troduced during the 2-hour periods between the first and second and between the third and fourth sessions of food reinforcement. Hence, sessions of food reinforcement immediately preceding avoidance sessions were accompanied by the houselight; those immediately following avoidance sessions were accompanied by the tone.

As before, similar results for the two groups of daily sessions permitted pooled data as for Fig. 1. Again, responding reinforced by food during days 26 to 30 of avoidance conditioning was suppressed relative to that before avoidance conditioning (Fig. 2). In the 30-minute food sessions preceding avoidance sessions, the pattern of suppression was comparable to that in the 30-minute periods before avoidance sessions in the first experiment. In Fig. 2, less suppression is shown in the postavoidance food sessions than in preavoidance sessions, a result suggesting that the postavoidance suppression of the first experiment constituted at least partial stimulus generalization with sessions preceding avoidance sessions. Whether postavoidance suppression is entirely explained as generalization with preavoidance sessions is an open question, for the continuing partial suppression may have resulted from stimulation provided by the food-reinforcement schedules themselves as well as by static stimuli present in all conditioning sessions.

The slightly reduced suppression in this second experiment, compared to the first, accompanied more effective avoidance behavior, as revealed by lower shock rates. However, in neither experiment did an analysis of within-subject data reveal any simple relation between amounts of suppression in the food sessions and the animals' performances in the adjacent avoidance sessions.

The present experiments extend research on "conditioned anxiety" and suggest a redefinition of aversive events, even within the context of laboratory studies. They also present a technique for evaluating variables that affect not only behavior in aversive situations but behavior that is outside those sessions but is still affected by them. In addition, these studies raise questions regarding experiments where endocrine changes or other physiological variables are studied in subjects that are under the influence of avoidance conditioning or other stressful procedures. In most experiments each animal is tested at a given time each day and is exposed to the cyclical activities of laboratory routine. This could result in conditioned suppression on a grand scale, in the presence of whatever stimuli characteristically preceed the avoidance sessions, perhaps of a magnitude that dwarfs the within-session events that are usually observed.

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Independence of Short- and Long-Term Memory: A Neural System Analysis

Abstract. *Rats were given electrical stimulation to the midbrain reticular formation or to the hippocampus 4 seconds after they received shocks contingent on the animals' bar-press responses. They were retested for memory of the shocks 64 seconds or 24 hours after the shocks. The animals that received stimulation to the midbrain reticular formation showed amnesia at the 64-second retest and memory at the 24-hour retest. In contrast, animals that received stimulation to the hippocampus showed memory at the 64-second retest and amnesia at the 24-hour retest. The data support a dual, parallel-processing model of memory.*

A major issue in the study of memory is the number of memory systems necessary to process newly acquired information, and the interrelations among these systems. There are probably at least two processes, one for short-term memory (STM) and one for long-term memory (LTM) (*1–3*). If STM and LTM constitute different processes, then different neural systems should be involved. In order to identify the neural systems that subserve the two processes, relatively localized brain stimulation of subseizure intensity should be used to disrupt ongoing neural activity. Low-intensity electrical stimulation of either the hippocampus, amygdala, or centre median in cats or of the caudate in rats disrupts LTM of aversive information (*4, 5*). However, the effect of electrical brain stimulation on STM has not been elucidated. The purpose of the present study was to stimulate various neural structures of animals after they had an aversive experience and to test for retention at short and long intervals.

In the first experiment, 41 male Long-Evans rats, 230 to 270 g at the start of the experiment, were subjects. The animals were divided into three groups—two for brain stimulation, either to the midbrain reticular formation (MRF) ($N = 13$) or to the hippocampus ($N = 12$), and a nonstimulation control group ($N = 16$). All animals were anesthetized with Nembutal, 35 mg/kg, and had bilateral implants of bipolar electrodes, into the MRF (coordinates: 6.5 mm posterior Bregma, 1.5 mm lateral, 6.4 mm vertical), into the hippocampus (coordinates: 40 mm posterior Bregma, 2.0 mm lateral, 4.3 mm vertical), or into the skull for the control group. The electrode assembly was fixed to the skull with acrylic cement. After recovery from surgery all animals were reduced to 80 percent of their initial weights and maintained at these weights. They were then tested, by an ascending method of limits, for the intensity of current required to produce a behavioral withdrawal response for subjects with MRF implants or a behavioral seizure response for subjects with hippocampus implants.

