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THE PSYCHOLOGY AND NEUROSCIENCE OF FORGETTING

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■ **Abstract** Traditional theories of forgetting are wedded to the notion that cue-overload interference procedures (often involving the A-B, A-C list-learning paradigm) capture the most important elements of forgetting in everyday life. However, findings from a century of work in psychology, psychopharmacology, and neuroscience converge on the notion that such procedures may pertain mainly to forgetting in the laboratory and that everyday forgetting is attributable to an altogether different form of interference. According to this idea, recently formed memories that have not yet had a chance to consolidate are vulnerable to the interfering force of mental activity and memory formation (even if the interfering activity is not similar to the previously learned material). This account helps to explain why sleep, alcohol, and benzodiazepines all improve memory for a recently learned list, and it is consistent with recent work on the variables that affect the induction and maintenance of long-term potentiation in the hippocampus.

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INTRODUCTION

The question of why people forget what they once knew has been continuously investigated for more than a century, and standard accounts of what we have learned about this fundamental issue can be found in almost any general psychology textbook. Unfortunately, the story that those books tell has changed over the years from a theoretically coherent (but ultimately incorrect) interference-based account of forgetting to an atheoretical laundry list of factors that may or may not play a role. Once, the standard story was that the lion's share of forgetting is caused by interference and that the main culprit is interference from prior learning (i.e., proactive interference) rather than subsequent learning (i.e., retroactive interference). Theoretical mechanisms such as unlearning, spontaneous recovery, and response competition initially offered a compelling theoretical explanation for the interfering effects of prior learning, but this way of thinking unraveled in the face of disconfirming evidence more than 30 years ago (Tulving & Madigan 1970). Because no new theory emerged to take its place, authors now typically claim that retroactive and proactive interference may both be important and that forgetting probably also involves retrieval failure due to changed or otherwise inadequate retrieval cues. The notion that decay might play a role, which was once almost universally rejected, has also been resurrected to the status of a possible contributing factor. The tentative and atheoretical nature of the modern account of forgetting is somewhat disappointing after so many years of diligent effort. In the pages to follow, I review findings from psychology, psychopharmacology, and neuroscience in an effort to extract a more compelling theoretical message from the large body of research on forgetting that has accumulated over the years.

When grappling with fundamental questions about the nature of memory and retrieval, psychological theories have often been informed by progress in related fields. For example, theories concerned with the distinction between implicit and explicit memory have relied heavily on developments not only in psychology but also in neuropsychology and neuroscience (e.g., Gabrieli 1998, Schacter 1992). By contrast, theories of forgetting have rarely ventured beyond the traditional boundaries of experimental psychology. As a case in point, consider the notion that memory traces consolidate over time (an idea that will figure prominently in the account of forgetting that is presented below). According to this idea, memories become less fragile and, therefore, more resistant to interference as time passes. Consolidation theory is a standard account in neuroscience, but it is scarcely even mentioned in the psychology literature. To see that this is true, consider the numbers shown in Table 1. This table shows, for various journals, the number of articles that include "memory" as a keyword relative to the number of articles that include both "memory" and "consolidation" as keywords (according to PsychInfo). For six standard psychology journals, an average of 379 articles about memory appears for every one article that happens to mention both memory and consolidation. For six standard neuroscience journals, the corresponding number is 14. Evidently, Keppel (1984) accurately expressed the attitude that implicitly pervades the study

TABLE 1 Number of articles that list "memory" as a key word (A), number that list "memory" and "consolidation" as key words (B), and the ratio of those two values (A/B) for each of six cognitive and six neuroscience journals

Cognitive journal	A	B	A/B	Neuroscience journal	A	B	A/B
Journal of Verbal Learning & Verbal Behavior	546	1	546	*Journal of Neuroscience*	363	26	14
Journal of Memory & Language	262	1	262	*Neuroscience*	216	18	12
Journal of Experimental Psychology	3816	9	424	*Learning & Memory*	158	27	6
Journal of Experimental Psychology: Learning, Memory & Cognition	875	3	292	*Behavioral Neuroscience*	493	29	17
Cognitive Psychology	189	1	189	*Brain Research*	817	60	14
Memory & Cognition	1142	3	381	*Brain*	1691	70	24
Mean cognitive	**1137**	**3**	**379**	**Mean neuroscience**	**623**	**46**	**14**

of forgetting when he said, "For a cognitively oriented psychologist, I find little connection between the behavioral evidence obtained from human learning experiments on the one hand and neurophysiological theory on the other" (p. 157). What I hope to show here is that the situation has changed rather dramatically in that respect, and I begin with a more detailed consideration of what was once the standard theoretical account of forgetting.

