can mediate feeding reflexes independently of the hypothalamus (*23*). Therefore, injected norepinephrine may excite or inhibit a part of the feeding system which passes between the limbic forebrain and the midbrain along an epithalamic route which lies parallel to the well-established lateral hypothalamic connection (*17*, *24*) and may be cross-linked to it by transthalamic fibers (*12*, *17*). This postulated part of the feeding system may be involved in the hyperphagia which results from some lesions in the dorsal thalamus or stria medullaris (*25*). The epithalamic pathway might also mediate the recovery from aphagia shown by rats with lateral hypothalamic lesions: indeed the feeding patterns of such rats show abnormalities (*11*) similar to those associated with feeding elicited by norepinephrine (*3*, *26*). In both cases, the rats are hypersensitive to the flavor of food and unwilling to expend muscular effort to obtain it.

A search should be made ipsilaterally to effective injection sites for areas which, when destroyed, eliminate the eating elicited by norepinephrine. Such experiments would both test the scheme outlined above and clarify the afferent and efferent connections (*18*, *19*, *21*, *24*) of this rostral hypothalamic part of the feeding system. As axonal uptake mechanisms and alpha adrenergic receptor sites mediate the elicitation of eating by norepinephrine injected into the substantia innominata (*27*), connections with adrenergic systems related to that area (*2*, *28*) are of particular interest. Whether the adrenergic portion of the feeding system is afferent, efferent, or in parallel to the lateral hypothalamic portions of the system, its role in normal hunger remains to be determined.

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5. The hydrochloride was equally effective (*27*), but the bitartrate was the purest available salt.
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7. Intakes of chow after norepinephrine were consistently large with repeated injections (range for all tests of effective cannulas: 0.8 to 4.7 g; median difference between first and second norepinephrine injections: 0.8 g). Water intakes were small—often nil—at most 5 ml in an hour after injection. A choice between water and relatively unpalatable dry chow was given to increase the strength of data on food intake as evidence for effects on a feeding system rather than less specific oral effects.
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14. Unlike cholinergic elicitation of drinking [A. E. Fisher and J. N. Coury, *Science* **138**, 69 (1962)], injection of norepinephrine into Papez' circuit structures consistently failed to elicit eating under the present conditions (see Fig. 2), although 1 to 3 μg or more of solid norepinephrine in these and all tested structures occasionally causes augmented intake of foodstuff which is also eaten under control conditions [J. N. Coury, *Science* **156**, 1763 (1967)]. The pattern of effective sites in Fig. 2 also indicated that the drug could not merely be acting directly on VMH structures to inhibit satiety [J. Olds, A. Yuwiler, M. E. Olds, C. Yun, *Amer. J. Physiol.* **207**, 242 (1964)].
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## Motivated Forgetting Mediated by Implicit Verbal Chaining: A Laboratory Analog of Repression

Abstract. *After learning an* A-B *paired-associates list, college students read a list of* D *words, several of which were consistently accompanied by unavoidable electric shock. The* D *words were members of implicit* B-C, C-D *chains, inferred from published word-association norms. In a subsequent recall test of the original* A-B *list, the* B *words that were implicitly associated with the shocked* D *words were forgotten significantly more often than control words.*

Are memory items which are specifically associated with unpleasant events more readily forgotten than affectively neutral items? Despite the wealth of empirical and theoretical interest in this question, particularly with respect to the psychodynamic concept of repression, no simple and effective techniques for the study of motivated forgetting have been reported (*1*). Our purpose was to demonstrate that forgetting does occur as a function of unpleasant associations.

We adapted Russell and Storms's (*2*) four-stage mediation paradigm for this purpose. In their study, subjects first learned an *A-B* paired-associates list, where *A* is a nonsense syllable and *B* is an English word. Associations *B-C* and *C-D* were inferred from word association norms. For example, if the *A-B* pair were *cef-stem*, then the *B-C* association would be *stem-flower*, and the *C-D* association, *flower-smell; A* and *D* are thus associated by way of the *B-C* and *C-D* links (see Table 1). Russell and Storms found that learning an *A-B* pair facilitated the subsequent learning of a related *A-D* pair.

If such implicit verbal chains do operate, then saying the *B* word implicitly elicits the *C*, and in turn, the *D* words. If the *D* word is associated with an unpleasant event, such as electric shock, then the likelihood of saying, or thinking of, the associated *B* word should be reduced, because the *B* response has an unpleasant consequence, namely, thinking of the *D* word, which presumably elicits fear. Thus, pairing specific *D* words with electric shock should cause differential forgetting of *A-B* pairs learned prior to the *D* word presentations. The *B* words associated with shock-paired *D* words should be forgotten more often than *B* words associated with neutral *D* words.

