

CHANGES OF BEHAVIORAL STRATEGY AS A MECHANISM OF RELEARNING INSTRUMENTAL RESPONSES AFTER CEREBRAL LESIONS

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Abstract. Several examples of functional recovery of instrumental responding after cerebral lesions are reviewed. Small incision on the boundary between the sensory and the motor cortex of a dog changed the saliency not only of the tactile but also of the auditory conditioned stimuli, eliciting the preoperatively acquired alimentary instrumental response. Prefrontal lesions in cats affected differently each of the several learning processes involved in the performance of the avoidance response: they enhanced the classically conditioned component, attenuated the motivational role of fear but left the motor component intact. Preoperatively acquired interrelations of these components underwent substantial changes, leading to the restoration of the previous level of avoidance of pain. However, a highly adaptive mode of behavior, namely avoidance of the state of fear, was permanently impaired. After small electrolytic lesions of amygdalar nucleus centralis cats were handicapped in the acquisition of escape from pain responses. The responses to low shock intensities were especially diminished. Improvement of the escape from pain responses was observed after a prolonged increase of the intertrial response frequency. The effectiveness of both escape and avoidance learning depended on the duration of the postoperative recovery period prior to instrumental training. All these data indicated that ce-

rebral lesions changed the interactions between different parts of the neuronal circuitry participating in the learning and performance of a given instrumental response.

INTRODUCTION

Functional recovery from brain damage has commonly been explained as a result of either changes of the features of the structural elements or changes of the wiring diagram. In this paper changes of behavioral strategies by lesioned adult animals will be considered. The factors influencing the effectiveness of such changes may be independent of the recovery processes taking place within the damaged structure (16). Most often one employs retention and relearning of rather complex kinds of behavior to measure the extent and amount of functional recovery. Interactions between the brain structures involved in such kinds of behavior are also complex. The patterns of those interactions are formed during the ontogeny and specific preoperative training. A large number of data indicate that the severity of the behavioral impairment after the same brain injury depends on the history of the adult subjects (11). The reason why preoperative experience changes the susceptibility to the effects of specific brain lesions is not sufficiently clear. It has been postulated that diversified associative learning, similarly to enriched environment, generally increases the adaptive potentiality of the individual (1, 12, 13). Independently of the secondary cell loss problem, a lesion placed in one structure changes its interactions with others and, consequently, may severely damage the complex neuronal circuit established during the preoperative training. Some specific patterns of interactions between brain structures established in preoperative learning may be more influenced, and some other patterns less influenced by the distractions caused by a given lesion. The most efficient adaptive response of the lesioned subject consists in a change in behavioral strategy and in using preserved links in the neuronal circuitry for the emission of the required response. It is obvious that the possibility of changing the behavioral strategy is dependent on the task, and also on the extent and place of the lesion. Maze learning provides much more opportunity for changing the behavioral strategy than do specific sensory tasks (6).

PROPERTIES OF THE "SPECIFIC TACTILE STIMULUS"

A very instructive analysis of the behavioral consequences of a limited lesion in sensory-motor cortex was made by Konorski and his coworkers in the sixties (2-4, 10, p. 427-432). Dogs were trained in the

alimentary instrumental response of lifting the right foreleg. A typical food reward method was used. For each dog several conditioned stimuli were used. The elicited response was the strongest to the auditory and the weakest to the tactile stimulus applied to the trunk. Then the same tactile stimulation, but acting on the distal part of the leg performing the instrumental movement, was introduced. This conditioned stimulus was termed the "specific tactile stimulus" because of its unique properties. It elicited instrumental responses with the shortest latencies, being effective even in fully satiated dogs and very resistant to extinction (2-4). Moreover, the introduction of the "specific tactile stimulus" resulted in the weakening of the instrumental response to all other conditioned stimuli. Then a small lesion was done, namely an incision within the contralateral posterior sigmoid gyrus extending from the postcruciate to the coronal sulcus. As was shown later (7), that incision separated only those parts of the "precentral" motor area and the somatosensory area I from which arm movements are elicited under electrical stimulation.