The electrical stimulation was delivered bilaterally via two Nuclear-Chicago constant-current stimulators and consisted of a 5-second train of biphasic symmetrical pulses. Pulses lasted 0.1 msec and were at 100 hz for animals with MRF implants and at 30 hz for the animals with hippocampus implants. For the critical treatment, the current intensity for each animal was half the observed threshold intensity. Current intensity for treatments fell between 20 and 45 μa for the MRF group and between 15 and 32 μa for the hippocampus group.

Each rat was trained to press a bar in a Skinner box on a continuous reinforcement schedule for 15 minutes daily. The Skinner box was equipped

with retractable lever, liquid feeder, house lights, speaker, and exhaust fan, and each was enclosed in a sound-attenuating chamber. Reinforcements were 0.01-ml portions of a 30 percent sucrose solution, delivered to the animal by a liquid dipper. Relay circuitry and digital printing counters automatically programmed and recorded the events during the experiment.

After animals learned to respond within 15 seconds and behavior was stable, they were habituated to cords attached to the electrodes. When stable bar-pressing behavior was reestablished, the critical brain stimulation was delivered for 5 seconds in the middle of the 15-minute session to ensure that the level of stimulation did not interfere with bar-pressing activity. Brain stimulation produced invariably no more than a 10 percent decrement or increment in the rate of pressing the bar. Two days after the brain stimulation test, the animals received a 5-ma, 60-hz, 1-second shock to the feet (FS). The FS contingent was on a bar-press response at the end of minute 10 of the session. Immediately after the FS, the bar was withdrawn. After 4 seconds, animals received 5 seconds of bilateral brain stimulation in either the MRF or the hippocampus. The control group received no brain stimulation. At 55 seconds after the offset of brain stimulation, half of each group were retested for 10 minutes for retention of the foot-shock, and the other half were removed from the boxes. The latter animals were retested similarly 24 hours later.

The electrode placements, as verified histologically, were in the dorsal hippocampus or in the MRF region ventral to the superior colliculus and lateral to the central gray.

Suppression of bar-pressing behavior was used as an indication of retention for the FS experience. This suppression was indexed by the ratio $B/(A + B)$, where A is the number of bar presses during the first 5 minutes of the session before treatment and B is the number of bar presses during the first 5 minutes of the retest session. Thus, a ratio of .50 indicates no relative change in rate after the treatment, and a ratio of .00 indicates complete cessation of response on the retest.

Results are shown in Fig. 1. For the control group, there was suppression of bar-pressing (that is, memory of the FS) whether the interval between FS and retest was short or long. In contrast, there was no suppression (that is, there was amnesia for the FS) when the MRF group was tested at the short interval and when the hippocampus group was tested at the long interval. For these two groups, there was suppression (memory of the FS) at the opposite intervals.

A two-way analysis of variance revealed that the mean suppression of bar-pressing on the retests was influenced by site of brain stimulation ($F = 3.80$, d.f. $= 2/35$, $P < .05$), time of retest ($F = 4.46$, d.f. $= 1/35$, $P < .05$), and the interaction between site of brain stimulation and time of retest ($F = 11.60$, d.f. $= 2/35$, $P < .001$). A Newman-Keuls comparison test showed that for a short interval between FS and retest, the MRF group differed significantly from the control group ($P < .01$) and from the hippocampus group ($P < .01$); the control and hippocampus groups did not differ from each other. For the long interval, the hippocampus group differed significantly from the control group ($P < .01$) and from the MRF group ($P < .01$); the control and MRF groups did not differ from each other. For each group, the suppression at the two intervals was compared. For the hippocampus group, suppression was significantly less at the long interval ($P < .01$); and for the MRF group, suppression was significantly greater at the long interval ($P < .05$). For the control group, the difference between the intervals was not significant.

