The cerebral cortex: Its roles in memory storage and remembering

DONALD R. MEYER

The Ohio State University, Columbus, Ohio

In the last dozen years, I have repeatedly expressed my conviction that the traces of long-term memories are almost incredibly resilient, and I have also presented some unpopular opinions with respect to their temporal dynamics. Thus, when I was president of the Midwestern Psychological Association, the theme of my address was that injuries to the cortex will rarely, if ever, affect them, although such injuries will frequently suppress remembering of stored information (D. R. Meyer, 1972a). Thereafter, I suggested that long-term memories are formed within a few milliseconds and then become almost immediately resistant to changes as a function of time (D. R. Meyer & Beattie, 1977). Hence, since my answers to the questions before us have long been a matter of record, I perceive my task to be to show why I believe them, and then to discuss their implications.

Where Are Memories Stored Within the Brain?

When I first began my studies of the brain, I was taught that the neocortex was its main memory bank. Hence, I supposed that it would be an easy matter to show that different kinds of injuries to the cortex affect different kinds of memories. However, when I looked at the works of other people who had previously examined that idea, I was startled to find that there was very little solid evidence for such a proposition. Instead, the typical result of an inquiry that asked if a habit could be permanently destroyed by a cerebral cortical ablation had been that performance of the habit was impaired but the animal would then exhibit savings if retrained after surgery.

However, and remarkably, nobody thought that the theory could possibly be wrong. Instead, the results were interpreted as meaning that the traces of memories were modifications either of the cortex as a whole or of large subsectors of the cortex (e.g., Lashley, 1929, 1935; Pavlov, 1927), or else that when an animal was trained on a task, it learned a variety of habits whose traces were stored in different places in the cortex (Hunter, 1931). Thus, it was supposed

that if postoperative recoveries of performances of habits were observed, those recoveries were functions of memories that were stored in the undamaged regions of the cortex.

It was for those reasons that many of the studies of our group at The Ohio State University have dealt with the effects of huge injuries to the cortex upon the performance of learned tasks. Most of them have been concerned with the performance of a task involving a black-white discrimination problem (see D. R. Meyer & P. M. Meyer, 1977, for a review of the program's early phases). As we first showed many years ago, a normal rat that is trained on a problem and then prepared with an injury to the posterior half of its cortex will forget the problem, but will then relearn it in about as many trials as it takes for a naive posterior subject to learn the problem after surgery. The number of trials required in either instance will vary with the training conditions, but the near-equivalence of learning and relearning rates has been observed in many different contexts (Glendenning, 1972; Gray & D. R. Meyer, 1981; Horel, Bettinger, Royce, & D. R. Meyer, 1966; Lashley, 1935).

We began with the question of whether a rat with an injury to its posterior cortex could either learn or relearn the problem because it still has its anterior cortex (Horel et al., 1966). Our strategy was simply to destroy the latter region before we trained subjects on the problem, to train them, and then to prepare them with second-stage ablations of the posterior cortex. Then we retrained them on the problem, and compared the rate at which they relearned the problem with the rate at which the problem was relearned by rats that learned the task as normals and were then prepared with ablations of the posterior cortex. We found that the rates were exactly the same, and we have recently confirmed that observation (Hata, Diaz, Gibson, Jacobs, P. M. Meyer, & D. R. Meyer, 1980). Hence, since it didn't seem to matter in the slightest whether rats with injuries to the posterior cortex still had their anterior cortex, we concluded that recoveries from posterior injuries are functions that depend upon subsocortical systems.

We also have found that the same thing is true for injuries sustained in the opposite order (Horel et al., 1966; Howarth, D. R. Meyer, & P. M. Meyer, 1979).

This contribution was presented as part of an invited symposium at the Midwestern Psychological Association meetings, Chicago, May 1984, entitled "Long Term Memory: How Durable, and How Enduring?" The author's mailing address is: Laboratory of Comparative and Physiological Psychology, Ohio State University, 1314 Kinneau Road, Columbus, OH 43212.

That is, if naive rats are prepared with injuries to the posterior cortex, are trained on the problem, and are then prepared with second-stage anterior injuries, they will relearn the problem at exactly the same rate as rats that are trained on the task while they are normal and then are subjected to first-stage injuries that destroy the anterior cortex. In passing, I shall note that the generalization is for injuries inflicted in adulthood; a somewhat different picture is obtained if the first-stage injuries are sustained in infancy (Hata et al., 1980; Howarth et al., 1979).

We next asked whether the apparently complete forgetting of the problem that a posterior injury brings about is due to destruction of the memory for the problem or, instead, is an impairment of remembering. We addressed that question by studying the effects of treatments with d-amphetamine (Braun, D. R. Meyer, P. M. Meyer, 1966). We found that if the task had been learned prior to surgery, posterior subjects would relearn it very quickly if they were treated with the drug. However, we found that comparable treatments had no effect whatever upon the rate at which a preoperatively naive posterior animal would learn the problem after surgery. Hence, it was plain that posterior injuries do not destroy preoperative memories, and the only question that remained was whether the memories were partially stored by the cortex and partially by lower level systems.

