THE EFFECTS OF EARLY EXPERIENCE ON THE EMOTIONAL RESPONSES TO PAIN

by

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There has recently been an increase of theoretical interest in the effects of early experience on behavior, together with an increasing number of experimental studies. In one area, however, there is a marked discrepancy between theoretical emphasis and amount of empirical investigation: the area of avoidance behavior, and pain. It has long been assumed that a major factor in adult behavior is the individual's early exposure to noxious stimulation, so that the threat or expectancy of such stimulation can dominate behavior in many situations without any actual fulfilment of the threat. But there has been little systematic study of such early experience.

The purpose of the present study is to make a beginning at closing this gap between theory and empirical knowledge, and also to bring together the literature on the nature of pain itself, together with that on the related topic of the development of avoidance behavior in animals. Recent reviews (Edwards,1950; Hall,1953) have noted the perplexing difficulty of simply defining the term "pain" adequately for contemporary psychology. But more important is the lack of a consistent theoretical framework which is broad enough to include the large number of apparently unrelated and contradictory experiments and hypotheses on pain.

It is the purpose of the present review of the literature to indicate that psychology is in need of an approach to the problem of pain other than the one it has hitherto taken. Most psychologists have either entirely disregarded the physiological mechanisms which have been discovered recently, or have attempted to solve the problem primarily in terms of the autonomic nervous system. As we shall see, neither procedure is adequate for a theoretical treatment of the facts.

THE PROBLEM IN PSYCHOLOGY

One of the fundamental assumptions prevalent in contemporary psychology is that behavior is a series of responses to stimulation. Repeated stimulation is believed to provide an opportunity for the response to become strengthened, or "reinforced", so that it will be retained by the organism. If the stimulus is discontinued for a period of time, however, the response is expected to weaken, and finally extinguish.

The common observation that "a burnt child dreads the fire" has presented a complex problem for this stimulus-response model of behavior. Its basic assumption requires that the child be burned periodically, at least, so that he may retain the avoidance response to the flames. However, the fact is that organisms often persistently avoid a noxious stimulus after a single

painful contact. Two separate stimulus-response theories have been advanced specifically to account for this fact, that avoidance responses do not extinguish in the absence of further painful reinforcement. While investigations oriented around these theories have provided an extensive amount of factual data on the response to noxious stimuli, they have not yet been able to provide sufficient evidence to support the theories.

Mowrer's theory. In an extensive paper on the acquisition of avoidance responses, Mowrer (1950, chap.9) has proposed that painful stimulation innately elicits an autonomic discharge which gives rise to the emotion of fear. To account for the prolonged avoidance of noxious stimuli without periodic painful contacts, Mowrer has maintained that neutral stimuli become conditioned to the autonomic fear response during the painful stimulation. These conditioned stimuli can subsequently elicit the autonomic discharge, which warns the organism by giving rise to the emotion of "anxiety". Motor responses which are instrumental in reducing the anxiety, such as avoidance behavior, become strengthened and are retained by the organism.

The most pertinent evidence against this type of theory has been provided by Solomon, Kamin and Wynne (1953), who have performed experiments with the explicit purpose of proving the theory's fundamental assumptions. These authors calculated the latencies of both the autonomic discharge and the avoidance response

to an auditory signal which had been conditioned to a strong electric shock. About ten trials after conditioning was completed, they found that the autonomic discharge occurred after the avoidance response. Nevertheless, the avoidance response continued for the entire 200 test trials without any change, despite the fact that the latencies precluded anxiety-reduction. The authors noted, too, that most of the dogs continued to make avoidance responses long after the disappearance of any signs of autonomic discharge.

Further evidence against a peripheral theory for avoidance behavior has been provided by the investigations of Solomon and Wynne (1950) and Auld (1951). These investigators found that blocking the afferent autonomic fibres by surgery or chemical agents did not prevent avoidance responses from becoming established. They were obliged to conclude that the autonomic nervous system plays a part in the acquisition of avoidance responses, but that it is not absolutely necessary.

Miller's theory. A theory that is comparable to the one above has been proposed recently by Miller (1951). To account for "the obvious fact that people learn to fear an experience, such as a severe burn, that produces prolonged pain" (p.446), Miller has maintained that the avoidance response becomes strengthened because of a rapid reduction in pain impulses. The present writer is not clear on the manner in which a reduction in impulses is supposed

to strengthen the nerve connections which mediate the avoidance response. In any case, the suggested mechanism has no support from physiology of the nervous system; there is no evidence of a rapid reduction of impulses in peripheral nerve after a severe burn (Fulton, 1946, p.313).

According to Miller's theory, people who are insensitive to pain ought never to acquire avoidance responses, since there is no pain to reduce. Furthermore, the lack of pain from birth, prevents any generalization from earlier experience with pain--the mechanism which Miller uses to explain avoidance behavior when there has been no observable contact with a painful stimulus. Nevertheless people insensitive to pain do acquire avoidance responses, and, in fact, learn to avoid physical damage by noxious stimulation almost as well as normal pain-sensitive individuals (McMurray, 1950).

None of the arguments presented above are intended to show that the reduction of pain and anxiety do not contribute to the acquisition of avoidance responses. They undoubtedly do participate. But the attempts by Miller and Mowrer to ascribe all responses to pain to these mechanisms alone must be considered unsuccessful in view of the behavioral data. Part of the difficulty may lie in the assumption of a direct, uncomplicated relationship between the noxious stimulus, the pain (or innately-aroused fear of pain) and the response, since the treatment of pain as a reinforcing agent for stimulus-response connections does not provide an adequate

framework for integration of the facts to be presented in the next section.

THE CENTRAL NERVOUS MECHANISMS OF PAIN

The complexity of the nervous system which is directly involved in the mediation of pain is seen even in studies of pain receptors by anatomical and physiological means (Bishop, 1943, 1944; Tower. 1943; Feindel et al, 1948). Earlier conceptions of the afferent process maintained that excitation of a pain spot in the skin directly innervated a fiber in the spinal cord, which in turn connected with successive fibers in a simple chain formation, until the impulses reached the cortex and were immediately localized (see Tower, 1943). Recent studies of the morphology and patterning of the receptors for pain have made it increasingly clear that the nervous system can not operate in such a simple chain fashion. In any normal area of the skin, the pain-receiving terminals from many neurons overlap and interlock intricately, so that a multiplicity of nerve fibers supply information to the highest centers in the brain for interpretation. Impulses from a small area of the skin must be synthesized into the concept of a point which is then projected onto the periphery in the course of localization (Tower, 1943).

Similarly, studies on the various distinctive qualities

of pain indicate that the central nervous system is also designed to integrate the temporal patterns of impulses passing along many pain fibers (Bishop, 1944, 1946, 1948). A variety of sensory effects, such as non-painful pain, itch, painful prick, ache, and the different qualities of pain, are "induced within the modality of pain depending apparently only on the pattern, spatial and temporal, of the impulses in nerve fibers induced by various patterns of stimulation" (Bishop, 1948, p.151).

While these data describe the nature of the information which is transmitted by pain fibers, the fact that non-painful prick and itch can be elicited by stimulating "pain"-endings prevent us from saying simply that pain is the result of stimulation of these receptors, in the same way that vision is the result of stimulation of the retina. This type of difficulty has led Bishop (1946) to suggest that the term "sense modality" is a straight-jacket from which the study of cutaneous sensation may profitably be released. This does not imply, however, that pain may be considered to be the result of excessive stimulation of the fibers transmitting touch, pressure and temperature, as some authors have maintained (Nafe, 1934). There is considerable evidence that pain is a distinctive physiological process with characteristic receptors, fiber-conduction groups and central pathways. A specific type of nerve ending which functions only to produce the variety of sensory qualities associated with pain has been isolated and studied

carefully (Bishop, 1943; Bishop, 1944). Furthermore, electrophysiological studies have shown that the fibers which carry pain fall into two distinctly characteristic size ranges, although there is overlap at the margins with fibers subserving other sensations. The large, fast-conducting A fibers carry sharp pain, while the smaller, slow-conducting C fibers transmit dull, throbbing pain (Gasser, 1943; see Bishop, 1946 and others there cited).

The pain pathways in the central nervous system have been thoroughly investigated (Walker, 1943). There is still controversy, however, on the main site at which pain is appreciated, (that is, the level at which the afferent process determines the distinctive type of response which marks the perception of pain). The early suggestion by Head and Holmes (1912) that the thalamus is the site in question has received severe criticism (Lashley, 1938; Walker, 1943; Marshall, 1951). The evidence provided by clinical investigations (Michelsen, 1943; Marshall, 1951 and others there cited) and by a careful ablation study in the monkey (Peele, 1944) leaves no doubt that pain has some kind of cortical representation. But the pain pathways alone, including the facts of localization, are insufficient to account for the complexity of pain phenomena. The lack of precise knowledge has made it necessary to infer the nature of the physiological mechanisms involved from indirect evidence. Tower (1943) and Bishop (1943) have conjectured that the

evidence on the patterning of the pain-receptors strongly suggests that the higher centers have the role of integrating and analyzing the impulses which arrive along the many paths. In short, it is unlikely that a single area is involved in pain sensation, but rather that large areas of cortex, thalamus and possibly even lower structures are directly concerned. Recent anatomical evidence, to be reviewed later, supports this contention. Tower has further suggested that the pathways for pain are, in all likelihood, comparable to the visual pathways described by Marshall and Talbot (1942), with a succession of levels at which pain impulses undergo modification, integration and repatterning, until the highest centers are finally reached. These writers conceive of pain as having its own distinct pathways in the cerebrum to account for localization and interpretation of the complex patterns of impulses.

A similar suggestion has been made by Marshall (1951), based on other evidence. Marshall found that small lesions in the somesthetic cortex usually produce dramatic impairment of pain sensation, while larger ones, including hemispherectomy, frequently have little effect after an initial disturbance during the week or so after operation. The implication of this evidence is that pain pathways involve widespread, two-way circuits between somesthetic cortex and thalamus.

The characteristically slow rate of pain impulses passing along the small C fibers has led Hebb (1949) to suggest recently

that pain actually involves a disruption of the pathways employed by the other senses, rather than having pathways of its own. But it is difficult to believe that disruption of pathways can account for the complex synthesis and integration of impulses that is necessary for the localization of pain, or that disruption can so neatly produce the forty different sensory blends and nuances of pain listed by Dallenbach (1939). It seems much more likely that the varieties of sensation related to pain, depending as they do on spatial and temporal patterns of impulses, require cerebral pathways of their own.

Before coming to any conclusions on pain mechanisms, however, we shall first consider the clinical and experimental evidence from which neural mechanisms of pain have been inferred.

The effects of hyperstimulation. An impressive array of evidence has been presented recently by Gerard (1951) to show that a brief, excessive barrage of sensory impulses is an adequate condition to produce the prolonged intense suffering of causalgic pain, such as pain of the phantom limb. Especially convincing is the experiment by Reynolds and Hutchins (1948). Tooth cavities on opposite sides of a patient's mouth were filled in this study, but maximal care was taken to prevent pain in one tooth, while treatment was rather rough, with no anesthesia, in filling the other. More than thirty patients were treated in this way; all developed a severe pain which lasted for many months in the roughly handled

tooth, but not in the one anesthetized during operation. The pain was abolished permanently, however, by a single procaine block. It therefore appears that, once the initial excessive sensory barrage had functionally modified the central nervous system, only normal sensory stimulation was needed to maintain the pain process. Further supporting evidence for initial hyperstimulation as a cause of prolonged pain has been reported recently by Kennard (1950), showing that brief, local irritation of the spinal cord, directly applied, may set up the conditions for prolonged causalgic pain.