The results show that MRF stimulation interferes with retention of the aversive experience at the 64-second retest but not at the 24-hour retest, while hippocampal stimulation interferes with retention of the aversive experience at the 24-hour retest but not at the 64-second retest.

In order to ensure that these amnestic effects were not due to lesions induced by implantation of electrodes, additional rats had electrodes implanted in the MRF ($N = 3$) or hippocampus ($N = 4$). These animals were not stimulated 4 seconds after the FS. The MRF group was tested for retention after 64 seconds, while the hippocampus group was tested after 24 hours. The nonstimulated animals retained the aversive experience (that is, there was a marked suppression of bar pressing), a result that eliminates the possibility that the amnestic effect was due to the lesions. Possible aversive, rewarding, or seizure effects of brain stimulation also cannot account for these results, since brain stimulation alone had no effect on bar-press rates.

Instead, our results suggest a structural basis for a dual processing of

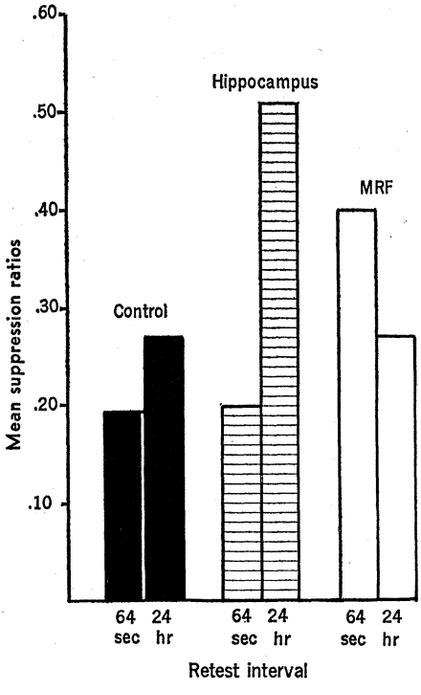

Fig. 1. Mean suppression ratios for bar-pressing activity 64 seconds and 24 hours after the foot shock. Electrical stimulation was given for 5 seconds to the hippocampus or the midbrain reticular formation (MRF) 4 seconds after the foot shock; controls received no brain stimulation.

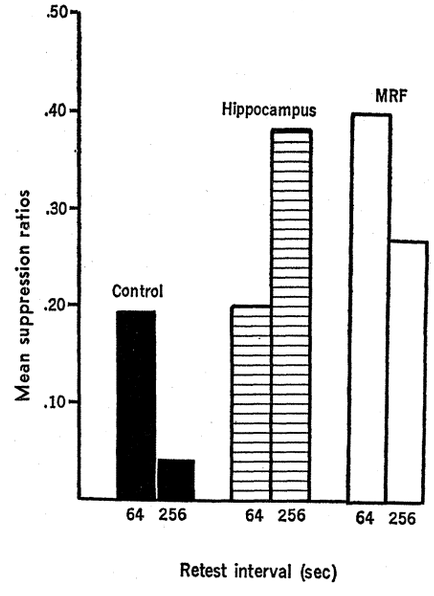

Fig. 2. Mean suppression ratios for bar-pressing activity 64 and 256 seconds after the foot shock. Electrical stimulation was given for 5 seconds to the hippocampus or the midbrain reticular formation, either 4 or 196 seconds after the foot shock, and animals were retested 55 seconds after stimulation. Controls received no brain stimulation.

memory of aversive information, where the MRF is involved in STM and the hippocampus is involved in LTM. (It is assumed that STM is being measured 64 seconds after an FS and that LTM is being measured 24 hours after an FS.) That stimulation of the hippocampus after trials disrupts LTM in rats is consistent with similar results for cats (*5*). Thus, with the use of brain stimulation after trials it is possible to dissociate STM from LTM, a result that suggests that STM and LTM are operating independently. Further support for this view is provided by Milner (*2*), who found that patients with bilateral hippocampal lesions had good STM but failed to store new information in LTM, and by Warrington and Shallice (*3*), who found that a patient with a parietooccipital lesion had deficient STM but good LTM.