I first became convinced that the multilevel theory was probably wrong by the results of a study in which a preoperative habit was pitted against a postoperative habit (LeVere & Morlock, 1973). In that investigation it was shown that if a rat is trained to choose a dim and not a bright door, is prepared with an injury to the posterior cortex, and then is trained to choose the bright door, the preoperative training markedly suppresses the rate of postoperative learning. That finding supported our conclusion that the injuries will not erase preoperative memories, but to me its most interesting aspect was the size of the effect. Thus, when we confirmed it with our own procedures, we found that black-positive preoperative training would double the number of trials required for a posterior subject to learn to choose the white door. I view such results as inconsistent with the holographic theory of memory (e.g., Pribram, 1971) which says that memory traces are stored throughout the brain and are weakened in proportion to the scope of an injury to the brain.

Of course, one can argue that the black-white memory is only one of many kinds of memories. Also, it is widely believed that ablations of the frontal or temporal lobes of monkeys have yielded impairments which were evidently memorial (e.g., Goldman & Rosvold, 1970, and Mishkin & Pribram, 1954, respectively). We have looked at those impairments for ourselves, and have found, without exception, that the evidence at hand does not support the con-

cept that memories are stored by the associative cortex of primates (D. R. Meyer, 1972b; P. M. Meyer & D. R. Meyer, 1982). Animals with injuries to these regions have many difficulties, including perseverative impairments (Mishkin, 1964; Settlage, Zable, & Harlow, 1948), failures to get information into storage if distracted (Bartus & LeVere, 1977; Blake, D. R. Meyer & P. M. Meyer, 1966), constrictions of fields of attending (Butter & Hirtzel, 1970; Meyer, 1972b), and impairments of formation of concepts (Horel & Keating, 1972; Riopelle, Alper, Strong, & Ades, 1953). However, if those problems are coped with in appropriately designed investigations, the so-called memorial impairments of frontal and temporal preparations disappear.

We have also been urged to temper our conclusion that the cortex is not a memory bank on the grounds that it could be that memories for words are stored by human cerebral cortex. However, there is no support for that conjecture that I know of, and I know of one argument against it. In the study I refer to (Howes, 1964), the investigator listened to patients with aphasia until each had produced a sample of 5,000 words. That often took a very long time. Thereafter, the samples were analyzed to see if the patients were using very common words more frequently than normal people do, at the expense of utterances of uncommon words. It was found that they were not, and hence that the first-order structure of their language was intact. I interpret that to mean that their memory banks for words were still intact, even though the patients had impairments of word retrieval and utilization.

In summary, then, the theory that at least some memories are stored as modifications of the neocortex has no empirical foundation. That idea was plausible 50 years ago. It is not plausible today. Hence, I suggest that we forget it, and that workers with interests in the nature of engrams should look for them within the brain's core. I have no idea what an engram is like, but I know from behavioral studies of the question that memory traces cannot be produced via syntheses of protein or peptide molecules. The reason is simply that structural traces were formed very rapidly indeed, much too rapidly for synthetic processes to have a role in their assembly (D. R. Meyer & Beattie, 1977).

Are Long-Term Memories Continuously in Flux?

Now I shall turn to the question of whether the substrates of memories in long-term storage undergo continuous changes as a function of time. Our commonsense conception is that memories decay unless they are refreshed through rehearsal. The idea is nicely expressed by the motto that you have to use them or you'll lose them. But at least some neurologists have come to the opposite conclusion, namely that memories congeal while in storage and become more resil-

ient as they age (Ribot, 1885; Russell, 1959). My own conclusion is that structural traces are very stable entities, regardless of whether they have been in storage for seconds, minutes, hours, days, or years.

I first began to consider that conclusion approximately 30 years ago. The process began when I encountered a paper that dealt with the effects of severe hypothermia upon retention of a maze habit (Andjus, Knopfmacher, Russell, & Smith, 1956). In that investigation, the core temperatures of rats had been reduced to a point at which the animals had neither an EEG nor an EKG. Some of them did not survive the treatment, but the wonder was that most of them did, and that when they were rewarmed they were able to remember the task they had learned prior to treatment.

It had previously been shown that electroconvulsive shock treatments (ECT) would bring about complete impairments of performance of a simple passive-avoidance task provided the treatments were given immediately after the problem had been learned (Duncan, 1949). It was not clear whether the effect was an impairment of storage or retrieval of the memory, but almost everybody took it to mean that the memory had not been stored. Thus, it was presumed that the treatments had disrupted a short-term dynamical encoding process that permitted the development of structural traces in the period following the training (Hebb, 1949).

While I saw nothing wrong with that idea, I knew that ECT had some other effects that could not be explained in such a manner. My principal source of information was my mother, who had had an episode of involuntal depression and was given electroshock treatments. The treatments were successful, but she nonetheless was bothered by the fact that she was no longer able to remember certain memories. Some of those memories were for things that she had learned long before, which implied that the effects were either failures of retrieval or else were disruptions of structural traces that were long-term by any theorist's standards.