These results support the hypothesis proposed by Livingston (1943) on the basis of clinical evidence of the causalgias. The theory is an attempt to explain not only causalgic pain, such as the pain of the phantom limb but also the extreme hyperalgesia found in regions of skin which have recovered completely from an earlier small lesion. Livingston suggests that, at the time of the lesion, continued sensory bombardment of internuncial pools of neurons in the spinal cord effects synaptic changes, giving rise to self-perpetuating reverberatory circuits. Occasional sensory input from "trigger points" maintain these circuits, or under their own impetus they may continue to fire efferent neurons long after recovery from the lesion. In this way they modify the intensities of perceived pain and produce muscle spasm, localized sweating and other conditions found in causalgia.

The effects of hypostimulation. The evidence cited by Gerard (1951) for hypostimulation as an adequate cause of causalgic pain is as impressive as the arguments for hyperstimulation. In experiments using a pressure cuff on the arm, touch and temperature were lost after about a half hour of inflation, And, at that time, slow pain from normal stimulation suddenly acquired the "peculiar, unpleasant quality of causalgic pain" (Gerard, 1951, p.6). It seems fully possible, however, that this causalgic pain may be due simply to the block of slow pain fibers rather than the faster A fibers of touch. Such an interpretation is possible since Weddell and his associates (1947, 1948) have shown that stimulation of single pain endings brings about an excessive and distorted awareness of pain, while normal pain sensation occurs with the stimulation of many pain receptors.

One of the most striking examples of hypostimulation as a cause of intense, burning pain has been recorded by Rothman (1943). Patients suffering from severe itch in the anal and vulvar regions were relieved of the itch by surgical section of the fibers of the entero-lateral columns which carry pain. But section of the posterior tracts, destroying fine touch and pressure fibers (Larsell, 1951) produced still greater itch and the burning pain of causalgia.

There have been many interpretations of such facts. Kendall (1939) and Wortis et al (1942) have inferred that

The fast-moving A fibers of touch or pain normally produce a relatively refractory state centrally, preventing the upward passage of impulses from the slow-conducting pain fibers. Lesions of the A fiber tracts would therefore allow an excess of slow impulses to pass into the cortex and produce causalgic pain. Weddell et al (1948) have criticized this hypothesis and refer to the work of Gordon and Whitteridge (1943), who found that there were sufficient fast-pain fibers to abolish alpha rhythm in the cortex of many patients who had severe causalgic pain as a result of peripheral nerve lesions. This argument, however, is not convincing. Only partial destruction of the A fibers may be necessary to bring about causalgic pain.

A second hypothesis has been presented by Hebb (1949) to account not only for causalgic pain such as phantom limb pain, but also the pain resulting from lesions in any part of the nervous system. Hebb suggests that the slow rate of impulses entering the thalamus. after optimal stimulation of the C fibers; disrupts spatially- and temporally-organized activity in the brain stem and in the cortex. this disruption per se constituting the physiological basis of pain. In developing his argument, Hebb lays stress on the fact that "a conceivable source of pain is a decrease or absence of peripheral sensory activity, even in pain fibers" so that "if higher somesthetic nuclei are deafferented . . . . the cells must eventually fire, and fire more or less in synchrony,

with massive jolts to other nuclei connected with them" (pp. 184 -185). Thus, pathological changes in the somesthetic tracts up to and including the thalamic relay nucleus may reduce normal somesthetic A fibers to the efficiency of C fibers, and elicit prolonged, intense pain.

Hebb (1949) has been careful to point out that his hypothesis for pain is incomplete, since syringomyelia, a degenerative disease of the pain and temperature tracts in the cord (Larsell, 1951) is an outstanding case of hypofunction without pain. There are other similar instances; phantom limb pain probably occurs in no more than five or ten percent of cases of phantom limb after amputation (Henderson and Smyth, 1948). In fact, there are no reported instances of loss of somesthetic tracts accompanied by pain where some pain fibers have not been left intact. If the pain tracts are cut, as in the operations for itch described by Rothman, or in the case of syringomyelia, there is no pain.

Hebb's hypothesis, then, needs further qualification. Disruption of pain fibers is, as the experiments of Weddell et al have indicated, an adequate cause of causalgic pain. Lesions in other somesthetic tracts may be considered as contributory to the pain, but not a necessary cause.

If Hebb's type of hypothesis is applied to the spinal cord, as Gerard (1951) has done, there is no difficulty in conceiving of pain as hypersynchronous neural activity, and yet the

cerebrum may still be considered as having specialized pathways for pain impulses. Gerard suggests that "under causalgic conditions, a hypersynchronization, a firmer locking together of a larger than normal number of neurons, has occurred to form a pulsating pool, and that this synchronization has become exaggerated by virtue of the lack of disturbing impulses to disrupt the synchrony and by reinforcement with those specific pain afferents that are feeding in to lock the neurons (just as cortical neurons become locked in their beat by a flickering light). Such a pulsing pool could recruit additional units, could move along in the grey matter, could be maintained by impulses different from and feebler than those needed to initiate it, could discharge excessive and abnormally patterned volleys to the higher centers" (p.10).

Such are the current ideas about the mechanisms of abnormal pain. Let us now return to the enquiry concerning the normal pain process.

The nature of pain. That pain is, in part, functionally dependent on touch, pressure and other somesthetic sensory impulses has been shown earlier by the evidence that lesions of the somesthetic tracts make pain more likely to occur in abnormal form, and that a lack of normal somesthetic stimulation can provide the conditions for severe causalgic pain (Lashley, 1938; Hebb, 1949). In a study of the phenomena of phantom limbs, Henderson and

Smyth (1948) have suggested that there is normally a balanced integration of many impulses from the various senses which impinge on the somesthetic cortex. In the case of patients who have lost a limb. however, the lack of normal somesthetic sensory impulses gives rise to a hyper-excitability of the sensori-motor cortex. producing the normal phantom limb without pain.

It seems very likely, however, that it is in the spinal cord that the conditions for causalgic pain occur rather than in the somesthetic cortex. The fact that urination and defecation may often cause excruciating pain in the phantom limb suggests that pain impulses from the excitation of sympathetic nerve fibers (Walker, 1943), which normally are not sufficient to elicit conscious pain, can now cause an upward discharge of impulses in a large number of pain fibers.

These data indicate that there is. in the spinal cord, a type of transmission that is not unlike the patterns of neurons described by Lorente de Nó (cited by Bishop, 1946), where the axon terminals of a given neuron may connect with the dendrites of many other neurons. In this way, the interneurons which lie between (A) the pain fibers entering the cord, and (B) the spinal tract which transmits pain impulses centrally, would be susceptible to the modifying influences of many impulses. Thus there may be interneurons which receive the axon terminals of pain fibers only, while other internucial cells have synaptic connections

with the terminals of touch, pressure and temperature fibers as well; the excitatory threshold of the latter internuncial group would be determined in part by the other somesthetic fibers which may be considered as normally having inhibitory effects on these cells. It is conceivable, then, that normal sensory excitation, including pain, could send patterned impulses into the spinal cord which would be able to maintain their relative intensities without distortion as they pass through the internuncial cells toward the higher centers for integration, synthesis and, ultimately, localization. If touch and pressure fibers are cut, however, many of the neurons which are normally inhibited after the passage of rapid impulses from A fibers would be available to the slow pain impulses, allowing spatial and temporal summation of excitatory potentials to fire many of the spinal neurons and sending abnormally patterned impulses toward the cerebrum. It is possible, of course, to consider this process, as Gerard has done, as a hypersynchrony of electrical potentials, sending up bursts of impulses after activation from pain fibres.

Bishop (1946) has noted that poorly localized pain usually has a particularly unpleasant quale; but touching or pressing the pain spot makes it more easily localized, and at the same time, less unpleasant. This could be explained if we think of touch and pressure impulses passing through the cord as normally having a desynchronizing effect, and preventing summation of

lectrical activity in the interneurons transmitting pain mpulses. The unpleasant character of the pain would thus dimiish and the pain impulses would ascend in more distinctly-patterned form for precise localization.

hese ideas concerning pain mechanisms are very like hose presented by Livingston, Hebb and Gerard. But by looking rimarily to the spinal cord instead of higher up they provide a basis for combining the three hypotheses with respect to bnormal pain, and still permit us to consider normal pain as a pecific physiological process with its own pathways in the erebrum.

HE RESPONSE TO PAIN

he complexity of the neural mechanisms discussed bove has been recognized for human subjects, but not in investigations employing animals. A systematic attack on the problem of the response to pain must now take explicit account of the nown complexities of pain considered as a receptive or sensory vent.

he effects of emotion. One of the outstanding characeristics of both pain sensation and response is their susceptibility to change with emotional states concurrent with the painful timulation. It has been frequently demonstrated that fear and

anxiety intensify the disruptive effects of a noxious stimulus (Estes and Skinner, 1941; Hunt and Brady, 1951a, 1951b; Mowrer and Viek, 1948). The opposite may also occur. Intense emotional excitement during war or sports may block pain sensation and response completely, despite severe bodily damage (Beecher, 1953; Livingston, 1943, 1953). These data stress the necessity, if pain phenomena are to be understood, of recognizing central states which are capable of modifying the sensory impulses passing through the cerebrum, or blocking their passage entirely.

In their discussion on the anticipation of pain, Mowrer and Viek (1948) make the assumption that the disruptive effects of anxiety during painful stimulation are simply added to the disturbance of the pain itself. Recent evidence on the effects of anxiety demonstrates that the state of affairs is not so simple. Clinical and experimental evidence has shown that the effect of prefrontal lobotomy (Landis et al, 1950; King et al, 1950) and morphine (Wikler, 1950; Keats and Beecher, 1950) in cases of intractable pain is primarily on the emotional states which influence pain sensation and response, and not on the pain thresholds (Chapman et al, 1948, 1950). Before lobotomy, the patients report that the quale of intractable pain is not just pain, not anxiety, nor even an admixture of the two. It is a unique state of mind that has been referred to as "the big pain", "suffering" or "anguish" (Landis et al, 1950; Keats and Beecher, 1950). After lobotomy, however, the

patients usually report that the pain is gone. They sometimes admit they still have their pain, but that "it does not bother them" (Dynes and Poppen, cited by Keats and Beecher, 1950). It appears that the emotions intensify pain (Hill et al, 1952a, 1952b), and give it a particularly disagreeable quality.

It is obvious that these emotional effects on pain are a function of earlier experience with painful stimulation. Apparently, the frontal lobes are a focal area in modifying the pain impulses in their passage through the cerebrum. It is interesting, then, that Bousfield and Orbison (1952), on the basis of psychological, anatomical and neurophysiological evidence, have suggested that the association areas of the frontal lobes play a major role in the acquisition of emotional behavior during childhood. The dramatic effects of prefrontal lobotomy could thus very likely be due to the prevention of early emotional learning from having an effect on the elaboration of pain impulses in the cerebrum.