After the lesion, the instrumental response, namely lifting the right foreleg, was elicited without any difficulty by all conditioned stimuli used before. However, this type of lesion immediately abolished all of the features of the "specific tactile stimulus", including its high resistance to extinction. Moreover, after the lesion the auditory conditioned stimulus ceased to be suppressed by the "specific tactile stimulus" and regained its previous resistance to extinction.

Two peculiarities of these data have to be stressed. First, the introduction of a very salient stimulus, presented in separate trials but interspersed between other conditioned stimuli, weakened the effectiveness of less salient stimuli. Clearly, there is interaction between processes elicited by different conditioned stimuli. Secondly, cutting of the U-shaped fibers connecting the somatosensory and the motor cortical areas considerably weakened responding to the tactile stimulus applied to the foreleg and without any additional training enhanced responding to the auditory stimulus. The lesion intervening in a small fragment of the neuronal circuitry changed the efficacy of the intact fragments of the wiring diagram.

DEFENSIVE RESPONSES AFTER PREFRONTAL LESIONS

Avoidance responding provided very convincing evidence of the change of the behavioral strategy after prefrontal lesions (21). We will consider here the basic results obtained from cats trained in the bar-pressing avoidance response. After reaching the required criterion of performance,

the preoral and dorsomedial areas of the prefrontal cortex were removed. This lesion resulted in decrease of avoidance performance, but postoperative training was effective in the recovery of the original level of shock avoidance. As shown in Fig. 1, the short-latency avoidance responses permanently deteriorated and the recovery was due to the compensa-

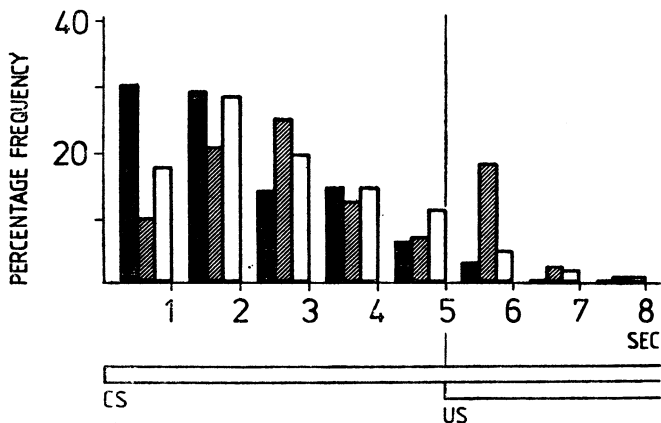


Fig. 1. Frequency histograms of the latencies of bar-pressing responses before (black bars), after (hatched bars) prefrontal lesions, and after postoperative re-learning (open bars) in cats trained in the avoidance responses to the 70 dB white noise CS with the 5 s CS-US interval. Note the pronounced postoperative deterioration of the avoidance responses performed with latencies shorter than 1 s and only partial recovery of this class of responses after postoperative relearning.

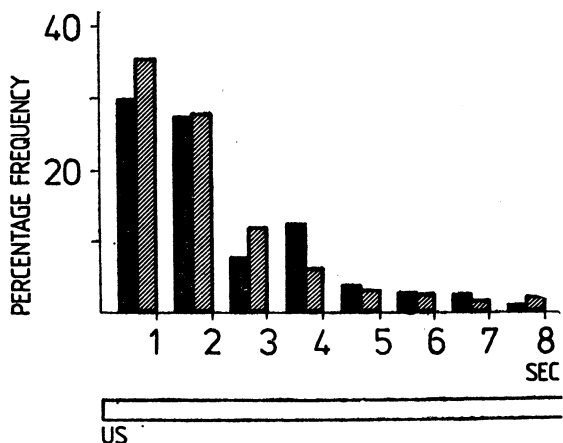


Fig. 2. Frequency histograms of the latencies of bar-pressing responses before (black bars) and after (hatched bars) prefrontal lesions in cats trained in the escape responses from unsignalled shock. Note the absence of the effect of lesion on any class of escape responses.

tory increase of the avoidance responses performed with longer latencies (17). The decrease of short-latency avoidance responding cannot be related to a motor disfunction, since the same lesions do not change the ability to perform short-latency escape from unsignalled shock responses (Fig. 2).