Importantly, she rarely mentioned the concerns that had occupied essentially the whole of her being immediately before she was treated. My first impression from my conversations with her was that her memories for things that bothered her had been destroyed. But a few years later, I abandoned that idea when she mentioned them during a relapse. Instead, I began to suspect that the treatments had principally affected her recall of memories she had learned in relation to her illness, and that ECT had therapeutic effects because it has a motivationally selective effect upon memorial retrieval.

My conviction was subsequently strengthened when I read a paper that suggested that individuals with mild concussive injuries to the head will forget what they were doing at the time of the injury but not what they were doing before they had embarked

upon their last course of action (Bucklew, 1956). Hence, we undertook a series of studies which showed that if rats are trained on several tasks, and the tasks are learned for different incentives, a single ECT that is given immediately following completion of training on the last task will suppress performance of an earlier task if, and only if, the same incentives were employed for training on both the earlier and the last task (Howard, Glendenning, & D. R. Meyer, 1974; Howard & D. R. Meyer, 1971; Robbins & D. R. Meyer, 1970).

The studies also showed that if a memory had been in storage for a period of days, the effect of the treatment upon its recall was independent of its age. Thus, manipulations of the motives for which a last task was learned before the treatment was given would bring about selective suppressions of retrieval such that older habits would be forgotten and newer ones would be unaffected. Hence, it was apparent that the age of a memory had nothing to do with its resilience, and that the findings posed a serious problem for the long-term consolidation theory.

However, that idea is still defended. Its most recent support has come from studies of patients who were treated with ECT (Squire, Slater, & Chace, 1975). The patients were tested for recall of TV shows that had been on for one season, 1, 2, or 3 years prior to the time of the treatment. As the long-term consolidation theory would predict, the patients exhibited a differential impairment of recall of the shows of the previous season but not of the shows that had been aired 3 years before the treatments. From my perspective, however, the most interesting result was that the treatments did not suppress recall of the shows that had appeared in the second season prior to the treatments. Thus, it seemed to me that the consolidation theory would also predict that memories for those shows would be affected, albeit to a lesser extent than the newest group of memories.

Our motivationally selective theory accounts for that result, and for the others. First, an individual does not ordinarily become a candidate for ECT until he has been sick for some time. When the treatments are given, they will yield suppressions of memories that were learned while he was ill but will not affect remembering of memories that had been learned while he was well. The affected memories will be newer than the unaffected memories, but the difference in their ages will have nothing to do with the result. Moreover, our theory denies the proposition that the treatments destroy the newer memories; all they will do is make the memories unavailable, or latent. If the problem recurs, those memories will return, whether they are wanted or not, and in such a circumstance it may be necessary for the patient to be treated once again.

I believe that our theory has important implications for the management of patients with depression. At present, impairments of the kinds just described

are viewed as side effects of ECT that have nothing fundamentally to do with the therapeutic consequences of the treatments. However, I suggest that the treatments work because the treatments render the patients unable to remember the problems that depressed them. That is, the desirable effects of ECT are products of the same laws as those which determine when the side effects will be observed. The concept implies that a therapist who wishes to target the unwanted memories should administer the treatments when a patient is disturbed, but not when he is feeling just fine. Thus, if he is feeling just fine, the treatments will suppress wanted memories but will have no appreciable effect upon recall of memories that the patient should be rid of. But, with that proviso, I have no doubts at all about the efficacy of the treatments, and certainly do not share the views of many people that their use should be condemned as barbaric.

With What Do We Remember Memories?

Although it is sometimes better to forget than to remember, for most of us the opposite is true. Hence, I shall now discuss some principles that govern protections of the remembering of memories. I shall not reiterate the findings of our studies of drug-induced reversals of amnesias, for in those investigations we were mainly concerned with the question of whether a completely latent memory can exist. Although the results supplied a rationale for the use of pharmacotherapies in treatments of clinical amnesias, we believe that explorations of the leads we first developed are not our own business, but the business of pharmacologists. Hence, at this juncture, I shall limit the discussion to studies of recoveries from amnesias that are brought about by various kinds of training and retraining of animals with injuries to the cortex.

Many years ago, we undertook a program of quantitative studies of impairments of performance of the brightness discrimination problem. First, after having gained a general idea of the kinds of results we might expect, we standardized our principal behavioral and surgical procedures. Thereafter, we explored the effects of different combinations of the standardized procedures and continuously controlled our use of the procedures by frequent replications of the studies. Now we have data from several thousand rats, of which the very great majority were trained on the problem and then were retrained after having been subjected to two successive injuries to the cortex.

The data have taught us many things. Among them is the fact that impairments of performance of the black-white discrimination problem are due in large part to disturbances of functions that have nothing to do with the processing of visual information. To illustrate, a radical bilateral ablation of the posterior half of the cortex will serve to destroy all the zones of termination of projections from the eyes to the

cortex (Hughes, 1977). The injury will also induce an apparently complete forgetting of the problem. However, only a third of that impairment is a visually specific effect. The rest of it is due to an impairment that can be produced by any injury to the cortex. Much of our recent work has dealt with the question of the nature of the larger impairment, and has led us to believe that the neocortex has an equipotential function in remembering of memories.

