell impedance changes to produce the pattern of wave rhythms necessary to trigger the learned response. It is further postulated that impedance shifts arise in a redistribution of ionic material in the various tissue compartments comprising the cerebral systems.

The glial tissue compartment comprises a significant portion of the volume of the dendritic zone of the hippocampal pyramidal cell layer. True extracellular space has been found to be quite small [72]. It therefore is probably not significantly available even assuming it could be a low resistance shunt path that might mask any changes attributable to altered resistance and impedance in physiologically active neuronal membranes [5]. The glial compartment has been found to be an intervening structural enclosure around most of the neuronal cells. Thus the glia intervene between the neurones and their vascular system in metabolic exchanges [3]. The evidence available on the low membrane resistance of glial cells and the long-lasting characteristics after electrical stimulation give strong support to the postulate that the glial cells exercise a modulating role on electrotonic dendritic processes in respect to both their rate and regularity in rhythmicity. Finally the site of the actual laying down of the memory trace in the RNA structures of both the glia and the neurones may be at the functional interface between neuronal and glial tissue. The mechanism involved may perhaps be found by the application of techniques for impedance measurements with coherent electromagnetic sources (radioactive materials) in the spectral absorption range of substances such as the nucleic acids [3]. The mechanism for the storage of learning and the mechanism for ionic transport responsible for restablishment of cellular excitation or inhibition remain the crucial targets of investigation.

Summary

The key to the theoretical framework of the present investigation is the division of the isocortex and related thalamic nuclei into an extrinsic division receiving large tracts of extra-thalamic sensory afferents and an intrinsic division receiving its major direct afferents from within the thalamus. The Lashley-Jacobsen-Kluver-Pribram-Mishkin line of investigation of cognitive processes of the isocortex organizes both the extrinsic and intrinsic sectors into neurally distinct portions serving distinct visual, aural, and other classes of cerebral function. Employing ablation and resection techniques, these studies have shown that functionally separable parts of the isocortex do not necessarily serve different basic functions but rather may serve the same function in hierarchical organization.

Neuropsychological experiments with animals having distinct intrinsic sector lesions have shown the separate characteristic functions of the frontal and posterior intrinsic systems in problem solving. Posterior intrinsic system lesions cause a deficit in differentiative behavior in problem-solving impairing a systematic search for relevant clues. On the other hand, frontal intrinsic system lesions cause a deficit in intentional behavior, impairing guidance of new action by the behaviorally relevant consequences of prior actions. The frontal and posterior intrinsic systems interact to shape behavior by means of a hierarchical neural organization involving the convergence of efferents from the two systems at subcortical loci.

Young's theory of memory as a function of classifying cells at

many levels (perhaps embracing a hierarchy of extrinsic, posterior intrinsic, and frontal intrinsic systems) capable of making mnemonic records of which responses to different circumstances are likely to be good for the organism has been accepted for the present study. The Hyden-Babich line of investigation of the relationship between the base ratios in the ribosenucleotides in neural and glial molecules and the functions of genetic and psychic memory in the neurones has been taken as the key to the brain mechanisms of memory. A composite model of these two approaches to memory requires the suggested systems of classifying cells that close the unneeded channels of behavioral output under given circumstances and the storage of encoded programs for action in long-lasting changes in the base ratios in the ribosenucleotides in the molecular structure of the neurones and their associated glia.

The mechanism of classifying and encoding programs has, for purposes of the present study, been taken to be one of neuronal signal analysis of electrical brain wave processes. By this method of handling of information in the brain, the neurons in the cerebral systems function as phase comparators of the patterns of electrical waves which sweep across neuronal surfaces in complex spatiotemporal patterns that arise both from extrinsic sensory systems and from intrinsic systems and autogenic sources. The wave rhythms are modified by impedance shifts which arise in a redistribution of ionic material at the functional interface of the neuronal and associated glial tissue compartments. The chemical mechanisms of ionic transport in impedance shifts and transduction of ionic flux into RNA molecular storage interact continuously with the ongoing electrical brain wave processes to influence behavioral output.

III. RELATED RESEARCH ON LEARNING AND PROBLEM SOLVING IN MENTAL RETARDATION

From the neuropsychological point of view of the present study [140, 192, 193], it is not feasible to limit this review and subsequent interpretations of findings strictly to the topics of discrimination and problem solving. The fundamental brain mechanisms involved in the basic neurophysiological processes in learning inevitably overarch and derogate any such arbitrary topical divisions [42]. Accordingly, discrimination and problem solving research will be given primary but not exclusive consideration here as two of the many topics embraced by the larger basic subject of learning research [24, 25, 42, 88, 204].

Neither is it feasible to restrict the present review to those studies which expressly include mentally retarded subjects although such studies will occupy the bulk of attention. It is not surprising to discover that psychologists have tended to utilize types of problems and methodology that have been developed with other types of Ss, including animals, and to interpret the performance of the mentally retarded in terms of these previously developed theoretical formulations [211]. The development of the experimental science of neuropsychology in the field of learning in mental retardation, while it may eventually be expected to take somewhat specialized directions, will continue to draw upon problems, methods, and theory developed by psychologists and neurophysiologists with all kinds of Ss involved in various types of learning experiments, particularly those designed to reveal the underlying basic brain mechanisms [33, 60, 62, 83, 101, 150, 153, 154, 210].

The topics of discrimination learning and problem solving have been included in several reviews of learning research within the past decade [50, 53, 64, 111, 209]. Most recently, Davis [40] reviewed the major theoretical trends in the interpretation of problem solving, as well as concept learning, originality and creativity. He presented a learning approach to problem solving in terms of "overt" and "covert" trial-and-error behavior [48, 204]. He pointed out that the concept of "transfer" has reappeared with substantial regularity in recent research to express the effects on problem solving performance of such factors as "practice," "pre-training," "direction," "centering," "set," "fixation," "learning set," and "available functions." Recent experiments which demonstrate that pretraining can result in positive, negative, or zero transfer were cited [19, 90, 141].

Several writers since the middle of the twentieth century have attempted to organize and develop the topic of problem solving [50, 64, 107, 110, 186, 202]. Rosenberg [204] considered the attempt to define problem solving a frustrating task but avowed a general preference for broad definitions rather than a multitude of operational definitions. Davis [40] expressed the even stronger view that problem solving is "the most chaotic of all identifiable categories of human learning." From a neuropsychological point of view, it has become apparent that problem solving has become an anachronism along with "animal spirits," "soul," "mind," "insight," and other such elusive concepts developed long before the modern assault upon actual brain mechanisms and learning [24, 25, 42]. "Problem solving" is not an apt description of a definitive type of experimental procedure such as "conditioning," "operant conditioning," or "discrimination." It has rather developed as a label of convenience for a miscellaneous collection of procedures in learning experiments. As long as the subject of learning remains organized largely on the basis of experimental procedures, this miscellaneous chaos will continue in the literature.

Since problem solving will doubtless remain with us indefinitely as such a collection, it is probably best defined in terms identical in nature with the scientific method for the building of a body of knowledge. Gagne [64] thus defined problem-solving behavior in terms of five stages of response development, viz., (1) apprehending the stimulus situation, (2) forming the relevant concepts, (3) searching the alternative responses, (4) selecting the effective responses, and (5) verifying the outcomes. Various writers seem to stress different stages of this complete definition of what one must recognize at once as the definition of complex learning. For example, Skinner [202] emphasized manipulation of the relevant conditions to facilitate effective response. This would seem to place stress on stages two and three, perhaps in a systematic recycling until the solution is found. Such systematic flexibility is generally found to be severely impaired in the mentally retarded. Duncan [50] would stress stages three and four by placing importance on the discovery of the correct solution. Ray [186] would stress stages three and four in his concern with solution-oriented behavior. Johnson [107] would stress stages one, three, and four by emphasizing complex stimulus situations, varied behavior, and solution processes. Kendler, Glucksberg, and Keston [110] placed emphasis upon stages three and four by distinguishing problem solving from conditioning on the basis of the longer response chains involved.

While there is no neuropsychological evidence for stressing any particular stage of complex learning as peculiar to, or characteristic of, a separate entity known as problem-solving behavior, there is abundant neurophysiological evidence that complex learning, requiring longer response chains, e.g., in problems requiring successions of learning set discriminations, is impaired by resection or ablation of the posterior intrinsic sector [32, 175, 176]. There is likewise evidence that very simple learning, e.g., a simple position discrimination, may not be impaired by such resection or ablation of the posterior intrinsic sector of the forebrain. Hence, although there is no basis in brain mechanisms for a problem-solving category of learning as such, there are very fundamental anatomic, histological, and functional reasons for separate categories of simple versus complex learning. The precise nature of simple learning remains to be developed in much greater neurophysiological specificity for all the sensory modalities, particularly the kinaesthetic, tactile, and olfactory receptors [157]. It has been determined that the capacity to discriminate difficult visual patterns (complex learning) is permanently lost after bilateral total ablation of the visual cortex, but discriminations of simple visual patterns (simple learning) can be mediated by small remnants of the visual projection areas [126, 157]. Ablation of preoccipital cortex will cause deficiencies, limiting the animal to simple learning, in visual pattern discrimination [1, 2]. Ablation of association cortex in the parietotemporo-preoccipital region will also cause deficiencies, reducing the subject to simple learning, in visual pattern discrimination [34]. Neff [157] clearly described what happens when an animal capable only of simple learning is presented with a test of complex learning:

> "Any investigator who has tested the sensory discriminations of animals in a multiple choice situation is familiar with a pattern of behavior which often occurs when the discrimination is made very difficult. The experimental animal which has been making appropriate responses in less difficult discriminations suddenly appears to ignore the stimulus cues and adopts a position habit such as always selecting the stimulus

on the right. This behavior can be described most readily by saying that the animal acts as if it does not 'attend' to the stimulus cues which the experimenter is presenting."

In research with mentally retarded subjects, it is of great practical importance that one goal of experimentation be the delineation of the basic nature of simple versus complex learning in the several sensory modalities [191]. The resulting hypothetical constructs [138] can then be used to provide the logical framework for the subject of learning and perhaps bring some order out of the chaotic condition in the anachronistic category of problem solving.

In the meantime, some logical organization might start with the working hypothesis that the cortical neuron by a stochastic mode of operation functions as a phase comparator of extrinsic sensory input and intrinsic mnemonic and electrotonic processes, including glial modulation by nonlinear impedance loading, that sweep across the neuronal membrane in complex spatiotemporal patterns [3]. By this stochastic process the classifying neurones make a determination that these extrinsic and intrinsic brain wave processes are similar or different for purposes of behavioral response.

Bearing in mind that the above postulated mechanism for the mediation of behavior by the central nervous system is a working hypothesis only, the following constructs and interpretations of research are suggested knowing they will go the way of all other working hypotheses as soon as they have generated new evidence upon which better constructs can be built.

Phase comparison of brain wave processes and stochastic classification into a binary plan of similar versus different patterns on a probability basis must lead logically to the recognition of certain basic neuropsychological constructs as underlying principles of learning. One of these constructs is DIFFERENTIATION, the basic function of classifying the information carried by brain wave processes as similar or different, the underlying discriminating mechanism for all learning, and the peculiar function of the posterior intrinsic sector of the forebrain [175, 176]. This function is not limited to any particular sensory modality. The stochastic differentiation of the information carried by extrinsic and intrinsic brain wave processes as different (NOVEL) is a form of simple learning when the novel is positive, presenting little difficulty even for animals with frontal lesions [150], but such animals are severely impaired if the novel element is negative and requires inhibition of approach [150, 206].

Another basic construct is INTENTION, the basic function of classifying the information in brain wave processes as indicating "approach" or "avoidance," the underlying mechanism of motivation for all learning, and the peculiar function of the frontal intrinsic sector of the forebrain. Intention is the result of the stochastic comparison of patterns of brain wave processes in the extrinsic and mediobasal forebrain systems with patterns in the frontal intrinsic sector to produce more and more precise behavior from moment to moment. Strategic problem solution involves the economic restriction of the range of systems of transformations of the input and outcome of neural events into increasingly complex hierarchies of intention [44, 84, 124, 175, 176].

The definition of these first two constructs required the use of a third basic construct of CLASSIFICATION, the function of neurons in the intrinsic systems of the forebrain (telencephalon). In the interest of parsimony in neurophysiology, there seems to be good reason to postulate that the process of classification of neural events as "similar" or "different" in the posterior intrinsic system is electrophysiologically identical with the process of classification of neural events as indicative of "approach" or "avoidance" in the functions of the frontal intrinsic system. The neurons in both cerebral systems have the integrative capacity to act as phase comparators of the patterns of electrophysiological waves that sweep across the neuronal surface in complex spatiotemporal patterns from both extrinsic and intrinsic sources [3, 7]. Consistent phase patterns in hippocampal wave trains, for example, are intimately related to this process of classification in the learning process. The conveyance of information on the basis of graded analog wave processes in these cerebral systems is the current thesis on the mechanism of classification [3]. Continued activity in these intrinsic systems results in a sequence of patterns of information of increasing complexity, i.e., partitions of elements or sets and subsets of elements of neural events [218], which in turn allow more and more precise specification of particular elements in the set of events occurring in the extrinsic systems.

The fourth basic construct is MEMORY, the function of setting up a representation of learning in cerebral systems, consisting of a record of the classifications made in reaction to elements of the external environment involved in the learning situation. In the evolulution of the memory mechanism of the brain, there has been a gradual release from dependence on the classifying systems built in by heredity [233]. Hyden [97] postulates that the record is written by changes in the base ratios of ribosenucleotides in the classifying neurons of cerebral systems in response to volleys of specifically timed impulses. Adey [3] postulates more precisely that the record is written at the functional interface between neuronal and glial tissue by means of ionic exchanges involving glial tissue acting as a varying nonlinear impedance on the electrotonic wave processes in dendritic structures and chemically specifying corresponding changes in the base ratios of ribosenucleotides.

Specialized small cells in the spinal cord produce an inhibitory enzyme that blocks transmission in a pathway [51, 222]. Young [233] suggests that this biological mechanism has been extended into the higher cerebral systems as the fundamental electrophysiological process of classification of neural events in learning. Combining this formulation with the findings of Adey [3] on impedance shifts in glial tissue, it may be postulated that an inhibitory enzyme involved in the ionic exchanges between neuronal and glial compartments may be the chemical mechanism for impedance shifts, which in turn become the electrical mechanism for the blocking of an unneeded pathway in a sequence of more and more precise differentiations and intentions.

In view of the present neuropsychological approach to mental retardation, the nature of embryonic and postnatal growth and development of the isocortical structures and functions becomes important. A cytological analysis of the cortex of a human fetus of eighteen weeks revealed sufficient cellular differentiation to distinguish five local areas. One area of pyramidal cells (Betz cells), which corresponds roughly to the pre-central motor area of the mature brain, showed the most advanced development [20]. A study of the brain stem of fetuses between six months of age and birth, using the Weigert-Pal method of staining post-mortem sections to bring out the medullation of nerve tracts, revealed that the tracts become medullated in the order of their phylogenetic development in general. However, reflex activity may be elicited prior to myelinization, and the inception of function advances the medullary process [121, 122]. These studies show that the infracortical centers are the ones best prepared by the most advanced medullation to govern neuromuscular activities of the neonate.

Myelinization is not essential for a large share of the pyramidal tract fibers involved in the pathways for voluntary motor impulses [128, 129, 194]. Whether the unmyelinated fibers differ in function from the myelinated ones is not yet known. The large fibers originating in the Betz cells, confined to a small locus in the motor cortex, appear to be only one component of the motor pathways. Other cortical areas, such as the post-central gyrus and others, give origin to the pyramidal tract [128, 194]. Tracts other than the pyramidal are involved in voluntary activity, viz., the so-called "extra-pyramidal system" consisting of complex descending chains of fibers, interrupted synaptically in the nuclei of the basal ganglia and the brain stem, that finally reach the spinal cord. The cerebellum is also involved in the extra-pyramidal system in such a manner that the basal ganglia and the cerebellum may influence cortical activity by "feedback" circuits [129]. It seems probable that the reticular system of the brain stem, consisting of clusters of cells interwoven with fibers, may also be involved in voluntary activity [194].

Granting the complexity of the motor systems of the central nervous system, it is useful to know the pace of development of the motor tracts. General cortical differentiation, characterized by a series of migratory laminations resulting in a six-layered neocortex, has begun in the brain of the seven-week-old embryo [215]. The six cortical layers are all basically laid down in this process of lamination by the four-month-old stage of fetal development, but the migratory process is not fully completed until the seventh month. Local cortical differentiation, distinguished by the rearrangement and disposition of cells which produce such local areas as the motor, auditory, and visual, develops rapidly during the last stages of fetal development [215]. Conel [37] studied the development of cytological structures of the cortex of the newborn infant and reported that even in the newborn cortex the cells have achieved their adult arrangement, but not medullation or proliferation, which is in agreement with the findings of Tilney [215] that the cellular division and migratory processes are complete at birth. According to the four basic criteria of advancing structural development used by Conel [37], the cells in the most advanced stage of development at the time of birth are those in that part of the anterior-central gyrus which mediates movements of the neck and shoulders. After one month, the greatest change from the condition of the neonate's brain has taken place in the motor area of the gyrus centralis that mediates movements of the hands [38].

Postnatal maturation of the infant's brain cannot be attributed to multiplication of subdivision of the cells, for they are all in place at the time of birth. Hence, cell proliferation has been considered as the fundamental criterion of postnatal structural maturation. Using the Golgi method of staining to determine advancement in proliferation of dendritic processes, de Crinis [41] analyzed sixty-eight human brains, varying in age from five days to thirteen years. He found dendritic processes clearly developing in the brain of a ten-week-old infant. He found that the motor region of the eleven-month-old infant's cortex has reached an advanced stage of development, characterized by pyramidal cells that have become strikingly long and dendritic processes that are well matured. This motor region and the primary auditory and visual projection areas of the human cortex were found to be the first to mature. Broca's area does not achieve a comparable stage of development until about the age of seventeen months. It is not until approximately the fourth year that the apical processes of the frontal lobe develop to a stage comparable to that of the motor area at eleven months. Thus, the crucial portions of the frontal intrinsic system, as well as the posterior intrinsic system, that are required for the most complex differentiations and intentions in behavior are the latest maturing areas of the human cortex [41, 147, 175, 176].

Acquisition of sensorimotor skills is of primary importance during early childhood in view of the preceding pattern of development of the central nervous system. It seems fairly evident that certain neurostructural growth must take place prior to the onset of overt function, but it seems equally evident that completion of all neurostructural growth is not a prerequisite for the onset of function [144, 147]. As a matter of fact, an influential factor in determining the structural growth of one neural component may be the stimulation received from the functioning of other structures which are interrelated. Obviously, in such an interrelated system, rigid demarcation between structure and function as two distinct processes of growth is quite impossible. However, it is possible to determine grossly those periods (1) when neuromuscular function is predominantly under nuclear, subcortical control, (2) when the cortex begins to suppress or inhibit the nuclear activities, and (3) when the cortex takes a dominant role in controlling neuromuscular behavior patterns [146, 147].

Many of the typically newborn behavior patterns of neuromuscular activity appear to be of phyletic origin. The course of development shows that nuclear, subcortical control reaches maximum manifestation about the end of the first postnatal month and then declines progressively between the first and fourth months. There is a gradual onset of cortical inhibitory influences, starting with the muscular activity of the upper part of the body and developing essentially in a cephalocaudal pattern [36, 146]. Complete cortical control of neuromuscular behavior patterns is achieved generally by the eleventh month and is exemplified in the achievement of erect locomotion [145]. At the inception of cortical control of sensorimotor activities, the movements are usually poorly coordinated. Further development of cortical control is indicated by increasing integration of the necessary movements [69, 147].

The integrative capacity of the human cortex is an amazingly variable function, both in onset and subsequent development, depending upon antecedent physiological and mnemonic (stored experiential) conditions of a given individual's cortical growth and development. Since growth in structure in terms of postnatal cell proliferation is not complete until the fourth year for the latest maturing intrinsic sectors of the telencephalon, since the motor area and certain primary sensory projection areas are the earliest maturing sectors at eleven months, and since the acquisition of sensorimotor as well as problem solving skills logically seems to require at least some limited function of all these different sectors, it is useful to consider the course of acquisition of skills between eleven and forty-eight months. The performance of older retarded children with MA's in this range is also important.

Ketterlinus [113] used three groups of Ss in the age groups of 2, 3, and 4 years to determine whether very young normal children were able to adapt to mirror reversal. For example, one problem was to pick up objects and put them in a cup when both the objects and cup could be seen only in a mirror. Acquisition curves showed a decrease in time to perform the task with practice, but the rate of decrease was greatest for the youngest group and least for the oldest. In other words, the youngest group began the experiment with the poorest level of efficiency but made the best rate of improvement. Since growth in structure and function are inextricably interwoven at this age range, the initial differences between age groups in favor of successively higher levels of CA may logically be attributed to greater growth in this structure-function complex. However, the greater rates of improvement in favor of the successively lower levels of CA, on the other hand, should logically be attributed to the stimulation of growth produced by the functioning of interrelated neural components.

Another investigation of the age factor in maze learning by very young normal children revealed the same pattern of initial differences in growth and subsequent differences in rates of improvement [143]. There were twenty children, ten boys and ten girls, in each of three age groups, averaging approximately 3, 4, and 5 years. The design of this study bridged the important fourth year when the latest maturing cortical areas reached an advanced stage of structural growth. Each child in the experiment was required to push a stylus along a grooved pathway five trials daily for five days per week until a total of fifty trials had been completed. Learning curves based upon median time scores showed that the five-year age group differed significantly from both younger groups in greater efficiency of initial performance on the first five trials. Final levels of performance were similar, however, for all age groups. Thus, the course of learning during the experiment was quite similar regardless of age, but the younger the group the greater the rate and absolute amount of gains. Hence, advanced growth in structure and function improves initial efficiency by better cortical integration of sensorimotor skills but is not a necessary prerequisite for learning such skills. The necessary growth in function may be stimulated by training during the age range embraced by this study.

Efficiency of initial or final performance levels on sensorimotor skills is not dependent entirely upon the single factor of growth in cortical structure and function. The factor of complexity of the learning required of a given individual operates interdependently with growth to affect the level of efficiency of performance. Simple learning and complex learning are not discrete categories but represent a continuous variable interacting with growth on a phylogenetic as well as an ontogenetic scale [115, 217]. Mattson [142] investigated the relative influence of maturation and practice on sensorimotor skills at three levels of complexity. Simple, intermediate, and complex rolling-ball maze patterns were used with twenty-four boys and twenty-six girls having an average age of about 5 1/2 years and an average IQ of 103. Practice and control groups were matched for age, sex, IQ, and maze scores for the initial four days of testing. Then, the practice group was given training on all three maze patterns each day for twenty-six days in such counterbalanced order that, in nine trials per day, each pattern came once as first, once as second, and once as third. Thus, the two groups differed only in that the practice group had twenty-six more days of tests than the control group. Although the practice and control groups began the experiment with approximately matched efficiency, the practice group manifested significantly superior performances on each of the three mazes in the test at the end of training and also in the retest given sixty days later. The advantage of the practice group over the control group in the test at the end of training was significantly greater as the complexity of the task increased. Matching groups on initial performance and subjecting one group to growth in function (another way of saying learning) on three levels of complexity, revealed the interacting effects of level of growth and level of complexity. The effect of growth in function was significantly greatest for the most complex task, but in terms of relative efficiency at any given period of life the final level of performance on the most complex task may be worse, or at least no better, than the initial level of performance on the simplest task.

Several investigators have been unable to find any significant differences between normals and retardates on learning simple sensorimotor skills [106, 187, 228, 231]. Woodrow [231] used a variety of sorting tasks that were simple enough to pose no new discrimination learning for the retardates. He selected a retarded group (CA = 13-14; MA = 8-9 years) and a normal control group matched on MA and compared their performance in terms of speed of sorting sticks of varying lengths, pegs of different colors, letters of the alphabet, and simple geometric forms. There were no significant differences between the groups on speed of sorting, amount of improvement in speed, or in amount of transfer among tasks. A discrimination task such as these easy sortings amounts to no more than simple learning of a sensorimotor skill for normal or retarded Ss with an MA level of more than eight years.