The visual and auditory association areas of the cortex must also have an important influence in the modification of pain impulses. The response to pain is determined in large part by perception of the kind of agent that causes the damage, and by the perceived possibilities of adjusting to the situation. Pain impulses convey little information about either of these. With duration of stimulation held constant, pain resulting from contact with a heated wire, a pulse of electric current, pulling of the

hair. or a jab with a fine needle all feel exactly alike to a subject if care is taken to keep away from him any supplementary information concerning the stimuli employed (Lewis, 1942). Furthermore. "it is plain that the reaction of the man in a sick-bed. where his pain may be a warning of disaster, will not be the same as the reaction of a well and comfortable man in the laboratory subject to a momentary pricking sensation" (Beecher, 1953, p. 166; Hall, 1953). The meaning of the pain, and the emotional effects of the context in which it is received are both a function of earlier learning, including experience with painful stimulation (but not this alone, since verbal communications, for example, play a part). These factors must affect the response to noxious stimuli at all times. so that the modifying cortical influences must involve the whole cerebrum.

One of the central points of the thalamus at which pain impulses may possibly be influenced by these many areas is the diffuse projection system (Jasper, 1949; Jasper and Ajmone-Marsan, 1950). This area is known to receive especially strong projections from the frontal association areas. which increases with increasing mammalian development (Starzl and Whitlock, 1952). It seems possible, then. that the effects of lobotomy and morphine are primarily on the long internuncial fibers that pass from the frontal lobes to the diffuse projection system of the thalamus (Keats and Beecher, 1950; Wikler. 1950 and others there cited). It is known that pain impulses

can pass into the diffuse projection system along two different paths (Walker, 1943), presumably "awakening" the association areas so that impulses from these areas would then be able to pass back to the intralaminar region of this system. Since it has been shown that stimulation of the intralaminar region can modify sensory impulses in the somato-sensory areas of the cortex to a striking degree, it would thus be possible for impulses from the association areas to modify pain impulses and affect the quale and the response which they produce, as they pass through widely spread circuits in the cortex and thalamus.

What these mechanisms are actually involved in the modification of pain impulses as a result of earlier experience has not been ascertained. But the behavioral evidence indicates, at least, that these types of mechanisms are closer to psychological description than the conception that anxiety is the result of autonomic activity alone.

The response to pain. Any consideration of the emotional response to pain must recognize the central nervous mechanisms which provide the basis for emotional response. Excellent reviews of these mechanisms (Bard, 1950; Lindsley, 1951; Gellhorn, 1953) have outlined the role of the hypothalamus, the limbic system, and the reticular system and tegmentum of the midbrain in emotional response. More recently, "pain suggestive" reactions such as crying, struggling and flight have been produced in cats by

lectrical and mechanical stimulation of the midbrain (Spiegel t al, 1954). Since stimulation of this region also evokes a ow order of pain sensation in human patients (Reyes et al, ited by Spiegel et al, 1954) and sensitizes animals to visual, uditory and tactual stimuli (Spiegel et al, 1954), it is ossible that the midbrain tectum is an important integrating enter in the response to noxious stimulation. Most important, owever. is the fact that all these regions of the brain are unctionally inter-related so that the nerve impulses along many racts. which ultimately bring about overt response, can be odified and influenced at a series of different levels.

he anatomical description, unfortunately, does not nswer the psychologist's question: how does the organism acquire urposive responses to pain, and how are they retained? It has een suggested recently that stable neural changes may occur in he central nervous system following the response to a noxious timulus (Hebb, 1949). This permanent modification would allow the rganism to respond again in the same situation without the con- inual evocation of fear. and the cerebral circuits would be open o the modifying influences resulting from earlier experience. here is no unequivocal neurophysiological evidence with regard to earning phenomena. Nevertheless, the behavioral evidence requires he assumption that permanent changes occur after the acquisition f avoidance responses. The complexity of the nervous system

related to pain makes untenable the assumptions that the response to noxious stimulation is determined simply by events at the time of stimulation, and that pain serves solely as a reinforcing agent. Emotional learning which has been acquired in childhood appears to have highly variable effects on pain sensation and response (Livingston, 1953). Thus, simply the threat of pain may have overwhelming results on the emotional response, and may determine to a large degree the effects of noxious stimuli in learning situations.

EARLY EXPERIENCE AND THE RESPONSE TO PAIN

Earlier clinical formulations of the problem of early experience by Freud and his followers (Greenacre, 1945 and others there cited) have not led to any relevant experimental studies, although the effects of early experience on adult behavior was recognized. More recently, Scott and his associates (Scott et al, 1951) have arrived at a new hypothesis of the effects of early experience. They maintain that during the development of the organism there are specific critical periods after which sufficient maturation has occurred for various types of experience to have lasting effects on adult behavior. Two separate studies indicate the possibility of such a critical period in the rat. Noxious stimulation in early infancy was found by one study to have no

demonstrable effects on problem solving and pattern discrimination at maturity (Griffiths and Stringer, 1952). Significant effects were found in another experiment in which the rats were subjected to intense electric shocks in late infancy (Ried, cited by Griffiths and Stringer, 1952). In neither of these investigations, however, were the animals tested for response to noxious stimulation at maturity.

The relationship between age and the response to pain has been demonstrated in experiments on one-trial learning in the rat (Hudson, 1950). The number of rats displaying avoidance responses toward a metal plate which gave them an electric shock was found to increase with increasing age. That the increase of avoidance responses may be partly acquired, and not due solely to maturation. is suggested by the study of a chimpanzee deprived of normal somesthetic stimulation during infancy and early maturity (Nissen et al, 1951). After removal from somesthetic restriction, the chimpanzee was found to be strikingly poor in localizing sites of noxious stimulation on its body. Furthermore, the animal appeared to have a heightened pain threshold, since "he 'panted' as chimpanzees do when they are being tickled" (Nissen et al,1951, p.502) when his legs or lower ventral trunk were poked with a pencil or pin.

The method of sensory deprivation, or restriction, has proven successful in ascertaining the effects of early sensory

experience on adult behavior (Clarke et al,1951; Hymovitch, 1952). Observations of everyday behavior suggest that earlier experience with noxious stimuli has a marked influence on the response to pain or the threat of pain. The experiments reported in this paper were an attempt to study, by means of the technique of sensory restriction, the effects and interaction of both early non-specific experience and early experience with noxious stimuli on the adult response to painful stimulation.

EXPERIMENT I

The purpose of the first experiment was to ascertain the effects of two variables on the adult response to noxious stimulation in a learning situation: 1) type of early environment, and 2) age of first experience with noxious stimulation. Rats were reared from infancy to maturity in two different types of environment. Some of the rats were given experience with electric shock at different ages, while others received no shock. The rats were then trained in a Yerkes discrimination apparatus according to the general method of Muenzinger (1934), to see whether the relation of punishment to learning differed according to the type of earlier experience.

METHOD

A total of 104 albino rats (from the Sprague-Dawley strain) were divided into two groups. Fifty-two were reared separately in narrow cages in order to restrict the total sensory experience of the animals. The restriction cages are shown in Figure 1. The second group, containing 52 rats, were reared in a "free environment": 7 to 8 rats were kept together in a large cage, which is illustrated in Figure 2. There were five barriers in each cage, as well as large windows at the ends and on the side.

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FIGURE 1.

Restriction Cage for Rats

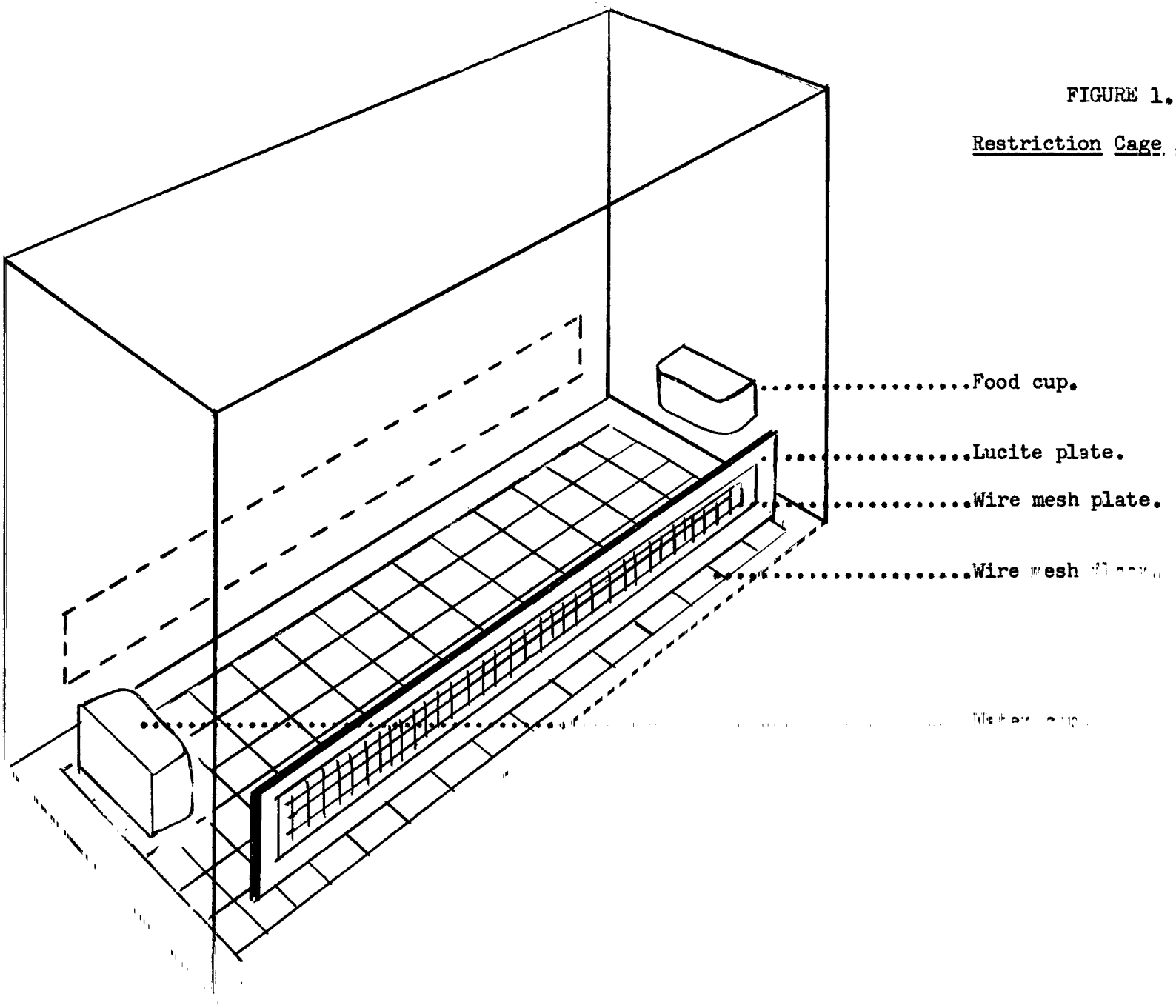
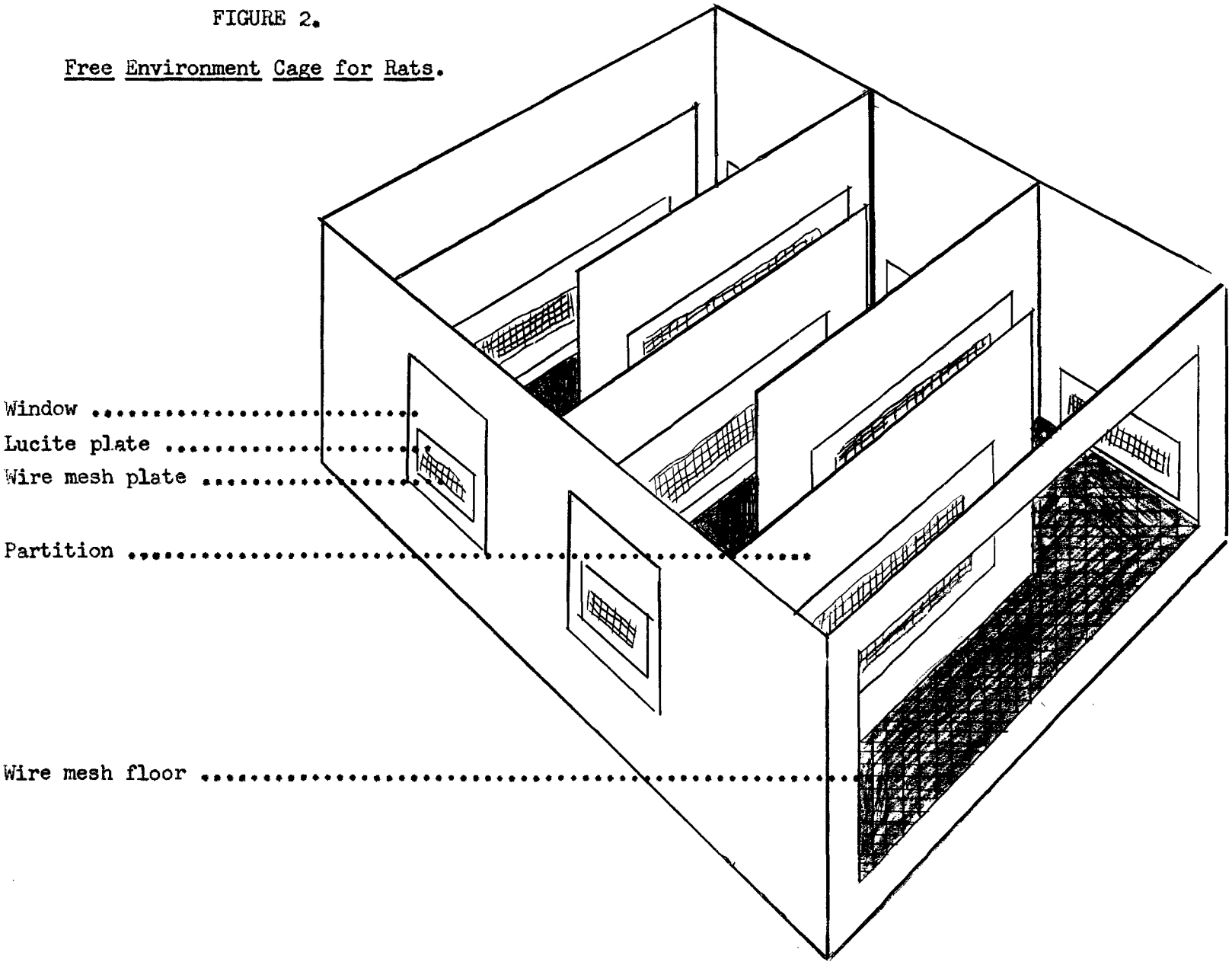