Many neuroscientists are of the opinion that neuropsychological research has yielded data that are only qualitative, fuzzy, and unlikely to be reproducible. However, we have shown that that is nonsense. Thus, we have developed our program to a point at which we are no longer interested in whether an injury to the cortex will have an effect upon performance. Instead, we measure the effect in terms of how many mean trials of training are required for a subject to recover from the injury. Then we compare that measurement with scores for subjects with other kinds of injuries. For many kinds of injuries, we know what the scores will be within one or two trials; for others, we know that the measurements are accurate to within 10% of the mean score. Moreover, our procedures for maintaining long-term quality control of the program have worked well enough that we can still replicate the findings of studies that we carried out 20 years ago.

With those facts in mind, I shall now describe our uses of the scores. A rat that is trained with our contemporary methods (Glendenning, 1972) will usually learn the black-white problem in approximately 25 mean trials. If the rat is then prepared with an ablation that destroys both posterior quadrants of its cortex, that is, an injury that bilaterally destroys the posterior half of its cortex, it will relearn the problem in approximately 25 mean trials. However, if the posterior quadrants are spared, and both of the anterior quadrants of its cortex are destroyed, the animal will relearn the problem in 17 mean trials. Hence, the extra cost of a posterior injury is approximately 8 mean trials, or about half the cost of an injury that destroys the extravisual regions of the cortex.

Significantly, the regionally specific impairment does not appear unless and until both quadrants of the posterior cortex are destroyed. Thus, a rat prepared with a unilateral injury to the posterior cortex will relearn the problem in approximately 8-9 trials. So will a rat with a unilateral anterior injury. Those numbers are very interesting. Thus, if we double the cost of an injury to one anterior quadrant of the cortex, we find that $2 \times 8.5 = 17$ mean trials. Also, if we double the cost of an injury to one posterior quadrant and add 8 trials to the product, we find that $(2 \times 8.5) + 8 = 25$ trials. The findings thus suggest that injuries to quadrants have linearly additive effects if we discount the 8-trial cost of an ablation that destroys the visual projections.

The same rules apply to other combinations of injuries to quadrants of the cortex. Thus, a hemidecorticated rat, that is, a subject with a two-quadrant injury that destroys an anterior and an ipsilateral posterior quadrant, will take about as many trials to relearn the problem as a subject with a bilateral injury that destroys both anterior quadrants of the cortex (D. R. Meyer & P. M. Meyer, 1977). Similarly, a subject with a crossed-quadrant injury, in which destruction of an anterior quadrant is combined with destruction of the contralateral posterior quadrant, will also relearn the problem in 17 mean trials (D. R. Meyer & P. M. Meyer, 1977). According to our concept, the number is 17, and not 25 for those groups because, in each instance, the operations spared a quadrant of the posterior cortex.

We have shown that regionally specific and summable impairments can be dissociated through the use of serial-lesion paradigms (see Finger, Walbran, & Stein, 1973). To illustrate, a rat that has been trained on the problem and has been retrained after having been subjected to ablation of a posterior quadrant will relearn the problem in approximately 8-9 trials. If the animal is then prepared with an ablation of its still-remaining posterior quadrant, it will relearn the problem in approximately 8 further trials. At first glance, the finding suggests that an injury to a quadrant always has a fixed cost, and that is how we first interpreted the two observations. However, although the same amount of training is required for recovery after each of the successive operations, the training does not correct the same impairment but, instead, two successive impairments. Thus, the interoperative retraining corrects the nonspecific summable impairment and the final re-retraining corrects the posteriorly specific, or visual, impairment.

Our first hint of that was from a study of serial anterior preparations (Glendenning, 1972). The animals were trained and then prepared with a first-stage ablation of an anterior quadrant. Their impairments were corrected by about 8 trials of interoperative retraining. However, when their contralateral anterior quadrants were destroyed, the animals relearned the problem in about 2 trials. That was not very different from the scores of subjects which were retrained following a one-quadrant injury and were not reoperated but merely tested for subsequent forgetting of the problem. We now interpret that result to mean that retraining after any injury to the cortex will induce a complete protection of remembering after any further injury that does not complete the destruction of the posterior cortex. In the latter circumstance, a second-stage injury will have a cost of 8 mean trials, and that is because interoperative training will not protect the subject from impairments which result from its not being able to perceive forms (Horel et al., 1966; Lavond & Dewberry, 1980;

Lavond, Hata, Gray, Geckler, P. M. Meyer, & D. R. Meyer, 1978).

We have played the same game in other ways. One has been to study the performances of serial preparations which were left with one anterior or one posterior quadrant following their second-stage injuries to the neocortex (Cloud, D. R. Meyer, & P. M. Meyer, 1982). The procedures involved combinations of first-stage two-quadrant injuries which were followed by one-quadrant injuries, or else of first-stage one-quadrant injuries which were followed by two-quadrant injuries. The study thus provided us with replications of our previous findings with respect to the effects of first-stage injuries to the cortex, and were generally consistent with our observations that two-quadrant injuries have twice the effect of one-quadrant injuries, provided the injuries do not destroy both posterior quadrants.