THE (ONCE) STANDARD STORY OF FORGETTING

Is Forgetting Due to Interference or Decay?

The question of whether forgetting is due to interference or to natural decay commanded a great deal of attention early in the twentieth century. To answer this question, an ideal experiment to perform is one in which a learning phase is followed by a retention phase during which the subject's brain remains biologically active (allowing for natural decay processes to unfold) while his or her mind remains in a quiescent state (so that no new learning interferes with the prior learning). A classic study by Jenkins & Dallenbach (1924) approximated this ideal experiment by comparing memory for nonsense syllables when subjects slept through the retention interval compared to when they remained awake. Interference theory would predict less forgetting during sleep due to the absence of new learning, whereas decay theory would predict no difference between the two conditions. Because Jenkins & Dallenbach (1924) found that subjects recalled more items when they slept than when they remained awake, interference theory won the day and decay

theory was essentially abandoned. Contributing to the demise of decay theory was McGeoch's (1932) oft-cited observation that the passage of time, per se, is not the cause of forgetting anymore than it is the cause of the physical deterioration associated with aging. Thus, a version of decay theory that attributes a causal role to the passage of time itself (a curious notion, to be sure) is conceptually flawed from the outset. A more reasonable decay theory might hold that forgetting is due to the deterioration of organic traces due to natural metabolic processes, but McGeogh (1932) was openly dubious about this possibility as well (though he obviously could not rule it out). His views were quite influential in the 1930s and 1940s, and they helped to cement the case in favor of interference theory.

The interference account of forgetting changed somewhat with the publication of a classic paper by Underwood in 1957. In that paper, Underwood considered the question of why it was that after subjects learned a list to a criterion of one perfect recall, the amount of forgetting that occurred over the next 24 hours varied greatly from study to study even though the studies in question involved similar stimulus materials (usually nonsense syllables) and similar subject populations (usually college students). Some studies reported that only 20% of the memorized items were forgotten after one day, whereas others reported that nearly 80% were forgotten. What Underwood discovered was that nearly all of the variability across studies could be explained by the number of prior lists the subjects had been asked to learn in the experimental setting. Studies in which subjects learned only a few prior lists reported much less forgetting than those in which subjects learned many prior lists. The inescapable conclusion was that forgetting was largely attributable to proactive interference (PI). Underwood (1957) went so far as to argue that retroactive interference (RI) is probably a minor cause of forgetting and that even the 20% of a memorized list that is forgotten over 24 hours when no prior lists are learned is more likely to be caused by proactive rather than retroactive interference. After all, subjects are more likely to have encountered similar interfering material at some point in their long past than in the 24 hours following their participation in a psychology experiment.

The realization that prior learning could profoundly affect the forgetting of subsequently learned material required a new theory, and interference theorists of the day developed one based on principles derived from the animal learning literature (Underwood & Postman 1960). Imagine that subjects learn a list of paired associates (the A-B list) and then later learn another list with the same stimulus terms but different response terms (the A-C list). When memory is later tested for the A-C list by presenting the A term as a retrieval cue and asking for the corresponding associate from the second list (i.e., C), performance will be worse than it would have been had the A-B list not been previously learned (i.e., PI will be observed). Moreover, the degree of proactive interference will be minimal shortly after the A-C list is learned but will increase as the retention interval following A-C learning increases. Theoretically, this occurs because while the subject is learning the A-C list, the B terms covertly come to mind. Because those terms are no longer correct, they are, in a sense, placed on an extinction schedule

(i.e., recalling those items is not reinforced). Eventually, the A-B associations extinguish, which is to say that they are unlearned. It has been well documented in the animal learning literature that extinguished responses (e.g., extinguished bar presses in rats) eventually spontaneously recover, and that was assumed to be true of the A-B associations as well. When the A-B associations spontaneously recover at some point well after the A-C list is learned, they will compete with the retrieval of the C terms on a recall test (hence, PI).