In a series of experiments we have shown that different mechanisms are involved in the performance of the short- and long-latency avoidance responses. In the course of regular avoidance training several different modes of responding are acquired (8, 20). At the very beginning the animals execute bar-pressing responses, which terminate the action of the footshock (escape from shock responses). Since each painful stimulation is preceded by a warning stimulus, the classical defensive response was rapidly acquired evidenced by the enhancement of fear after the onset of the conditioned stimulus. Only then the avoidance of shock responses occurs. When they were performed on a substantial percentage of the trials we observed a rapid increase in the performance of another class of responses: avoidance executed with very short latencies. All avoidance responses are reinforced by the prevention of painful stimulation but the short-latency avoidance responses are particularly reinforced by the termination of the fear-evoking conditioned stimulus (22). Consequently, we applied to these short-latency bar-presses the term avoidances of fear in contrast to the long-latency avoidance of pain.

The above classes of defensive responses differ in the ways in which they are affected by prefrontal lesions. Escape from shock and long-latency avoidance responses are undisturbed. Defensive responses acquired according to the classical conditioning paradigm are even enhanced after prefrontal lesions. On the contrary, short-latency avoidance responses, the most effective in protecting an organism from the danger situation, are permanently impaired after prefrontal lesions. Prefrontal lesions destroy only one fragment of the neuronal circuitry involved in avoidance responding, whereas the other fragments are left intact and may be used for behavioral recovery.

The interrelations within the neural circuitry are formed in the original learning. We may force the subjects to use different strategies for learning a given task. One group of cats was trained first to escape from the shock and only later, after the introduction of the warning stimulus, to avoid the shock. Another group was first confronted with the warning stimulus and inescapable brief footshock (classical defensive training), and only then the prolongation of the action of both stimuli made it possible to terminate them performing escape or avoidance response. The final preoperative level of the avoidance per-

formance was the same in both groups. However, as shown in Fig. 3, prefrontal lesions produced much smaller impairment of avoidance responses in the escape group than in the classical pretraining group (18). This kind of interaction between intervening and training variables is very often neglected by researchers.

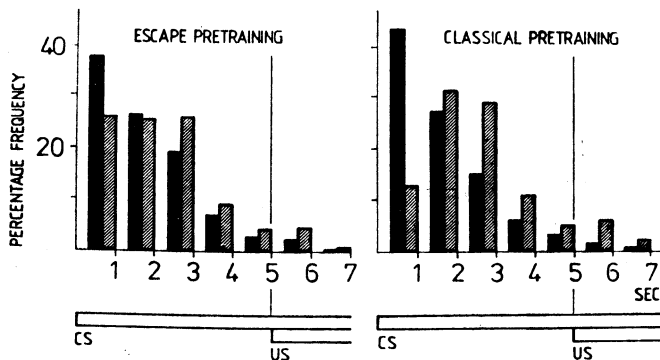


Fig. 3. Frequency histograms of the latencies of bar-pressing responses before (black bars) and after (hatched bars) prefrontal lesions in cats trained in the avoidance responses to 2,000 Hz tone of 60 dB intensity CS with the 5 s CS-US interval. Escape pretraining means that the cats were first trained for three days to escape unsignalled shock and only then the CS was introduced. Cats from the classical pretraining group were presented during the first three days with the CS signalling an unavoidable brief shock, and from the fourth day both stimuli were prolonged until the performance of the instrumental response. Note the marked differences between the groups in the amount of postoperative deterioration of short-latency avoidance responses.

DEFENSIVE RESPONDING AFTER AMYGDALAR LESIONS

Apart from the deterioration of specific functions, limbic lesions typically produce numerous changes in the spontaneous activity level and the organism's reactivity to its environment, in emotional responsiveness and fearfulness (5, 9). In the course of behavioral experiments the "spontaneous" activity of an organism may be realized in a form similar to the behavior evoked by a discrete conditioned stimulus but performed in intertrial intervals (intertrial response, ITR). An analysis of the data from experiments on intact animals indicates that increase in task difficulty leads to the enhancement of intertrial responding, and the change to more adequate behavioral strategy is followed by the decay of intertrial responding (19). The frequently observed enhancement of intertrial responding after prefrontal lesions has been considered in early papers as an index of the disinhibition syndrome. Our other data

obtained from cats and dogs after prefrontal lesions (21), and from cats and rats after lesions in the central nucleus of amygdala suggest other interpretation.