The methods were somewhat complicated, but the findings were remarkably simple. Thus, the groups that still had one posterior quadrant following their second-stage ablations re-learned the problem in approximately 3 mean trials. The groups that still had one anterior quadrant, and had been prepared with second-stage injuries that destroyed or completed the destruction of both posterior quadrants, re-learned the problem in 8 trials. Moreover, the outcomes were independent of the loci and the scopes of the first-stage and second-stage ablations, which showed that interoperative retraining on the problem induced a protection from the summable impairment regardless of how large the first-stage injury had been.

When taken as a whole, our quantitative studies constitute a proof of the existence of an equipotential function of the cortex. There are two facets to the proof. The first is that initial injuries to the cortex result in impairments which are governed by a law of mass action, provided, once again, that the regionally specific effects of the injuries are discounted. We have data which suggest that the law is valid for any combination of injuries to the cortex, including complete decortications. Thus, we have observed that the sum of the costs of bilateral anterior and bilateral posterior injuries will predict, with an error of about 10%, the cost of a one-stage ablation of the entire neocortex (Horel et al., 1966; P. M. Meyer, Yutzey, Dalby, & D. R. Meyer, 1968). However, that result was obtained through the use of methods we no longer employ (cf. D. R. Meyer & P. M. Meyer, 1977), and hence we believe that it needs further close examination.

The second facet of the proof is our finding that any kind of injury to the cortex will permit retraining to induce a protection of performance of the problem after any further injury to the cortex. A dramatic illustration is provided by the fact that an anterior-quadrant preparation, if retrained on the problem

and subjected to a second-stage bilateral posterior ablation, will take only 8 trials, instead of 25, to overcome its second handicap. We find ourselves unable to account for such an outcome, except by supposing that the anterior injury damages a system that also has components in the posterior sectors of the cortex, which, if destroyed by a first-stage ablation, would result in an impairment with a cost of 17 retraining trials. That, once again, is the cost of a first-stage bilateral anterior injury, even though the two subregions of the cortex each have their own specific functions.

We also have shown that a holistic system is involved in remembering memories. Our proof of that conclusion is as follows. First, the impairments of serial preparations are exactly the same as those of one-stage subjects unless they are given interoperative retraining on the problem (Kircher, Braun, D. R. Meyer, & P. M. Meyer, 1970; Petrinovitch & Carew, 1969; Thompson, 1960; see D. R. Meyer & P. M. Meyer, 1977, for review). Second, that is not an overtraining effect, for a comparable amount of overtraining on the problem, if given before a first-stage injury, conveys no protection of ultimate performance of the problem (Glendenning, 1972). Third, interoperative practice with the problem conveys no protection of ultimate performance unless the subject had been trained on the problem prior to its first-stage ablation (Bodart, Hata, D. R. Meyer, & P. M. Meyer, 1980; Hata et al., 1980; Horel et al., 1966; Howarth et al., 1979). In ordinary language, then, protection is observed if the rat has a memory to remember and has then recalled it after having first sustained a partial injury to the cortex.

At present, we are studying the question of how large a first-stage injury has to be for retraining on the problem after surgery to have a detectable effect. We now have a modicum of data from rats that were given preoperative training and were then subjected to injuries that destroyed a cylinder of cortex about 2 mm wide. As I think that our colleagues would expect, those first-stage injuries did not produce detectable impairments. However, when the subjects were subsequently prepared with second-stage two-quadrant injuries, they relearned the problem at a faster rate than animals with first-stage ablations of comparable extents. The savings were smaller than we have observed after first-stage injuries to at least one quadrant of the cortex, and why that was so is a problem that has yet to be resolved. I think that the likeliest reason for the difference is that the subjects with the tiny ablations received a much smaller amount of interoperative retraining than the quadrant preparations.

I believe that the findings are a partial explanation for Hughlings Jackson's (1873) law of momentum. Neurologists have long been puzzled by the fact that slowly growing lesions of the cerebral cortex may be

asymptomatic until they are astonishingly large (see Finger & Stein, 1982, for a review). Of course, an individual who has a small stroke with a focus in Broca's convolution is going to know at once that something is amiss and will promptly seek advice from a physician. However, an injury that makes him forgetful, but only transitorily forgetful, is not very likely to signal an alarm until it is very far advanced. I suggest that once the process has begun, memories that the patient can retrieve and utilize will thereafter be rememberable, and that unused memories will not be.

How Are We to Fight Senility?

I shall close this discussion by giving you my thoughts about the implications of our observations for reasonably normal human beings. As I have observed, I think they have a bearing on the logic of convulsive therapy, and also on the problem of why traumatic injuries to the cerebral cortex have effects that are different from injuries that develop very slowly. However, most of us are not very likely to be treated with ECT, and equally happily most of us are not very likely to expire from brain tumors. Hence we can ask: Why should we care where memories are stored within the brain, or whether they are durable, or what kinds of laws affect their availabilities?