A similar lack of basic difference in sensorimotor function by the retardate was found in a simple type of maze learning that permitted the Ss to see the entire maze as they worked. DeSanctis [45] used a paper and pencil maze with three groups of children. One group consisted of twelve retarded children (CA = 7-13 years), only one of whom was severely retarded. The other two were normal groups that approximated MA and CA controls. All except the one severely retarded child solved the maze. The performance of the retarded group in general was comparable to the normal control groups. The visual apprehension of the maze as a whole by the retardates at the beginning of a trial was closer to that of the CA controls than that of the MA controls, a younger group. Basically comparable function was thus affirmed for a sensorimotor skill of relatively low complexity. Sloan and Berg [203] used the open stylus maze with a sample of retarded adults covering a wide range of intelligence ($\overline{IQ}$ = 57; SD = 11.3; MA ≈ 8 years). Ten trials were given on the maze and time was the measure of performance. Again, this maze was of such low complexity for a relatively high MA group that it amounted to use of sensorimotor skills. There was, consequently, no significant relationship between MA and performance.

Inadequate or deficient neural growth in mental retardation is generally accepted and is becoming increasingly well established by experimentation. This deficit becomes apparent even in sensorimotor skills when the complexity of the learning required is increased. Ellis, Pryer, Distefano, and Pryer [54] made an extensive investigation with the shielded, high-relief finger maze with eight choice points (RLRRLLRL). They employed a total of one hundred fifty-one male and female Ss covering a very wide range of intelligence (IQ = 40-139), matching all groupings of Ss on CA, all being older adolescents or young adults. The Ss were first given training on a practice maze to a criterion of nine correct responses out of ten trials. This promptly eliminated twenty-two Ss, fifteen from the 40-59 IQ category, reducing the total group to one hundred twenty-nine Ss for the regular maze. During the main experiment, the intertrial interval was twenty seconds,and the criterion of learning was two consecutive errorless trials or a maximum of one hundred trials. The measure of performance was the number of trials to criterion for five groups of Ss. The mean number of trials to criterion for the five IQ groupings revealed a significant difference in speed of learning at each successively higher level of IQ, but the greatest improvement was achieved by the 80-99 IQ group over the 50-79 IQ group.

It is apparent that the syndrome of reduced growth in neural function _per se_ is not a unique characteristic of mental retardation. It rather characterizes the whole range of intelligence in successive levels from the highest to the lowest, but the degree of reduced growth is sharply accentuated at significantly subnormal levels of IQ. So severe was the reduced growth in the lowest IQ category here that task complexity of a shielded, high-relief finger maze prevented successful performance. The Ss that are eliminated from complex tasks should be a challenge to the experimenter seriously interested in trying to understand severe mental retardation.

Thus, it becomes apparent that the severely reduced growth in neural function in severely retarded children (MA < 4 years) is basically, functionally _different_ from a comparable MA level of normal ontogeny in at least one fundamental neurophysiological respect, viz., the inherent potential for stimulation of growth of one neural component by the functioning of an interrelated neural component, as discussed previously in regard to onset of cortical function. This great neurophysiological potential, so wonderfully characteristic of the normal infant [113, 143], is somehow severely reduced in its neurophysiological effectiveness in the retardate (MA < 4 years). The manner in which the various constellations of pathology somehow truncate both neural growth and potential stimulation of growth remains an important area of research in basic neurophysiology.

A study that portrayed severe mental retardation in a most unflattering, yet coldly scientific, phylogenetic perspective found that retardates (MA = 2-4 years) learned amazingly slower than the naive monkeys that Harlow [76] trained on discrimination problems. House and Zeaman [92, 93] trained severely retarded Ss (IQ = 31) on discrimination problems with the modified Wisconsin General Test Apparatus (WGTA). The two food wells were covered with erect tridimensional stimuli differing in both shape and color, e.g., a red triangle vs. a black cross. The bait was candy, and the positive stimulus was moved randomly from right to left food wells. Twenty-five massed trials were given daily until the criterion of 20 out of 25 correct was attained. The median number of errors for the retarded Ss was 119, compared with only 4 for the monkeys on the very same problems. Several investigators have found that normal children in a comparable CA range can learn these discrimination problems as well as or better than monkeys and chimpanzees [43, 66, 82, 224]. The fact that retardates with very low mental age (MA = 2-4 years) learn at such a significantly lower level of efficiency than normal children of the same age range, and even slower than primates, poignantly emphasizes the unique double handicap of the retardate who has not achieved the essential four years of neural growth in structure and function, despite his greater chronological age. Reduced growth is compounded by the severely reduced neurophysiological potential for stimulation of growth of one neural component by the functioning of interrelated neural components. Hence, learning, which is basically new growth in both structure and function at this level, fails to show the great rate of improvement found in very young normal children [113, 143].

Growth is so dramatically reduced in retardates (MA < 4 years) that the complexity of the task to be learned must be held at a very simple level. House, Orlando, and Zeaman [91] were able to use the form-color discrimination problems in the modified WGTA with a wider range of MA ($\overline{IQ}$ = 37; MA = 2-6 years) embracing the critical early years. They found with a large group of retardates (N = 66) that MA was significantly correlated with errors on the task (r = -0.54; p = 0.01), holding CA constant. Hence, growth in neural structure and function is taking place at a significant, even if doubly truncated, pace for simple learning in discrimination.

However, the severe double handicap of the very young retardate (MA < 4 years) diappears at MA = 5-6 years in simple discrimination learning. House and Zeaman [94, 95] compared thirty normal children with thirty-two retardates of the same mental age (MA = 4-6 years) on this same kind of discrimination learning. Each group was divided into two MA levels (MA = 4-5 vs. MA = 5-6 years). At the lower MA level, the retardates made significantly more errors than the normal controls, revealing the continuing effect of the double handicap in growth even at the threshold of advanced neuronal maturation in terms of cell structure and proliferation of dendritic processes in the latest maturing intrinsic sectors. At the upper MA level, the learning of simple discriminations was so rapid in both retardates and normal controls that no difference was found. At this level of growth and complexity of learning, the reduced growth of the retardate is again comparable in function to that of the younger normal child of the same mental age. Again when low complexity in discrimination learning for a given age level amounts to little more than learning a sensorimotor skill, the retardate shows no real difference in function from the younger normal child of comparable mental age, exemplifying the variable growth phenomenon characteristic of the entire range of intelligence.

The delayed response task is a very effective technique for studying the factor of variable complexity of the learning task. Holding the discrimination required by the task to a constant level, the factor of complexity can be varied in precise gradations of time of delay. Ablation studies [175, 176] have related the delayed response to the constructs of intention, in particular, and memory, in general. Ablation of the frontal intrinsic sector seriously impairs the function of intention. The constellations of pathology in mental retardation seem to impair this function rather seriously as well. Harlow and Israel [80] used candy bait and opaque containers to study delayed response for position in twenty-five severely retarded Ss (CA = 5-60; MA = 1-3 years). As long as the period of delay was not more than fifteen seconds, probably requiring no more memory than the burst of self-sustained firing in the sensory projection neurons, the retardates performed quite successfully with minimal practice. But the complexity of the problem seemed to increase markedly beyond thirty seconds of delay, when errors increased rapidly. Very short periods of delay less than fifteen seconds apparently require little more than the simple learning of a sensorimotor skill, for which the earliest maturing cortical sectors are quite adequate. But the latest maturing intrinsic sector with its function of intention and memory is required for periods of delay beyond thirty seconds. Skalet [200] found that a group (N = 46) of normal children (CA = 2-5 years) learned a three-choice delay for place for maximum periods ranging from one day to at least thirty-four days. In another similar group (N = 27), the range of maximum delay remained exactly the same for a six-choice problem using familiar forms, such as rabbit, automobile, etc. However, when six geometrical forms were used with another group of normal children (N = 37), the reduced periods of maximum delay ranged from only ten minutes to four days. This introduced another dimension of complexity into the delayed response problem; i.e., discrimination of geometrical forms.

Growth in structure and function enables the retardate to increase delay response intervals. Pascal et al. [164] tested twenty-seven male retardates (CA = 6-32; MA = 2-7 years) in a five-choice delayed response for place. Maximum delay was defined as the longest period of delay after which a subject made three successive correct responses ($p = 0.20^3$). The range of maximum delay was from two seconds to fourteen days, but most subjects beyond an MA of 3 years could delay responses for hours or days. The evidence seems to point to retarded inception of function of the intrinsic system in comparison with normal ontogeny. Further research designed to explore the course of onset and growth in initial function of the intrinsic system and the effects of various dietary or metabolic influences on this course of growth is needed.

Some interesting possibilities for research along these general lines have been demonstrated by Amos [11]. Three-year-old children with and without anoxic conditions at birth were compared on response for periods of one, two, three, four, five, and ten minutes of delay. The nineteen Ss in the anoxic group got progressively worse as the delay interval increased. At the ten-minute interval, the twenty control Ss were significantly superior in performance ($p = 0.05$). In view of the electrophysiological nature of mnemonic function, the research design could have been improved by addition of shorter delay periods of fifteen seconds, thirty seconds, or more, and by addition of longer delay periods of one hour, one day, or more. Nonetheless, the impairing effect of anoxia on the early growth of neural function in the intrinsic system was demonstrated.

In addition to delayed response, another useful experimental method for studying the growth of the frontal intrinsic sector and its function of intention is the double alternation problem. The successful solution of the double alternation problem requires the adaptation of intention to the outcomes of the immediately preceding responses, requiring at least some short-term memory for the learning process. Some investigators [165, 213] have used a double alternation task that requires the Ss to select the extreme door at the right end of a line of five doors twice in succession and then select the extreme door at the left end twice in succession (RRLL). The positive door is baited on each trial. These investigators studied the behavior of groups of cerebral palsiedchildren with a wide range of intelligence (IQ = 35-111) and mentally retarded children of a broad characteristic range of intelligence in retardation. They found significant correlations between double alternation performance and intelligence ranging from r = 0.64 to r = 0.83 in different groups. Growth in function of the frontal intrinsic system is a variable phenomenon significantly related to general intelligence.

One of the most useful methodologies for study of the function of intention comes from early classical trial-and-error studies of how children attack insoluble problems [73, 74]. The task was to learn, in a four-choice situation, that the last correct response was never correct on the next trial and that a systematic three-choice search without clues must be made on every trial. The Ss, a variety of retarded and normal children and adults as well as animals taken one at a time, were each confronted by four doors, only one of which was unlocked to allow escape from the enclosure on any given trial. If a given door had been unlocked in one trial, it was always locked on the next trial. Which door of the remaining three would be unlocked from trial to trial was determined by chance. Hamilton found that two retardates, a man and a boy, could perform almost as well as normals and better than animals. However, the main purpose of the study was to analyze trial-and-error behavior on an insoluble task, rather than behavior in mental retardation. The method is significant for future research on the separate functions of intention and differentiation by the separate frontal and posterior intrinsic systems. It requires a subject to learn an intention to avoid the last successful choice on the next trial and to learn a systematic differentiation among the three remaining choices without benefit of any clues. An error of intention may be defined very precisely as the choice of the door that was positive on the last successful trial. An error of differentiation may likewise be precisely defined as the choice of a continuing negative door that was among the three negative doors on the last successful trial. The probability of an error of intention as defined is one out of four choices on each trial ($p = 0.25$). The probability of an error of differentiation as defined is, on the other hand, always two out of the four choices on each trial ($p = 0.50$). In fact, it is this level of probability that represents the asymptote of learning in such a problem. Analysis of the separate error curves could have diagnostic significance for individual records of performance. Longitudinal studies of individual growth records and the characteristic course of onset and growth of the separate functions of differentiation and intention could be carefully studied with this technique.

The discrimination reversal problem is another excellent method for isolating the function of intention from the separate function of differentiation in behavior. During original learning (OL), both functions of differentiation and intention are involved by necessity in discrimination learning, but during reversal learning (RL) no new learning in differentiation is required. In the RL stage, a new intention must be learned for a problem that presents no change whatsoever in differentiation. The simplest form of discrimination reversal learning is the left-right position reversal problem with the WGTA used with rats in classical experiments [49]. House and Zeaman [94, 95] used this problem with their severely retarded Ss ($MA \approx 3$ years; $IQ \approx 30$). They found that retardates at this mental level could solve the problem and could achieve "learning set" [77], but the efficiency of their performance was not significantly better than the performance of rats. Longnecker and Ferson [133], however, found that a group of mongoloids (Down's Syndrome) at this same IQ level achieved one-trial reversal learning better than rats. In these critical growth years ($MA < 4$ years), significant differences in performance reflect increments in basic growth in neural structure and function of the frontal intrinsic sector that makes reversal of intention possible. Several investigators [170, 210, 212] have reported no differences in discrimination learning between retardates and normal controls when matched on higher levels of mental age (e.g., $MA = 7$ years). Two of these studies used a standard discrimination task, e.g., stimulus variation along a size dimension only, but Plenderlith [170] used a problem requiring reversal of initial preferences. Her Ss were confronted with the same pair of pictures for six trials only, then presented with a new pair for six trials, and so on until they learned that the first one chosen of a newly presented pair was never correct and that for the next five trials the other picture would always be correct. The positive picture was baited with "charms" or candy, and the positive position was varied randomly. The criterion of learning was five successive correct choices out of six trials for three pairs of pictures in succession. Reduced growth in function, not otherwise different from normal ontogeny at a comparable level of mental age, was again emphasized by the performance of the retardates. The methodology used in this study required the subject to learn an intention to avoid his initial preferences for five trials in each block of six trials, during which the function of differentiation was held constant. An error of intention in this kind of problem may be defined as the choice of the initial preference on any of the remaining five trials in a block. However, this problem must be preceded in an OL stage by learning to differentiate the same stimuli as a standard discrimination task in order to prevent the possible confounding effect of difficulty of differentiation of the stimuli during the RL stage [75].

The technique of the classical stimulus generalization study is the most useful way to analyze the mechanism of differentiation [39, 57, 197]. Barnett and Cantor [15] found no significant differences in the stimulus generalization gradients for spatial stimuli between normal children and retardates ($\overline{IQ} = 50$). In view of the postulated stochastic mode of operation of the intrinsic system, the probabilities of the classification of any given stimuli as same or different should vary along the classical stimulus generalization gradient for normal and retarded alike. The efficiency of learning to make a differentiation, however, depends upon the interacting effects of level of growth in neural structure and function and level of complexity of the task. Bialer [18] has shown that verbally mediated stimulus generalization is greater the higher the intelligence level (another way of saying level of growth). This is consonant with the reduced growth in language function characteristic of retardates. Barnett, Ellis, and Pryer [16] found that training retardates to attach sensible verbal symbols to the discriminative stimuli to mediate an improved delayed response resulted in the best performance for those with the highest levels of mental age.

An interesting methodology for analysis of the comparative efficiency of learning to make differentiations of varying complexity by groups of varying growth is the use of a concurrent series of discriminations. In the concurrent series technique, each pair of a series of different pairs of object-quality discrimination stimuli is presented only once, until all pairs have been presented, and then the pairs are repeated in exactly the same order. One series of discriminations learned to criterion is followed by another similar series. Learning set is measured by the amount of improvement in performance on succeeding series. Wischner and O'Donnell [229] compared the performance of groups of normal and mildly retarded children on this type of growth in function. Both groups achieved learning set rapidly and efficiently, but the normals reached a higher level of efficiency. Analysis of the serial position effects showed a more pronounced serial position effect for the retardates. Hence, with complexity of task held constant, the serial position effect reflects variations in growth, and conversely, with growth held constant by groupings, the serial position effect reflects the complexity of the tasks.

Mental retardation and normality are not discrete categories of growth, and more precise understanding of the characteristics of reduced growth in neural structure and/or function as a phenomenon of the whole range of intelligence, of which mental retardation by definition happens to be the sector of most reduced growth, requires better techniques than continued gross comparisons of retardates and normals. One example of a better technique is a statistically robust study of large groups of retardates by Ellis [52]. He compared fifty high mental age Ss ($\overline{\text{MA}} = 8$; $\overline{\text{CA}} \approx 15$ years) with forty low mental age Ss ($\overline{\text{MA}} = 5$; $\overline{\text{CA}} \approx 13\ 1/2$ years) on efficiency of performance on a single series of ten successive object-quality discrimination problems. Each problem was learned to a criterion of twenty successive correct responses. Analysis of errors to criterion for the two groups yielded an F-ratio significant at the .001 level of confidence. The high MA group achieved a significantly higher level of efficiency, even though both groups learned rapidly. Looking at the results from the point of view of reduced growth in neural function, it is evident that significant growth in the function of differentiation takes place in the interval between MA = 5 and MA = 8 years. The function of differentiation at MA = 5 years seems basically comparable to that at MA = 8 years in every way except level of efficiency.

Another example of improved technique is a study of large groups of subjects by Ellis and Sloan [55]. They tested one hundred thirty-nine mentally retarded and forty normal children on a form-oddity problem in discrimination to a criterion of twenty successive correct responses or a total of two hundred trials. The mentally retarded group was divided into four levels according to mental age ($\overline{MA}$ = 4.1, 6.1, 7.7, and 9.7). The two normal control groups had mean chronological ages in the middle of the range of mental ages of the retardates used for comparison ($\overline{CA}$ = 6.2 and 7.3). Two different pairs of object-quality discrimination stimuli were employed to make four possible combinations of four objects taken three at a time for the form-oddity problem. The four possible combinations were presented randomly with the odd or unpaired object always hiding a marble to designate a successful choice. No extrinsic rewards were given for correct responses. They found a significant product-moment correlation ($r = 0.48$) between the number of correct responses and mental age in the retardates. The higher the MA level, the more efficient the learning of the retardates, both in terms of asymptote and rate of approaching asymptote. The three highest MA groups of retardates demonstrated typical negatively accelerated curves of learning. However, learning by the lowest retarded group ($\overline{MA}$ = 4.1 years) was negligible. Differentiation of the odd (NOVEL) form in a discrimination problem should be a relatively simple kind of learning [150], but the lowest retarded group here affirmed the crucial importance of those early years (MA < 4 years) of growth of neural structure and function. It is clear that the interval from MA = 4.1 to MA = 6.1 is the period of transition from the threshold of advanced structural growth of the intrinsic systems to the typical form of integrated functioning characteristic of higher levels of mental growth. It would be very useful indeed to explore the course of growth in this transition zone with even greater precision of experimental design.

IV. ROTATION DISCRIMINATION COMPLEX (RDC-1) AND SIZE DISCRIMINATION SIMPLE (SDS-2)

Method

Thirty mentally retarded children ($\overline{IQ} = 41$; $\overline{MA} = 5.3$ years) enrolled in summer classes for the trainable mentally retarded at Southern Wisconsin Colony and Training School, Union Grove, Wisconsin, were selected from an initially available forty-two children (Appendix I). The thirty Ss who qualified by completing both of the experiments ranged in chronological age from ten to sixteen years ($\overline{CA}$ = 13-1/2 years) and satisfactorily bridged the transitional zone between the threshold of advanced structural maturation of the cortex and the achievement of integrated cortical control by the intrinsic systems of the telencephalon, although at reduced efficiency.

Two visual discrimination experiments were conducted in daily tandem arrangement. The first problem, designated as rotation-discrimination-complex (RDC-1), required the multiple-choice matching of an odd block in a sample pattern of three wooden blocks. The second problem, designated as size-discrimination-simple (SDS-2), required the multiple-choice fitting of a wooden ring at a certain designated place on a tapered post. Three choices were presented in a randomly varied array for each trial. Correct responses were reinforced with M & M candy in assorted colors, one little M & M candy given, and promptly consumed, per correct response for an average total of about ten candies per daily testing. The criterion of learning was set at five successive correct responses. The probability in a three-choice problem of making a correct choice simply by chance on each trial was one out of three ($p = 0.333$). Hence, the probability of making five successive correct responses by chance alone was about four out of a thousand choices ($p = 0.004$).

In the first problem (RDC-1), the odd element in the three-element pattern of identical wooden blocks consisted of the rotational relationship of the middle block to the remaining pair. The middle block in the pattern was rotated one hundred eighty degrees out of phase with the pair. All the blocks were identical in size, shape, natural finish, texture, and a single orienting clue, viz., a pair of notches at one corner of each block. The blocks in both the pattern and the choice array were partially recessed into fitted wells in circular aluminum discs mounted in an orderly arrangement on a platform. Each disc could be rotated and set at any desired angle. The apparatus was fitted with a shield that could be lowered into place to prevent the subject from seeing the experimenter change the choice array in accordance with two different randomized tables for alternate days of testing (Appendix II). The sample pattern was arranged along the side of the apparatus farthest from the subject and was never changed. Original plans for the experiment had provided an elaborate series of patterns ranging from major to minor variations in degree of rotation, but the first pattern described above proved to be at a complex conceptual level of learning for this experimental group.

There was an empty disc-tray at the right end of the pattern to receive the choice of the subject. The three-choice array was placed along the side of the apparatus nearest the subject, who was instructed to pick up the block of his choice from the array and place it correctly in the empty tray. Verbal instructions and one demonstration by the experimenter preceded each ten-trial session. After each correct response, the experimenter placed the candy reward on the corner of the correct block where the orienting clue was located to try to center attention on the relevant clue.

Both experiments (RDC-1 and SDS-2) were continued in thirty-trial stages until half of the experimental group (15 Ss) had achieved the criterion of learning on the first experiment (RDC-1), the more difficult one of the two for these Ss. This necessitated four stages, making a total of one hundred twenty trials. The experiment was terminated after each subject had completed the necessary twelve daily sessions on both experiments. By this time, all but one (29 out of 30 Ss) had achieved criterion on a series of at least two (and in one case up to seventeen) variations of the second experiment (SDS-2).

For the second experiment (SDS-2), one of the ring assemblies with five rings used in a previous study [105] was modified for a three-choice size discrimination. The initial presentation here consisted of the bottom (#1), middle (#3), and top (#5) rings from the first ring assembly set used in that earlier study. The three selected rings were randomly arranged in a straight-line, left-to-right array across the front of the post to be fitted. Again, two different randomized tables for alternate days of testing were used to vary the array from trial to trial in such a manner that each position would be positive no more than five times out of ten trials nor more than three times in succession (Appendix III). The experimenter (E) designated the place on the post to be fitted, which happened to be the bottom (#1) position in the initial variation employed, by placing his finger at the appropriate place on the post and giving the following instruction: "Choose the ring that fits right here." One demonstration by E on the first day of the experiment for each subject was all that was necessary. After a subject reached the criterion of learning on this first variation, another variation was introduced without demonstration at the next daily session. The second variation used was the top (#5) position on the same post with the same three rings used for the first presentation. Because the post tapered sharply from bottom to top, the ring (#5) for the top position had a very small hole in it in comparison with the ring (#1) for the bottom position. This size discrimination task was considered to be very simple in the original plans for the experiment. The fact remains that one subject failed in one hundred twenty trials to learn the initial differentiation of the bottom ring (#1) with the largest hold in it. Many subjects in the non-criterion group for experiment RDC-1 learned the first and second variations of experiment SDS-2 only with great difficulty. See Appendix IV for all seventeen variations that were used in one case.

For purposes of scoring responses, the positions in the three-choice array were designated as right (R), middle (M), and left (L) for both experiments. Detailed scoring was practiced to facilitate statistical tests for position or distractor preferences. For example, a wrong choice made at the LEFT position when the RIGHT position was positive was scored as L - R. A sample scoring protocol for a daily session with one subject is given in Appendix V. The distractors for the first experiment (RDC-1) were randomly programmed from six equally spaced angles of rotation away from the correct position for making the match and were designated by the number of degrees of rotation (Appendix II). The distractors for the second experiment (SDS-2) were designated by the selected ring position numbers in each of the five-ring assembly problems borrowed from the earlier study (Appendix III).