FIGURE 2.

Free Environment Cage for Rats.



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Each of these two main groups was further subdivided into three groups. One group received electric shocks during 21 to 40 days of age. The second group was shocked during 101 to 120 days of age, while the third group did not receive electric shock prior to testing.

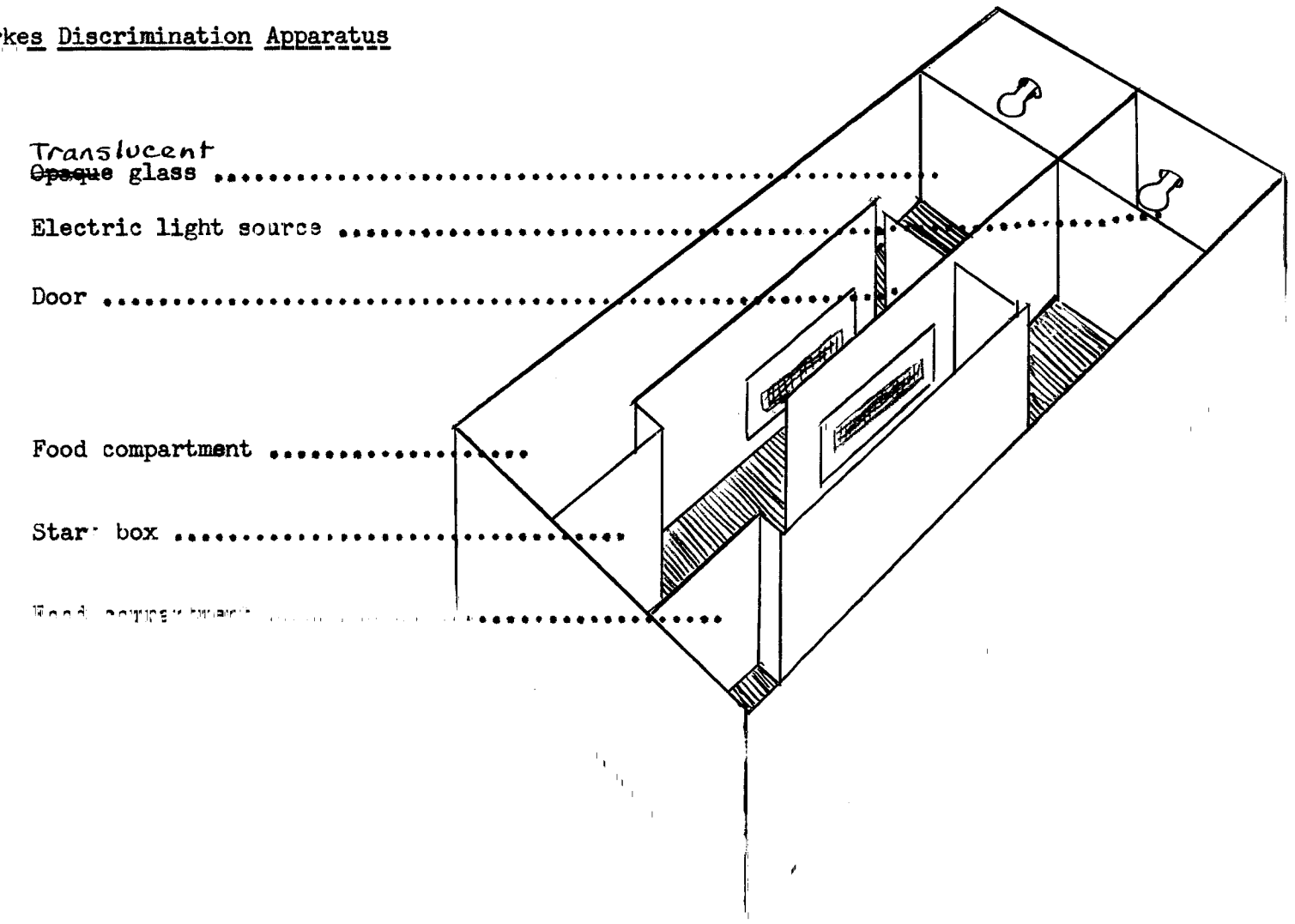
A split-litter technique for genetic control was achieved by dividing each of the twelve litters randomly among the six groups. The rats were placed in the experimental cages after weaning was completed at 18 days of age.

Electric shock was administered in the home cages by means of a modified Licklider (1951) conductance-capacitance shock circuit. The source of electrical energy was a Sola neon-tube transformer capable of converting 110 volt current to 15,000 volts of low amperage.

Beck et al (1953) have demonstrated that the nature of the circuit is such that rats are able to avoid the shock by remaining motionless in the cage during the electrical discharge. During the age period in which the rats were to receive experience with electric shock, an intense shock of a half second duration was presented every hour for 20 hours each day. Thus all rats had an equal opportunity to learn to avoid the noxious stimulus.

FIGURE 3.

Yerkes Discrimination Apparatus



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Testing apparatus. All the rats were tested in a modified Yerkes visual discrimination apparatus, illustrated in Figure 3. Five-watt bulbs provided a diffuse light for the brightness discrimination problem. By means of a manual control the door to the dark or negative side could be locked securely, while the door to the light or positive side was free to fall open when the rat pushed it lightly. The apparatus was wired to the same shock circuit as the home cages, and was controlled manually to provide a shock lasting 1/10 second. The shock used here was weaker than that given in the home cages, since a preliminary experiment showed that intense shock often prevented some of the animals from attempting a second discrimination for hours after they received the first shock.

Procedure. Training was started when the rats were 175 days of age, immediately after they were removed from their rearing cages. They were numbered randomly so that the experimenter was unaware, during testing, of the past history of each animal. The rats were kept in large metal cages, with fifteen or sixteen rats in a cage, until testing was completed.

All the rats received five days of preliminary training in the apparatus before the discrimination problem was presented. The rats were tested every second night, after about twenty-three hours of food deprivation. On the first night of formal training,

The rats received five trials, but were not shocked for the first three. During the last two trials, half of each of the six groups of rats received an electric shock when they touched the door that led to the wrong side. The other half was shocked for pushing the correct door, which opened to the food compartment. The lighted side was alternated from left to right according to the Gellerman series (described by Munn, 1950). This procedure was repeated for five trials during the second training period, and for ten trials each training night until the fiftieth and final trial. The rats were fed wet bran cereal as reward for tem seconds after each trial, and were given fifteen minutes of feeding in their cages on alternate non-training nights.

RESULTS

The mean time and error scores made by each of the six main groups of rats on the discrimination problem are listed in Table I. The analyses of variance for time and errors are summarized in Tables II and III respectively. The statistical computations of these analyses were made in accordance with the procedures outlined by Johnson (1949).

TABLE I

Mean Time in Seconds and Error Scores for Various Groups in Solving a Brightness Discrimination Problem

	Restricted		Free Environment	
	Time	Errors	Time	Errors
Shock for Wrong Response				
Early shock: N:	8		7	
Mean:	831.8	7.6	1441.0	5.9
Range:	338-1815	5-9	413-2563	3-8
Late shock: N:	8		8	
Mean:	2937.3	8.6	3421.8	7.4
Range:	1114-7320	6-18	1240-5083	6-10
No shock: N:	8		8	
Mean:	2933.1	8.5	691.4	3.8
Range:	1415-6168	5-13	289-1156	3-6
Shock for Correct Response				
Early shock: N:	8		8	
Mean:	1177.8	16.6	11303.3	21.8
Range:	726-2009	12-24	816-27664	13-26
Late shock: N:	8		7	
Mean:	24156.3	26.4	30090.6	32.7
Range:	6274-54998	23-32	13522-49800	23-38
No shock: N:	7		8	
Mean:	7305.4	24.1	2845.8	16.9
Range:	5407-10577	17-28	640-7483	9-23

TABLE II

Summary of Analysis of Variance for Time Score Values

SOURCE	df	MEAN SQUARE	F	p
Environment:	1	22,875.16	1.89	-
Age of shock experience:	2	358,840.75	29.60	.001
Testing shock:	1	726,197.81	59.91	.001
E x A:	2	43,157.70	3.56	.05
A x T:	2	293,062.75	24.18	.001
Residual:	84	12,122.04		
Total:	92			

TABLE III

Summary of Analysis of Variance for Error Score Values

SOURCE	df	MEAN SQUARES	F	p
Environment:	1	3.57	-	-
Age of shock experience:	2	292.85	21.60	.001
Testing shock:	1	5,974.12	440.57	.001
E x A:	2	171.57	12.65	.001
E x T:	1	102.81	7.58	.01
A x T:	2	212.39	15.66	.001
E x A x T:	2	58.47	4.31	.01
Residual:	81	13.56		
Total:	92			

The analyses of variance, then, show that the age at which the rats first had experience with electric shock determined, in part, the differences in time- and error-scores among the various groups during discrimination training in which electric shock was used as a punishing agent. Graphs I to VIII and the probability values in Tables IV and V elaborate the effects of each age period at which the rats first encountered repeated electric shocks. Among the restricted groups of rats, the animals that were shocked during infancy made lower time- and error-scores than the rats that first encountered electric shock at maturity. The results are different for the free environment rats: the groups shocked at infancy made higher time- and error-scores than the rats that had no previous experience with shock. The most errors, however, and the highest time-scores were made by the free environment rats that first received continued experience with electric shock in their home cages at maturity.