The stimulus materials we used are shown in Table 1. Note that, as in the Russell and Storms (*2*) study, the *C* words are never presented, but are assumed to occur as implicit associative responses linking *B* with *D*. Subjects first learned the *A-B* pairs (List 1). After attaining a specified criterion, the *D* words were presented (List 2), with electric shock paired with three of the ten words. Finally, the *A-B* pairs were presented once to test retention.

Sixteen male Princeton University undergraduates served as volunteers. General paired-associates instructions were provided, and subjects learned List 1 by the method of anticipation. The list was presented in three random orders, by use of a slide projector controlled by interval timers. Each nonsense syllable appeared for 1 second, followed, after a 0.75-second slide change, by the syllable and the response word for 1 second. The next syllable appeared immediately after, and 2 seconds elapsed between successive list presentations. List 1 learning continued to one perfect trial in which all pairs were correctly anticipated.

Table 1. Stimulus words used and inferred associative responses. The *D* words followed by (1) were the experimental words for half the subjects; those followed by (2) were the experimental words for the remaining subjects.

| List 1 | | Inferred chained word | List 2 | |
|---|---|---|---|---|
| *A* | *B* | *C* | *D* | |
| CEF | stem | flower | smell | (1) |
| DAX | memory | mind | brain | (2) |
| YOV | soldier | army | navy | |
| VUX | trouble | bad | good | (2) |
| WUB | wish | want | need | |
| GEX | justice | peace | war | (1) |
| JID | thief | steal | take | (2) |
| ZIL | ocean | water | drink | |
| LAJ | command | order | disorder | |
| MYV | fruit | apple | tree | (1) |

Electrodes were then placed on the third and fourth fingers of the left hand, and a key-operated buzzer was provided for the right hand. Subjects were told that a list of words (List 2) would be projected on the screen, and that each time a word appeared they were to pronounce that word aloud. Some words would be accompanied by shock, and subjects were to press the buzzer key whenever one of these words appeared. The key press did not avoid or escape the shock; it simply indicated that subjects had learned which words led to shock, and which were safe. Each word was presented for 2 seconds, and shock, when presented, occurred during the last second of word presentation. The shock source delivered 1.25 ma at 250 volts a-c, 60 hertz.

For half the subjects, the shock-paired *D* words were *smell, war,* and *tree*; for the other half, *brain, good,* and *take*. List 2 presentations, in three random orders, continued until subjects had correctly anticipated shock for three consecutive trials with no incorrect anticipations. The mean number of presentations of List 2 was 7.2, standard deviation = 3.6. Subjects were then given a single relearning trial of List 1, with electrodes left in place. The measure of motivated forgetting was the percentage of shock-associated *B* words forgotten relative to the percentage of control *B* words forgotten. Finally, we asked subjects to state the purpose of the experiment, to recall the words associated with shock, to recall the words of List 2, and finally, to state any connections they could think of between shocked words and List 1 words, and between any List 2 and List 1 words.

Since the two groups of subjects did not differ significantly in any experimental measures, their data were pooled. No subject correctly stated the purpose of the experiment. Recall of the shock-associated *D* words was perfect, while mean percent recall of the control *D* words was 71.4. This difference is significant at the .01 level, as evaluated by a Wilcoxon matched-pairs signed-ranks test. This result is to be expected, since subjects' task was to learn which words anticipated shock.

Ten subjects reported that they could not think of any specific connections between any *D* word and the *A-B* pairs. Of the remaining six subjects, four reported two correct associations each, but none involved the experimental (shocked) stimuli. The other reported connections were incorrect. These data indicate that any shock-related forgetting is not attributable to verbalizable associations between *D* and *B* words, nor to the demand characteristics of the experiment. The original List 1 learning data indicate that any subsequent differences in recall cannot be attributed to differential initial learning. The mean numbers of trials to learn experimental *A-B* pairs (4.1, S.D. = 2.8) and control *A-B* pairs (3.4, S.D. = 2.0) did not differ significantly. Similarly, the mean number of correct anticipations (repetitions) of experimental and control pairs did not differ (experimental, 5.1, S.D. = 5.8; control, 5.9, S.D. = 3.1).

We turn now to the recall data relevant to our hypothesis. For the subjects with *smell, war,* and *tree* as the shocked *D* words, 20.8 percent of the associated *A-B* pairs were not anticipated correctly, compared to 3.6 percent of the control pairs forgotten. The other group of subjects forgot 37.5 percent of the experimental pairs, and 8.9 percent of the control pairs. A Wilcoxon matched-pairs signed-ranks test applied to the pooled data indicated that the difference in percent retention between experimental and control pairs is significant at the .01 level, $T$ (*13*) = 5. Three subjects forgot none of the pairs, and only two subjects forgot more control than experimental pairs. No subject substituted an experimental *B* word incorrectly. Perhaps because of the rapid pacing of the paired-associates list, subjects either anticipated correctly in the recall test or failed to answer.