Problems with the Standard Story of Forgetting

A major difficulty with the PI-based account of forgetting is that the Jenkins & Dallenbach (1924) sleep study appeared to establish the importance of RI in forgetting. If PI were responsible for most forgetting, why would sleep (which eliminates RI) have such a positive effect on retention? Underwood (1957) readily admitted that he had no explanation for this, and he simply hypothesized that the mechanisms responsible for PI may not be active during sleep. Although there was no obvious basis for this claim, it proved to be a testable hypothesis, and Ekstrand (1967) performed the relevant experiment. In this study, subjects first learned two lists, an A-B list of paired associates followed by an A-C list of paired associates. Half the subjects learned these lists late at night and then slept for eight hours, whereas the other half learned these lists in the morning and then remained awake for eight hours. The key finding was that memory for both lists was enhanced by sleep. That is, memory for the C items was enhanced following sleep even though memory for the supposedly proactively interfering B items was also enhanced. This led Ekstrand to conclude that the mechanisms of PI (which presumably involve competition between the B and C items) are, if anything, enhanced by sleep. As such, the beneficial effects of sleep are more likely to be due to a reduction in RI, which is what Jenkins & Dallenbach (1924) had originally assumed.

In a comprehensive overview of the interference literature, Postman (1971) noted that sleep studies point to ". . .retroactive interference produced by the subject's normal waking activities as a condition of forgetting" (p. 1123), but he went on to note that researchers who subsequently investigated the specific sources of interference did not capitalize on this observation. As Postman (1971) put it: "As it turned out, the systematic analysis of the latter problem focused on proactive rather than retroactive effects" (p. 1123). Unfortunately, that analysis ultimately suggested that PI might not account for much normal forgetting in spite of its large effects in the laboratory. Underwood & Postman (1960), for example, set out to show that a subject's preexperimental learning history could affect the rate of forgetting for lists learned in the laboratory. Their basic strategy involved comparing the rates of forgetting over a period of one week for a list of three-letter words (like *age*, *end*, *him*, and so on) versus a list of three-letter trigrams (like *ati*, *est*, *han*, and so on). The idea was that the words would have many more preexperimental associative connections to other words than the trigrams would. Those associations would, according to the standard theory of the day, need to be unlearned in order to

form associations between the words on the list presented in the laboratory. No such unlearning would need to take place for the trigrams. Theoretically, the unlearned associations to the words would, in time, spontaneously recover and interfere with memory for the list of words. Thus, the rate of forgetting for words should exceed that for nonwords. Contrary to this prediction, the rates of forgetting were the same.

Conceivably, this failure to demonstrate the applicability of PI to real-life learning and forgetting was due to an inadequate theory. Indeed, as indicated above, the theory of unlearning followed by spontaneous recovery is no longer accepted, and if that theory is wrong, perhaps it does not follow that the rate of forgetting for words should exceed that for nonwords. More troubling for the notion that PI explains everyday forgetting, though, was a later study reported by Underwood & Ekstrand (1966, 1967). They showed that, in the laboratory, PI could be easily demonstrated if (as usual) the prior learning trials were massed. But if they were spaced over four days, which is the condition that presumably more closely reflects the way that extraexperimental learning has taken place, no PI was observed. If learning is typically distributed, these results suggest that PI may not be a major source of forgetting in everyday life. In a single sentence that appears without explanation in his earlier classic article, Underwood (1957) mentions that he included studies for consideration in his review only if the prior learning was massed. This curious (at the time) inclusion criterion makes sense in light of his later work suggesting that the effects of PI are not otherwise apparent.

As the work of Underwood and his colleagues began to cast doubt on the importance of PI in everyday forgetting, other studies continued to accumulate showing that the major assumptions of interference theory were simply untenable. Slamecka (1966), for example, showed that subjects do not unlearn previously acquired associations when learning new associations in the laboratory. By 1970, the field had clearly lost its patience with increasingly complicated interference theories of forgetting (e.g., Tulving & Madigan 1970), and little theoretical headway has been made since that time.