The two major objectives of these visual discrimination experiments were to analyze the intrinsic system functions (1) of a mentally retarded group whose growth in neural structure and/or function by standardized measures centered on the transition zone (MA = 4 - 6 years) between the threshold of advanced structural maturation of the cortex (MA = 4 years) and the achievment of integrated cortical control by the intrinsic systems of the telencephalon (MA = 6 years), and (2) of upper and lower sub-groups within this transitional group precisely divided by their achievement or non-achievement of the criterion of learning on a relatively complex task. Two basic types of visual discrimination (rotation vs. size) at inherently different levels of task complexity were designed to probe for contrasting performance characteristics of the separate functions of differentiation and intention by the intrinsic systems.

An error of differentiation was defined as a wrong choice that followed next after a wrong informing choice. Errors of differentiation indicated the level of efficiency of search for relevant clues. An error of intention was defined as a wrong choice that followed next after a correct informing choice. Errors of intention indicated the level of efficiency of use of relevant clues to guide behavior. The null hypothesis was posed for the following factors isolated by analysis: (1) initial position preferences, (2) final position preferences, (3) distractor preferences, (4) reliability of performance, (5) task related differences in differentiation and intention, (6) sub-group related differences in differentiation and intention, and (7) task by sub-group interactions in differentiation and intention.

Results

The analysis of the data for irrelevant position and distractor preferences was concentrated upon experiment RDC-1, which was not successfully negotiated by half of the subjects (Ss). When the discrimination problem is made too difficult, Ss may be expected to resort to irrelevant and idiosyncratic preferences and seemingly to ignore the relevant stimulus clues that E presents to them [157]. Such an analysis for preferences was not considered useful for the other experiment (SDS-2), since all but one of the Ss successfully negotiated two or more variations of the problem. In order to analyze the RDC-1 data for initial and final preferences for position (L-M-R) in the choice array, the data were divided into initial and final halves of the experiment for a robust statistical test. The first two stages of thirty trials each were combined for the sixty-trial initial half; and similarly, the last two stages were combined for the final half. The same plan of dividing the data was used to analyze it for initial and final preferences for the six distractors (rotations of 045°, 090°, 135°, 225°, 270°, and 315°).

At the outset, errors were tabulated for each subject in each group (criterion vs. non-criterion) by position of the wrong choices in the choice array (Appendix VI). An analysis of variance was performed separately on each array of data, i.e., each array of three columns of the positions ($n_j = 3$), by fifteen rows of subjects ($n_k = 15$). The error score (Y_{kj}) placed in each of the cells ($N = 45$) was the sum of errors made by a given subject at a given choice position during the course of sixty trials. Since the data consisted of sixty repeated measurements on the same two groups of subjects, it was decided to use the appropriate F ratio for testing the hypothesis of zero slope (null hypothesis) when the samples are related. After appropriate derivation and development, explained by Lewis [132], the equation for testing the hypothesis of zero slope when the samples are related is as follows:

$$F = \frac{\Sigma_k n_k \bar{y}_k^2}{n_j - 1} \; \frac{(n_j - 1)(n_k - 1)}{SS_{XxI}}$$

It will be observed that the error term in this particular equation is the interaction sum of squares (SS_{XxI}). This type of error term is related to the differential effects of the experimental values of X_k on the various individual subjects. Thus,

$$SS_{XxI} = \Sigma_k \Sigma_j (Y_{kj} - M_k - M_j + M_y)^2$$

We expect to find real differences among the row means and use this method of correcting for them in tests of significance of the experimental treatments.

The results are summarized in Tables 1 and 2. The analysis did not reveal any significant preferences for position in the choice array during either the initial half of the experiment (vid., Table 1) nor during the final half (vid., Table 2) for either group of Ss. Position habits were readily observed by E in the daily test perform-

Table 1

Summary of Initial Position Preferences

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between positions - bg	10.85	2	5.43	0.04*	
Between individuals - bI	304.21	14	21.73	0.18**	
Interaction - XxI	3475.52	28	124.13		
Total	3790.58	44			
Non-Criterion Group:					
Between positions - bg	102.19	2	51.10	1.18*	
Between individuals - bI	81.88	14	5.85	0.13**	
Interaction - XxI	1216.51	28	43.45		
Total	1400.58	44			

* = When df = 2 and 28, p = .05 requires F = 3.33.

** = When df = 14 and 28, p = .05 requires F = 2.06.

Experiment RDC-1

Table 2

Summary of Final Position Preferences

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between positions - bg	56.85	2	28.43	0.53	*
Between individuals - bI	637.22	14	45.52	0.85	**
Interaction - XxI	1498.51	28	53.52		
Total	2192.58	44			
Non-Criterion Group					
Between positions - bg	248.72	2	124.36	2.08	*
Between individuals - bI	122.27	14	8.73	0.15	**
Interaction - XxI	1673.99	28	59.79		
Total	2044.98	44			

* = When df = 2 and 28, $p = .05$ requires $F = 3.33$.

** = When df = 14 and 28, $p = .05$ requires $F = 2.06$.

Experiment RDC-1

ances of several Ss in the non-criterion group, but there was clearly no group bias for any particular position. The different individual position habits of several of the Ss tended to counterbalance themselves and to nullify any group effect. A careful inspection of the large magnitude of the interaction sum of squares (SS_{XxI}) in comparison with the total sum of squares in both initial and final performances (vid., Tables 1 and 2) shows that the interacting effects are really the only important factor to be considered for either group. The expected real differences among the row means did not materialize in the error data (vid., Appendix VI).

Next, the error data were tabulated for each subject in each group separately by the distractors involved in the wrong choices. A similar analysis of variance was performed on each of these four arrays of data, i.e., each array of six columns of distractors ($n_j = 6$) by fifteen rows of subjects ($n_k = 15$) for each group by each of the initial and final conditions. The results of this analysis again showed a similar pattern of no significant variance between distractors or between individuals. And again there were large interacting effects of distractors by subjects in each of the initial and final conditions and in each group (vid., Tables 3 and 4).

The error data for the combined groups ($n_k = 30$) by the experimental stages of thirty trials each ($n_j = 4$) were next subjected to correlational analysis. The results are shown in Table 5. There appeared to be very little or no mutual relationship between the sets of errors of the combined groups made during the different stages of the experiment. In other words, no general group factor appeared in this combined error data array. Rather, the idiosyncracies of the Ss in the way they made errors were indirectly affirmed by this absence of a general relationship. Even the errors made by the successful criterion group were highly idiosyncratic, as was shown by the large interaction mean square of the criterion group previously discussed in Table 1.

Having found the error data to be dominated by the idiosyncracies of the Ss, it seemed appropriate to determine the strength of relationship, if any, between the sets of correct response data for each stage of the experiment (RDC-1). A similar analysis by the method of correlation was made of the cumulative raw scores of all Ss in the combined groups for each of the four stages of the experiment. The resulting matrix of correlations is shown in

Table 3

Summary of Initial Response Deviation Patterns

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between positions - bg	62.41	2	31.21	0.79	*
Between individuals - bI	3724.78	14	266.06	6.77	<.01
Interaction - XxI	1099.61	28	39.27		
Total	4886.80	44			
Non-Criterion Group:					
Between positions - bg	96.40	2	48.20	3.95	<.05
Between individuals - bI	644.95	14	46.07	3.78	<.01
Interaction - XxI	341.45	28	12.19		
Total	1082.80	44			

* = When df = 2 and 28, p = .05 requires F = 3.33.

Experiment RDC-1

Table 4

Summary of Final Response Deviation Patterns

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between positions - bg	149.38	2	74.69	2.37	*
Between individuals - bI	1049.75	14	74.98	2.38	<.05
Interaction - XxI	883.31	28	31.55		
Total	2082.44	44			
Non-Criterion Group					
Between positions - bg	23.25	2	11.63	0.46	*
Between individuals - bI	1233.43	14	88.10	3.49	<.01
Interaction - XxI	705.90	28	25.21		
Total	1962.58	44			

* = When df = 2 and 28, $p = .05$ requires F = 3.33.

Experiment RDC-1

Table 5

Intercorrelation Matrix of Errors by Experimental Stages

Stages	A	B	C	D
A		.07	.23	-.12
B	.07		-.13	.22
C	.23	-.13		.11
D	-.12	.22	.11	
	.18	.16	.21	.21

Experiment RDC-1

Table 6. There were low positive relationships among the four stages. All the obtained values of r were significant at the .05 level or better, but they were too small to account for any important per cent of the variance in the scores. Whatever the factor (or factors) operating to produce the mutual positive relationships among the four stages of performance, the effect of it was weak, probably indicating that the non-criterion sub-group's scores were not contributing to the strength of the relationships. The reliability of performance of a group, half of whose members did not solve the problem, would not be expected to be high.

An analytical attack upon the nature of the idiosyncracies in the performance of these two groups of Ss separately was then undertaken. The responses of each subject for each of the initial and final conditions of performance were tabulated in the form of frequencies of choice at each of the three positions in the choice array. These frequency scores were then subtracted from the programmed frequencies of reinforcement (M & M candy reward) available at each of the three positions, and the positive or negative deviations from the programmed pattern were used as the cell values (Y_{kj}) in each data array, i.e., three columns of choice positions ($n_j = 3$) by fifteen rows of subjects ($n_k = 15$). The details of the analysis are shown in AppendixVII. For purposes of analysis of variance, using the formula for related samples discussed above deviation signs were ignored and the phenomenon of deviation frequency per se was analyzed as a way of examining the idiosyncracies of Ss. It is readily apparent in the nature of data array in Appendix VII that the algebraic sum of each row is zero, and it is very interesting to note that the algebraic sum of most of the columns is diminished in varying patterns by the canceling effects of the diverse idiosyncracies of the different Ss.

The results of this analysis of deviation frequency as a revealing phenomenon in itself are summarized in Tables 7 and 8. Only the non-criterion group evidenced any significant choice position effect, and this effect occurred only in the initial stages of the experiment (vid., Table 7). It would be expected that, in the absence of learning to make the relevant differentiations among the stimuli, the non-criterion group would learn to match the probabilities of reinforcement at the choice positions. Much research with animals on schedules of reinforcement [201] indicates the feasibility of this expectation. Tending to support this argument, their performance

Table 6

Intercorrelation Matrix of Correct Responses by Experimental Stages

Stages	A	B	C	D
A		.45	.40	.30*
B	.45		.38	.31
C	.40	.38		.64
D	.30*	.31	.64	
	1.15	1.14	1.42	1.25

* = With df = 29, p = .05 requires r = .302.

Experiment RDC-1

Table 7

Summary of Analysis of Errors of Differentiation

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between stages - bg	252.18	3	84.06	5.28	<.01
Between individuals - bI	596.60	14	42.61	2.68	<.01
Interaction - XxI	668.07	42	15.90		
Total	1516.85	59			
Non-Criterion Group					
Between stages - bg	32.20	3	10.76	0.71	*
Between individuals - bI	407.10	14	29.08	1.91	**
Interaction - XxI	639.30	42	15.22		
Total	1078.60	59			

* = When df = 3 and 42, p = .05 requires F = 2.83.

** = When df = 14 and 42, p = .05 requires F = 1.94.

Experiment RDC-1

Table 8

Summary of Analysis of Errors of Intention

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between stages - bg	6.32	3	2.11	0.54	*
Between individuals - bI	186.50	14	13.32	3.40	<.05
Interaction - XxI	164.43	42	3.91		
Total	357.25	59			
Non-Criterion Group:					
Between stages - bg	12.93	3	4.31	1.55	*
Between individuals - bI	49.73	14	3.55	1.27	**
Interaction - XxI	117.07	42	2.79		
Total	179.73	59			

* = When df = 3 and 42, p = .05 requires F = 2.83.

** = When df = 14 and 42, p = .05 requires F = 1.94.

Experiment RDC-1

changed during the final stages to show practically no position effect at all in their deviation scores; thus they were achieving a good group matching of the probabilities of reinforcement at each position by the way they distributed their deviations.

However, the most important findings of this analysis were the significant variances between individuals in both groups and in both initial and final conditions of performance. High deviation row means were an indication of radical departures from the programmed frequencies of reinforcement. Such radical departures were revealingly characteristic of individuals with (1) a very rigid, unsystematic search for relevant clues, or (2) a very rigid, perseverative position habit without regard for clues. Such individuals were found in both groups, but apparently an all-or-none sort of intrinsic function of differentiation of the relevant stimuli determined the destiny of a given individual in the experiment. The high deviation individual in the non-criterion group seemed to belong to the second type of individual described above. On the other hand, the very low deviation row means were indicative of good individual matching of the programmed frequencies of reinforcement. These individuals either (1) learned to use relevant clues successfully with few resulting errors and close adherence to the program or (2) learned to match the frequencies of reward at each position without any differentiation of clues. Such individuals were also found in both groups, but predominantly in the criterion group. The criterion group showed a very marked reduction in variance between individuals, though still at a significant level, in the final conditions of performance, reflecting their overall improvement in performance by reduction of errors. The non-criterion group showed about the same ratio of significant variance, at an even higher absolute level of variance, between individuals in the final stages of performance as compared with the initial stages, reflecting the absence of differentiation of relevant clues. These unsuccessful individuals showed increasingly diverse efficiency levels in trying to match the programmed schedule of rewards at each position.

Finally, an analysis of the error data was undertaken to relate this data to the learning function characteristics of the posterior and frontal intrinsic sectors of the forebrain for both groups and for both experiments (RDC-1 and SDS-2). The error data were separated into two basic types for each experiment, viz., (1) an error of differentiation of clues, i.e., a wrong choice immediately following a wrong informing choice. The data arrays that were developed by this procedure are shown in Appendix VIII and Appendix IX.

An analysis of variance was performed separately for each data array by the method previously described for related samples. The main data derived from each analysis are also shown in Appendix VIII and Appendix IX. The results of these analyses for both experiments are grouped and summarized in Tables 7, 8, 9, and 10.

The results of the analyses with respect to significant variance between individuals will be considered first in view of the previously discussed idiosyncracies. Significant variance between stages of the experiments, reflecting changes or growth in the functions of differentiation and intention, will be considered last and will constitute the most important outcome of these analyses. In the first experiment (RDC-1), there were significant differences between individuals in the criterion group in both the functions of differentiation and intention (Tables 7 and 8). In differentiation, high row means (high error condition) meant a difficult and very inefficient search for the positive clues. In intention, such high row means mean a very inefficient integration of the outcomes of previous responses with differentiations of clues to produce better intentions. There were no significant differences between individuals in the non-criterion group in either differentiation or intention. Both of these functions were characterized by a purely random pattern of performance.

In the second experiment (SDS-2), there were again significant differences between individuals in the criterion group in both differentiation and intention (Tables 9 and 10.) Individuals that made the greatest numbers of errors of each type were very inefficient in searching for clues and in integration of outcomes of responses for more precise intentions. The non-criterion group failed to show any significant differences between individuals in the function of differentiation in the second experiment but did show significant differences between individuals in intention (Tables 9 and 10). It seems plausible that the members of this group were all about uniformly handicapped in ability to make differentiations though still able to solve at least a few variations of the problem (size discrimination). The members of this group were not, however, uniformly handicapped in the function of intention and showed significant variability between individuals in efficiency of use of outcomes of previous responses.

Table 9

Summary of Analysis of Errors of Differentiation

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between stages - bg	131.26	3	43.75	1.78	*
Between individuals - bI	844.73	14	63.19	2.46	<.05
Interaction - XxI	1030.99	42	24.55		
Total	2006.98	59			
Non-Criterion Group:					
Between stages - bg	35.52	3	11.84	0.56	*
Between individuals - bI	477.43	14	34.10	1.62	**
Interaction - XxI	885.23	42	21.08		
Total	1398.18	59			

* = When df = 3 and 42, p = .05 requires F = 2.83.

** = When df = 14 and 42, p = .05 requires F = 1.94.

Experiment SDS-2

Table 10

Summary of Analysis of Errors of Intention

Source of Variation	SS	df	ms	F	P
Criterion Group:					
Between stages - bg	47.81	3	15.94	2.59	*
Between individuals - bI	228.23	14	16.30	2.65	<.01
Interaction - XxI	258.69	42	6.16		
Total	534.73	59			
Non-Criterion Group:					
Between stages - bg	4.72	3	1.57	0.33	*
Between individuals - bI	163.93	14	11.71	2.44	<.05
Interaction - XxI	201.53	42	4.80		
Total	370.18	59			

* = When df = 3 and 42, $p = .05$ requires $F = 2.83$.

Experiment SDS-2

Discussion

A few individuals in the criterion group manifested a rather striking initial use of outcomes of previous responses but solved the problem nonetheless surely than the others in that group. They revealed to the experimenter that they had solved the problem by first putting together a long succession of wrong responses that indicated systematic use of negative feedback. Such a long succession could not be reasonably expected to occur by chance in this three-choice problem. After a long period of non-reward (candy), they abruptly switched over to a succession of correct responses and enjoyed the rewards. They appeared to cling to the negative clues until they were quite sure they would not fail any further when they switched to the positive clue. In a multiple-choice design, the criterion of learning should be defined in terms of a statistically improbable succession by chance alone of either right or wrong responses. Some individuals may profit most from such negative feedback at a critical stage in learning and may be considered to have reached criterion by this route.

Growth in the functions of differentiation and intention during the course of the experiments would be expected to appear as a significant main effect of variance between stages. The criterion group demonstrated very significant growth in differentiation ($p = <.01$) across the four stages of the first experiment (RDC-1), but the non-criterion group showed no such growth (Table 7). The criterion group achieved the rotation pattern discrimination after considerable training (120 trials); the non-criterion did not achieve it. This criterion group's average mental age of 5 years 6 months proved barely adequate for the task. The non-criterion group with an average mental age of 4 years 11 months failed to show any significant trend. Apparently, the insight threshold for this type of task lies above CA = 4-11 and perhaps very close to CA = 5-6 for most mentally retarded groups of the trainable level.

On the second experiment (SDS-2), neither group showed any significant growth in differentiation between stages (Table 9). The absence of any further neural or functional integration on this type of size discrimination task may be accounted for by the fact that all but one subject readily solved not only the first variation of the problem, but also at least one or more additional variations as well. Significant growth would not be expected where the neural functional integration and performance are already asymptotic for the group.

Any significant growth in the function of intention across experimental stages failed to occur in both experiments (Tables 8 and 10). A significant improvement in intention would not be expected where growth in differentiation came too late in the experiment to produce any gradual effect on improved integration of intention, as seemed to be the case in the first experiment (RDC-1). However, there was plenty of room across stages for gradual improvement in intention in the second experiment (SDS-2) where successful differentiation and problem solution generally occurred early and where the general level of efficiency of intention was not markedly better than for the first experiment (Appendix I). Such gradual but significant trend for improvement in intention, however, was absent or certainly not significant. The level of efficiency in intention was poor in both experiments, leaving plenty of room for improvement (Appendix I).

In view of these findings, it seems most plausible to consider that the frontal intrinsic sector for intention actually functioned less adequately in both experiments for the criterion group than the posterior intrinsic sector for differentiation, so much so that it was not amenable to significant improvement. Neither sector would yield to improvement in the non-criterion group. Reduced efficiency, such as was manifested by this absence of significant improvement during intensive training, may be accounted for either (1) by inadequate development of, or (2) by injury to, the frontal intrinsic sector of the telencephalon.

Summary

The present study explored the nature of a quasi-four-to-six-year level of isocortical function in problem solving of retardates in late childhood or early adolescence. The neurally distinct functions of the posterior and frontal intrinsic systems in problem solving were examined in the behavior of two such groups of retardates classified by success or failure on a relatively difficult problem. The results showed that significant improvement in the function of differentiation by the posterior intrinsic system was achieved by training of retardates whose mental age on the average had reached

the equivalent of about 5 1/2 years of normal ontogeny. The lower group, averaging about 7 months less in mental age, were unable to make any improvement and could not solve the problem after 120 trials. The attainment of the equivalent of about six years of normal growth in structure and function of the posterior intrinsic system is a critical factor in making the differentiations required for the more complex problems. On the other hand, on a very simple problem for both groups no significant improvement in differentiation was found.

The results also showed an absence of improvement in the function of intention by the frontal intrinsic system for both groups. The function of intention for both groups was very inefficient on both problems and certainly left much to be desired. Rather than speculate extensively on the absence of a significant result in this area, it seems logically better to suggest a test of the pattern of growth in function of the frontal intrinsic system in retardates of successively higher levels of mental age with problems of successively greater complexity.

V. DISTANCE-DISCRIMINATION COMPLEX (DDC-3)

After extensive study of Normal children, Piaget postulated the existence of five "stages" in the development of childrens' mental operations: (1) Sensory-motor, (2) Pre-operational, (3) Intuitive thought, (4) Concrete operations, and (5) Propositional, or formal operations [168, 169].

In their study of the development of geometric concepts, in particular, Piaget and Inhelder [169] traced the development of the notion of distance and its measurement. They found that preschool children perceived two equal intervals as measuring the same distance only when the endpoints were in alignment. "The key feature of all responses at these earlier levels is that children fail to conceive of the length of a journey as an interval between the point of arrival and the point of departure but think of it only in terms of the former."

In discussing the implications of Piaget's work for the teaching of the mentally retarded, Wolinsky [230] suggested that perception, too, is developmental and that experimentation with the mentally retarded should not be confined to presentation of structural material. There should be an exploration of what the child sees and what he does with the material. "Is the child's retardation a deficiency in structure or is it an unsuccessful mastery of a part of a particular developmental stage in cognition?" [230]

In an attempt to investigate aspects of the structure of distance perception, boys and girls in special classes for the retarded in the Madison, Wisconsin public schools were presented with a distance matching task in which the positions of the endpoints of the same interval were systematically varied. Two groups of children, of "high" and "low" mental age were included.

Method

Five boys and five girls participated in the "low" MA group, I, (3, 0 - 5, 11) and five boys and five girls in the "high" MA group, II, (6, 0 - 10, 11). The average I. Q. was 63, with a range of 53-76, largely WISC IQ's obtained by a specialist. CA's in group I ranged from 7-6 to 13-0 and those in group II from 11-7 to 16-4. These children were chosen randomly from five classes in the same school. School authorities had graded the classes by age and development so it is not surprising that the children in group I all came from two lower level classes and those in group II from three higher level classes. In this school there were 51 boys and 22 girls, and the girls chosen almost exhausted the number available in group I.

The apparatus consisted of one 30" x 30" plywood board, painted white, in which holes were drilled to accomodate four wooden dowels, 3/8" in diameter and 12" in length. The holes were arranged so that the dowel-pairs could be presented in four different ways as shown in figure 2. The dowels in each pair were 20" apart. For convenience the positions were called Displaced (D), Angled (A), Perpendicular (P), and Front-Back (F).

A continuous piece of elastic was stretched between the dowels 5" above the board. The elastic passed through the windshield and back window of a small, plastic car about 1 1/8" in length so that the car could be freely moved between the dowels but would hold position when released. E moved his car a certain distance from a position against a dowel (always from S's right or away from S), and then S was instructed to move his car, also from an extreme position, matching the distance covered by E's car. All testing was done individually in an empty classroom of the school.

Each S received two to four practice trials at each position with no correction for errors. Initial instructions were to "move your car the same distance as my car. If I move my car just a little bit, then you move your car just a little bit. If I move my car a long way, then you move your car a long way." During the practice trials, after S moved his car E would say, "Is it the same as mine?" or "Did I move mine a long (little) way?" After S's reaction, E would then say, "Did you move yours a long (little) way?"