Pairing of shock with associates of memory items clearly interfered with their subsequent retrieval. Two interpretations of this finding may be considered. First, the shock may have resulted in differential retroactive interference mediated by the superior retention of the experimental *D* words. If learning a list of such *D* words between initial learning and recall produces retroactive interference, then the particular form of motivation employed may be irrelevant. The same effect may be obtainable with positive reinforcement, and, indeed, with any operation that produces superior retention of experimental words. This possibility, however, seems unlikely in view of the retroactive facilitation effects reported by Horton and Wiley (*3*). Using a three-stage chaining paradigm, they found that, after learning an *A-B* and a *B-C* list, learning an *A-C* list facilitated *A-B* retention.

Nevertheless, the experiment was repeated with an independent sample of 40 subjects drawn from a different college population: paid volunteers attending summer session at Dickenson College, Carlyle, Pennsylvania. Half of these subjects received shock associated with the experimental *D* words; the other half received money reward associated with the experimental *D* words. As in the original experiment, trials to learn List 1 and number of correct anticipations during List 1 learning did not vary as a function of any experimental conditions. Again, as in the earlier experiment, recall of the experimental *D* words was significantly superior to recall of control *D* words (100 percent versus 49 percent correct recall for the shock group; 95 percent versus 55 percent of the money group; $P < .01$ in both cases). In terms of these variables, this second experiment replicated the first.

Differential forgetting as a function of shock was similar to the data obtained earlier. Fifteen percent of the experimental *A-B* pairs were forgotten, compared to 5 percent of the control pairs, and this difference is significant at the .05 level. In contrast, no significant difference in forgetting was obtained between experimental and control pairs in the money-reward condition (10 and 11 percent, respectively). In this money condition, 22 pairs were forgotten, 6 experimental and 16 control. This is very close to the distribution that would be expected by chance, namely 6.6 and 15.4.

These additional data are unambiguous. The differential forgetting shown is specific to an unpleasant event, shock, and is not attributable to the differential recall of shock-associated words.

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## Reversal Learning and Forgetting in Bird and Fish

Abstract. *Pigeons and goldfish were trained in red-green discrimination in daily sessions, with the rewarded color changed every 2 days. Improvement in the performance of the pigeons could be traced to decrements in retention from each day to the next. The goldfish showed no improvement and no decrements in retention. The results suggest that progressive improvement in habit reversal is a product of proactive interference, and that the absence of improvement in the fish is due, not to the lack of some higher-order process which operates to produce improvement in higher vertebrates, but to a difference in learning-retention mechanisms.*

Suppose that on each of a series of trials we offer a rat or a pigeon a choice between two stimuli, *A* and *B*, rewarding it today for choosing *A*, tomorrow for choosing *B*, the day after for choosing *A*, and so forth. With each change in the training conditions, the animal changes its preference—today it develops a preference for *A*, tomorrow for *B*, the day after for *A*, and so forth. The first reversals are accomplished with some difficulty, the animal persisting during the early trials of each day in the choice of the rewarded alternative of the preceding day, but, as training continues, the number of errors made each day declines progressively. This is the phenomenon of "progressive improvement in habit reversal," known for many years (*1*), but until now little understood.

One explanation of progressive improvement has been that the animal comes to adopt a "win-stay, lose-shift" strategy, with response on each trial based on the sensory aftereffects or short-term memory of the events of the immediately preceding trial; but the aftereffects interpretation is contradicted by the fact that reversal performance is not impaired by substantial increases in the intertrial interval (*2*). Another explanation has been that the animal comes to attend more and more readily to the relevant (reward-correlated) dimension of stimulation, but the attentional interpretation is contradicted by the fact that improvement takes place concurrently in two different dimensions of stimulation which are equally often relevant and irrelevant (one relevant when the alternative is irrelevant) in a long series of problems (*3*). That the improvement is due to some higher-order process has been suggested by the fact that it does not occur in the fish—although the fish is capable of repeated reversal, it shows no decline in errors per reversal as training continues—but the nature of the process has not been specified (*4*).

We shall contend here that the process is after all a simple one—that improvement in reversal results from decrements in retention which are produced by proactive interference. From experiments on human memory, it is well known that learning of *X* may impair the retention of subsequently learned *Y* (proactive interference), just as the retention of *X* may be impaired by the subsequent learning of *Y* (retroactive interference), both effects being due apparently to the competition of antagonistic response tendencies; the greater the amount of potentially competing material learned before *Y* is learned, the poorer the retention of *Y* (*5*).

Data from some recent experiments on habit reversal in rat and pigeon indicate that the preferences which are established in each experimental ses-