The differences between the two types of environment appear, in the variance tables, not to be significant. However, since the results above show that each type of environment produced different

TABLE IV

Significance of the Differences between Time Scores for the Six Groups Taken Two at a Time

Shock Wrong

	RE	RL	RN	FE	FL	FN
Restricted						
Early (RE)	-	.01	.01	*	.001	*
Late (RL)		-	*	*	*	.01
No shock (RN)			-	*	*	.01
Free environment						
Early (FE)				-	.01	*
Late (FL)					-	.001
No shock (FN)						-

Shock Correct

	RE	RL	RN	FE	FL	FN
Restricted						
Early (RE)	-	.01	.001	.02	.001	*
Late (RL)		-	.02	*	*	.01
No shock (RN)			-	*	.001	.01
Free environment						
Early (FE)				-	.01	.05
Late (FL)					-	.001
No shock (FN)						-

*Not significant.

TABLE V

Significance of the Differences between Error Scores for the Six Groups Taken Two at a Time

Shock Wrong

	RE	RL	RN	FE	FL	FN
Restricted						
Early (RE)	-	*	*	*	*	.01
Late (RL)		-	*	*	*	.01
No shock (RN)			-	.05	*	.001
Free environment						
Early (FE)				-	.05	.01
Late (FL)					-	.001
No shock (FN)						-

Shock Correct

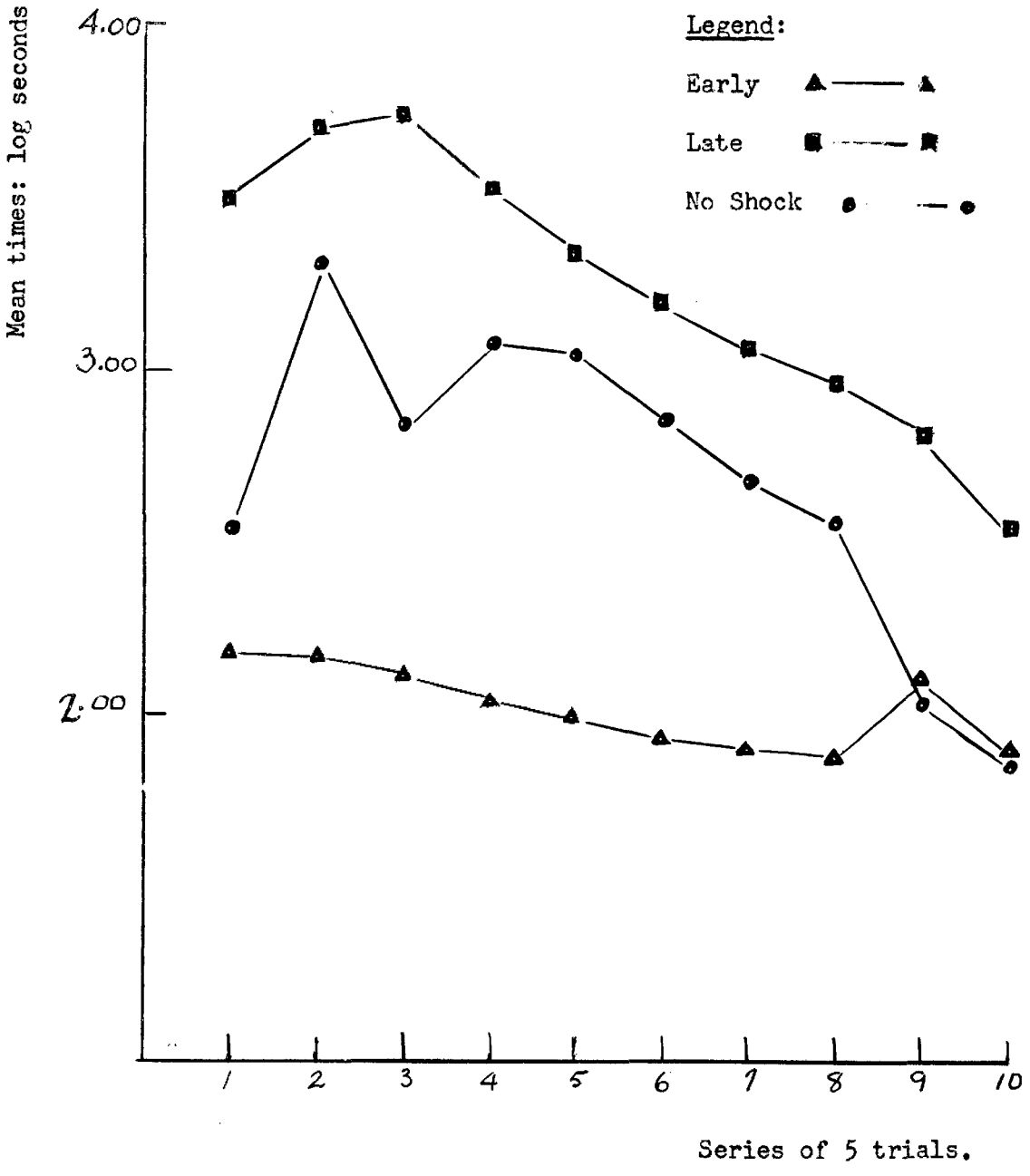
	RE	RL	RN	FE	FL	FN
Restricted						
Early (RE)	-	.001	.01	.05	.001	*
Late (RL)		-	*	.05	.02	.01
No shock (RN)			-	*	.01	.02
Free environment						
Early (FE)				-	.001	*
Late (FL)					-	.001
No shock (FN)						-

*Not significant.

GRAPH I.

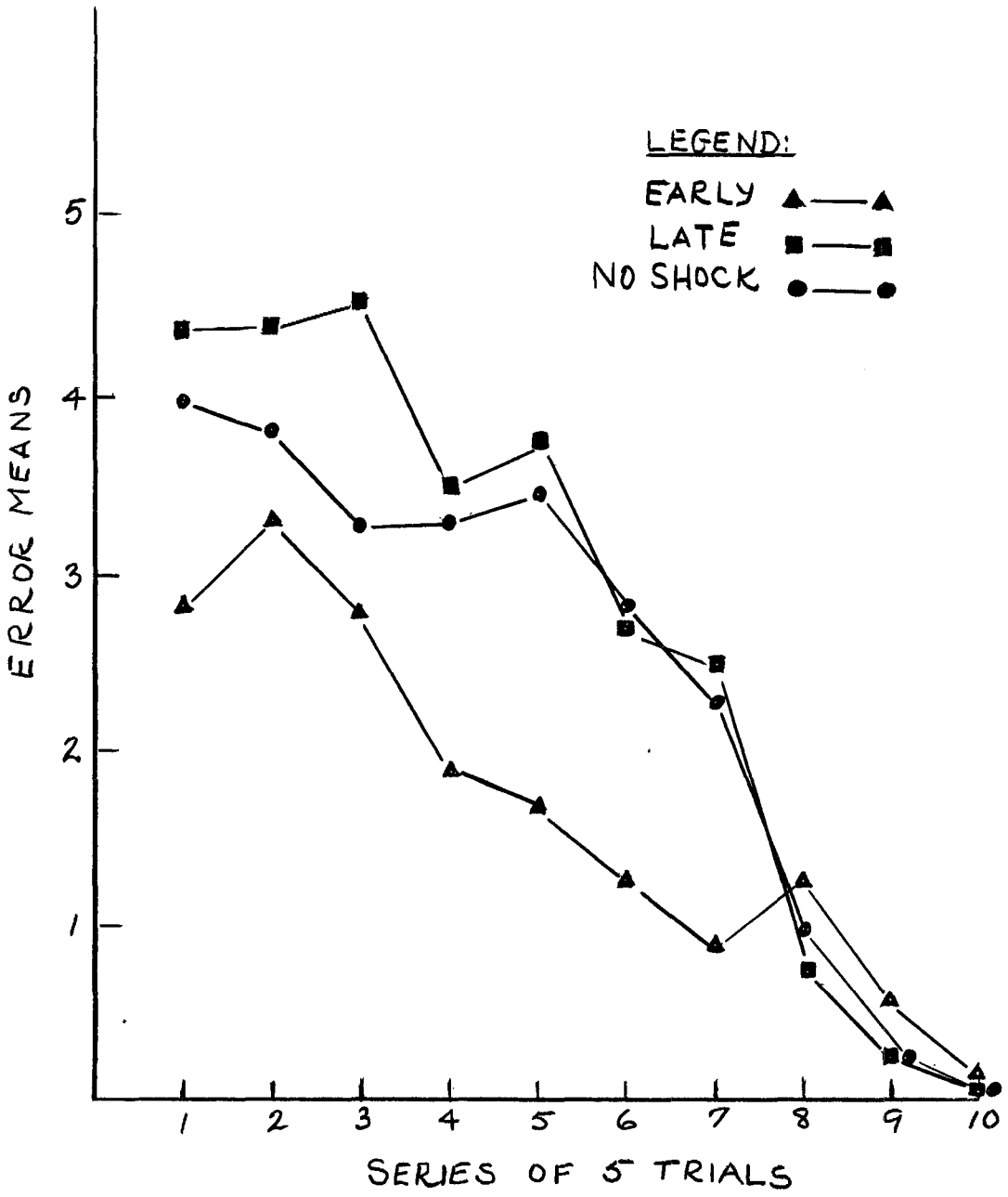
Mean Times for Restricted, Shock Correct Groups

for Series of Five Trials.



GRAPH II.