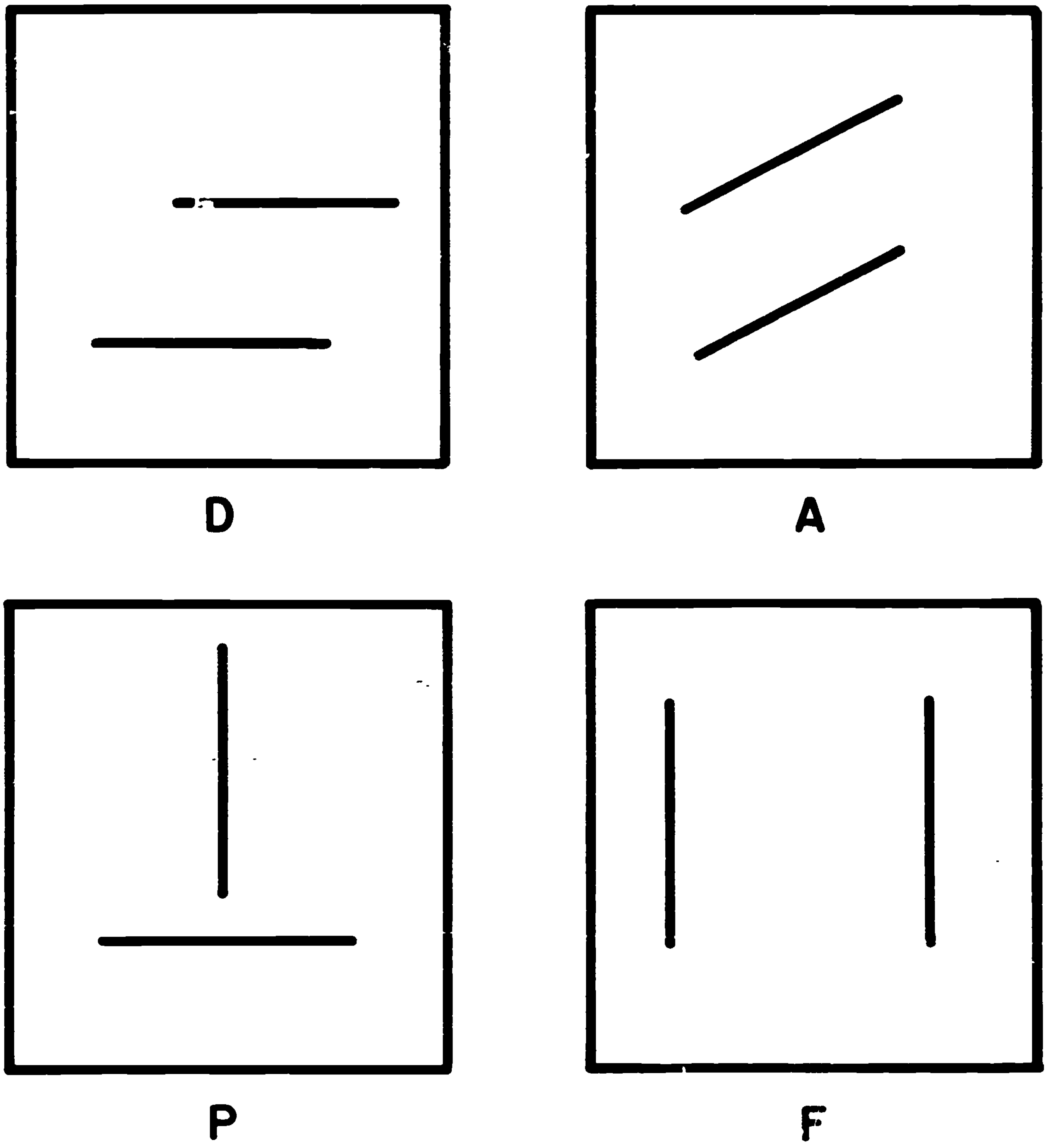

Figure 2

SCHEMATIC VIEW OF POSITIONS FOR DDC-3

Every S was then tested three times at each of the four positions, the order of presentation being randomized individually in advance. E had trained himself to choose randomly a distance between 5" and 16", measured from the dowel to the front of the car, and a subsequent check of distances actually chosen shows that this procedure was followed. Distance was not manipulated since a pilot study had indicated it was most likely not a significant factor. Only after S indicated satisfaction with his action did E measure the distance to S's and E's car These measurements were taken to the nearest 1/8" and recorded without comment to S. Few Ss attempted to watch the measurement and fewer still could read the numbers.

Results

The absolute value of the difference between E's and S's distances was used as a measure of error. The absolute value was chosen since the position of E's car sometimes encouraged S to overestimate and sometimes to underestimate the distance. These errors were interpreted as an overall measure of the difficulty of these tasks.

In addition to the difficulty measure, a measure termed "consistency" was calculated from the three observations taken at each position. This measure was the unbiased sample variance estimate, $s_c^2 = \Sigma(x_i - \bar{x})^2/(n - 1)$, where x_i is the absolute difference, i.e. error. Clearly, s_c^2 will be least when errors are either all large or all small and greatest when some are large and some are small. Also s_c^2 should behave as a variance and hence its logarithm should be approximately normally distributed.

If S's considered only the endpoints of two distances then they should have made consistent errors, at least at some positions, e.g., D, A, and perhaps F. Misunderstood directions or complete ignorance of the task, however, should result in inconsistent responses.

Analysis of the Absolute Values of the Errors

The curve of these absolute values appears unimodal and not excessively skewed so a preliminary analysis of variance was done on the absolute values. The analysis assumed a mixed model, the effects of position (P), age (mental age)(MA), and sex (S) taken as fixed and individuals nested within sex-age combinations (I/s-a) taken as random. The analysis of variance summary is shown in Table 11 and appropriate means are presented in Table 12. No adjustment was made for lack of independence, which must be suspected where repeated measures are taken on subjects, because it was possible to present the repeated measures in random order. Box [21] notes that in many such situations the nonindependence among the errors takes a special form, all are equally correlated, and that the usual F-tests are still valid.

The position means shown in Table 12 suggest that D and A may both be more difficult than P and F, there being little difference between D and A and between P and F. If μ_D, μ_A, μ_P and μ_F are the population means corresponding to the above sample means, then the contrast $\psi = \frac{1}{2}(\mu_D + \mu_A) - \frac{1}{2}(\mu_P + \mu_F)$ is nonzero if the above hypothesis is correct. A Scheffé contrasting procedure [196] yields the sample contrast $\hat{\psi} = \frac{1}{2}(M_D + M_A) - \frac{1}{2}(M_P + M_F) = 1.313$, where the M's are sample means from Table 12. The resulting 99% confidence interval for ψ is $0.085 \leq \psi \leq 2.541$, indicating that this comparison is likely the source of the significant P effect. This finding proved to be very valuable in interpreting the P x MA x S interaction. The average response at positions D and A can be considered as one "difficult" task and the average at P and F as one "easy" task. The cubical figure (Figure 3) can then be constructed with dimensions difficulty, mental age, and sex. The numbers at the vertices are averages of pairs of means from Table 12.

Thus, a compelling interpretation of the interaction P x A x S is that the lower mental age boys did significantly poorer than girls on the more difficult tasks (see the large value for male, lower mental age, difficult).

Analysis of the Consistency Measures

The consistency measures were first transformed to common logarithms and then subjected to an analysis of variance. The results are shown in Table 13.

Table 11

Analysis of Variance of Absolute Values of Error Scores

Source	df	Mean Square	F
Position	3	37.068	15.562*
Mental Age	1	25.026	2.657
Sex	1	12.950	1.375
Individuals/(S-MA)[b]	16	9.418	9.780*
PxMA	3	1.920	< 1
PxS	3	2.070	< 1
PxI(S-ma)[a]	48	1.382	2.474
SxMA	1	19.781	2.100
PxMAxS	3	27.439	11.519*
Replication s[c]	160	0.963	

[a] Error term for P, PxMA, PxS, and PxMAxS.

[b] Error term for MA, S, MAxS.

[c] Error term for I(S-ma) and PxI(S-ma).

Experiment DDC-3

Table 12

Means for the Position Effect and the PxAxS Interaction

Positions	D	A	P	F
	2.744	2.251	1.253	1.117
Group I				
Males	4.033	2.875	2.300	1.525
Females	1.925	1.853	1.125	1.675
Group II				
Males	2.266	2.225	0.712	0.650
Females	2.750	2.050	0.875	0.617

Experiment DDC-3

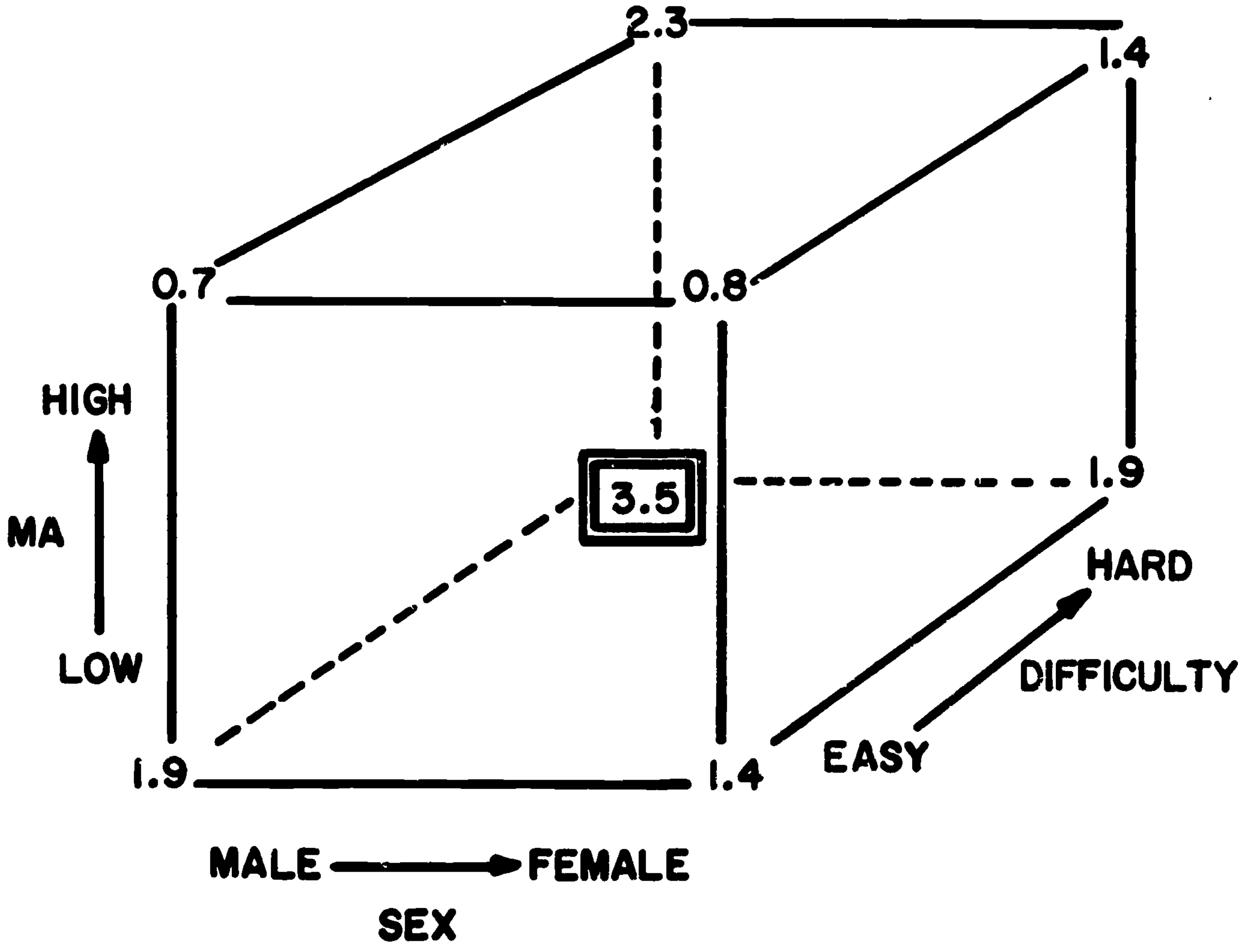

FIG. 3 Means (pooled for p) shown to illustrate the PxMAxS interaction.

Table 13

Analysis of Variance of the Consistency Estimates of Each Individual's Responses at Each Position

Source	df	Mean Square	F
Positions	3	176.384	6.543**
Mental Age	1	558.989	10.318*
Sex	1	0.090	<1
Individuals/Sex-MA[a]	16	54.273	
PxMA	3	98.262	3.645*
PxS	3	16.894	<1
PxI(s-ma)[b]	48	26.957	
MAxS	1	116.010	2.138
PxMAxS	3	127.675	4.736*

Note: The analysis was performed on the logarithms of the consistency estimates.

[a] Error term for MA, S, MAxS

[b] Error term for P, PxS, and PxMAxS

*Significant at the .001 level

**Significant at the .01 level

Experiment DDC-3

Note particularly the much greater role played by mental age in this second analysis. Both MA and P x MA are shown to be non-chance sources of variation in addition to P x MA x D as before. Positions again are a strong source of variation. Since there is now only one observation per cell, the I(s-ma) and P x I(s-ma) have no proper error term and no inference about them is offered.

The means for groups I(-1.261) and II(-6.548) (mean logarithms) show group I to be significantly less consistent than group II. In addition, the position means for groups I and II (see Table 14) show that the difference is small at F and grows progressively greater through A, P, and D. The consistency of group II as contrasted with group I at the position D is striking.

The consistency means at the four positions do not exhibit the previously observed dichotomy of the mean errors. From Piaget's observation about the importance of endpoints and from observation of the children doing similar tasks during pretesting, it was predicted that the errors would be most consistent for the position F and would differ progressively more through P then D then A, i.e., $\sigma_F^2 < \sigma_P^2 < \sigma_D^2 < \sigma_A^2$. This assertion will be discussed in more detail later, but suffice to say it is based on the tendency of some S's to "line up" the two cars with reference to lines perpendicular to the elastic bands. This was nearly impossible in the F position (see Figure 2) because the problem board was placed in the middle of a long classroom table.

The above argument can be phrased as a "linear hypothesis" of ordered means as an alternative to the null hypothesis of equal means. In terms of the logarithms of the consistency scores, let σ_F^2 be the log consistency at the position F (front-back). The hypotheses then take the following form:

$$H_0 \ : \ \sigma_F^2 = \sigma_P^2 = \sigma_D^2 = \sigma_A^2$$

$$H_1 \ : \ \sigma_F^2 < \sigma_P^2 < \sigma_D^2 < \sigma_A^2$$

The hypothesis H_0 can be tested against the class of linear alternatives, H_1, with the statistic L [162]. The <u>ranks</u> of the logs of the logs of the individual variance estimates are shown in Table15. L is calculated by summing the observed ranks at each position,

Table 14

Consistency Means for Positions and for the PxA and PxAxS Interactions

Sources	Mean			
	D	A	P	F
Positions	-4.927	-0.892	-2.221	-7.579
Group I				
Males	2.244	4.255	3.992	-10.769
Females	-1.749	-3.851	-0.827	-3.385
Combined	0.247	0.202	1.583	-7.977
Group II				
Males	-12.495	-4.579	-7.120	-6.867
Females	-7.707	+0.609	-4.930	-9.295
Combined	-10.101	-1.985	-6.025	-8.081

Note—These are the means of the *logarithms* of the consistency estimates. Positive logarithms indicate quantities greater than one while large negative logarithms indicate positive quantities near zero.

Experiment DDC-3

Table 15

Individual Means Ranked Across Positions to Test a Linear Hypothesis

Ss	Predicted Ranks			
	D = 3	A = 4	P = 2	F = 1
	Observed Ranks			
1	4	3	1	2
2	3	4	2	1
3	2	3	4	1
4	2	3	4	1
5	2	3	4	1
6	4	2	3	1
7	1	3	4	2
8	1	3	2	4
9	2	3	4	1
10	3	1	2	4
11	1	4	3	2
12	2	4	1	3
13	1	2	4	3
14	1	2	3	4
15	1	3	4	2
16	1	3	4	2
17	3	4	2	1
18	2	4	3	1
19	2	4	3	1
20	3	4	1	2

Experiment DDC-3

multiplying each sum by the predicted rank and adding the four products. Here, $L = 526$, and (using Page's notation) $m = 20$, $n = 4$, so that the observed value of L is significant at the .05 level. [$P(L > 522) = 0.05$, $P(L > 531) = 0.01$.] Hence, the null hypothesis can be rejected and support is given to the linear hypothesis.

Interpretation of the P x MA x S interaction is more difficult for these consistency estimates. The boys in group I, however, showed a very different sort of response at position F—exceptional consistency. These lower mental age boys showed the most variable responses at D, A, and P and the most consistent responses at F (Figure 4).

Discussion

Guided by Piaget's observations, four positions were chosen in an attempt to test various levels of the structure of distance perception. The results showed that the tasks had different difficulty levels, and that the order was in essential agreement with predictions based on Piaget's studies.

The task F should be easiest, and in fact the smallest average error occurred here. D should be most difficult with A and P somewhere intermediate. The largest mean error did occur at D, and it was shown that D and A can be considered apart from P and F. The same sort of miscalculation by Ss led to large errors on the tasks D and A, however. Especially in D, Ss would align S's and E's car with reference to a line perpendicular to the elastic bands. That the reference was not to the edge of the board is clear since Ss did <u>not</u> use the edge of the board in the task A, a strategy which would have led to a correct placement. Repeatedly E observed S carefully sighting along the cars, aligning them rather than equating distances. Task P did not allow this strategy, and insofar as it was possible it led to a correct placement in F.

These findings seem to support Piaget's observation that children who function at "preschool" levels perceive equal intervals as measuring the same distance only where the endpoints are in alignment. A possible conclusion is, therefore, that distance perception is developmental, the development corresponding more

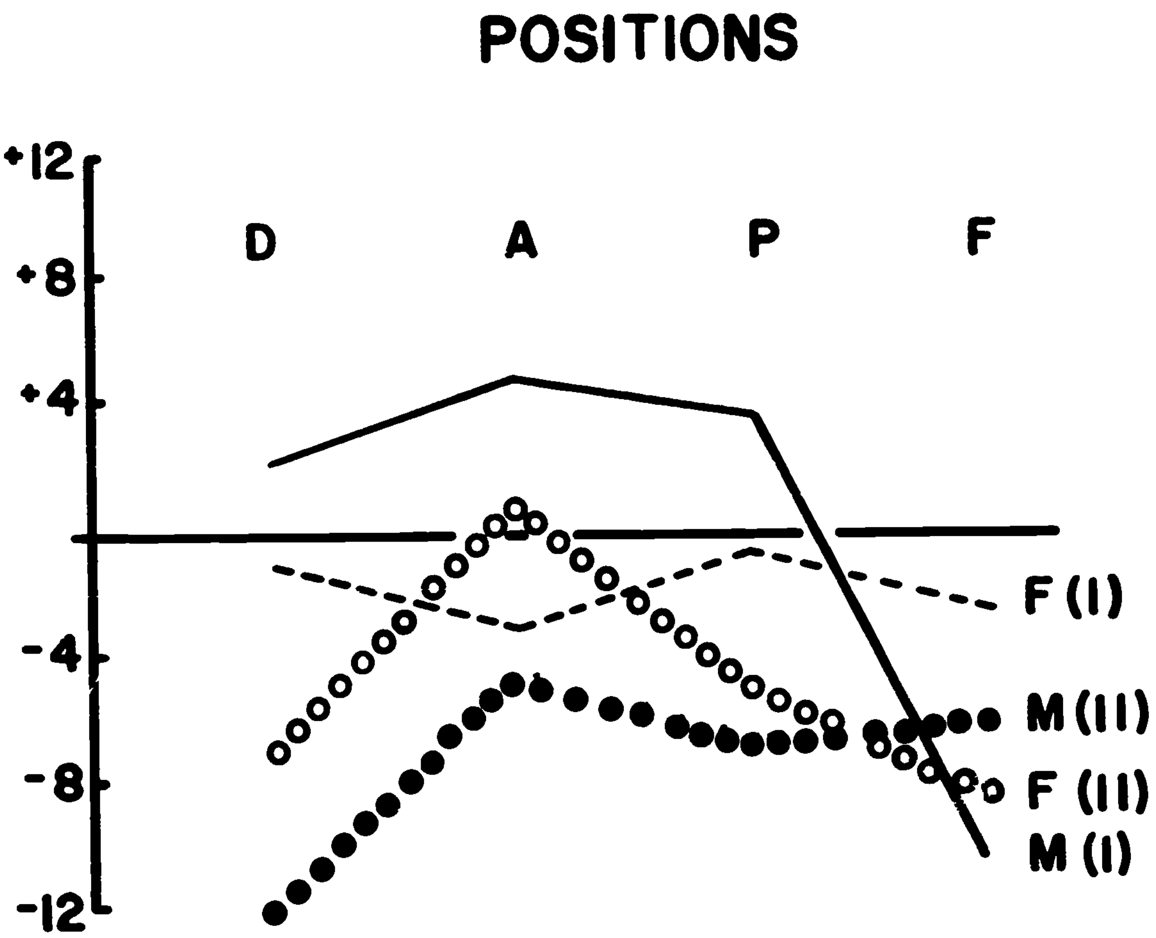

Fig. 4 Graph of the mean log consistencies for males and females, groups I and II, across positions.

to MA than to CA. There was a cognitive dimension to the task, since the construct, same distance ("as far as"), was presented verbally with only a few practice trials. Apparently, if the equal distances were not perceived correctly, the children referred to a simple space of parallels and perpendiculars, the reference lines not being the walls or table but the lines (elastic bands) of the task itself.

The complex relationship among position, mental age, and sex was completely unexpected. If the poor performance of boys at the lower levels on more difficult tasks is confirmed by other studies it would argue for some special treatment (of M-Rs) during the early years in school.

As noted before, the consistency studies are valuable chiefly in delineating the role of increasing mental age. Whereas there is no significant difference between the mean absolute errors of groups I and II, group II was significantly more consistent. The children in group II were older, and those who made large errors tended to perseverate and rarely achieve a close match (see especially the consistency of group II at position D). The remainder were essentially successful and rarely made large errors.

The prediction of maximum inconsistency at position A may seem strange, but the angled elastic bands were expected to confuse the children. This was partially true, and the fact that some children could do well at this task also increased the variability. Task P offers little chance to be confused by the initial and terminal points, and those children who seemed not to understand how to match the distances in this case usually moved their car to the center of their path (opposite the initial point of E's path). The effect of this was to minimize error in a sense because E could place his car only so far from the center, and random placements were bound to make S correct some of the time. The observed greater average consistency at position D leads to the conjecture that Ss employed a more systematic approach in D, though some approaches yielded larger errors.

Summary

Twenty children in special public school classes for the mentally retarded were given verbal instructions to match distances set by E in four different comparative situations (positions). Practice was given only to acquaint them with the elements of the task. Equal numbers of boys and girls were tested in low(MA 3, 0 - 5, 11) and high MA (6, 0-10, 11) groups, three trials at each position.

Analysis of absolute errors showed that low MA boys did significantly worse than girls at the more difficult positions, whereas lower order comparisons showed only positions to be significantly different.

The high MA group was more consistent in its responses, and predictions about the consistencies at the four positions were confirmed.

It seems that perception of distance is maturational, the levels of maturation being related to MA rather than CA, at least in the present situation. Incorrect responses tended to be made with reference to a rigid, orderly space of parallels and perpendiculars.

VI. HYPOTHESIS-DISCRIMINATION-COMPLEX (HDC-4)

These experiments were designed to confront a subject with a situation in which two equally valid hypotheses were available for choice from the outset. Once a selection was made the experimental situation was changed so that the chosen hypothesis broke down, either gradually or abruptly, while the originally nonchosen hypothesis continued to operate. Both original learning and subsequent modification of response, if it occurred, were studied.

There have been a number of studies in which simultaneously operating hypotheses have been used at stages other than the first. An example of these is one by Bruner, Matter, and Papanek [26] in which the simultaneously relevant cues were color and single alternation. An important distinction between this study and the present one relative to the use of the simultaneously relevant cues was, however, that Bruner et al.'s subjects were taught the color cue discrimination before the single alternation cue was introduced. A second difference was removal of the color cue when the single alternation cue alone became relevant. Bruner et al. were concerned with the effects of different degrees of both motivation and learning on the degree to which incidental cues were learned.

Iwahara and Sugimura [102, 214] held the stimulus setting constant even though the relevant hypothesis changed; in one case, from a size discrimination to a color discrimination, and, in the other, from a position discrimination to a color discrimination. In contrast to the present experiment and to Bruner et al.'s, at no time were the two hypotheses simultaneously relevant. Also, in this case, the experiment was a study of effect of different degrees of learning of the first hypothesis on ability to exchange it for the second.

In Luchin's water jar tests [135] there were also stages during which two hypotheses were available to the subjects. But, again, this was only after the subjects had first been taught one solution.