As an example, data will be presented from normal cats and cats with electrolytic lesions of the amygdalar central nucleus trained in bar-pressing escape from unsignalled footshock (14). In all subjects bilateral lesions were symmetrical and included only a fragment of the dorsolateral part of the central nucleus without injury of the stria terminalis (Fig. 4). Prior to training, one group of the cats rested after lesions in their home cages for 10 days and the other group — for 35 days.

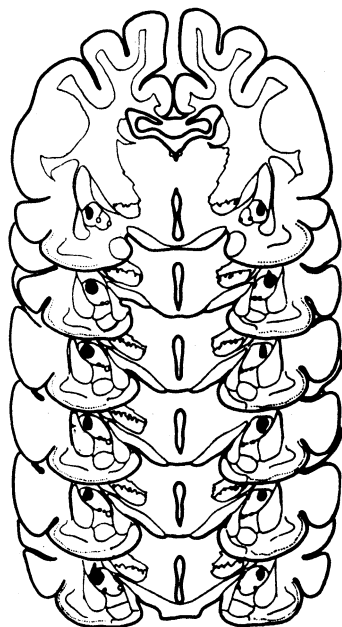


Fig. 4. Reconstruction of lesions in the amygdalar central nucleus through areas of maximum tissue destruction in individual cats of one experimental group.

The best index of the efficacy of escape training is the shortening of the latencies of responses terminating the shock. This process was rapid early in the training and more gradual afterwards. As shown in Fig. 5, at the beginning of training escape latencies and the frequency of intertrial responses were the same in all groups. In the second block of 50 trials there were marked differences in the response latencies, which were the shortest in the normal cats, longer in the cats starting training 35 days after lesions, and very long in the cats starting training 10 days after the lesion. The rapid shortening of the escape latencies was accompanied by a marked increase of ITR frequency in all groups.

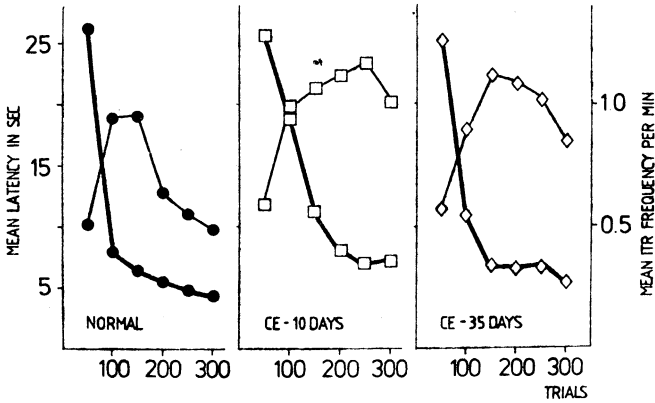


Fig. 5. Changes of the mean response latency (thick line) and mean frequency of intertrial responses (thin line) in 50 trial blocks in the normal cats (the left panel), cats which started escape training 10 days after amygdalar central nucleus lesions (the middle panel), and cats which started training 35 days after such lesion (the right panel).

Such enhancement of intertrial responding indicates the elevation of behavioral arousal. The most rapid rise of ITR was observed in the normal cats. The cats starting training 10 days after the lesions, the same group which had been particularly handicapped in shortening the escape latencies, was the slowest in attaining maximal ITR frequency. The subsequent decline of intertrial responding in each group followed the time course necessary to reach the asymptotic level of response latency

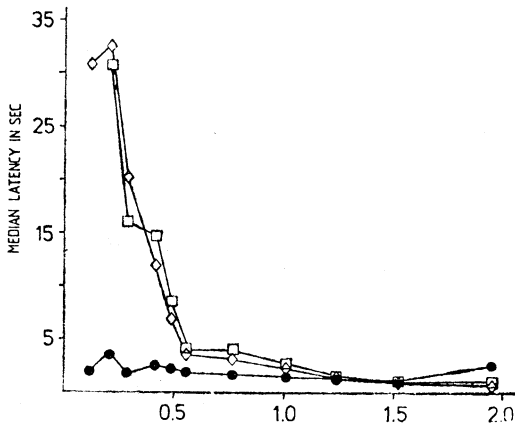


Fig. 6. Median latencies of escape responses to footshocks of different intensities applied during the test sessions. Circles denote the group of normal cats, squares denote the group starting training 10 days after amygdalar central nucleus lesion and diamonds denote the group starting training 35 days after such lesion.

duration. The asymptotic escape response latency was shorter in normal and longer in amygdalar cats.