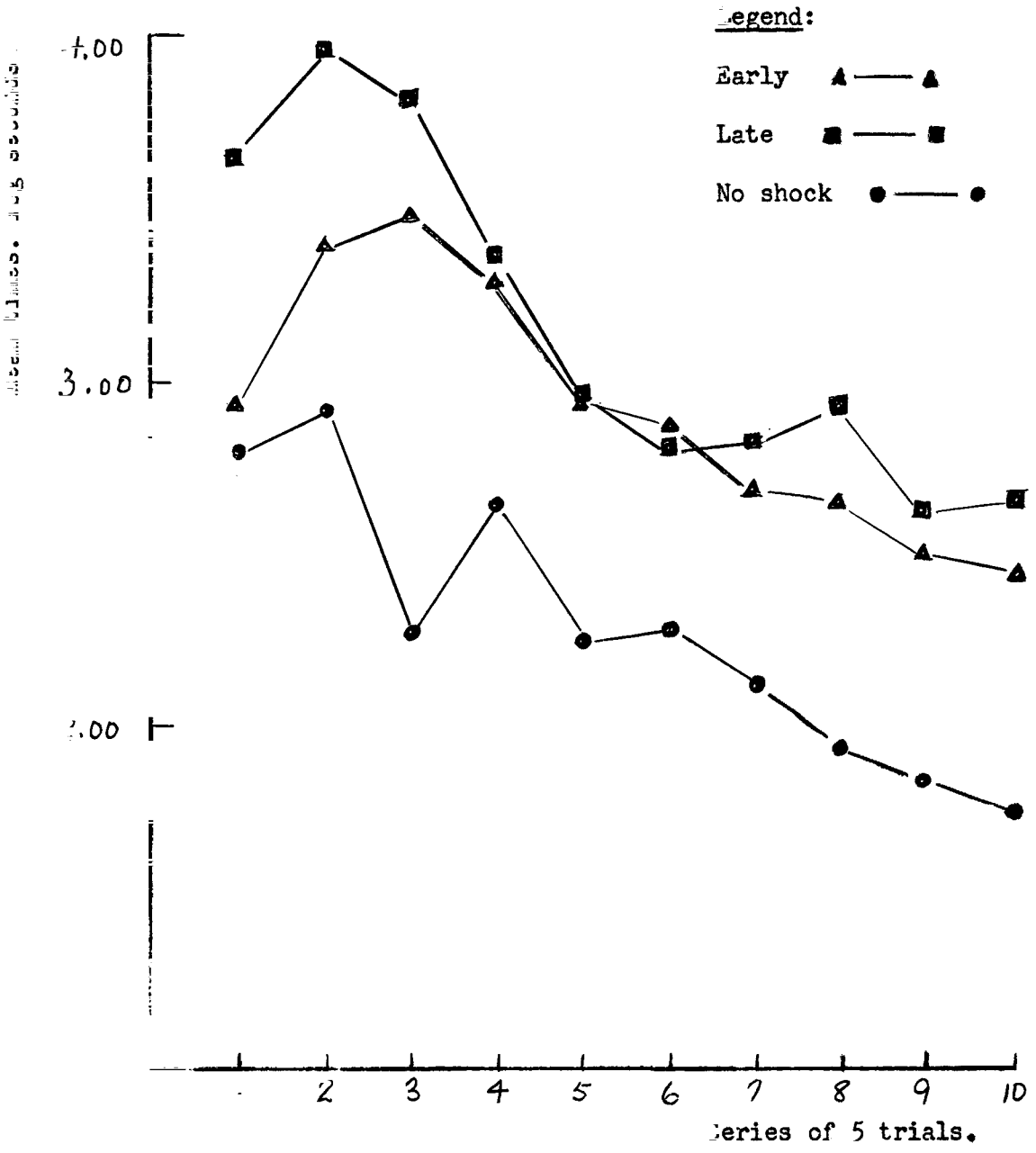
Mean Errors for Restricted, Shocked Correct Groups for Series of Five Trials



GRAPH III.

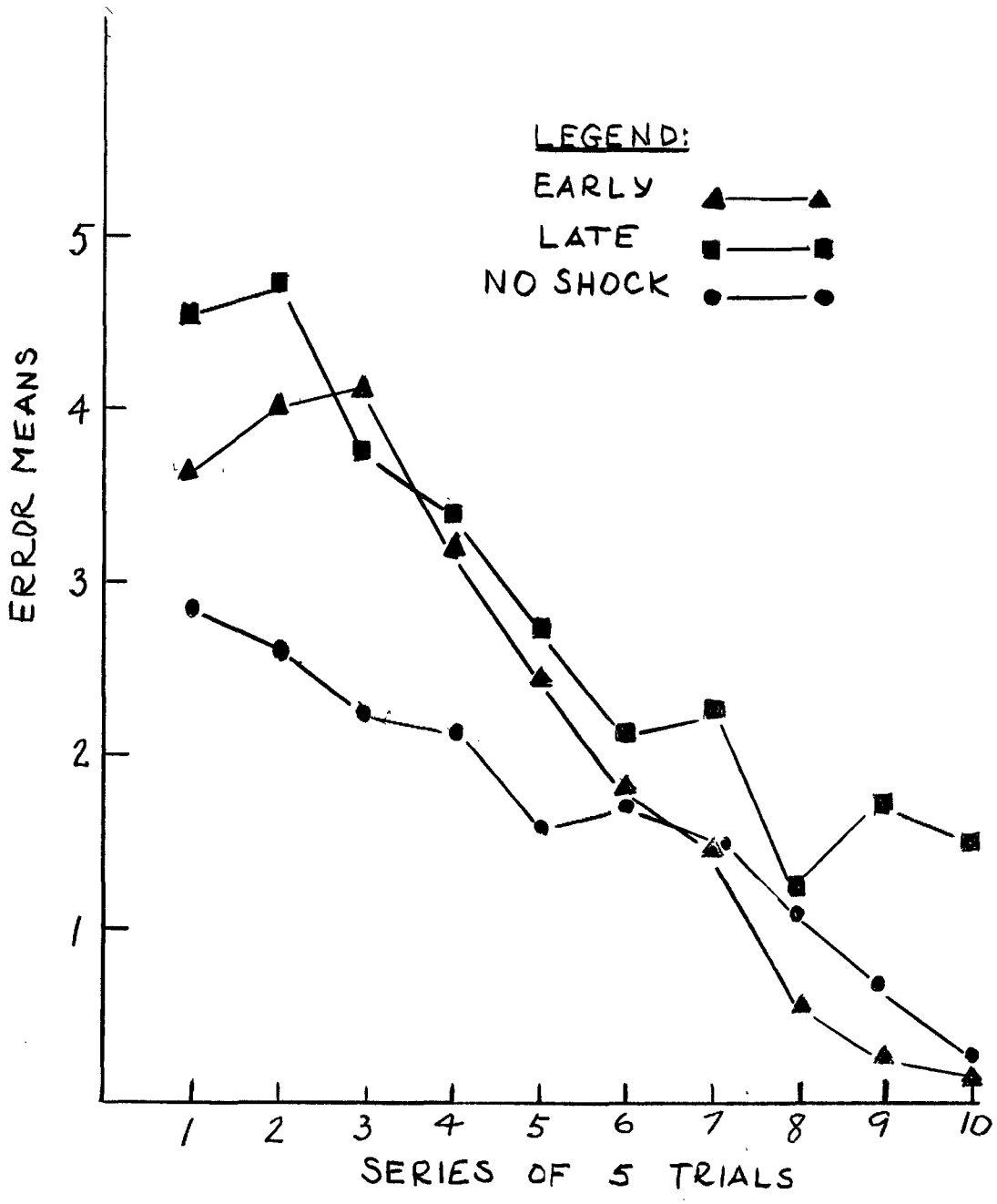
Mean Times for Free Environment, Shock Correct Groups

for Series of Five Trials.



GRAPH IV.

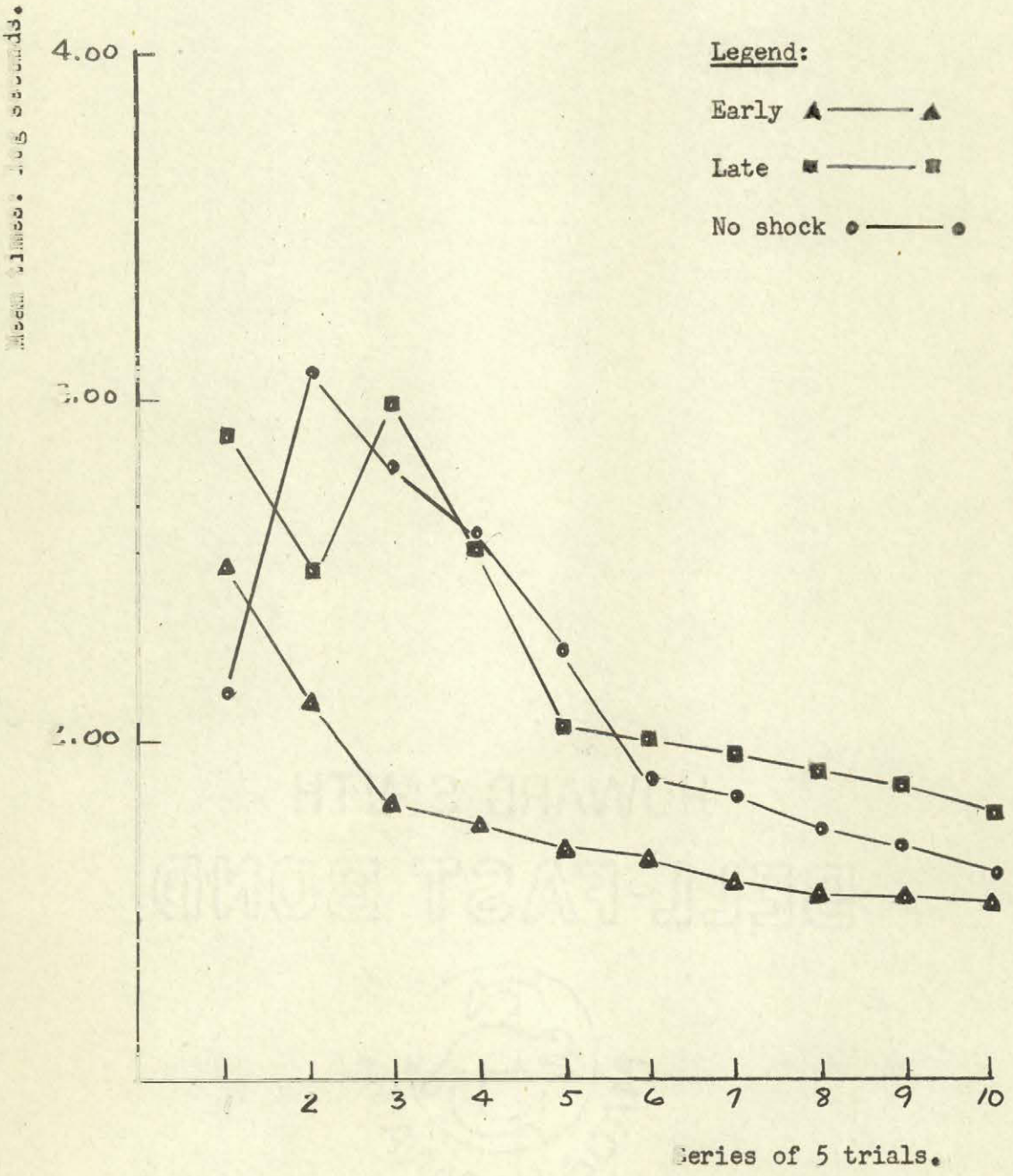
Mean Errors for Free Environment, Shock Correct Groups for Series of Five Trials.



GRAPH V.