No studies in which two hypotheses were available to subjects from the outset seem to have been made and none were found in which the primary concern was to compare the relative effectiveness of different rates of failure of an earlier learned hypothesis as a means of inducing change to a different hypothesis.

Method

Subjects in these experiments were sixty-five mental retardates, most of whom were classified as severely retarded, from the Central Wisconsin Colony and Training School at Madison, Wisconsin. They ranged in chronological age from approximately four years, eight months, to forty-two years, ten months; in mental age from approximately one year, two months, to twelve years, ten months; and in I. Q. from eleven to sixty-nine. The ratio of males to females was forty-eight to seventeen.

Before the experiments proper a number of patients, as well as a number of children at a privately operated playschool, were exposed to various forms of the procedure and combinations and arrangements of the props for the purpose of standardizing procedures and determining the kinds of reactions which might be expected under the various experimental conditions.

Apparatus

The apparatus used for gathering the data for this study was quite simple. Subjects were confronted with three paper cups one of which was predominately red, one predominately green, and the third yellow in color. A cardboard box of suitable size, with one side and flap cut away was used to screen the cups and the experimenter's arms, hands, and shoulders from the subject as the cups were being prepared between trials. Another cardboard box, with one side and all flaps cut away, was used to screen the subject from other materials needed to conduct a series of trials. These other materials consisted of a pencil, a clipboard on which the instrument outlining a series of trials for one sitting was placed, a container of rewards, and the instruments needed for a period or day of sittings. With the exception of two subjects for whom raisins were employed, rewards were M & M's randomized as to color.

The "Instruments" or data and checksheets, were 8-1/2" by

11" size sheets of paper, each containing the information needed to provide a program of trials and a record of the performance of each subject for a given day.

The six types of instruments used in the study were (1) Teaching Instruments, (2) Tests for Hypothesis Adopted, (3) Abrupt Failure Instruments—Always Middle to Always Green, (4) Abrupt Failure Instruments—Always Green to Always Middle, (5) Gradual Failure Instruments—Always Middle to Always Green, and (6) Gradual Failure Instruments—Always Green to Always Middle. Samples of each are included in Appendix X.

A subject could make a correct response each time by using either the hypothesis Always Middle or Always Green. On the Teaching Instruments this was accomplished by placement of the symbol for the green cup (typewritten capital letter "0") and the symbol for the reward (typewritten "+") in the middle of the three positions on each trial. Randomization was then used to determine on which of the opposite sides of the symbol for the green cup the symbols for the yellow and red cups (typewritten capital letter "U" and typewritten "_" respectively) were to be placed on each trial. Randomization of the placement of these symbols (as was the case with randomization of certain aspects of the design of the other instruments in this study) was accomplished by the use of a table of random numbers. The number of trials for each instrument was 48.

The two hypotheses programmed into the Teaching Instrument (Always Middle or Always Green) operated simultaneously throughout the entire series of trials. Five such instruments with the pattern of trials on each uniquely randomized were utilized. Choice of either hypothesis enabled the subject to make a correct response on each trial. The design of the Test for Hypothesis Adopted was such that a single administration provided a reliable indication of the hypothesis originally chosen. A criterion of ten consecutive responses consistent with either hypothesis was used.

The Abrupt Failure Instruments—Always Middle to Always Green also consisted of a set of five so designed that use of the first learned hypothesis (in this case Always Middle) resulted in immediate total failure whereas the previously unlearned hypothesis continued to provide rewards at the 100 per cent level. Each instrument in the set differed from the others in that the order in which the equal number of the four possible different individual trial arrangements randomly appeared was according to a pattern unique for that instrument.

The Abrupt Failure—Always Green to Always Middle Instruments were, except for one feature, identical in design to the Abrupt Failure Instruments—Always Middle to Always Green. This likeness between the two sets included the attribute of having the same respective patterns of randomization. The single distinguishing feature was provision so that the hypothesis Always Green was never rewarded and the hypothesis Always Middle was rewarded 100 per cent of the time.

The Gradual Failure Instruments—Always Middle to Always Green were programmed in such a way as to constitute a series of trials on which the rate of exceptions to the originally learned hypothesis accelerated from one in twelve trials to twelve exceptions for the last twelve. Ordinal placement of the exceptions within each series of twelve trials was randomized, as was which of the two possible styles of exceptions that was employed in each case.

The Gradual Failure Instruments—Always Green to Always Middle were identical to the instruments described immediately above except that in this instance the failing hypothesis was Always Green. (In the pilot study both gradual failure instruments were programmed for a maximum of fifty per cent failure.)

Because of the peculiar nature of the gradual Failure Instruments, and the fact that their structure made a criterion of ten consecutive correct responses inappropriate (particularly during the early stages), this criterion was modified for these instruments. Instead of simply ten consecutive correct responses, the requirement was established as ten or more consecutive responses with at least two of these ten being trials where the correct response constituted a departure from the first learned hypothesis.

Subjects were escorted between the wards and the experimental room by the experimenter. After subjects had been brought to the experimental room for the first time they were seated on a chair opposite the experimenter and steps were taken to determine their reactions to the rewards. This was done by showing them a reward and then attempting to elicit, by whatever means seemed appropriate, some type of indication as to whether or not it was something they found desirable. Only one subject was dropped because of not liking either of the rewards.

The second step was to try to determine whether the subjects could distinguish between the colored cups. Several different ways of ascertaining this were used. First of all, if it seemed that the subjects could use words they were asked to name the colors of the different cups several times as each cup was indicated. If this failed, an attempt was made to get them to point to the cup which was the same color as the one indicated by the experimenter.

As these steps were completed, the subjects were given a brief oral explanation, accompanied by a very short demonstration, of the procedural mechanics of the problem and of what they would be expected to do. They were then started on the first trial of the Teaching Instrument.

These trials were presented by removing a corrugated box screen from over the three cups. After the subject had responded by looking under one of the cups (and acquiring the reward if it was a correct response) the screen was replaced. The response was then recorded on the instrument being used for that subject for that day. With the screen still in place the experimenter then prepared the cups for the next trial.

As trials progressed whatever oral and/or physical encouragements or admonitions were necessary to help the subject catch on to the proceedings were given (short of giving away the correct response). These continued until a smoothly working, desirable routine was established. The subject was then given one Teaching Instrument's worth of trials (forty-eight) each day until the criterion of ten consecutive correct responses was reached. Regardless of the point in a day's trials at which the criterion was reached the Test for Hypothesis Adopted was always given on that day.

Only two subjects failed to reach criterion within a cutoff point of 480 trials on the first or Teaching Instrument Stage. A cutoff point of 720 trials was used in the second stage of the experiment because a pilot study had shown that none of the subjects who changed from one hypothesis to the other needed more than

623 trials to do so. (In the pilot study the cutoff point for the second stage had been 1000 trials.)

The type of instrument used for a subject after the subject had reached criterion on the Teaching Instrument, and on one of the tests on the Test for Hypothesis Adopted, was determined by two considerations. The first of these was which of the two hypotheses the subject had used in reaching criterion on the Teaching Instrument. For example, if the Test for Hypothesis Adopted indicated that the hypothesis Always Green had been used, the type of instrument employed next was one of the two designed to cause the subject to abandon this hypothesis and adopt Always Middle.

Since there were two tracks in this category (the abrupt and gradual) which of these two was used was determined by assigning subjects alternately to each track on the basis of the order in which they reached criterion on the Teaching Instrument with the end result of having the same number of subjects in each track.

If the Test for Hypothesis Adopted indicated the subject had used the hypothesis Always Middle to reach criterion on the Teaching Instrument the next type of instrument utilized was from one of the two tracks designed to cause the subject to abandon this hypothesis and to adopt Always Green. Which of the two tracks in this category was used for a subject was determined on the same basis as that employed in the other category; namely, the order in which subjects reached criterion on the Teaching Instrument.

Adoption of the Always Middle hypothesis was checked on by confronting the subject with more than three cups (five and seven) to check on second from right, third from right, second from left, third from left, and similar hypotheses.

Results

For this group of mental retardates the hypotheses used in the pilot and main study ranged in difficulty from the easiest Always Middle (position) through Always Green (color) to Alternately Right and Left (two cups). The pilot study revealed that simple alternation was too difficult in both the learning and shifting stages for these subjects so it was supplanted with the hypothesis Always

Middle in the experiment proper. Actually most of the subjects in the switching process after giving up the color hypothesis resorted to consistently choosing a particular side regardless of the color of the cup and the fact that rewards were uncovered only on every other trial. This rather striking phenomenon, which has been commented on previously, was in evidence in all our experiments whenever the complex learning proved too difficult.

Of the sixty-five subjects used in this experiment, sixty-one mastered the Teaching Instrument. Of these, fifty did so by use of the Always Middle (or position) hypothesis and 11 did so by use of the Always Green (color) hypothesis. (In the pilot study twenty-seven subjects chose color and five a right-left alternation hypothesis.)

Twenty-five of the fifty subjects who solved the Teaching Instrument by use of the Always Middle (or position) hypothesis were given the abrupt failure sequence in the second stage, and twenty-five were given the gradual failure sequence. Of the twenty-five subjects who experienced the abrupt failure sequence, seventeen changed to the hypothesis Always Green, and eight failed to make this change. Of the twenty-five subjects who experienced the abrupt failure sequence, seventeen changed to the hypothesis Always Green, and eight failed to make this change. Of the twenty-five who had been given the gradual failure sequence seventeen changed to the hypothesis Always Green and eight did not make this change.

Six of the elven subjects who solved the Teaching Instrument by use of the color hypothesis were given the abrupt failure sequence in the second stage, and five were given the gradual failure sequence. Six of these subjects changed successfully to the Always Middle (or position) hypothesis.

Learning and shift scores were computed for all subjects and were then subjected to both correlational and multivariate analysis. In the multivariate analysis the symbolic basis vectors were High CA vs. Low CA, Shift - Abrupt vs. Gradual, Sex - Female vs. Male, Age x Shift, Age x Sex, and Age x Shift x Sex for the variates learning score and shift score (Tables 16 and 17).

Table 16

Multivariate Tests of Equality of Mean Vectors for the Dependent Variates of Learning Score and Shift Score
(DF = 2 and 47)

Base Vector	F-Ratio	P Less Than
High C.A. vs. Low C.A.	15.9383	0.0001
Abrupt vs. Gradual Mode	0.5396	0.5866
Female vs. Male	1.1266	0.3328
Age x Shift	0.0956	0.9091
Age x Sex	1.5267	0.2267
Shift x Sex	1.4774	0.2387
Age x Shift x Sex	0.7660	0.4706

Experiment HDC-4

Table 17

F-Ratios for Indicated Factors and Variables Learn and Shift
DF = 1 and 48

	Univariate F	P Less Than
High C.A. vs. Low C.A.		
Learn	0.3739	0.5438
Shift	32.2274	0.0001
Abrupt vs. Gradual Mode		
Learn	0.0322	0.8584
Shift	1.1008	0.2994
Female vs. Male		
Learn	1.4719	0.2310
Shift	0.4104	0.5248
Age x Shift		
Learn	0.0396	0.8431
Shift	0.1824	0.6713
Age x Sex		
Learn	2.4531	0.1239
Shift	00.2256	0.6370
Shift x Sex		
Learn	0.0221	0.8825
Shift	2.9729	0.0912
Age x Shift x Sex		
Learn	0.0108	0.9176
Shift	1.4357	0.2368

Experiment HDC-4

Correlations were computed among the variables available for each subject (MA, IQ, CA, abrupt and gradual modes, learning scores and shift scores) for the total group, and for each of five different diagnostic categories (Tables 18 and 19). The groupings employed were based on the expanded medical classification of the American Association on Mental Retardation [87]. Category I included diseases and conditions due to infections, intoxication, and trauma, II included unknown prenatal influences including mongolism, III covered unknown prenatal influences excluding mongolism, IV Mongolism, and V included mental retardates whose condition was diagnosed as due to uncertain (or presumed psychologic) causes with the functional reaction alone manifest.

Means for characteristic phases of the study are shown in Tables 20, 21 and 22.

With respect to age the multivariate test of equality of mean vectors for the dependent variates of learning score and shift score was F (2,47) = 15.94, P less than .0001. The univariate analysis of the age effect indicated that the two groups did not differ, F (1,48) = .3739. However the shift scores for the effect was significantly different F (1,48) = 32.2274, P less than .0001.

When groups were matched for MA, IQ or CA by far the greatest factor was age. MA F(1,48) = 3.8954, IQ F(1,48) = 6.979, and CA F(1,48) = 19.002. In the learning phase of the experiment neither mode of shift, sex, MA, IQ or CA were significant. In shifting neither mode of shift, sex, nor MA were significant. IQ played a role, but CA was significant at a probability level of less than .0001.

The correlations (Tables 18 and 19) also reveal the powerful overall effect of age and this age effect remains clearly in evidence when young and old are compared on a basis of sex (Table 22).

Although the scores for the abrupt and gradual modes were not significantly different for the overall group, the correlations for Diagnostic groups II, III, and IV; and the total group were higher for the abrupt than for the gradual mode (Table 19). Perhaps the reason the age effect did not appear in Diagnostic Group V was that it was composed of those frankly diagnosed as uncertain or unknown as to etiology and this may have caused inadvertent inclusion

Table 18

Correlation Matrix

	MA	IQ	CA	Abr Mode	Grad Mode	Learn Score	Shift Score
MA		.365	.366	.179	.259	.085	.238
IQ	.365		-.542	-.479	-.465	-.108	-.437
CA	.366	-.542		.756	.547	.096	.635
Abr Mode	.179	-.479	.756			.244	
Grad Mode	.259	-.465	.547			.118	
Learn Score	.085	-.108	.096	.244	.188		.182
Shift Score	.238	-.437	.635			.182	

Experiment HDC-4

Table 19

Correlations for Various Diagnostic Groups and Total Group

Diagnostic Group	Abrupt Mode	Gradual Mode	Learning Score	Shift Score
I	.766	.765	-.050	.758
II	.951	.463	.242	.622
III	.966	.228	.237	.576
IV	.994	.565	.373	.654
V	.583	.624	.061	.139
Total Group	.756	.547	.096	.635

Experiment HDC-4

Table 20

Learning Means for C.A.

High C.A. 40.5	Abrupt 40.1	Female 14.3
		Male 47.1
	Gradual 40.9	Female 19.0
		Male 53.1
Low C.A. 33.9	Abrupt 32.4	Female 40.5
		Male 31.1
	Gradual 35.4	Female 37.0
		Male 34.2

Experiment HDC-4

Table 21

Shift Means for C.A.

High C.A. 500.3	Abrupt 483.3	Female 720.0
		Male 418.7
	Gradual 517.2	Female 455.6
		Male 551.6
Low C.A. 162.5	Abrupt 117.1	Female 157.0
		Male 110.4
	Gradual 207.9	Female 201.0
		Male 213.1

Experiment HDC-4

Table 22

Learning and Shift Means for Mode and Sex

	Learning	Shift
Abrupt Mode	36.2	300.2
Gradual Mode	38.2	362.6
Female	27.6	372.4
Male	41.1	315.0
High C.A. Female	17.3	554.7
High C.A. Male	49.8	478.5
Low C.A. Female	37.9	190.0
Low C.A. Male	32.4	151.5
Abrupt Mode Female	24.8	494.8
Abrupt Mode Male	38.7	257.9
Gradual Mode Female	28.8	316.7
Gradual Mode Male	44.2	392.3

Experiment HDC-4

of children with central nervous systems not so subject to the foreshortening effect of retardation.

For those who shifted with difficulty the abrupt mode was the better and in general the gradual mode seemed the better differentiating condition. The theoretical advantages of the abrupt mode seem better utilized by the lower CA subjects.

Discussion

The most significant finding of the study, derived from a correlational and multivariate analysis of the data is the crystalizing and handicapping effect of age. Whether the abrupt or the gradual method of attempting shifting was used the younger individuals were more flexible. This marked effect was not discernible in the simple learning phases, but was clearly revealed when shifting was called for. Moreover, inflexibility or rigidity seems to set in at a much earlier age in retardates than it does in normals or supernormals. It seems clear that in mental retardates appropriate education should be begun at an early age, especially as regards basic thinking habits. This is in complete accord with the basic psychoneurological position set forth in the preliminary chapters and our behavioral findings. Just as the language development of the deaf must be dealt with at an early age if best results are to be achieved, so must the education of the mental retardate be initiated at an early age.

Present practice of relying on Mental Age for starting purposes seems clearly contraindicated. Early initiation of the educational process for the mentally retarded is at least as important as it is for the gifted though the curricular content would be quite different. Indeed postponement is much more apt to be harmful for those of lower intelligence than it is for the bright. The hypothesis that the early period of dendritic proliferation is lengthened or shortened and the decline in RNA deposition hastened or postponed in accordance with the degree of intelligence is an intriguing one well worth further examination.

Summary

Mental Retardates were confronted with an experimental situation in which they had an opportunity to choose either of two equally valid hypotheses. Conditions were then changed so that the chosen hypothesis ceased to work, either gradually or abruptly, in an effort to bring about a switch to the originally nonchosen hypothesis which continued to work.

Inability to shift, or difficulty in shifting, was strongly associated with chronological age. This led to the recommendation that the education of the mentally retarded be initiated at an early age and be not so closely tied in with mental age.

VII. GENERAL SUMMARY AND CONCLUSIONS

This investigation employed a neuropsychological approach to research on learning and problem solving in mental retardation. After discussing neurological bases in some detail, simple and complex learning and problem solving situations were employed in a variety of experimental settings:

1. Rotation-Discrimination Complex (RDC-1)
2. Size-Discrimination Simple (SDS-2)
3. Distance-Discrimination Complex (DDC-3)
4. Hypothesis-Discrimination Complex (HDC-4)

In the RDC-1 and SDS-2 experiments upper and lower subgroups, within a transitional group with respect to maturation of the cortex, divided by their achievement or nonachievement on a relatively complex task were subjected to two basic types of visual discrimination at inherently different levels of task complexity to probe for performance characteristics of the separate intrinsic systems' functions of differentiation and intention. The results showed that significant improvement in the function of differentiation by the posterior intrinsic system was achieved by training of retardates whose mental age on the average had reached the equivalent of about 5 - 1/2 years of normal ontogeny. The results are in accord with the point of view that the equivalent of about six years of normal growth in structure and function of the posterior intrinsic system is required for making the differentiation required for the more complex problems. Those averaging about 7 months less in mental age were unable to solve the problem. Both groups readily solved a very simple problem and no significant improvement in differentiation occurred. The function of intention was very inefficient on both problems for both groups.

The Distance-Discrimination Complex (DDC-3) showed boys doing significantly worse than girls at the more difficult positions, whereas lower order comparisons showed only positions to be significantly different. The high M.A. group was more consistent in its responses, and predictions about the consistencies at the four positions were confirmed. Perception of distance seems to be maturational, the levels of maturation being related to M.A. rather than C.A. Incorrect responses tended to be made with reference to a rigid, orderly space of parallels and perpendiculars.

The Hypothesis-Discrimination Complex (HDC-4) experiments confronted the subjects with an experimental situation in which they had an opportunity to choose either of two equally valid hypotheses. Once a choice was made conditions were changed so that the originally chosen hypothesis ceased to work, either gradually or abruptly, in an effort to bring about a switch to the originally non-chosen hypothesis which had operated from the outset. Inability to shift, or difficulty in shifting, was strongly associated with chronological age, much more so than with any other variable.

The results of all simple and complex learning situations were in accord with the theoretical considerations advanced as to basic brain functioning. Mental retardates suffer from a double handicap. The brain doesn't develop as extensively and such growth as ther is occurs over a shorter period of time. Reduced growth is compounded by the severely reduced neuro-physiological potential for stimulation of growth of one neural component by the functioning of interrelated neural components. This effect may go undetected if only simple learning, or any learning that follows previously established patterns, is studied.

The overall results led to the conclusion that the present practice of relying on M.A. for starting purposes seems clearly contraindicated. It was suggested that continued or protracted flexibility is positively associated with the degree of intelligence. Early initiation of the educational process for the mentally retarded is indicated at least as much as it is for the gifted though the curricular content would be quite different. Actually, early initiation of the educational process for the mentally retarded is probably more important than it is for those of higher intelligence because not only does growth cease earlier, but flexibility is much shorter lived.

VII. REFERENCES

1. Ades, Harlow W. "Effect of Extirpation of Parastriate Cortex on Learned Visual Discriminations in Monkeys." Journal of Neuropathology and Experimental Neurology 5: 60-65; 1946.

2. Ades, Harlow W., and Raub, David H. "Effect of Preoccipital and Temporal Decortication on Learned Visual Discrimination in Monkeys." Journal of Neurophysiology 12: 101-108; 1949.

3. Adey, W. Ross. "Hippocampal Mechanisms in Processes of Memory: Thoughts on a Model of Cerebral Organization in Learning." In: Brain Function; Volume II. RNA and Brain Function, Memory and Learning. Edited by Mary A. B. Brazier. Berkeley: University of California Press, 1964. Pp. 233-276.

4. Adey, W. R.; Dunlop, C. W.; and Hendrix, C. E. "Hippocampal Slow Waves; Distribution and Phase Relationships in the Course of Approach Learning." Archives of Neurology 3: 74-90; 1960.

5. Adey, W. R.; Kado, R. T.; and Didio , Jr. "Impedance Measurements in Brain Tissue of Animals Using Microvolt Signals." Experimental Neurology 5: 47-66; 1962.

6. Adey, W. R.; Kado, R. T.; Didio, Jr.; and Schindler, W. J. "Impedance Changes in Cerebral Tissue Accompanying a Learned Discriminative Performance in the Cat." Experimental Neurology 7: 259-281; 1963.

7. Adey, W. R., and Walter, D. O. "Application of Phase Detection and Averaging Techniques in Computer Analysis of EEG Records in the Cat." Experimental Neurology 7: 186-209; 1963.

8. Adey, W. R.; Walter, D. O.; and Hendrix, C. E. "Computer Techniques in Correlation and Spectral Analyses of Cerebral Slow Waves during Discriminative Behavior." Experimental Neurology 3: 501-524; 1961.

9. Adey, W. R.; Walter, D. O.; and Lindsley, D. F. "Subthalamic Lesions: Effects on Learned Behavior and Correlated Hippocampal and Sub-Cortical Slow-Wave Activity." Archives of Neurology 6: 194-207; 1962.

10. Aldrich, C. G., and Doll, E. A. "Problem Solving Among Idiots: the Use of Implements." Journal of Social Psychology 2: 306-336; 1931.

11. Amos, Iris E. Delayed Response Performance at Three Years of Age Among Children with Anoxic and non-anoxic Experiences at Birth. Unpublished Ph.D. Dissertation. Louisiana State University, 1959.

12. Anderson, V. Elving. "Genetics in Mental Retardation." In: Mental Retardation. Edited by Harvey A. Stevens and Rick Heber. Chicago: The University of Chicago Press, 1964. Pp. 348-394.

13. Babich, Frank R.; Jacobson, Allan L.; Bubash, Suzanne; and Jacobson, Ann. "Transfer of a Response to Naive Rats by Injection of Ribonucleic Acid Extracted from Trained Rats." Science 149: 656-657; 1965.

14. Bagshaw, M., and Pribram, K. H. "Cortical Organization in Gustation (Macaca Mulatta). Journal of Neurophysiology 16: 499-508; 1953.

15. Barnett, C. D., and Cantor, G. N. "Pursuit Rotor Performance in Mental Defectives as a Function of Distribution of Practice." Perceptual and Motor Skills 7: 191-197; 1957.

16. Barnett, C. D.; Ellis, N. R.; and Pryer, Margaret W. "Learning in Familial and Brain-Injured Defectives." American Journal of Mental Deficiency 64: 894-901; 1960.

17. Benoit, E. Paul. "Toward a New Definition of Mental Retardation." American Journal of Mental Deficiency 63: 559-565; 1958.

18. Bialer, I. "Primary and Secondary Stimulus Generalization as Related to Intelligence Level." Journal of Experimental Psychology 62: 395-402, October 1961.

19. Blumenfeld, W. "Practice with Repetition and Reward without Improvement." Journal of General Psychology 71: 313-321; 1964.