Furthermore, when acetoxycyclohexamide (AXM), an inhibitor of protein synthesis, was injected before or immediately after a learning trial, the drug interfered with LTM but not STM (*6*). However, AXM that was injected 15 or 30 minutes after a learning trial did not interfere with LTM (*6, 7*). The fact that an inhibitor of protein synthesis interferes with LTM if it is given immediately after training but not if it is given 15 minutes later also suggests a parallel processing of information in STM and LTM. Finally, repeated presentations of FS and electroconvulsive shock (ECS) in short intervals (at least 0.5 second between FS and ECS) are sufficient to lead to LTM of the FS (*8*). Our experiment and the above-mentioned studies indicate that parallel rather than sequential processing of STM and LTM must occur for aversive information.

In another experiment, we tested a retention interval between 64 seconds and 24 hours to determine the time course of decay of STM and growth or consolidation of LTM. Subjects were 19 male Long-Evans rats, divided into groups for MRF stimulation ($N = 6$), hippocampal stimulation ($N = 7$), and no stimulation ($N = 6$). The animals were prepared surgically, trained, and given the FS as in the earlier experiment. The animals received 5 seconds of brain stimulation 196 seconds after the FS; and 55 seconds after the offset of stimulation they were retested for 10 minutes for retention of the FS. The suppression ratios for brain stimulation 196 seconds after FS are shown in Fig. 2, with corresponding data (from the earlier experiment) for brain stimulation 4 seconds after FS. For both groups the interval between brain stimulation and retest was the same (55 seconds). In the control group there was greater suppression at 256 seconds after the FS than at 64 seconds after the FS. However, at the 256-second retest there was suppression for the MRF group, but little suppression for the hippocampus group.

A two-way analysis of variance revealed that the mean suppression on the retests was influenced by site of brain stimulation ($F = 14.1$; d.f. $= 2/34$; $P < .001$) and the interaction between site of brain stimulation and time of retest ($F = 8.88$, d.f. $= 2/34$, $P < .01$). A Newman-Keuls comparison test showed that at the 256-second retest, the MRF and hippocampus groups showed significantly less suppression than did the control group ($P < .01$). Furthermore, for the control and MRF groups, suppression was significantly greater at the 256-second retest than at the 64-second retest ($P < .05$); in contrast, for the hippocampus group suppression was significantly less at the 256-second retest than at the 64-second retest ($P < .01$).

Thus, MRF stimulation that is applied 196 seconds after an FS produces marked suppression of bar-pressing at the 256-second retest. If it is assumed that MRF stimulation interferes only with STM processes, rapid growth and consolidation of LTM are indicated. Hippocampal stimulation that is applied 196 seconds after an FS produces little suppression at the 256-second retest. If it is assumed that hippocampal stimulation interferes only with LTM processes, then this result indicates decay of STM. Thus, it appears that 256 seconds after an FS the animal's memory is primarily LTM, with some slight involvement of STM. The data also support a differential neural basis for independent processing of STM and LTM.

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Selective Dissemination

Schneider (*1*) has presented an interesting and complete account of the implementation of a system for the selective dissemination of information (SDI) for cancer-related literature. Unfortunately, enthusiasm for his own approach —use of an enumerative classification —has led him into some rather sweeping claims regarding the superiority of this method over a whole host of others, which he lumps under the general heading "keyword-based" and treats in a somewhat cavalier and irresponsible fashion. Among the "keyword-based" systems are those using uncontrolled keywords (humanly assigned) and those based on subject headings and thesauri, as well as systems operating on free text (that is, searching the natural language of a machine-readable text with a computer).

In actual fact, a retrieval or dissemination system, if properly designed, can function effectively via any of these methods. Under a certain set of conditions one method will be preferable to another, but all can be made to work. Schneider's criticism of existing systems ("imprecise indexing," "a high level of 'noise,'" and "occasionally provide a useful item of information to users" are among statements used) is exaggerated and highly subjective. Moreover, he fails to cite a single study to justify his criticism. Indeed, he chooses to dismiss lightly the results of the ASLIB-Cranfield Project (*2*), the most complete study of indexing languages yet undertaken, presumably because these results do not fit his own view of the universe.

Instead, Schneider refers to a recent