Mean Times for Restricted, Shock Wrong Groups

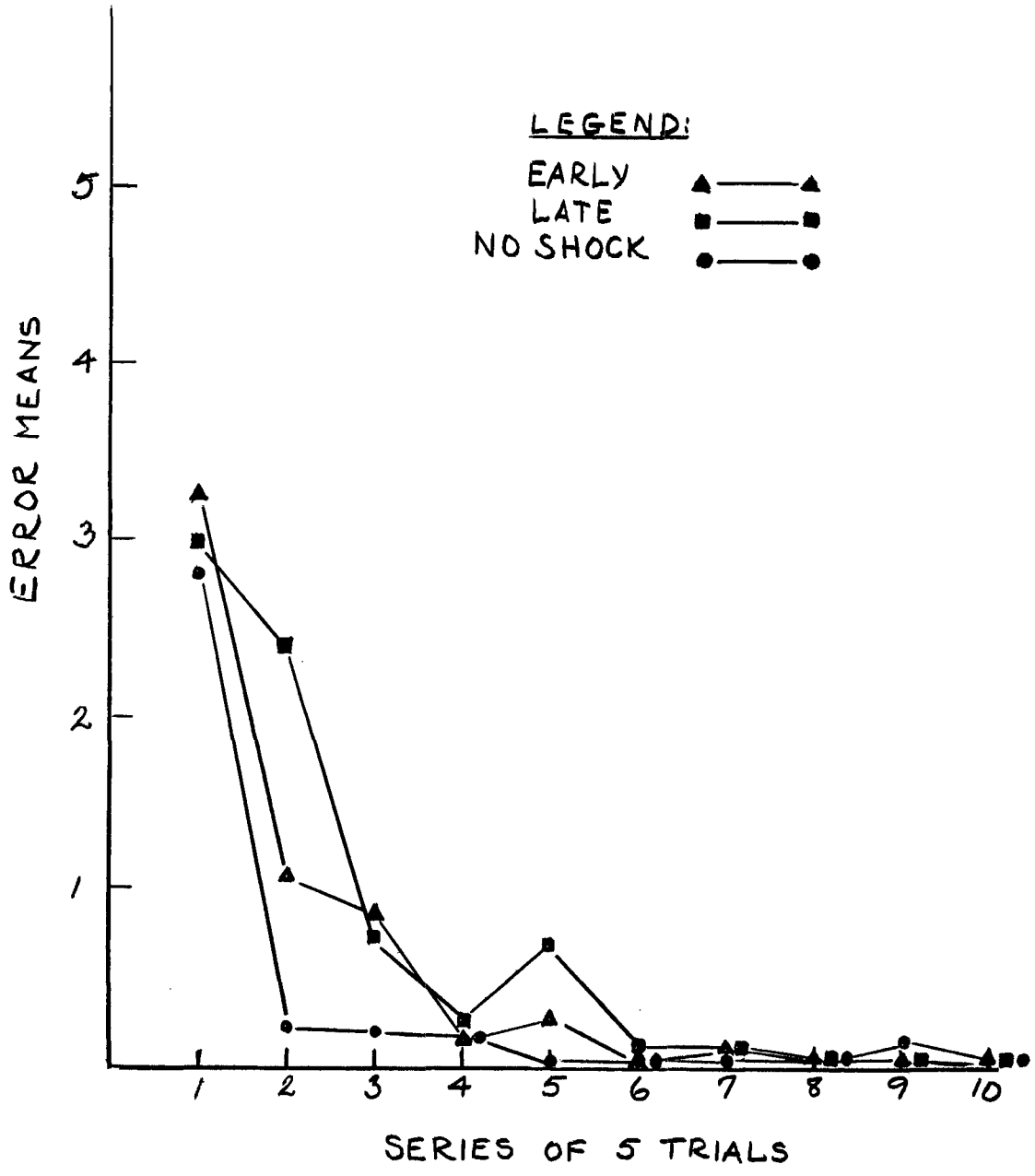
for Series of Five Trials.



GRAPH VIII

Mean Errors for Free Environment, Shock Wrong Groups

for Series of Five Trials.



and opposite effects in the early-shock and in the no-shock groups, the differences had the effect of canceling each other out. The probability values presented in Tables IV and V. which were obtained from t scores calculated from the time- and error-score variances, indicate that the restricted and free environments had significant effects on the learning scores. Thus, in the groups of rats that had no shock experience prior to testing, the restricted rats spent significantly more time in discrimination training and made more errors than the free environment rats. The opposite effect, however, was obtained in the groups that received electric shock in infancy: the free environment rats that were shocked during training for making the correct choice made significantly higher time- and error-scores than the restricted rats. There were no consistent differences between the restricted and free environment rats that received repeated electric shocks in their home cages at maturity.

Finally, the analyses of variance show that strong electric shock for correct response produced higher time- and error-scores than the same intensity of shock for the wrong response. The fact that there was a significant interaction among all the variables

indicates that the manner in which punishment was administered. that is, for correct or wrong response, had different effects depending on the earlier experience of the animals.

Supplementary observations. The groups that were shocked for the correct response were observed frequently to run to the correct door and make hesitant, approach-avoidance movements for a long period of time. They would then turn quickly and give the wrong door a brief push, returning immediately to the correct side and continue the approach-avoidance movements until they finally ran through. These observations suggested strongly that, despite the larger time and error scores made by the rats shocked for the correct choice, these animals had learned to make the discrimination as quickly as the groups that were shocked for the wrong response.

The time and error scores of a small number of control restricted and free environment rats, which were tested without shock in the discrimination apparatus, are presented in Table VI. There were no significant differences between the two groups in learning the brightness discrimination, indicating that the results that were obtained reflect real differences in the effects of a punishing electric shock on the various groups reared

TABLE VI

Mean Time in Seconds and Error Scores for Control Groups in Solving a Brightness Discrimination Problem

	Restricted		Free Environment	
	Time	Errors	Time	Errors
N:	6		5	
Mean:	609.8	23.8	722.4	22.4
Range:	421-816	18-33	490-1057	18-30

p for time difference: not significant.

p for error difference: not significant.

under different conditions.

DISCUSSION

The time and error scores that have been obtained with the discrimination apparatus may be used as indices of emotional disturbance due to electric shock stimulation. The differences among the groups, then, may be interpreted as representing the effects of the experimental variables on the emotional response of the adult rat to noxious stimulation.

The results have shown that the restricted rats that had no previous experience with noxious stimuli were significantly more disturbed by the electric shock than the comparable free environment group. The results of Mowrer and Viek (1948) and Hunt and Brady (1951a, 1951b) suggest that the free environment rats have learned to perform an instrumental response to noxious stimulation. Fighting, being bitten, and possibly other forms of noxious stimulation that occur in a free, social environment may have provided an opportunity for the rats to acquire responses which are capable of decreasing emotional disturbance following noxious stimulation. The restricted rats, who were alone in their rearing cages, did not have an opportunity to acquire similar

esponses. The results indicate that a multi-stimulus, ocial environment. without repeated intense stimulation, s an optimal situation for acquiring the appropriate esponses to noxious stimuli. Furthermore, the absence r somesthetic noxious stimulation during the development of the restricted rats may have resulted in fear r the strangeness of the new form of stimulation, as ell as of the noxious stimulus itself.

n the free environment groups, the rats hat were shocked in late infancy were significantly ore disturbed during discrimination training than the ree environment rats that had no previous experience ith electric shock. These results support the findings r Ried (cited by Griffiths and Stringer, 1952) that ntense noxious stimulation in late infancy produced decrement in learning at maturity.

he results with the rats that received ontinued shock experience at maturity suggest that the pportunity to learn to avoid repeated noxious stimuation is more advantageous if it occurs in infancy han at maturity. The older rats may have received any more shocks than the early-shock groups, before hey learned to avoid the noxious stimulus, and this

may have led to a greater generalization of the disturbing effects of the electric shock.

In view of the recent criticism by Beck et al (1953) of the Licklider apparatus (1951), the possibility remains that the smaller, young rats may have got a less intense shock, and that it may have been easier for them to learn to avoid the noxious stimulation. The lack of dependable shock apparatus prevents a general conclusion regarding the relative effects of early- and late-shock experience.

The significant interaction between age and environment indicates that the adult response to noxious stimulation is, in part at least, a function of the age at which the organism acquires experience with noxious stimuli, and the environment in which the experience is obtained.

These results reflect the complexities of pain mechanisms which were discussed earlier in the review of the literature on pain. Previous experiments in psychology that used animal subjects have tended to neglect the effects of early sensory experience, and treated the response to noxious stimuli simply in terms of the strengthening or weakening of stimulus-response connections. The early sensory experience of the organism, including experience with painful

stimuli, has been shown in this experiment to determine to a large degree the effects of noxious stimulation on the mature organism in a learning situation.

EXPERIMENT II

The purpose of the second experiment was to make a further investigation of the effects of early sensory experience on the adult response to noxious stimulation. The differences between groups of rats raised in restricted and in free environments were sufficiently significant in the first experiment to suggest that it would be desirable to make similar observations with a higher species, such as the dog. The dog's greater expressiveness, or variety of modes of response, should provide a better opportunity for analyzing the behaviour in first exposure to noxious stimulation. The technique which was used was to deprive the dogs, as far as possible, of some kinds of normal sensory experience, particularly pain.

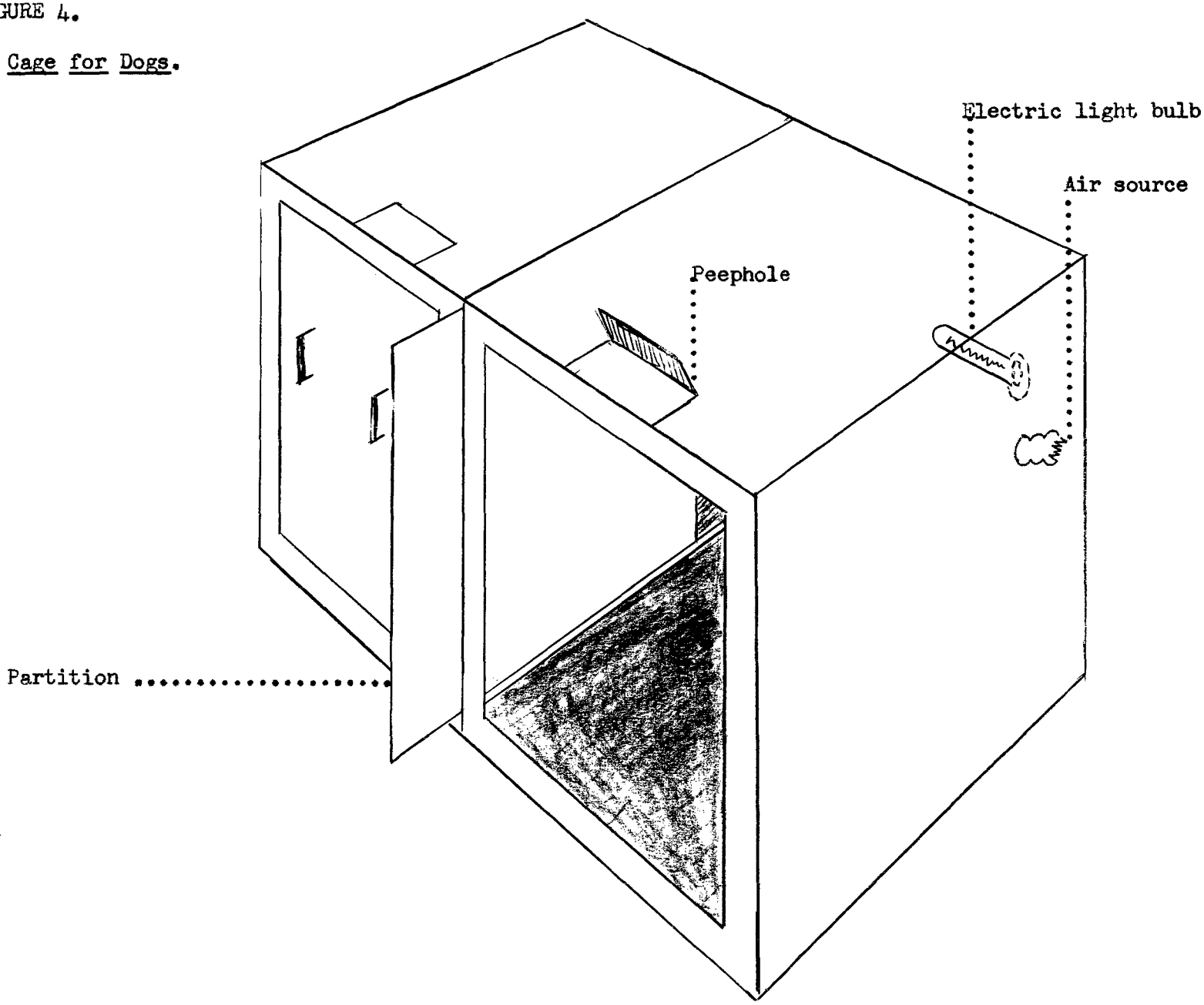
METHOD

Subjects. A litter of three puppies and three other laboratory-reared dogs, all from an inbred Scottish terrier strain, were used. Two of the puppies, Sandy and Clipper, were each placed in a specially constructed cage. The remaining littermate, Lochie, and the other three control dogs were reared normally.