20. Bolton, J. S., and Moyes, J. M. "The Cyto-Architecture of the Cerebral Cortex of the Human Fetus of Eighteen Weeks." Brain 35(I): 1-25; 1912.

21. Box, G. E. P. "Some Theorems on Quadratic Forms Applied in the Study of Analysis of Variance Problems II. Effects of Inequality of Variance and Correlation in the two-way Classification." Annals of Mathematical Statistics 25: 484-498; 1954.

22. Brazier, Mary A. B. (Ed.) The Central Nervous System and Behavior. New York: Josiah Macy, Jr., Foundation, 1960. Pp. 359-379.

23. Brazier, Mary A. B. "The Problem of Periodicity in the Electroencephalogram: Studies in the Cat." Electroencephalography and Clinical Neurophysiology 15: 287-298; April 1963.

24. Brazier, Mary A. B. "Evoked Responses Recorded from the Depths of the Human Brain." Annals of the New York Academy of Sciences 112: 33-59; 1964.

25. Brazier, Mary A. B. (Ed.) Brain Function. Volume II. RNA and Brain Function, Memory and Learning. UCLA Forum in Medical Sciences, Number 2. Berkeley: University of California Press, 1964. 360 p.

26. Bruner, Jerome S.; Matter, Jean; and Papanek, Miriam Lewin. "Breadth of Learning as a Function of Drive Level and Mechanization." Psychological Review 62: 1-10; January 1955.

27. Bruner, Jerome S.; Wallach, Michael A.; and Galanter, Eugene H. "The Identification of Recurrent Regularity." American Journal of Psychology 72: 200-209; 1959.

28. Brutkowski, S. J.; Konorski, J.; Lawicka, W.; Stepien, I.; and Stepien, L. "The Effect of the Removal of Frontal Poles of the Cerebral Cortex on Motor Conditioned Reflexes." Acta Biologiae Experimentalis 17: 167-188; 1956.

29. Carmichael, Leonard (Ed.) Manual of Child Psychology. New York: John Wiley & Sons, 1954. 1295 p.

30. Chow, K. L. "A Retrograde Cell Degeneration Study of the Cortical Projection Field of the Pulvinar in the Monkey." Journal of Comparative Neurology 93: 313-340, 1950.

31. Chow, K. L. "Further Studies on Selective Ablation of the Associative Cortex in Relation to Visually Mediated Behavior." Journal of Comparative Physiology and Psychology 45: 109-118; 1952.

32. Chow, K. L. "Lack of Behavioral Effects following Destruction of Some Thalamic Association Nuclei in Monkey." Archives of Neurology and Psychiatry 71: 762-771; 1954.

33. Chow, K. L. "Changes of Brain Electropotentials during Visual Discrimination Learning in Monkey." Journal of Neurophysiology 24: 377-390; 1961.

34. Chow, K. L., and Hutt, P. J. "'Association Cortex' of Macaca Mulatta: Review of Recent Contributions to its Anatomy and Functions." Brain 76: 625-677; 1953.

35. Chow, K. L., and Pribram, K. H. "Cortical Projection of the Thalamic Ventrolateral Nuclear Group in Monkeys." Journal of Comparative Neurology 104: 57-75; 1956.

36. Coghill, G. E. Anatomy and the Problem of Behavior. New York: Macmillan, 1929. 113 p.

37. Conel, J. L. The Postnatal Development of the Human Cerebral Cortex. Vol. I. The Cortex of the Newborn. Cambridge: Harvard University Press, 1939. 117 p.

38. Conel, J. L. The Postnatal Development of the Human Cerebral Cortex. Vol. II. The Cortex of the One-Month Infant. Cambridge: Harvard University Press, 1941. 136 p.

39. Czehura, W. S. "The Generalization of Temporal Stimulus Patterns on the Time Continuum." Journal of Comparative Psychology 36: 79-90; 1943.

40. Davis, Gary A. "Current Status of Research and Theory in Human Problem Solving." Psychological Bulletin 66: 36-54; 1966.

41. De Crinis, M. "Die Entwicklung der Grosshirnrinde nach der Geburt in ihren Beziehungen Zur Intellektuellen Ausreifung des Kindes." Wiener Klinische Wochenschrift 45: 1163; 1932.

42. Delafresnaye, J. F.; Fessard, A.; Gerard, R. W.; and Konorski, J. Brain Mechanisms and Learning. A Symposium Organized by the Council for International Organizations of Medical Sciences. Oxford: Blackwell Scientific Publications, 1961, 702 p.

43. Denny M. Ray. "Research in Learning and Performance." In: Mental Retardation. Edited by Harvey Stevens and Rick Heber. Chicago: The University of Chicago Press, 1964. Pp. 100-142.

44. Denny-Brown, D. "Positive and Negative Aspects of Cerebral Cortical Functions" (Ross Herman Jennings Bryson Memorial Lecture) North Carolina Medical Journal 17: 295-303; July 1956.

45. De Sanctis, S. "Visual Apprehension in the Maze Behavior of Normal and Feebleminded Children." Journal of Genetic Psychology 39: 463-467; 1931.

46. Dethier, Vincent. "The Hungry Fly." Psychology Today 1: 64-72; June, 1967.

47. Deutsch, J. A. "A New Type of Behavior Theory." British Journal of Psychology 44: 304-317; 1953.

48. Deutsch, J. A. "A Theory of Insight, Reasoning, and Latent Learning." British Journal of Psychology 47: 115-125; 1956.

49. Dufort, Robert H.; Guttman, Norman; and Kimble, Gregory A. "One Trial Discrimination Reversal in the White Rat." Journal of Comparative and Physiological Psychology 47: 248-249; 1954.

50. Duncan, C. P. "Recent Research on Human Problem Solving." Psychological Bulletin 56: 397-429; 1959.

51. Eccles, J. C. The Physiology of the Synapse. Berlin: Springer-Verlag, 1964. 316 p.

52. Ellis, Norman R. "Object-Quality Discrimination Learning Sets in Mental Defectives." Journal of Comparative and Physiological Psychology 51: 79-81; 1958.

53. Ellis, Norman R. (Ed.) Handbook of Mental Deficiency: Psychological Theory and Research. New York: McGraw-Hill, 1963. 722 p.

54. Ellis, N. R.; Pryer, M. W.; Distefano, M.K. Jr.; Pryer, R. S. "Learning in Mentally Defective, Normal, and Superior Subjects." American Journal of Mental Deficiency 64: 725-739; January 1960.

55. Ellis, N. R., and Sloan, W. "Oddity Learning as a Function of Mental Age." Journal of Comparative and Physiological Psychology 52: 228-230; 1959.

56. Estes, W. K. "Component and Pattern Models with Markovian Interpretation." In: Studies in Mathematical Learning Theory. Edited by R. R. Bush and W. K. Estes. Stanford: Stanford University Press, 1959. Pp. 1-52.

57. Felsinger, J. M. "The Generalization of Extinction Effects within a Habit Pattern." Journal of Experimental Psychology 34: 477-485; 1944.

58. Fields, W. S., and Abbott, W. (Eds.) Information Storage and Neural Control. Springfield: Thomas, 1963. 411 p.

59. Flechsig, Paul. Die Localisation der Geistigen Vorgänge Insbesondere der Sinnesempfingunden des Menschen. Leipsig, 1896. 88 p.

60. Fletcher, Harold J. "Discrimination Learning by Retardates as a Function of Number of Implicit Response Trials." Psychonomic Science 4(4): 159-168; 1966.

61. Flexner, L. B.; Flexner, J. B.; and Roberts R. B. "Memory in Mice Analyzed with Antibiotics." Science 155: 1377-1383; March 17, 1967.

62. Forgus, R. H. "The Effect of Different Kinds of Form Pre-Exposure on Form Discrimination Learning." Journal of Comparative and Physiological Psychology 51: 75-78; 1958.

63. Fuller, J. L.; Rosvold, H. E.; and Pribram, K. H. "The Effect on Affective and Cognitive Behavior in the Dog of Lesions of the Pyriform-Amygdala-Hippocampal Complex." Journal of Comparative and Physiological Psychology 50: 89-96; 1957.

64. Gagné, R. M. "Problem Solving and Thinking." Annual Review of Psychology 10: 147-172; 1959.

65. Galanter, Eugene H., and Gerstenhaber, Murray. "On Thought: The Extrinsic Theory." Psychological Review 63: 218-227; 1956.

66. Gellerman, L. W. "Form Discrimination in Chimpanzee and Two-Year Old Children: I. Form (Triangularity) per se. II. Form vs. Background." Journal of Genetic Psychology 42: 3-27; 28-50; 1933.

67. Gerard, Ralph W. "Memory: Fixation in Experience. In Science and Human Affairs, Edited by Larson, Richard E. Palo Alto, California: Science and Behavior Books, Inc., 1965. 214 p.

68. Gerard, R. W., and Duyff, J. W. Information Processing in the Nervous System. Volume III. Proceedings of the International Union of Physiological Sciences. International Congress Series No. 49. New York: Excerpta Medica Foundation, 1962. 458 p.

69. Gesell, Arnold. "Maturation and the Patterning of Behavior." In: A Handbook of Child Psychology (2nd Edition). Edited by Carl Murchison. Worcester: Clark University Press, 1933. Pp. 209-235.

70. Gibson, James J., and Gibson, Eleanor J. "Perceptual Learning: Differentiation or Enrichment ?". Psychological Review 62: 32-41; 1955.

71. Glass, B. "The Establishment of Modern Genetical Theory as an Example of the Interaction of Different Models, Techniques, and Inferences." In: Scientific Change. Edited by A. C. Crombie, London: Heinemann, 1963. Pp. 521-541.

72. Green, J. D., and Maxwell, D. S. "Hippocampal Electrical Activity I. Morphological Aspects." E.E.G. Clinical Neurophysiology 13: 837-846; 1961.

73. Hamilton, G.V.N. "A Study of Trial and Error Reaction in Mammals." Journal of Animal Behavior 1: 33-66; 1911.

74. Hamilton, G.V.N. "A Study of Perseverance Reaction in Primates and Rodents." Behavior Monographs 3: No. 2. 1916. 65 p.

75. Harlow, Harry F. "Studies in Discrimination Learning by Monkeys: I. The Learning of Discrimination Series and the Reversal of Discrimination Series." Journal of General Psychology 30: 3-12; 1944.

76. Harlow, Harry F. "Studies in Discrimination Learning in Monkeys: V. Initial Performance by Experimentally Naive Monkeys on Stimulus-Object and Pattern Discriminations." Journal of General Psychology 33: 3-10; 1945.

77. Harlow, Harry F. "TheFormation of Learning Sets." Psychological Review 56: 51-65; 1949.

78. Harlow, Harry F. "Higher Functions of the Nervous System." In: Annual Review of Physiology. Edited by Victor E. Hall 15: 493-514; 1953.

79. Harlow, Harry F.; Davis, R. T.; Settlage, P. H.; and Meyer, D. R. "Analysis of Frontal and Posterior Association Syndromes in Brain Damaged Monkeys." Journal of Comparative and Physiological Psychology 45: 419-429; 1952.

80. Harlow, H. F., and Israel, R. H. "Comparative Behavior of Primates: Complicated Delayed Reaction Tests on Subnormal Humans." *Journal of Comparative Psychology* 14: 253-262; October 1932.

81. Harlow, H. F., and Woolsey, C. N. (Eds.) *Biological and Biochemical Bases of Behavior.* Madison: University of Wisconsin Press, 1958. 467 p.

82. Hayes, K. J.; Thompson, R.; and Hayes, C. "Discrimination Learning Set in Chimpanzees." *Journal of Comparative and Psysiological Psychology* 46: 99-104; 1953.

83. Heal, Laird W. "Discrimination Transfer in Normal and Mentally Retarded Children of Comparable Discrimination Ability: Partial Intradimentional and Extradimentional Shifts as a Function of Overtraining." *Dissertation Abstracts* 25(6): 3702; 1964.

84. Hebb, D. O. *Organization of Behavior: A Neuropsychological Theory.* New York: Wiley, 1955. 335 p.

85. Hebb, D. O. *A Textbook of Psychology.* Philadelphia: Saunders, 1958. 276 p.

86. Hebb, D. O. "Distinctive Features of Learning in the Higher Animal." In: *Brain Mechanisms and Learning.* Edited by J. F. Delafresnaye. Oxford: Blackwell Scientific Publications, 1961. Pp. 37-51.

87. Heber, Rick F. *A Manual on Terminology and Classification in Mental Retardation.* Pineville, La.: American Association on Mental Deficiency, 1961. 109 p.

88. Hilgard, E. R. *Theories of Learning.* New York: Appleton-Century-Crofts, 1948. 408 p.

89. Hoagland, H. "Potentialities in the Control of Behavior." In: *Man and His Future.* Edited by G. Wolstenholme. London: Churchill, 1963, pp. 299-314.

90. Hoffman, L. R.; Burke, R. J.; and Maier, N.R.F. "Does Training with Differential Reinforcement on Similar Problems Help in Solving a New Problem ?" Psychological Reports 13: 147-154; 1963.

91. House, B. J.; Orlando, R.; and Zeaman, D. "Role of Positive and Negative Cues in the Discrimination Learning of Mental Defectives." Perceptual and Motor Skills 7: 73-79; 1957.

92. House, B. J., and Zeaman, D. "Reward and Nonreward in Discrimination Learning of Imbeciles." Journal of Comparative and Physiological Psychology 51: 614-618; 1958.

93. House, B. J., and Zeaman, D. "Visual Discrimination Learning in Imbeciles." American Journal Mental Deficiency 63: 447-452; 1958.

94. House, B. J., and Zeaman, D. "Position Discrimination and Reversals in Low-Grade Retardates." Journal of Comparative and Physiological Psychology 52: 564-565; 1959.

95. House, B. J., and Zeaman, D. "A Comparison of Discrimination Learning in Normal and Mentally Defective Children." Child Development 29: 411-416; 1959.

96. Hyden, Holger. "Biochemical Changes in Glial Cells and Nerve Cells at Varying Activity." In: Biochemistry of the Central Nervous System, Vol. III. Edited by O. Hoffmann-Ostenhof and F. Brücke. London: Pergamon, 1959. Pp. 64-89.

97. Hyden, Holger. "The Neuron." In: The Cell, Vol. IV. Edited by J. Brachet and A. E. Mirsky. New York: Academic Press, 1960. Pp. 215-323.

98. Hyden, Holger. "A Molecular Basis of Neuron-Glia Interaction." In: Macromolecular Specificity and Biological Memory. Edited by F. O. Schmitt. Cambridge: MIT Press, 1962. Pp. 55-69.

99. Hyden, Holger. "Nuclear RNA Changes of Nerve Cells during a Learning Experiment in Rats." Proceedings of the National Academy of Sciences (U.S.A.) 48: 1366-73; 1962.

100. Hyden, Holger. "RNA—A Functional Characteristic of the Neuron and Its Glia." In: Brain Function, Volume II. RNA and Brain Function. Edited by Mary A. B. Brazier. UCLA Forum in Medical Sciences. Berkeley: University of California Press, 1964. Pp. 29-68.

101. Hyman, Lester M. "Symmetry, Numerosity, and Partial Identity as Stimulus Factors in Retardate Discrimination Learning." Dissertation Abstracts 26(7): 4090-4091; 1966.

102. Iwahara, Shinkuro, and Suzimura, Takeshi. "Studies on Shifts of Discrimination Learning: 1. The Number of Trials During Prior Learning." Japanese Journal of Educational Psychology 6: 42-48 (English abstract 71-72); October 1958.

103. Jacobsen, C. F. "The Effects of Extirpations on Higher Brain Processes." Physiological Review 19: 303-332; 1939.

104. Jacobsen, C. F., and Nissen, H. W. "Studies of Cerebral Functions in Primates. IV. The Effects of Frontal Lobe Lesions on the Delayed Alternation Habit in Monkeys." Journal of Comparative Psychology 23: 101-112; 1937.

105. Jensen, Kai. Conditions Influencing Insight and Problem Solving Behavior in the Mentally Retarded. Project No. S.A.E. 6437. Madison: University of Wisconsin, 1960. 158 p.

106. Johnson, B. "Practice Effects in a Target Test: A Comparison of Groups of Varying Intelligence." Psychological Record 26: 300-316; 1919.

107. Johnson, D. M. "Problem Solving and Symbolic Processes." Annual Review of Psychology 1: 297-310; 1950.

108. Kaada, B. R.; Pribram, K. H.; and Epstein, J.A. "Respiratory and Vascular Responses to Monkeys from Temporal Pole, Insula, Orbital Surface and Cingulate Gyrus: A Preliminary Report." Journal of Neurophysiology 12: 347-356; 1949.

109. Kaufman, M. E. "The Formation of a Learning Set in Institutionalized and Noninstitutionalized Mental Defectives." American Journal of Mental Deficiency 67(4): 601-605; 1963.

110. Kendler, H. H.; Glucksberg, S.; and Keston, R. "Perception and Mediation in Concept Learning." Journal of Experimental Psychology 61: 186-191; 1961.

111. Kendler, Tracy S. "Concept Formation." Annual Review of Psychology 12: 447-472; 1961.

112. Kennedy, Donald. "Introduction to Part II." In: Neural Theory and Modeling. Edited by Richard F. Reiss. Stanford: Stanford University Press, 1964. 427 p.

113. Ketterlinus, E. "Learning of Children in Adaptation to Mirror Reversals." Child Development 2: 200-223; 1931.

114. Kluver, H. "Visual Functions after Removal of the Occipital Lobes." Journal of Psychology 11: 23-45; 1941.

115. Köhler, W. The Mentality of Apes (Translated by Winter, E.). New York: Harcourt Brace, 1925. 336 p.

116. Konorski, J. "Recent Achievements in the Field of the Functional Organization of the Cerebral Cortex." Acta Physioliga Polonica 12: 611-629; July-August 1961.

117. Konorski, J. "The Physiological Approach to the Problem of Recent Memory." In: Brain Mechanisms and Learning. Edited by A. Fessard; R. W. Gerard; and J. Konorski. Springfield, Illinois: Charles C. Thomas, 1961. Pp. 115-132.

118. Konorski, J. "The Role of Central Factors in Differentiation." In: Information Processing in the Nervous System. Edited by R. W. Gerard, and J. W. Duyff. New York: Excerpta Medica Foundation, 1962. Pp. 318-329.

119. Konorski, J., and Lawicka, W. "Analysis of Errors in Prefrontal Animals on the Delayed Response Test." In: The Frontal Granular Cortex and Behavior. Edited by J. M. Warren, and K. Akert. New York: McGraw-Hill Book Company, 1964. Pp. 271-294.

120. Kruger, L. Observations on the Organization of the Sensorimotor Cerebral Cortex. Unpublished Doctor's Dissertation, Yale University, 1954.

121. Langworthy, O. R. "Medulated Tracts in the Brain Stem of a Seven-Month Human Fetus." Contributions to Embryology, Carnegie Institute, Washington 21: No. 120, 37-52; 1930.

122. Langworthy, O. R. "Development of Behavior Patterns and Myelinization of the Nervous System in the Human Fetus and Infant." Contributions to Embryology, Carnegie Institute, Washington 24: No. 139, 3-57; 1933.

123. Lashley, K. S. "Studies of Cerebral Function in Learning. V. The Retention of Motor Habits after Destruction of the So-Called Motor Areas in Primates." Archives of Neurology and Psychiatry 12: 249-276; 1924.

124. Lashley, K. S. "The Problem of Cerebral Organization in Vision." In: Biological Symposia. Edited by Jaques Cattell 7: 301-322; 1942.

125. Lashley, K. S. "The Mechanism of Vision. XVIII. Effects of Destroying the Visual "Associative Areas" of the Monkey." Genetic Psychology Monographs 37: 107-166; 1948.

126. Lashley, K. S. "In Search of the Engram." Society for Experimental Biology (Symposia) 4: 454-482; 1950.

127. Lashley, K. S. "Cerebral Organization and Behavior." Research Publications. Association for Research in Nervous & Mental Diseases 36: 1-14; 1958.

128. Lassek, A. M. "The Human Pyramidal Tract IV. A Study of the Mature, Myelinated Fibers of the Pyramid." Journal of Comparative Neurology 76: 217-225; 1942.

129. Lassek, A. M., and Evans, J. P. "The Human Pyramidal Tract XIV. A Study of the Representation of the Cortico-Spinal Components in the Spinal Cord." Journal of Comparative Neurology 84: 11-16; 1946.

130. Leary, R. W.; Harlow, H. F.; Settlage, P. H.; and Greenwood, D. D. "Performance on Double-Alternation Problems by Normal and Brain-Injured Monkeys." Journal of Comparative and Physiological Psychology 45: 576-584; 1952.

131. Levinson, Abraham. The Mentally Retarded Child. A Guide for Parents. New York: The John Day Company, 1952. 190 p.

132. Lewis, Don. Quantitative Methods in Psychology. New York: McGraw Hill, 1960. Pp. 51-136; 420-542.

133. Longnecker, E. D., and Ferson, J. "Discrimination Reversal Learning in Mongoloids." American Journal of Mental Deficiency 66: 93-99; 1961.

134. Lorente de Nó, R. "Cerebral Cortex: Architecture, Intracortical Connections, Motor Projections." In: The Physiology of the Nervous System, 2nd Edition. Edited by I. F. Fulton. London: Oxford University Press, 1943. Pp. 274-301.

135. Luchins, Abraham S. "Mechanization of Problem Solving: The Effect of Einstellung." Psychological Monographs 54: 1-95; 1942.

136. Luco, J. V. "Plasticity of Neural Function in Learning and Retention." In: Brain Function. Volume II. RNA and Brain Function. Edited by Mary A. B. Brazier. Berkeley: University of California Press, 1964. Pp. 135-159.

137. Lurin, A. R. Human Brain and Psychological Processes (Translated by Basil Haigh). New York: Harper and Row, 1966. 587 p.

138. Mac Corquodale, Kenneth, and Meehl, Paul E. "On a Distinction between Hypothetical Constructs and Intervening Variables." Psychological Review 55: 95-107; 1948.

139. Magoun, H. W. "Introduction." Brain Function. Volume II RNA and Brain Function, Memory and Learning. U.C.L.A. Forum in Medical Sciences. Edited by Mary A. B. Brazier. Berkeley: University of California Press, 1964, Pp. 1-4.

140. Maher, Brendan A. "Intelligence and Brain Damage." In: Handbook of Mental Deficiency, Psychological Theory and Research. Edited by Norman R. Ellis. New York: McGraw-Hill, 1963. Pp. 224-252

141. Mattson, D. E. "Three Kinds of Transfer in a Problem Solving Task." Journal of Educational Psychology 56: 73-80; 1965.

142. Mattson, M. L. "The Relation between the Complexity of the Habit to be Acquired and the Form of the Learning Curve in Young Children." Genetic Psychology Monographs 13: 299-398; 1933.

143. McGinnis, E. "The Acquisition and Interference of Motor Habits in Young Children." Genetic Psychology Monographs 6: 209-311; 1929.

144. McGraw, Myrtle B. Growth: A Study of Johnny and Jimmy. New York: Appleton-Century, 1935. 319 p.

145. McGraw, Myrtle B. "Neuro-muscular Development of the Human Infant as Exemplified in the Achievement of Erect Locomotion." Journal of Pediatrics 17: 750; 1940.

146. McGraw, Myrtle B. The Neuromuscular Maturation of the Human Infant. New York: Columbia University Press, 1943. 140 p.

147. McGraw, Myrtle B. Maturation of Behavior." In: Manual of Child Psychology. Edited by L. Carmichael. New York: John Wiley and Sons, Inc., 1946. Pp. 332-369.

148. McPherson, M.W. "A Survey of Experimental Studies of Learnings in Individuals Who Achieve Defective Ratings on Standardized Psychometric Measures." American Journal of Mental Deficiency 52: 232-254; 1948.

149. Mishkin, Mortimer. "Visual Discrimination Performance Following Partial Ablations of the Temporal Lobe: II. Ventral Surface vs. Hippocampus." Journal of Comparative and Physiological Psychology 47: 187-193; 1954.