My answer is simply that we all have brains that are steadily deteriorating. When we are young, the changes are asymptomatic, but as we grow older they are not. Thus, if we have not been put away for good by the time we are 50 or so, we are virtually certain to be as forgetful as our grandparents were when they were aged. Our children will wish that we wouldn't harp about the way things were when we were little, and will wonder why we can't remember where we put our slippers when we have no problem in recalling Ted Williams's batting average.

Of course, those problems would not descend upon us if we knew how to keep our brains from rotting. At present, the search for countermeasures to the process is a hot neuroscientific topic, largely because that is where a lot of federal money is. The media have anticipated the results of such inquiries, and we hear every day that if we eat right, don't smoke, exercise, and stay away from liquor, there is no good reason why we shouldn't be chipper until we are 135. However, I have recently been studying the effects of diets on forgetting in old age, and it doesn't seem to be to be especially likely that dietary therapies are going to be in place very soon. Hence, I believe that a middle-aged person can expect to be dead before we have them, and would be well advised to try to cope with the problems with methods our findings have suggested.

First, such a person should never suppose, nor should people who are in better shape, that a memory that cannot be recalled is ipso facto likely to have

gone the way of all flesh. There is no proof whatever that long-term memories decay (P. M. Meyer & D. R. Meyer, 1982), and I know of no survivable procedure that will serve to obliterate the trace of a memory (D. R. Meyer & Beattie, 1977). Nor is there any proof that the age of a memory determines its durability (Hebb, 1975; Lewis, 1979; Robbins, & D. R. Meyer, 1970), and the best work that yet has been done with respect to the problem of the permanence of human memories suggests that their traces will persist without rehearsal for periods of as long as several decades (Bahrick, Bahrick, & Wittlinger, 1975). Hence, while it sometimes is said that human beings can be sure only of death and taxes, I would add that they can also be reasonably sure that the memories they have formed are still with them.

Second, such a person should expect to find it harder to remember new memories than old ones. But that is not because of the difference in the ages of the memories. Instead, it is due to the fact that old memories have a great probability of having been recalled in different motivational contexts. The point is illustrated by a study of rats that were trained on the black-white problem for a given incentive, then prepared with injuries to the posterior cortex, and then trained on reversals of the problem for the same or a different incentive (LeVere & Davis, 1977). Postoperative interference was observed if the two incentives were the same, but not if the two tasks were learned for different incentives. Hence, when one's brain is going down the drain, it becomes ever harder to remember something that was learned in a particular context.

Now I shall observe that I am one of the members of the group to whom my counsel is addressed. Although I am not yet a doddering old fool, I am not a spring chicken and suspect that my cortex has its fair share of senile plaques. For years I have prided myself on my ability to lecture without the use of notes, and thus far have managed to continue to do so with only a few embarrassments. However, I can no longer run like a fire horse as soon as the class bell is sounded. Instead, I must rehearse what I am going to say, and am often surprised by things that I find in materials I wrote long ago. Thus armed, I usually am able to speak for a period of 48 minutes, and also to judge within a minute or so when the period will come to an end.

At one time I thought that the reviews were effective because I was relearning the materials. At one time I also thought that rats which are given retraining on the black-white problem recover from their injuries by forming new memories that replace their preoperative memories. But now I doubt the first proposition, because the second one has been disproved. Thus, if posteriorly decorticated rats are treated with a drug that suppresses learning, but not remembering, of a brightness discrimination problem, the treatment has the paradoxical effect of facil-

itating postoperative performance of the problem (Davis & LeVere, 1979; LeVere & Fontaine, 1978). Hence, for a brain-injured subject—like myself—it appears that the principal effect of retraining on a task is to facilitate retrieval of memories that are still intact, but latent.

Finally, I think that the foregoing studies suggest that a person who will soon be retiring should hesitate before he sells the old home place and moves down to St. Petersburg. There is simply no question that the weather there is better in the winter than it is in the Midwest, for Midwestern winters are cold, damp, gray, and dingy by anybody's reckoning. However, even those conditions are a part of the contexts in which we operate. Our personal digs are even more so, and although a change of scene is often good for a youngster, it can be a disaster for an oldster. Hence, in my opinion, which I formed long ago in places such as New York and Boston, Florida is certainly a pleasant place to visit, but I certainly wouldn't want to live there.