Rearing. The method of experimental sensory restriction has been described in earlier papers

FIGURE 4.

Restriction Cage for Dogs.



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(Clarke et al, 1951; Thompson and Heron, 1954) from this laboratory. The cage that was used for rearing each of the restricted dogs in the present experiment is illustrated in Figure 4. Each cage contained two compartments, and by opening a sliding partition, the dog was allowed to enter a freshly-cleaned compartment every day. The floor of the compartment was covered with a two- or three-inch bed of paper strips. All the edges inside the cage were lined with sponge rubber.

The dogs were placed in the cages after weaning was completed at the age of four weeks. They were removed at eight months of age. Since the electric light source was in one compartment only, the dog spent every second day in diffuse daylight which passed through the top of the cage.

Lochie was reared in a private home, and the other three control dogs were brought up in the laboratory. All four received the usual training and varied environment.

Test apparatus. A high-voltage and low-amperage electric-shock source was provided by a conductance-capacitance circuit described by Licklider (1951). A toy car which was controlled by hand through

a long wire was connected to the shock source. In this way, the dogs received a strong electric shock each time they were touched by the moving toy car. The electric shock source could also be connected to the metal floors of the restriction cages and laboratory testing enclosure.

Procedure. The restricted dogs were first tested with noxious stimuli in their home cages. The floor of each compartment was divided into two halves by a strip of black wood about three inches high. During a ten-minute period each day the dogs received an electric shock whenever they were on the left side of the dividing strip of wood. This procedure was carried out on five successive days with the restricted and normally-reared dogs.

The second test was made one week later. The restricted dogs were removed from their cages and tested one at a time in an enclosure, 6 feet by 3 feet, bounded by wire mesh two feet high. A long black strip of wood divided the metal floor into two halves. All the dogs received an electric shock for a full second when they were on the part of the floor that was closest to the experimenter. They received no shocks when they were on the other side

of the dividing strip. Each dog was tested for ten minutes a day for five days.

In the third test, the toy car was moved around the dog in the enclosure to observe the responses to the car itself. After five minutes, the dog received an electric shock each time the car touched him. Each of the restricted and control dogs were tested in this way for ten minutes a day for five days. The duration of the shock was usually one second, although dogs that moved away rapidly did not get the full shock.

Supplementary observations were made in a large empty room. The responses of the dogs to repeated pin pricks and to contact with lighted matches were recorded.

OBSERVATIONS AND RESULTS

The behaviour of the dogs was observed and classified in consultation with other psychologists who were working with the same colony of dogs. The responses of some of the dogs in the second and third tests have been recorded on film.

1. The first test in the restriction cages indicated that the restricted dogs were less disturbed by electric shock than the normally-reared dogs.

evels of electric shock that elicited squealing and umping in the normal dogs produced only increased ctivity in the restricted dogs. When the intensity r the shock was raised so that the restricted dogs squealed and jumped to the shock, the normal controls elped loudly long after the duration of the shock, nd jumped violently around in the compartment. None r the dogs learned to avoid the side of the compartment on which they received the shock.

. In the second test, the control dogs voided the side on which they received electric shock fter a mean of 4.5 shocks. Neither of the restricted ogs avoided the place of shock permanently after aving received fifty shocks. This difference is ignificant at the 0.1 percent level.

ualitative differences between the two roups were observed. The normal dogs avoided the hock-side after yelping and jumping to the shock. he restricted dogs jumped and squealed, but appeared ess disturbed by the same intensity of shock. Furthermore, their emotional disturbance diminished during he first five trials, and no emotional responses to he shock were observed from the sixth to the thirtieth rials. The intensity of the shock was raised twice

with the same sequence of events. At a very high intensity of shock, however, the restricted dogs squealed at every shock but, unlike the control dogs, did not avoid the side of the enclosure where the shock was administered.

3. In the test with the toy car, the normal dogs learned to avoid the noxious stimulus permanently, after having received a mean of two shocks.

The two restricted dogs showed different behaviour to the toy car. During the first twelve trials, Clipper squealed immediately with each shock, but continued to sit in the center of the enclosure without making any attempt to run away. On the thirteenth shock he jumped up after being shocked, and his behaviour with the following seventeen shocks was categorized as diffuse emotional excitement: he dashed away from the toy car for about two feet with highly excited and exaggerated movements of the whole body, and then rushed back into it, frequently receiving a series of shocks repetitively. Clipper exhibited this behaviour only after getting the shock, but not to the toy car itself before it shocked him. During the thirty-first to the fiftieth trials, however, Clipper responded to the approach of the car, and

succeeded in avoiding the shock continually. His excessive activity decreased during these trials, and the final responses were marked by the same precise avoidance behaviour of the normal control dogs.

For the first fifteen trials Sandy sat in the same place in the enclosure, and made no observable emotional response to each shock. From the sixteenth to the twenty-fourth trials, however, he squealed loudly with the shock, but did not run away from it. At the twenty-fifth trial, Sandy showed a type of freezing response to the toy car. Whenever the car started to move, the dog ran to the same corner of the testing enclosure and sat tensely in the same position with his back to the car, and his head facing 180 degrees away from its line of approach. This behaviour continued until the end of the fiftieth shock trial.

Supplementary observations. No avoidance responses were observed in the restricted dogs, after the above tests were completed, when they were pinched, hit on the head or legs with metal rods, or had their skin pierced by a large pin in the abdomen, chest or paws. They continued to prance excitedly near the

experimenter, with no evidence of increased excitement. Each of the 3 control dogs yelped at the first of each of these manipulations, and could not be tested a second time.

The restricted dogs also walked into lighted matches repeatedly. Each poked his nose into the flame, withdrew for a few inches. apparently reflexively, and then walked forward into the match again. They continued in this manner for five days of testing, walking five or six times daily into each of two matches.

One striking feature of the restricted dogs' behaviour when they were out of their cages was the high level of aimless activity. It was observed during rest periods that this resulted in their frequently striking their heads against the water pipes that ran along the walls just above the floor. One dog, by actual count. struck his head against these pipes more than thirty times in a single hour. This was never observed once in the normal dogs. Furthermore, the rapid movement of the restricted dogs and its unpredictability as to direction resulted a number of times in the dogs having a paw or the tail stepped on. There was no sign whatever that the dogs felt pain when this happened, though it would have elicited a howl from a normal dog, and no attempt was made to withdraw

from the place where the injury was received.

DISCUSSION

The present investigation indicates that early sensory experience determines, in part at least, 1) the direction and type of overt response to noxious stimulation, and 2) the threshold of response.

The behaviour of the restricted dogs to the toy car has demonstrated that the first overt motor response to noxious stimuli is undifferentiated emotional excitement. The emergence of avoidance behaviour in one of the dogs also indicates that adaptive emotional behaviour occurs only after the organism has had actual experience with the noxious object. The development of a type of freezing behaviour in the other, however, shows that maladaptive behaviour may emerge from the emotional excitement and remain as a consistent response to the noxious stimulus. The experiment with the toy car adds further support to the view that diffuse emotional excitement is a primitive disturbed response out of which avoidance and other forms of emotional behaviour develop (Bridges, 1932; Hebb, 1949; Melzack, 1952).

The heightened response threshold in the restricted dogs to various types of noxious stimuli may be due to a number of different reasons.

1. In an environment that is totally unfamiliar, the behaviour of the restricted dogs is such as to indicate a continuous and profound emotional excitement (Melzack, 1954). Thus, an unusually high intensity of noxious stimulation may be necessary for the noxious stimulus to have an effect that is distinct from that of the other unfamiliar stimuli present in the environment.

2. In their home cages, the restricted dogs may have responded at a threshold level that is uninfluenced by previous experience with noxious stimuli. Thus anticipatory fear may have been present in the control dogs, so that they responded at a subthreshold level of noxious stimulation and were more disturbed by it (see Mowrer and Viek, 1948; Hill et al, 1952b).

The results obtained with the dogs may be compared with those obtained with rats in Experiment I. When the restricted dogs were first tested out of their cages, the electric shock appeared to have little disturbing effect. However, once the dogs started to respond, they showed great emotional disturbance.

This then decreased after they learned to avoid the noxious stimulus. The sharp rise and fall in time scores for the restricted rats with no previous shock experience, shown in Graphs I and III, indicate a comparable sequence in behaviour. The fact that the dogs actually had to acquire an avoidance response to the electric shocks, as well as to grossly injurious stimuli such as contact with fire, also suggests that, with increasing mammalian development, early experience plays an increasingly important role in determining the animal's responses to painful stimuli. Everyday observations of human behaviour related to pain, such as those recorded by Livingston (1953), have suggested that experience with pain in childhood is an important determinant of the manner in which the individual will respond to pain at maturity. The results obtained in these experiments provide empirical evidence for this type of observation.

The historiaal review of the literature on pain indicated that most of the earlier treatments of the problem of pain in animal psychology have tended to neglect the material obtained by observations and studies of human subjects. The experiments which have been reported here indicate that early experience determines to a large extent the degree to which a

noxious stimulus will disrupt behavior at maturity. These results make it impossible to treat the response to painful stimuli simply in terms of frequency and intensity of stimulations, without regard to the physiological processes which intervene between stimulus and response. Animal studies of the response to noxious stimulation must also recognize that central states, largely acquired in early experience, determine the actual capacity of a given stimulus to disrupt behavior, so that stimuli which are normally capable of having disruptive effects may not elicit emotional disturbance, or stimuli which usually have little effect may produce intense emotional disruption. These results provide a beginning to the study of pain in animals which is consistent with the evidence and hypotheses on the nature of pain which has been achieved from the study of human subjects.

SUMMARY

Two experiments have been done on the effects of early experience upon the perception of and response to pain. Both experiments indicate great differences in these respects, depending upon the conditions of rearing; in particular, the behavior of dogs reared without opportunity for contact with noxious stimuli is grossly abnormal. The dogs appeared not to perceive pain as such: at least, not for a considerable period after being placed in a normal environment.

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