150. Mishkin, Mortimer. "Perseveration of Central Sets after Frontal Lesions in Monkey." In: The Frontal Granular Cortex and Behavior. Edited by J. M. Warren and K. Akert. New York: McGraw-Hill Book Co., 1964, pp. 219-241.

151. Mishkin, M., and Hall, M. "Discrimination along a Size Continuum Following Ablation of the Inferior Temporal Convexity in Monkeys." Journal of Comparative and Physiological Psychology 48: 97-101; 1955.

152. Mishkin, M., and Pribram, K. H. "Visual Discrimination Performance Following Partial Ablations of the Temporal Lobe: I. Ventral vs. Lateral." Journal of Comparative and Physiological Psychology 47: 14-20; 154.

153. Mishkin, M., and Pribram, K. H. "Analysis of the Effects of Frontal Lesions in Monkey: I. Variations of Delayed Alternation." Journal of Comparative and Physiological Psychology 48: 492-495; 1955.

154. Mishkin, M., and Pribram, K. H. "Analysis of the Effects of Frontal Lesions in Monkey: II. Variations of Delayed Response." Journal of Comparative and Physiological Psychology 49: 36-40; 1956.

155. Morin, G.; Gastant, H.; and Naquet, R. "Le Cycle D'excitabilité des Systèmes Afférents Corticaux Chez le Chat." Revue Neurologique 84: 605-606; June 1951.

156. Morison, Robert S. "Gradualness, Gradualness, Gradualness" (I. P. Pavlov). The American Psychologist 15: 187-197; 1960.

157. Neff, W. D. "Sensory Discrimination." In: Handbook of Physiology—Neurophysiology III. Edited by J. Field, H. W. Magoun, and V. E. Hall. Washington, D.C.: American Physiological Society; Baltimore: Williams and Wilkins, 1960. Pp. 1447-1470.

158. Nirenberg, M. W. "The Dependence of Cell-Free Protein Synthesis in E. Coli upon Naturally Occurring or Synthetic Template RNA." In: Biological Structure and Function at the Molecular Level. Edited by A. Engelhardt. Moscow: Vol. I of Proceedings of Fifth International Congress of Biochemistry, 1961. Oxford: Pergamon Press, 1963. Pp. 184-195.

159. Nirenberg, W. "Nucleic Acids in Relation to the Coding of Genetic Information." In: Brain Function, Volume II, RNA

and Brain Function, Memory and Learning. Edited by Mary A. B. Brazier. UCLA Forum in Medical Sciences. Berkeley: University of California Press, 1964. Pp. 5-28.

160. Nirenberg, M. W., and Jones, Jr., O. W. "The Current Status of the RNA Code." In: Informational Macromolecules. Edited by H. J. Vogel, V. Bryson, and J. O. Lanpen. New York: Academic Press, 1963. Pp. 451-465.

161. Ochs, Sidney. Elements of Neurophysiology. New York: John Wiley and Sons, 1965. 621 p.

162. Page, E. B. "Ordered Hypotheses for Multiple Treatments: A Significance Test for Linear Ranks." Journal of American Statistical Association 58: 216-230; 1963.

163. Palay, S. L. "The Structural Basis for Neural Action." In: Brain Function, Volume II, RNA and Brain Function, Memory and Learning. Edited by Mary A. B. Brazier. Berkeley: University of California Press, 1964. Pp. 69-108.

164. Pascal, G. R.; Stolurow, L. M.; Zabarenko, R. N.; and Chambers, C. S. "The Delayed Reaction in Mental Defectives." American Journal of Mental Deficiency 56: 152-160; 1951.

165. Pascal, G. R., and Zax, M. "Double Alternation Performance as a measure of Educability in Cerebral Palsied Children." American Journal of Mental Deficiency. 59: 658-665; 1955.

166. Pavlov, I. P. Lectures on Conditioned Reflexes. (Translated by W. H. Gantt.) New York: International Pub., 1928. 414 p.

167. Penfield, W. "Functional Localization in Temporal and Deep Sylvian Areas." Research Publications. Association for Research in Nervous and Mental Diseases 36: 210-226; 1958.

168. Piaget, Jean, and Inhelder, Barbel. The Child's Conception of Space. London: Routledge & Kegan Paul, 1956. 490 p.

169. Piaget, Jean; Inhelder, Barbel; and Szeminska, Alina. The Child's Conception of Geometry. New York: Basic Books, 1960. 411 p.

170. Plenderlith, Mavis. "Discrimination Learning and Discrimination Reversal Learning in Normal and Feebleminded Children." Journal of Genetic Psychology 88: 107-112; 1956.

171. Poliak, Stephen. Main Afferent Fiber Systems of the Cerebral Cortex in Primates. Berkeley: University of California Press, 1932. 370 p.

172. Pribram, Karl H. "Toward a Science of Neuropsychology (Method and Data)." In: Current Trends in Psychology. Edited by R. A. Patton. Pittsburgh: University of Pittsburgh Press, 1955. Pp. 115-142.

173. Pribram, Karl H. "Neucortical Function in Behavior." In: Biological and Biochemical Bases of Behavior. Edited by Harry F. Harlow and Clinton N. Woolsey. Madison: The University of Wisconsin Press, 1958. Pp. 151-172.

174. Pribram, Karl H. "On the Neurology of Thinking." Behavioral Science 4: 265-287; October 1959.

175. Pribram, Karl H. "A Review of Theory in Physiological Psychology." Annual Review of Psychology 11: 1-40, 1960.

176. Pribram, Karl H. "The Intrinsic Systems of the Forebrain." In: Handbook of Physiology. Section I, Volume II: Neurophysiology. Edited by John Field. Washington, D. C.: American Physiological Society. 1323-1344, 1960.

177. Pribram, K. H.; Chow, K. L.; and Semmes, J. "Limit and Organization of the Cortical Projection from the Medical Thalamic Nucleus in Monkey." Journal of Comparative Neurology 98: 433-448; 1953.

178. Pribram, K. H.; Konrad, K.; and Gainsburg, D. "Frontal Lesions and Behavioral Instability." Journal of Comparative and Physiological Psychology 62: 123-124; 1966.

179. Pribram, K. H., and Kruger, L. "Functions of the 'Olfactory Brain.'" Annals New York Academy Science 58: 109-138; 1954.

180. Pribram, K. H.; Lim, H.; and Poppen, R.; et al. "Limbic Lesions and the Temporal Structure of Redundancy." *Journal of Comparative and Physiological Psychology* 61: 368-373; 1966.

181. Pribram, K. H., and MacLean, P. D. "Neuronographic Analysis of Medical and Basal Cerebral Cortex. II. Monkey." *Journal of Neurophysiology* 16: 324-340; 1953.

182. Pribram, K. H., and Mishkin, M. "Simultaneous and Successive Visual Discrimination by Monkeys with Inferotemporal Lesions." *Journal of Comparative and Physiological Psychology,* 48: 198-202; 1955.

183. Pribram, K. H., and Mishkin, M. "Analysis of the Effects of Frontal Lesions in Monkey: III. Object Alternation." *Journal of Comparative and Physiological Psychology* 49: 41-45; 1956.

184. Pribram, K. H.; Mishkin, M.; Rosvold, H. E.; and Kaplan, S. J. "Effects on Delayed-Response Performance of Lesions of Dorsolateral and Ventromedial Frontal Cortex of Baboons." *Journal of Comparative and Physiological Psychology* 45: 565-575; 1952.

185. Pribram, K. H.; Rosner, B. S.; and Rosenblith, W. A. "Electrical Responses to Acoustic Clicks in Monkey: Extent of Neocortex Activated." *Journal of Neurology and Physiology."* 17: 336-344, July 1954.

186. Ray, W. S. "Complex Tasks for Use in Human Problem Solving Research." *Psychological Bulletin* 52: 134-149; 1955.

187. Reynolds, W. F., and Stacey, C. L. "A Comparison of Normals and Subnormals in Mirror Drawing." *Journal of Genetic Psychology* 87: 301-308; 1955.

188. Rich, A. "On the Problems of Evolution and Biochemical Information Transfer." In: *Horizons in Biochemistry*. Edited by M. Kasha and B. Pullman. New York: Academic Press, 1962. Pp. 103-126.

189. Riopelle, A. J.; Alper, R. G.; Strong, P. N.; and Ades, H.W. *Journal of Comparative and Physiological Psychology* 46: 145-149; 1953.

190. Rose, J. E., and Woolsey, C. N. "Organization of the Mammalian Thalamus and Its Relationships to the Cerebral Cortex." *Electroencephalography and Clinical Neurophysiology* 1: 391-404; 1949.

191. Rosenberg, Sheldon. "Problem-Solving and Conceptual Behavior." In: *Handbook of Mental Deficiency, Psychological Theory and Research.* Edited by Norman R. Ellis. New York: McGraw-Hill, 1963. Pp. 439-462.

192. Rosenzweig, M. R.; Krech, D.; and Bennett, E. L. "A Search for Relations between Brain Chemistry and Behavior." *Psychological Bulletin* 57: 476-492; 1960.

193. Rosvold, H. E. "Physiological Psychology." *Annual Review of Psychology* 10: 415-454; 1959.

194. Ruch, T. C. "Motor Systems." In: *Handbook of Experimental Psychology.* Edited by S. S. Stevens. New York: John Wiley and Sons, 1951. Pp. 154-208.

195. Russell, Roger W. (Ed.) *Frontiers in Physiological Psychology.* New York: Academic Press, 1966. 277 p.

196. Scheffé, Henry. *The Analysis of Variance.* New York: John Wiley and Sons, Inc., 1959. 477 p.

197. Schlosberg, H., and Solomon, R. L. "Latency of Response in a Choice Discrimination." *Journal of Experimental Psychology* 33: 22-39; 1943.

198. Schmitt, F. O. (Ed.) *Macromolecular Specificity and Biological Memory.* Cambridge: MIT Press, 1962. 119 p.

199. Settlage, P.; Butler, R.; and Odoi, H. "Perseverative Interference in Monkeys Following Bilateral Removal of the Prefrontal Areas." *Journal of General Psychology* 54: 255-262; 1956.

200. Skalet, M. "The Significance of Delayed Reactions in Young Children." Comparative Psychology Monographs 7: 1-82; 1931.

201. Skinner, B. F. The Behavior of Organisms. New York: Appleton, 1938. 457 p.

202. Skinner, B. F. Science and Human Behavior. New York: Macmillan, 1953. 461 p.

203. Sloan, W., and Berg, I. "A Comparison of Two Types of Learning in Mental Defectives." American Journal of Mental Deficiency 61: 556-566; 1957.

204. Spence, Kenneth W. "Theoretical Interpretations of Learning." In: Handbook of Experimental Psychology. Edited by S. S. Stevens. New York: John Wiley and Sons, 1951. Pp. 690-729.

205. Stamm, J. S. "Electrical Stimulation of Frontal Cortex in Monkeys during Learning of an Alternation Task." Journal of Neurophysiology 24: 414-426; 1961.

206. Stanley, Walter C., and Jaynes, Julian. "The Function of the Cerebral Cortex." Psychological Review 56: 18-32, 1949.

207. Stellar, Eliot, and Sprague, James M. Progress in Physiological Psychology Volume I. New York: Academic Press, 1966. 299 p.

208. Stevens, Harvey A. "Overview." In: Mental Retardation. Edited by Harvey A. Stevens and Rick Heber, Chicago: The University of Chicago Press, 1964. Pp. 1-15.

209. Stevens, Harvey A., and Heber, Rick. Mental Retardation. A Review of Research. Chicago: The University of Chicago Press, 1964. 502 p.

210. Stevenson, Harold W. "Learning of Complex Problems by Normal and Retarded S's." American Journal of Mental Deficiency 64: 1021-1026; 1960.

211. Stevenson, Harold W. "Discrimination Learning." In: Handbook of Mental Deficiency. Edited by Norman R. Ellis. New York: McGraw-Hill, 1963. Pp. 424-438.

212. Stevenson, H. W., and Zigler, E. T. "Discrimination Learning and Rigidity in Normal and Feeble Minded Individuals." Journal of Personality 25: 699-711; 1957.

213. Stolurow, L. M., and Pascal, G. R. "Double Alternation Behavior in Mental Defectives." American Psychologist 5: 273-274; 1950.

214. Sugimura, Takeshi,and Iwahara, Shinkuro. "Studies on Shifts of Discrimination Learning: II. Effect of a Wide Range of Trials in Prior Learning." Japanese Journal of Educational Psychology 7: 12-17 (English abstract 61); December, 1959.

215. Tilney, F. "The Structure and Development of the Brain." Unpublished lecture, New York. 1937. In: McGraw, Myrtle B. Chapter 7 in Manual of Child Psychology. Edited by Leonard Carmichael. New York: John Wiley and Sons, 1946. Pp. 332-369.

216. Tilney, F., and Casamajor, L. "Myelinogeny as Applied to the Study of Behavior." Archives of Neurology and Psychiatry 12: 1-66; 1924.

217. Tolman, E. C. Purposive Behavior in Animals and Men. New York: Appleton, 1932. 463 p.

218. Von Neumann, J., and Morgenstern, O. Theory of Games and Economic Behavior. Princeton: Princeton University Press, 1953. 641 p.

219. Wade, M. "Behavioral Effects of Prefrontal Lobectomy, Lobotomy, and Circumsection in Monkey (Macaca Mulatta)." Journal of Comparative Neurology 96: 179-207; 1952.

220. Waisman, Harry A., and Gerritsen, Theo. "Biochemical and Clinical Correlations." In: Mental Retardation. Edited by Harvey A. Stevens and Rick Heber. Chicago: The University of Chicago Press, 1964. Pp. 307-347.

221. Walker, A. E., and Weaver, T. A. "Ocular Movements from the Occipital Lobe in the Monkey." Journal of Neurophysiology 3: 353-357; 1940.

222. Wall, P. D. "Presynaptic Control of Impulses at the First Central Synapse in the Cutaneous Pathway." Progress in Brain Research 12: 92-118; 1964.

223. Warren, J. M., and Akert, K. (Editors) The Frontal Granular Cortex and Behavior. New York: McGraw-Hill Book Company, 1964. Pp. 126-148 and 353-371.

224. Weinstein, B. "Matching from Sample by Rhesus Monkeys and by Children." Journal of Comparative Psychology 31: 195-213; 1941.

225. Weiskrantz, L. Behavioral Changes Associated with Ablation of the Amygdaloid Complex. Doctoral Thesis. Boston: Harvard University, 1953.

226. Whitfield, I. C. "The Electrical Responses of the Unanaesthetized Auditory Cortex in the Intact Cat." Electroencephalography and Clinical Neurophysiology 9: 35-42; 1957.

227. Whitlock, D. G., and Nauta, W. J. H. "Subcortical Projections from the Temporal Neocortex in Macaca Mulatta." Journal of Comparative Neurology 106: 185-212; 1956.

228. Wilson, F. T. "Errors, Difficulty, Resourcefulness, and Speed in the Learning of Bright and Dull Children." Journal of Educational Psychology 22: 229-240; 1931.

229. Wischner, George J., and O'Donnell, James P. "Concurrent Learning-Set Formation in Normal and Retarded Children." Journal of Comparative and Physiological Psychology 55(4): 524-527; 1962.

230. Wolinsky, G. F. "Piaget and the Psychology of Thought: Some Implications for Teaching the Retarded." American Journal of Mental Deficiency 67: 250-256; 1962.

231. Woodrow, H. "Practice and Transference in Normal and Feebleminded Children." Journal of Educational Psychology 8: 85-96; 151-65; 1917.

232. Young, J. Z. "The Organization of a Memory System." Croonian Lecture of the Royal Society, delivered on May 6, 1965. Proceedings of the Royal Society, B 163: 285-320; 1965.

233. Young, J. Z. The Memory System of the Brain. Berkeley: University of California Press, 1966. 128 p.

234. Zigler, Edward. "Familial Mental Retardation: A Continuing Dilema." Science 155: 292-298; January 20, 1967.

APPENDIX Ia

CRITERION GROUP

No. & Subject Identification	CA	MA	Analysis of Errors			
			EXP. Dif.	RDC-1 - Int.	EXP. Dif.	SDS-2 - Int.
101.AA. M	9-8	5-6	45	28	22	20
102.AB. F	13-1	5-5	46	21	27	23
103.AC. M	14-6	4-1	55	19	54	21
104.AE. M	14-6	5-6	37	27	31	20
105.AF. F	10-6	6-3	51	25	14	6
106.AG. M	12-3	5-10	47	30	19	10
107.AH. F	11-9	4-5	27	40	40	34
108.AI. M	11-1	9-1	13	7	1	8
109.AM. M	11-6	6-3	52	24	7	13
110.AO. M	15-5	5-4	48	27	24	20
111.AT. F	14-6	6-0	36	30	18	19
112.BA. F	14-4	4-8	66	24	31	14
113.BH. M	14-2	6-4	36	19	12	27
114.BL. M	14-6	4-4	40	31	53	19
115.BO. F	13-7	3-11	58	23	39	32
M	13-0	5-6	43.8	25.0	26.1	19.1
SD			13.1	7.3	15.6	8.1

APPENDIX Ib

NON-CRITERION GROUP

No. & Subject Identification	CA	MA	Analysis of Errors			
			EXP. Dif.	RDC-1 - Int.	EXP. Dif.	SDS-2 - Int.
116. AJ. M	11-6	4-11	60	26	26	16
117. AK. M	13-10	4-4	41	30	45	27
118. AL. M	16-5	4-5	43	29	37	31
119. AN. M	13-8	5-6	70	21	9	14
120. AP. M	16-5	4-10	53	29	25	24
121. AQ. M	9-3	4-5	56	27	31	26
122. AR. M	15-1	5-1	43	31	23	27
123. AW. F	11-4	5-0	71	20	14	22
124. AX. M	14-5	5-1	45	29	29	22
125. AY. M	15-10	4-0	41	31	50	29
126. AZ. M	15-5	4-7	44	30	32	31
127. BG. F	16-4	5-4	54	27	20	18
128. BM. M	10-0	5-0	59	24	22	21
129. BN. M	13-2	6-5	47	28	9	8
130. BP. F	13-4	5-1	35	34	25	31
M	13-9	4-11	50.8	27.7	26.5	23.1
			10.8	3.8	11.7	6.8

APPENDIX II

ALTERNATE PROGRAMS FOR CHOICE ARRAY
Experiment RDC-1

Day One		Position	
Trial No.	L	M	R
1	270°	135°	*
2	135°	*	270°
3	045°	*	315°
4	045°	*	225°
5	*	045°	225°
6	045°	*	135°
7	*	135°	270°
8	225°	270°	*
9	090°	*	045°
10	*	315°	090°

Day Two		Position	
Trial No.	L	M	R
1	*	090°	270°
2	090°	225°	*
3	315°	045°	*
4	315°	*	090°
5	090°	315°	*
6	*	135°	045°
7	315°	*	090°
8	315°	225°	*
9	*	270°	045°
10	045°	225°	*

* = Location of correct choice.

APPENDIX III

ALTERNATE PROGRAMS FOR CHOICE ARRAY
Experiment SDS-2

Day One Trial No.	Position L	 M	 R
1	#3D+	#5D-	*
2	#5D-	*	#3D+
3	#3D+	*	#5D-
4	#5D-	*	#3D+
5	*	#3D+	#5D-
6	#5D-	*	#3D+
7	*	#3D+	#5D-
8	#3D+	#5D-	*
9	#5D-	*	#3D+
10	*	#3D+	#5D-

Day Two Trial No.	Position L	 M	 R
1	*	#5D-	#3D+
2	#5D-	#3D+	*
3	#3D+	#5D-	*
4	#5D-	*	#3D+
5	#3D+	#5D-	*
6	*	#3D+	#5D-
7	#5D-	*	#3D+
8	#3D+	#5D-	*
9	*	#3D+	#5D-
10	#3D+	#5D-	*

* = Location of correct choice.

#3 & #5 = Distractor ring number from a five-ring assembly used for the first variation of the problem.

D+ & D- = Generalized designation of the distractors for additional variations of the problem, i.e., considering only the two distractors in any given array, + indicates the larger, and - indicates the smaller, relevant size of the two without regard to the correct choice.

APPENDIX IV

SUCCESSIVE VARIATIONS IN SIZE DISCRIMINATION
Experiment SDS-2

Variations (SDS-2)	Assembly Problem**	CR*	Ring No. D-	D+
1	I	1	5	3
2	I	5	3	1
3	II	1	5	3
4	II	5	3	1
5	III	1	5	3
6	III	5	3	1
7	IV	1	5	3
8	IV	5	3	1
9	V	1	5	3
10	V	5	3	1
11	VI	1	5	3
12	VI	5	3	1
13	I	3	4	2
14	I	4	5	3
15	I	2	3	1
16	II	3	4	2
17	II	4	5	3

* = Ring Number of correct response.

** = Problem borrowed from previous study (Jensen, 1960).

APPENDIX V

SAMPLE SCORING PROTOCOL
(Subj. 120.AP.M - Stage D - Day 10)

Trial No.	Experiment RDC-1	SDS-2
91	L - L	L - L
92	π L - R	π L - R
93	π M - R	R - R
94	M - M	M - M
95	π L - R	π M - R
96	π R - L	L - L
97	π L - M	M - M
98	π M - R	R - R
99	L - L	L - L
100	π M - R	R - R*

π = A wrong choice.

* = The criterion was reached at this point on variation #2.