REFERENCES

- ANDJUS, R. K., KNOPFELMACHER, F., RUSSELL, R. W., & SMITH, U. (1956). Some effects of severe hypothermia on learning and retention. *Quantitative Journal of Experimental Psychology*, **8**, 15-23.
- BAHRICK, H. P., BAHRICK, P. O., & WITTLINGER, R. P. (1975). Fifty years of memory for names and faces: A cross-sectional approach. *Journal of Experimental Psychology: General*, **104**, 54-75.
- BARTUS, R. T., & LEVERE, T. E. (1977). Frontal decortication in rhesus monkeys: A test of the interference hypothesis. *Brain Research*, **119**, 233-248.
- BLAKE, M. O., MEYER, D. R., & MEYER, P. M. (1966). Enforced observation in delayed response learning by frontal monkeys. *Journal of Comparative and Physiological Psychology*, **61**, 374-379.
- BODART, D. J., HATA, M. G., MEYER, D. R., & MEYER, P. M. (1980). The Thompson effect is a function of the presence or absence of preoperative memories. *Physiological Psychology*, **8**, 15-19.
- BRAUN, J. J., MEYER, D. R., & MEYER, P. M. (1966). Sparing of a brightness habit in rats following visual decortication. *Journal of Comparative and Physiological Psychology*, **61**, 79-82.
- BUCKLEW, J. (1956). Complex behavioral units of the reactional biography. *Psychological Record*, **6**, 44-47.
- BUTTER, C. M., & HIRTZEL, M. (1970). Impairment in sampling visual stimuli in monkeys with inferotemporal lesions. *Physiology & Behavior*, **5**, 369-370.
- CLOUD, M. D., MEYER, D. R., MEYER, P. M. (1982). Inductions of recoveries from injuries to the cortex: Dissociation of equipotential and regionally specific mechanisms. *Physiological Psychology*, **10**, 66-73.
- DAVIS, N., & LEVERE, T. E. (1979). Recovery of function after brain damage: Different processes and facilitation of one. *Physiological Psychology*, **7**, 233-240.
- DUNCAN, C. P. (1949). The retroactive effect of electroshock on learning. *Journal of Comparative and Physiological Psychology*, **42**, 32-44.
- FINGER, S., & STEIN, D. G. (1982). Fast- versus slow-growing lesions and behavioral recovery. In *Brain damage and recovery* (pp. 153-173). New York Academic Press.
- FINGER, S., WALBRAN, B., & STEIN, D. G. (1973). Brain damage