In this experiment, the regular training sessions were interspersed with several test sessions, in which shocks of different intensities were applied. Normal cats responded to all intensities of shock with similar latencies provided that the shock threshold level was exceeded (Fig. 6). The shock threshold was higher for a large number of lesioned cats and, moreover, they responded to low shock intensities with longer latencies which decreased progressively with the increase of shock intensity. A similar rise of the shock threshold was observed in another study not only after nucleus centralis but also after amygdalar nucleus lateralis lesions (23).

The lowered shock sensitivity, or rather the ability, decreased after amygdalar lesions, to excite stimulus-motor associations when the evoking stimulus was weak, could be partially compensated by the rise of behavioral arousal and increase in the activity level evidenced by intertrial responses. The potential use of this mechanism of compensation seems to be dependent on the time span between the lesion and the beginning of training. The data presented in Fig. 5 demonstrate that the rise of ITR frequency is more rapid and the decline of the escape response latencies greater in amygdalar cats starting training 35 days after the lesion than in cats with only 10 days of postoperative recovery. Similarly, in another study the cats which started training in the bar-press avoidance of shock response 35 days after amygdalar nucleus centralis lesions showed the highest ITR frequency and only moderate impairment of avoidance performance (15). In the same study the cats which started training 10 days after lesions responded with the longest latencies, emitted a small number of intertrial responses and reached the required criterion after training twice as long as normal cats.

When the cats were trained to escape from shock of higher intensity (1.8-2.8 mA instead of 0.8-1.4 mA used in other experiments), the performance of the cats with amygdalar central nucleus lesions was in spite of their lower ITR frequency only slightly impaired in comparison with normal cats (23). Thus, stimulus characteristics seem to be important in tuning the intertrial responding level.

We suspect that the rise of intertrial responding is an adaptive response to the increase of the task difficulty. Such increase of ITR frequency is usually observed at the beginning of each consecutive stage of the training, when more complex interactions between the conditioned stimuli, the reward or punishment, and the required responses are introduced. For example, a marked rise of the ITR frequency is observed after the reversal of the signalling properties of the conditioned stimuli

(21). A similar increase of the task difficulty may be obtained without any change of the formal requirements of the task when the subjects' capabilities are lowered, e.g. owing to brain lesions. Thus, the increase of the intertrial responses frequency commonly observed after cerebral lesions ought to be considered in many instances as an adaptive response, whose effectiveness may be evaluated by a gradual improvement of performance and subsequent decline of the ITR frequency.

CONCLUSIONS

During the last decade considerable attention has been paid to the environmental attenuation of brain-lesion symptoms. The present paper considers this general problem from a somewhat different angle, concentrating on modulatory effects of the training situation. All the experimental data demonstrate complex interactions of neural processes involved in the learning and performance of behavioral acts. These interactions utilize inborn connections between centers but are formed in the original learning. The introduction of new conditioned stimuli, the order in which a subject has mastered a particular element of a complex task, all of them influence the formation of neuronal interactions. Differences in the originally established neuronal circuitry bring about clear effects on the postoperative performance of the lesioned subjects. It seems that comparisons between groups which differ not in the amount of learning (the overtraining effect) but in the kind of learning (the pretraining effect), leading to comparable task performance, provide an interesting approach to the problem of environmental preoperative effects on brain lesion symptoms. Similarly, very small lesions in the central nucleus of amygdala make it possible to retrace behavioral strategies employed by lesioned cats to learn the instrumental defensive responses. It is interesting to investigate whether similar modulatory effect of the increased intertrial responding would be observed after large subtotal lesions of the amygdalar complex routinely employed in most of the studies concerning the functions of that limbic structure.

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