APPENDIX VIa

INITIAL ERROR FREQUENCIES BY POSITION
Criterion Group - Exp. RDC-1

Subject Identification	Position			$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	L	M	R		
101.AA.M.	14	13	6	33	363.00
102.AB.F	46	0	0	46	705.33
103.AC.M	17	17	11	45	675.00
104.AE.M	1	0	39	40	533.33
105.AF.F	15	10	15	40	533.33
106.AG.M	16	11	11	38	481.33
107.AH.F	3	33	1	38	481.33
108.AI.M	6	5	4	15	75.00
109.AM.M	15	16	12	43	616.33
110.AO.M	5	15	18	38	481.33
111.AT.F	6	13	18	37	456.33
112.BA.F	23	18	8	49	800.33
113.BH.M	9	10	18	37	456.33
114.BL.M	13	15	9	37	456.33
115.BO.F	12	17	13	42	588.00
$\Sigma_j Y_{kj}$	201	193	183	577	7702.63
M_k	13.4	12.9	12.2		
$\Sigma_j Y_{kj}^2$	4337	3381	3471		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	2693.4	2483.3	2232.6		

APPENDIX VIb

INITIAL ERROR FREQUENCIES BY POSITION
Non-Criterion Group - Exp. RDC-1

Subject Identification	Position			$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	L	M	R		
116.AJ.M	13	12	19	44	645.33
117.AK.M	14	12	11	37	456.33
118.AL.M	15	18	10	43	616.33
119.AN.M	22	13	13	48	768.00
120.AP.M	4	20	15	39	507.00
121.AQ.M	19	12	11	42	588.00
122.AR.M	4	25	9	38	481.33
123.AW.F	9	16	19	44	645.33
124.AX.M	5	28	7	40	533.33
125.AY.M	24	4	6	34	385.33
126.AZ.M	7	16	12	35	408.33
127.BG.F	13	13	10	36	432.00
128.BM.M	18	13	11	42	588.00
129.BN.M	12	10	15	37	456.33
130.BP.F	15	15	4	34	385.33
$\Sigma_j Y_{kj}$	194	227	172	593	7896.30
M_k	12.9	15.1	11.5		
$\Sigma_j Y_{kj}^2$	3060	3925	2230		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	2509.1	3435.3	1972.3		

APPENDIX VIc

FINAL ERROR FREQUENCIES BY POSITION
Criterion Group - Exp. RDC-1

Subject		Position			
Identification	L	M	R	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
101.AA.M	17	11	12	40	533.33
102.AB.F	18	0	3	21	147.00
103.AC.M	12	8	8	28	261.33
104.AE.M	0	0	25	25	208.33
105.AF.F	18	4	14	36	432.00
106.AG.M	6	20	13	39	507.00
107.AH.F	11	8	11	30	300.00
108.AI.M	2	4	0	6	12.00
109.AM.M	15	12	7	34	385.33
110.AO.M	6	14	17	37	456.33
111.AT.F	5	20	4	29	280.33
112.BA.F	28	9	4	41	560.33
113.BH.M	1	0	2	3	3.00
114.BL.M	18	12	4	34	385.33
115.BO.F	14	16	9	39	507.00
$\Sigma_j Y_{kj}$	171	138	133	442	4978.64
M_k	11.4	9.2	8.9		
$\Sigma_j Y_{kj}^2$	2833	1902	1799		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	1949.4	1269.6	1179.3		

APPENDIX VId

FINAL ERROR FREQUENCIES BY POSITION
Non-Criterion Group - Exp. RDC-1

Subject Identification		Position		$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	L	M	R		
116.AJ.M	14	12	17	43	616.33
117.AK.M	7	17	10	34	385.33
118.AL.M	7	17	5	29	280.33
119.AN.M	14	16	13	43	616.33
120.AP.M	15	19	9	43	616.33
121.AQ.M	15	17	9	41	560.33
122.AR.M	2	22	12	36	432.00
123.AW.F	5	26	18	49	800.33
124.AX.M	10	15	9	34	385.33
125.AY.M	0	1	37	38	481.33
126.AX.M	17	9	12	38	481.33
127.BG.F	13	23	9	45	675.00
128.BM.M	10	19	11	40	533.33
129.BN.M	18	9	11	38	481.33
130.BP.F	3	14	18	35	408.33
$\Sigma_j Y_{kj}$	150	236	200	586	7753.29
M_k	10.0	15.7	13.3		
$\Sigma_j Y_{kj}^2$	1960	4262	3454		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	1500.0	3713.1	2666.7		

APPENDIX VIIa

INITIAL DEVIATIONS OF RESPONSE FREQUENCIES BY POSITION FROM PROGRAMMED FREQUENCIES OF CORRECT CHOICE LOCATION

Criterion Group - Exp. RDC-1

Subject Identification	Position L	Position M	Position R	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
101.AA.M	+ 8	- 3	- 5	16	85.33
102.AB.F	+46	-25	-21	92	2821.33
103.AC.M	+ 6	+ 3	- 9	18	108.00
104.AE.M	-14	-25	+39	78	2028.00
105.AF.F	+ 2	- 3	+ 1	6	12.00
106.AG.[illegible]	+ 8	- 5	- 3	16	85.33
107.AH.F	-12	+33	-21	66	1452.00
108.AI.M	+ 2	0	- 2	4	5.33
109.AM.M	0	+ 1	- 1	2	1.33
110.AO.M	-11	- 2	+13	26	225.33
111.AT.F	-12	+ 2	+10	24	192.00
112.BA.F	+10	+ 2	-12	24	192.00
113.BH.M	- 6	0	+ 6	12	48.00
114.BL.M	+ 3	+ 3	- 6	12	48.00
115.BO.F	0	+ 3	- 3	6	12.00
$\Sigma_j Y_{kj}$	140	110	152	402	7315.98
M_k	9.3	7.3	10.1		
$\Sigma_j Y_{kj}^2$	3038	2422	3018		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	1306.7	806.7	1540.3		

APPENDIX VIIb

FINAL DEVIATIONS OF RESPONSE FREQUENCIES BY POSITION FROM PROGRAMMED FREQUENCIES OF CORRECT CHOICE LOCATION

Criterion Group - Exp. RDC-1

Subject Identification	Position				
	L	M	R	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
101.AA.M	+ 2	+ 2	- 4	8	21.33
102.AB.F	+15	0	-15	30	300.00
103.AC.M	+ 2	+ 4	- 6	12	48.00
104.AE.M	-25	0	+25	50	833.33
105.AF.F	+ 2	+ 1	- 3	6	12.00
106.AG.M	-10	+20	-10	40	533.33
107.AH.F	- 2	+ 8	- 6	16	85.33
108.AI.M	+ 2	+ 4	- 6	12	48.00
109.AM.M	+ 5	+ 1	- 6	12	48.00
110.AD.M	- 9	0	+ 9	18	108.00
111.AT.F	- 9	+16	- 7	32	341.33
112.BA.F	+23	- 6	-17	46	705.33
113.BH.M	- 1	0	+ 1	2	1.33
114.BL.M	+11	+ 1	-12	24	192.00
115.BO.F	0	+ 6	- 6	12	48.00
$\Sigma_j Y_{kj}$	118	69	133	320	3325.31
M_k	7.9	4.6	8.9		
$\Sigma_j Y_{kj}^2$	1808	831	1719		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	928.3	317.4	1179.3		

APPENDIX VIIc

INITIAL DEVIATIONS OF RESPONSE FREQUENCIES BY POSITION FROM PROGRAMMED FREQUENCIES OF CORRECT CHOICE LOCATION
Non-Criterion Group - Exp. RDC-1

Subject Identification	Position L	Position M	Position R	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
116.AJ.M	+ 6	- 8	+ 2	16	85.33
117.AK.M	+ 2	- 3	+ 1	6	12.00
118.AL.M	+ 3	- 1	- 2	6	12.00
119.AN.M	+ 9	- 6	- 3	18	108.00
120.AP.M	-10	+ 8	+ 2	20	133.33
121.AQ.M	+ 7	- 6	- 1	14	65.33
122.AR.M	-13	+18	- 5	36	432.00
123.AW.F	- 8	+ 6	+ 2	16	85.33
124.AX.M	-11	+21	-10	42	588.33
125.AY.M	+20	-12	- 8	40	533.33
126.AZ.M	- 7	+ 7	0	14	65.33
127.BG.F	+ 2	+ 3	- 5	10	33.33
128.BM.M	+ 5	0	- 5	10	33.33
129.BN.M	- 1	- 4	+ 5	10	33.33
130.BP.F	+ 7	+ 5	-12	24	192.00
$\Sigma_j Y_{kj}$	111	108	63	282	2412.15
M_k	7.4	7.2	4.2		
$\Sigma_j Y_{kj}^2$	1161	1254	435		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	821.4	777.6	264.6		

APPENDIX VIId

FINAL DEVIATIONS OF RESPONSE FREQUENCIES BY POSITION FROM PROGRAMMED FREQUENCIES OF CORRECT CHOICE LOCATION
Non-Criterion Group - Exp. RDC-1

Subject Identification		Position			
	L	M	R	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
116.AJ.M	- 5	+ 7	- 2	14	65.33
117.AK.M	- 9	+17	- 8	34	385.33
118.AL.M	- 4	+14	-10	28	261.33
119.AN.M	+ 1	+ 1	- 2	4	5.33
120.AP.M	+ 2	+ 6	- 8	16	85.33
121.AQ.M	+ 1	+ 5	- 6	12	48.00
122.AR.M	-15	+16	- 1	32	341.33
123.AW.F	-13	+12	+ 1	26	225.33
124.AX.M	- 2	+ 5	- 3	10	33.33
125.AY.M	-18	-18	+36	72	1728.00
126.AZ.M	+ 8	- 6	- 2	16	85.33
127.BG.F	+ 1	+ 8	- 9	18	108.00
128.BM.M	0	+ 5	- 5	10	33.33
129.BN.M	+ 8	- 1	- 7	16	85.33
130.BP.F	-12	+ 4	+ 8	24	192.00
$\Sigma_j Y_{kj}$	99	125	108	332	3682.85
M_k	6.6	8.3	7.2		
$\Sigma_j Y_{kj}^2$	1123	1487	1802		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	653.4	1041.7	777.6		

APPENDIX VIIIa

ANALYSIS OF ERRORS IN DIFFERENTIATION
Criterion Group - Exp. RDC-1

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
101.AA.M	15	4	13	13	45	506.25
102.AB.F	18	16	12	0	46	529.00
103.AC.M	17	18	6	14	55	756.25
104.AE.M	15	10	6	6	37	342.25
105.AF.F	16	13	7	15	51	650.25
106.AG.M	11	15	12	9	47	552.25
107.AH.F	0	15	7	5	27	182.25
108.AI.M	4	4	2	3	13	42.25
109.AM.M	14	17	12	9	52	676.00
110.AO.M	14	13	11	10	48	576.00
111.AT.F	12	10	4	10	36	324.00
112.BA.F	20	22	14	10	66	1089.00
113.BH.M	12	12	0	12	36	324.00
114.BL.M	8	14	10	8	40	400.00
115.BO.F	15	15	11	17	58	841.00
$\Sigma_j Y_{kj}$	191	198	127	141	657	7790.75
M_k	12.7	13.2	8.5	9.4		
$\Sigma_j Y_{kj}^2$	2825	2938	1329	1619		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	2432.1	2613.6	1075.3	1325.4		

APPENDIX VIIIb

ANALYSIS OF ERRORS IN DIFFERENTIATION
Non-Criterion Group - Exp. RDC-1

Subject	Trial Blocks (Stages) A	B	C	D	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
116.AJ.M	20	11	10	19	60	900.00
117.AK.M	9	16	9	7	41	420.25
118.AL.M	21	14	5	3	43	462.25
119.AN.M	22	17	17	14	70	1225.00
120.AP.M	11	12	14	16	53	701.50
121.AQ.M	11	18	16	11	56	784.00
122.AR.M	8	15	9	11	43	462.25
123.AW.F	15	20	15	21	71	1260.25
124.AX.M	11	13	10	11	45	506.25
125.AY.M	13	6	12	10	41	420.25
126.AZ.M	12	8	13	11	44	484.00
127.BG.F	9	12	20	13	54	729.00
128.BM.M	18	14	14	13	59	870.25
129.BN.M	12	12	14	9	47	552.25
130.BP.F	11	7	13	4	35	306.25
$\Sigma_j Y_{kj}$	203	195	191	173	762	10084.50
M_k	13.5	13.0	12.7	11.5		
$\Sigma_j Y_{kj}^2$	3041	2677	2627	2331		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	2747.3	2535.0	2432.1	1995.3		

APPENDIX VIIIc

ANALYSIS OF ERRORS OF INTENTION
Criterion Group - Exp. RDC-1

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
101.AA.M	7	7	7	7	28	196.00
102.AB.F	6	6	7	2	21	110.25
103.AC.M	6	5	6	2	19	90.25
104.AE.M	6	8	6	7	27	182.25
105.AF.F	4	7	8	6	25	156.25
106.AG.M	6	6	8	10	30	225.00
107.AH.F	15	7	10	8	40	400.00
108.AI.M	1	5	1	0	7	12.25
109.AM.M	6	5	6	7	24	144.00
110.AO.M	6	5	8	8	27	182.25
111.AT.F	7	8	10	5	30	225.00
112.BA.F	4	3	7	10	24	144.00
113.BH.M	6	7	3	3	19	90.25
114.BL.M	9	6	8	8	31	240.25
115.BO.F	6	6	6	5	23	132.25
$\Sigma_j Y_{kj}$	95	91	101	88	375	2520.25
M_k	6.3	6.1	6.7	5.9		
$\Sigma_j Y_{kj}^2$	725	577	757	642		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	601.7	552.1	680.1	516.3		

APPENDIX VIIId

ANALYSIS OF ERRORS IN INTENTION
Non-Criterion Group - Exp. RDC-1

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
116.AJ.M	4	9	8	5	26	169.00
117.AK.M	6	6	9	9	30	225.00
118.AL.M	3	5	11	10	29	210.25
119.AN.M	3	6	6	6	21	110.25
120.AP.M	8	8	6	7	29	210.25
121.AQ.M	8	5	6	8	27	182.25
122.AR.M	8	7	8	8	31	240.25
123.AW.F	5	4	7	4	20	100.00
124.AX.M	9	7	8	5	29	210.25
125.AY.M	6	9	8	8	31	240.25
126.AZ.M	7	9	7	7	30	225.00
127.BG.F	8	7	5	7	27	182.25
128.BM.M	4	6	7	7	24	144.00
129.BN.M	6	7	6	9	28	196.00
130.BP.F	8	8	8	10	34	289.00
$\Sigma_j Y_{kj}$	93	103	110	110	416	2934.00
M_k	6.2	6.9	7.3	7.3		
$\Sigma_j Y_{kj}^2$	633	741	838	852		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	576.6	707.3	806.7	806.7		

APPENDIX IXa

ANALYSIS OF ERRORS IN DIFFERENTIATION
Criterion Group – Exp. SDS-2

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
101.AA.M	14	6	2	0	22	121.00
102.AB.F	10	12	5	0	27	182.25
103.AC.M	10	7	24	13	54	729.00
104.AE.M	12	13	5	0	30	225.00
105.AF.F	9	4	1	0	14	49.00
106.AG.M	9	6	3	1	19	90.25
107.AH.F	3	11	13	13	40	400.00
108.AI.M	1	0	0	0	1	0.25
109.AM.M	5	0	0	2	7	12.25
110.AO.M	12	1	8	3	24	144.00
111.AT.F	12	0	3	3	18	81.00
112.BA.F	2	2	15	12	31	240.25
113.BH.M	7	5	0	0	12	36.00
114.BL.M	9	7	24	13	53	702.25
115.BO.F	10	12	9	8	39	380.25
$\Sigma_j Y_{kj}$	125	86	112	68	391	3392.75
M_k	8.4	5.7	7.5	4.5		
$\Sigma_j Y_{kj}^2$	1259	794	1764	738		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	1041.7	493.1	836.3	308.3		

APPENDIX IXb

ANALYSIS OF ERRORS IN DIFFERENTIATION
Non-Criterion Group - Exp. SDS-2

Subject	Trial Blocks (Stages) A	B	C	D	$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
116.AJ.M	12	4	0	10	26	169.00
117.AK.M	4	18	11	12	45	506.25
118.AL.M	6	6	14	11	37	342.25
119.AN.M	5	1	3	0	9	20.25
120.AP.M	15	1	6	3	25	156.25
121.AQ.M	6	18	6	1	31	240.25
122.AR.M	6	2	10	5	23	132.25
123.AW.F	2	3	1	8	14	49.00
124.AX.M	10	3	7	9	29	210.25
125.AY.M	10	18	13	9	50	625.00
126.AZ.M	10	12	4	6	32	256.00
127.BG.F	2	5	1	12	20	100.00
128.BM.M	6	7	8	1	22	121.00
129.BN.M	7	1	1	0	9	20.25
130.BP.F	13	8	2	2	25	156.25
$\Sigma_j Y_{kj}$	114	107	87	89	397	3104.25
M_k	7.6	7.1	5.8	5.9		
$\Sigma_j Y_{kj}^2$	1080	1331	803	811		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	866.4	763.3	504.6	528.1		

APPENDIX IXc

ANALYSIS OF ERRORS IN INTENTION
Criterion Group - Exp. SDS-2

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
101.AA.M	8	9	2	1	20	100.00
102.AB.F	8	8	7	0	23	132.25
103.AC.M	7	9	2	3	21	110.25
104.AE.M	7	5	6	2	20	100.00
105.AF.F	3	0	3	0	6	9.00
106.AG.M	5	3	1	1	10	25.00
107.AH.F	11	10	8	5	34	289.00
108.AI.M	2	2	4	0	8	16.00
109.AM.M	5	4	2	2	13	42.25
110.AO.M	3	4	4	9	20	100.00
111.AT.F	4	5	2	8	19	90.25
112.BA.F	3	1	2	8	14	49.00
113.BH.M	8	8	3	8	27	182.25
114.BL.M	8	3	3	5	19	90.25
115.BO.F	7	9	9	7	32	256.00
$\Sigma_j Y_{kj}$	89	80	58	59	286	1591.50
M_k	5.9	5.3	3.9	3.9		
$\Sigma_j Y_{kj}^2$	621	576	310	391		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	528.1	426.7	224.3	232.1		

APPENDIX IXd

ANALYSIS OF ERRORS IN INTENTION
Non-Criterion Group - Exp. SDS-2

Subject	Trial Blocks (Stages)				$\Sigma_k Y_{kj}$	$\frac{(\Sigma_k Y_{kj})^2}{n_j}$
	A	B	C	D		
116.AJ.M	7	3	2	4	16	64.00
117.AK.M	8	6	7	6	27	182.25
118.AL.M	8	10	7	6	31	240.25
119,AN.M	1	3	7	3	14	49.00
120.AP.M	6	6	6	6	24	144.00
121.AQ.M	5	5	8	8	26	169.00
122.AR.M	9	6	5	7	27	182.25
123.AW.F	7	4	5	6	22	121.00
124.AX.M	8	8	6	0	22	121.00
125.AY.M	7	5	7	10	29	210.25
126.AZ.M	8	8	9	6	31	240.25
127.BG.F	6	3	1	8	18	81.00
128.BM.M	3	7	9	2	21	110.25
129.BN.M	4	2	2	0	8	16.00
130.BP.F	6	8	7	10	31	240.25
$\Sigma_j Y_{kj}$	93	84	88	82	347	2170.75
M_k	6.2	5.6	5.9	5.5		
$\Sigma_j Y_{kj}^2$	643	546	602	586		
$\frac{(\Sigma_j Y_{kj})^2}{n_k}$	576.6	470.4	516.3	448.3		

APPENDIX Xa

TEACHING INSTRUMENT

1. _ ⊕ U
2. U ⊕ _
3. _ ⊕ U
4. _ ⊕ U
5. U ⊕ _
6. _ ⊕ U
7. U ⊕ _
8. U ⊕ _
9. _ ⊕ U
10. _ ⊕ U
11. U ⊕ _
12. U ⊕ _
13. U ⊕ _
14. U ⊕ _
15. U ⊕ _
16. _ ⊕ U
17. U ⊕ _
18. _ ⊕ U
19. U ⊕ _
20. U ⊕ _
21. _ ⊕ U
22. U ⊕ _
23. _ ⊕ U
24. _ ⊕ U
25. _ ⊕ U
26. U ⊕ _
27. U ⊕ _
28. U ⊕ _
29. U ⊕ _
30. _ ⊕ U
31. U ⊕ _
32. _ ⊕ U
33. _ ⊕ U
34. _ ⊕ U
35. _ ⊕ U
36. U ⊕ _
37. U ⊕ _
38. U ⊕ _
39. U ⊕ _
40. U ⊕ _
41. _ ⊕ U
42. _ ⊕ U
43. U ⊕ _
44. _ ⊕ U
45. _ ⊕ U
46. _ ⊕ U
47. _ ⊕ U
48. _ ⊕ U

0 Green
_ Red

U Yellow
+ Reward

APPENDIX Xb

TEST FOR HYPOTHESIS ADOPTED

---

1. θ ± U
2. θ Ʉ _
3. _ Ʉ θ
4. θ Ʉ _
5. U ± θ
6. θ Ʉ _
7. U ± θ
8. U ± θ
9. θ + U
10. _ Ʉ θ
11. θ ± U
12. _ Ʉ θ

APPENDIX Xc

ABRUPT FAILURE INSTRUMENT #1
Always Green to Always Middle

---

1. 0 Ʉ _
2. _ Ʉ 0
3. _ Ʉ 0
4. 0 ± U
5. _ Ʉ U
6. 0 + U
7. U ± 0
8. 0 ± U
9. U ± 0
10. _ Ʉ 0
11. 0 ± U
12. U ± 0
13. _ Ʉ 0
14. 0 ± U
15. _ Ʉ 0
16. U ± 0
17. 0 Ʉ _
18. 0 ± U
19. U ± 0
20. _ Ʉ 0
21. 0 Ʉ _
22. _ Ʉ 0
23. 0 Ʉ _
24. U ± 0
25. U ± 0
26. 0 ± U
27. 0 ± U
28. U ± 0
29. 0 ± U
30. 0 Ʉ _
31. U ± 0
32. U ± 0
33. 0 Ʉ _
34. U ± 0
35. U ± 0
36. 0 ± U
37. _ Ʉ 0
38. 0 ± U
39. _ Ʉ 0
40. 0 Ʉ _
41. 0 Ʉ _
42. _ Ʉ 0
43. 0 ± U
44. 0 Ʉ _
45. _ Ʉ 0
46. 0 Ʉ _
47. 0 Ʉ _
48. 0 Ʉ _

---

0 Green

U Yellow

_ Red

+ Reward

APPENDIX Xd

GRADUAL FAILURE INSTRUMENT #2
Always Green to Always Middle
Exceptions 5/12 to 8/12

---

1. 0 Ʉ _
2. 0 ± U
3. _ ⊕ U
4. _ Ʉ 0
5. U ⊕ _
6. U ⊕ _
7. _ ⊕ U
8. U ⊕ _
9. 0 Ʉ _
10. 0 ± U
11. _ ⊕ U
12. _ ⊕ U
13. U ⊕ _
14. 0 ± U
15. U ⊕ _
16. _ Ʉ 0
17. 0 Ʉ _
18. 0 ± U
19. U ± 0
20. U ⊕ _
21. U ⊕ _
22. _ ⊕ U
23. U ⊕ _
24. U̲ Ʉ 0
25. 0 Ʉ _
26. 0 ± U
27. U ± 0
28. _ Ʉ 0
29. 0 Ʉ _
30. U ⊕ _
31. U ⊕ _
32. U ⊕ _
33. _ ⊕ U
34. 0 ± U
35. U ± 0
36. _ ⊕ U
37. _ ⊕ U
38. 0 ± U
39. U ± 0
40. _ Ʉ 0
41. 0 Ʉ _
42. _ ⊕ U
43. U ± 0
44. _ Ʉ 0
45. _ ⊕ U
46. _ ⊕ U
47. U ± 0
48. _ Ʉ 0

---

0 Green

_ Red

U Yellow

+ Reward

APPENDIX Xe

ABRUPT FAILURE INSTRUMENT #1
Always Middle to Always Green

1. ⊕ U _
2. _ U ⊕
3. _ U ⊕
4. ⊕ _ U
5. _ U ⊕
6. ⊕ _ U
7. U _ ⊕
8. ⊕ _ U
9. U _ ⊕
10. _ U ⊕
11. ⊕ _ U
12. U _ ⊕
13. _ U ⊕
14. ⊕ _ U
15. _ U ⊕
16. U _ ⊕
17. ⊕ U _
18. ⊕ _ U
19. U _ ⊕
20. _ U ⊕
21. ⊕ U _
22. _ U ⊕
23. ⊕ U _
24. U _ ⊕
25. U _ ⊕
26. ⊕ _ U
27. ⊕ _ U
28. U _ ⊕
29. ⊕ _ U
30. ⊕ U _
31. U _ ⊕
32. U _ ⊕
33. ⊕ U _
34. U _ ⊕
35. U _ ⊕
36. ⊕ _ U
37. _ U ⊕
38. ⊕ _ U
39. _ U ⊕
40. ⊕ U _
41. ⊕ U _
42. _ U ⊕
43. ⊕ _ U
44. ⊕ U _
45. _ U ⊕
46. ⊕ U _
47. ⊕ U _
48. ⊕ U _

0 Green

U Yellow

_ Red

\+ Reward

APPENDIX Xf

GRADUAL FAILURE INSTRUMENT #3

Always Middle to Always Green
Exceptions 9/12 to 12/12

---

1. ⊕ U _
2. ⊕ _ U
3. U _ ⊕
4. _ U ⊕
5. ⊕ U _
6. U ⊕ _
7. U ⊕ _
8. _ U ⊕
9. ⊕ U _
10. ⊕ _ U
11. U _ ⊕
12. U ⊕ _
13. ⊕ U _
14. ⊕ _ U
15. U _ ⊕
16. _ U ⊕
17. _ ⊕ U
18. ⊕ _ U
19. U _ ⊕
20. _ U ⊕
21. _ ⊕ U
22. ⊕ _ U
23. U _ ⊕
24. _ I ⊕
25. ⊕ U _
26. ⊕ _ U
27. U _ ⊕
28. _ U ⊕
29. ⊕ U _
30. ⊕ _ U
31. _ ⊕ U
32. _ U ⊕
33. ⊕ U _
34. ⊕ _ U
35. U _ ⊕
36. _ U ⊕
37. ⊕ U _
38. ⊕ _ U
39. U _ ⊕
40. _ U ⊕
41. ⊕ U _
42. ⊕ _ U
43. U _ ⊕
44. _ U ⊕
45. ⊕ U _
46. ⊕ _ U
47. U _ ⊕
48. _ U ⊕

---

0 Green
U Yellow
_ Red
+ Reward