- and behavioral recovery: Serial lesion phenomena. *Brain Research*, **63**, 1-18.
- GLENDENNING, R. L. (1972). Effects of training between two unilateral lesions of visual cortex upon ultimate retention of black-white habits by rats. *Journal of Comparative and Physiological Psychology*, **80**, 216-229.
- GOLDMAN, P. S., & ROSVOLD, H. E. (1970). Localization of function within the dorsolateral prefrontal cortex of the rhesus monkey. *Experimental Neurology*, **27**, 291-304.
- GRAY, T. S., & MEYER, D. R. (1981). Effects of mixed training and overtraining on recoveries from amnesias in rats with visual cortical ablations. *Physiological Psychology*, **9**, 54-62.
- HATA, M. G., DIAZ, C. L., GIBSON, C. F., JACOBS, C. E., MEYER, P. M., & MEYER, D. R. (1980). Perinatal injuries to extravisual cortex enhance the significance of visual cortex for performance of a visual habit. *Physiological Psychology*, **8**, 9-14.
- HEBB, D. O. (1949). *The organization of behavior*. New York: Wiley.
- HEBB, D. O. (1975). Science and the world of imagination. *Canadian Psychological Review*, **16**, 4-11.
- HOREL, J. A., BETTINGER, L. A., ROYCE, G. J., & MEYER, D. R. (1966). Role of neocortex in the learning and relearning of two visual habits by the rat. *Journal of Comparative and Physiological Psychology*, **61**, 66-78.
- HOREL, J. A., & KEATING, E. G. (1972). Recovery from a partial Kluver-Bucy syndrome in the monkey produced by disconnection. *Journal of Comparative and Physiological Psychology*, **79**, 105-114.
- HOWARD, R. L., GLENDENNING, R. L., & MEYER, D. R. (1974). Motivational control of retrograde amnesia: Further explorations and effects. *Journal of Comparative and Physiological Psychology*, **86**, 187-192.
- HOWARD, R. L., & MEYER, D. R. (1971). Motivational control of retrograde amnesia in rats: A replication and extension. *Journal of Comparative and Physiological Psychology*, **74**, 37-40.
- HOWARTH, H., MEYER, D. R., & MEYER, P. M. (1979). Perinatal injuries to the visual cortex enhance the significance of extravisual cortex for performance of a visual habit. *Physiological Psychology*, **7**, 163-166.
- HOWES, D. H. (1964). Application of the word-frequency concept to aphasia. In A. V. S. DeReuck & M. O'Conner (Eds.), *Disorders of language* (Ciba Foundation symposium; pp. 47-77). London: Churchill.
- HUGHES, H. C. (1977). Anatomical and neurobehavioral investigations concerning the thalamocortical organization of the rat's visual system. *Journal of Comparative and Neurology*, **175**, 311-336.
- HUNTER, W. S. (1931). Lashley on "cerebral control versus reflexology." *Journal of General Psychology*, **5**, 230-234.
- JACKSON, J. H. (1873). Lectures on the diagnosis of tumours of the brain. *Medical Times and Gazette*, **2**, 139ff.
- KIRCHER, K. A., BRAUN, J. J., MEYER, D. R., & MEYER, P. M. (1970). Equivalence of simultaneous and successive neocortical ablations in production of impairments or retention of black-white habits in rats. *Journal of Comparative and Physiological Psychology*, **71**, 420-425.
- LASHLEY, K. S. (1929). *Brain mechanisms and intelligence*. Chicago: University of Chicago Press.
- LASHLEY, K. S. (1935). The mechanisms of vision: XII. Nervous structures concerned in habits based upon reactions to light. *Comparative Psychology Monographs*, **11**, 43-79.
- LAVOND, D., & DEWBERRY, R. G. (1980). Visual form perception is a function of the visual cortex. II. The rotated horizontal-vertical and oblique-stripes pattern problems. *Physiological Psychology*, **8**, 1-8.
- LAVOND, D., HATA, M. G., GRAY, T. S., GECKLER, C. L., MEYER, P. M., & MEYER, D. R. (1978). Visual form perception is a function of the visual cortex. *Physiological Psychology*, **6**, 471-477.
- LEVERE, T. E., & DAVIS, N. (1977). Recovery of function after brain damage: The motivational specificity of spared neural traces. *Experimental Neurology*, **57**, 883-899.
- LEVERE, T. E., & FONTAINE, C. W. (1978). A demonstration of the importance of RNA metabolism for the acquisition but not performance of learned behaviors. *Experimental Neurology*, **59**, 444-449.
- LEVERE, T. E., & MORLOCK, G. W. (1973). The nature of visual recovery following posterior decortication in the hooded rat. *Journal of Comparative and Physiological Psychology*, **83**, 62-67.
- LEWIS, D. G. (1979). Psychobiology of active and inactive memory. *Psychological Bulletin*, **86**, 1054-1083.
- MEYER, D. R. (1972a). Access to engrams. *American Psychologist*, **27**, 124-133.
- MEYER, D. R. (1972b). Some features of the dorsolateral frontal and inferotemporal syndromes in monkeys. *ACTA Neurobiologiae Experimentalis*, **32**, 235-260.
- MEYER, D. R., & BEATTIE, M. S. (1977). Some properties of substrates of memory. In L. H. Miller, C. A. Sandman, & A. J. Kastin (Eds.), *Neuropeptide influences of the brain and behavior* (pp. 145-162). New York: Raven Press.
- MEYER, D. R., & MEYER, P. M. (1977). Dynamics and bases of recoveries of functions after injuries to the cerebral cortex. *Physiological Psychology*, **5**, 133-165.
- MEYER, P. M., & MEYER, D. R. (1982). Memory, remembering, and amnesia. In R. L. Isaacson & N. E. Spear (Eds.), *Expression of knowledge* (pp. 179-212). New York: Plenum Press.
- MEYER, P. M., YUTZEY, D. A., DALBY, D. A., & MEYER, D. R. (1968). Effects of simultaneous septal-visual, septal-anterior, and anterior-posterior lesions upon relearning a black-white discrimination. *Brain Research*, **8**, 281-290.
- MISHKIN, M. (1964). Perseveration of central sets after frontal lesions in monkeys. In J. M. Warren & K. Akert (Eds.), *The frontal granular cortex and behavior* (pp. 219-241). New York: McGraw-Hill.
- MISHKIN, M., & PRIBRAM, K. H. (1954). Visual discrimination performance following partial ablations of the temporal lobe. I. Ventral vs. lateral. *Journal of Comparative and Physiological Psychology*, **47**, 14-20.
- PAVLOV, I. P. (1927). *Conditioned reflexes*. Oxford: Oxford University Press.
- PETRINOVITCH, L., & CAREW, T. J. (1969). Interaction of neocortical lesion size and interoperative experience in retention of a learned brightness discrimination. *Journal of Comparative and Physiological Psychology*, **68**, 451-454.
- PRIBRAM, K. H. (1971). *Languages of the brain*. Englewood Cliffs, NJ: Prentice-Hall.
- RIBOT, T. (1885). *Diseases of memory* (pp. 1-52). London: Kegan Paul.
- RIOPELLE, A. J., ALPER, R. G., STRONG, P. N., & ADES, H. W. (1953). Multiple discrimination and patterned string performance of normal and temporal-lobe resected monkeys. *Journal of Comparative and Physiological Psychology*, **46**, 145-149.
- ROBBINS, M. J., & MEYER, D. R. (1970). Motivational control of retrograde amnesia. *Journal of Experimental Psychology*, **84**, 220-225.
- RUSSELL, W. R. (1959). *Brain, memory, learning: A neurologist's view*. Oxford: Clarendon Press.
- SETTLAGE, P. H., ZABLE, M., & HARLOW, H. F. (1948). Problem solution by monkeys following bilateral removal of the prefrontal areas. VI. Performance on tests requiring contradictory reactions to similar and to identical stimuli. *Journal of Experimental Psychology*, **38**, 50-65.
- SQUIRE, L. R., SLATER, P. C., & CHACE, P. M. (1975). Retrograde amnesia: Temporal gradient in very long-term memory following electroconvulsive therapy. *Science*, **187**, 77-79.
- THOMPSON, R. F. (1960). Function of auditory cortex in frequency discrimination. *Journal of Neurophysiology*, **23**, 321-334.