Summarizing the state of the art late in his career, Underwood (1983) said: "A relatively few years ago it seemed that a fairly comprehensive theoretical account of forgetting was close at hand, but that has slipped away. Some investigators have lost confidence in interference as a major cause forgetting, but none of the proposed replacements thus far has created a feeling that things are on a productive new track. But that will surely come" (p. 262). The productive new track that Underwood (1983) yearned for may be the track that interference theorists were on in the early part of the twentieth century. Indeed, the developments reviewed above suggest that the field may have made a wrong turn when it embraced PI as the primary cause of ordinary forgetting and when it adopted a cue-overload (A-B, A-C learning) approach to the study of the problem of forgetting. Quite possibly, the field was not very far off track even as late as 1951 when Carl Hovland, commenting on Jenkins & Dallenbach's (1924) classic sleep study, said "These experiments closely simulate the conditions of real life and indicate that intervening activity is a potent factor in producing forgetting" (p. 676). Note that the intervening activity

that is eliminated by sleep does not necessarily involve activities that are captured by A-B, A-C list learning methods. Indeed, as Underwood (1957) pointed out when making his case for the effects of PI, most of the intervening activity would involve materials unrelated to the original list. Even so, beneficial effects of sleep on memory are observed.

One of Underwood's students, Geoffrey Keppel, argued that, on the whole, the results point to what he called nonspecific RI as the major cause of forgetting in everyday life (Keppel 1968). I believe that Keppel was on the right track in spite of the later doubts he expressed about the utility of consolidation theory. However, his arguments were made at the end of the heyday of interference theory, and few have paid much attention to his case. The rather dramatic claim that the A-B, A-C list learning methods favored by experimental psychologists since the 1930s fail to capture some of the most important elements of the story of forgetting has certainly not penetrated the field's collective consciousness even though it was also endorsed by Ekstrand (1972) and Wickelgren (1977). In what follows, I try to pick up the story where Keppel (1968) left off many years ago.

Starting Over

One way to progress beyond the tentative laundry list account of forgetting that came to replace the once standard interference-based story is to assemble the basic facts and look for a common message. In what follows, I assemble some of the relevant facts from psychology, psychopharmacology, and neuroscience. A common theme running through that review is based on one of the oldest relevant neuropsychological considerations, namely, the temporal gradient of retrograde amnesia. Clinical reports dating back more than 100 years have suggested that brain damage leading to anterograde amnesia (i.e., to the inability to lay down new memory records) is also associated with temporally graded retrograde amnesia (Ribot 1881/1882). That is, memories formed prior to brain damage are impaired, but the effect depends on the age of the memory trace at the time the damage occurs, with more recently formed memories suffering the most. This phenomenon is known as Ribot's Law, and the results of later experimental investigations of retrograde amnesia generally agreed with its stipulations (Brown 2002, Squire et al. 1975).

For almost 50 years it has been clear that the medial temporal lobes, which include the hippocampus and adjacent cortex, play a critical role in the formation of new memories. When patient H.M. had those areas surgically removed in an effort to control his epileptic seizures, it eventually became clear that his ability to form new memories was severely and permanently impaired (Scoville & Milner 1957). It is perhaps not surprising, therefore, that studies have found that temporally graded retrograde amnesia is particularly likely to be observed if the brain damage in question involves the hippocampal region (e.g., Manns et al. 2003). Although studies involving human patients are fraught with interpretative complications because they necessarily rely on retrospective methods, a recent review of 13

more precisely controlled prospective animal studies corroborates the existence of temporally graded retrograde amnesia and its association with hippocampal lesions (Squire et al. 2001).

The temporal gradient of retrograde amnesia provides compelling evidence that memories consolidate over time and that the hippocampal formation (consisting of the hippocampus, dentate gyrus, subiculum, and entorhinal cortex) plays an important role in that process. If the hippocampal formation is damaged before the consolidation process is complete, recently formed memories that are still undergoing the consolidation process will be impaired. The idea that memories consolidate, which is nowhere to be found in recent cognitive theories of memory and forgetting, happens to be the standard story in the neuroscience literature, and it is often referred to as such (e.g., Dudai 2004, McGaugh 2000) even by those who disagree with it (e.g., Nadel & Moscovitch 1997, 2001). The contrast between the views of cognitive psychologists and cognitive neuroscientists is so complete and so striking that, paradoxically, it is almost easy to overlook. But the numbers presented in Table 1 suggest that the contrast is real. Keppel (1984) once again stated explicitly what many cognitive psychologists seem to say implicitly with the theories they propose: "I am simply not convinced that the concepts of perseveration and consolidation 'buy' the cognitive psychologist any explanatory power, except perhaps as a metaphor and as a reasonable explanation of retrograde amnesia" (p. 157). But three distinct domains of research, one conducted over the course of the last century by experimental psychologists and two others conducted much more recently by psychopharmacologists and neuroscientists, converge on the notion that much of what we forget has fallen prey to the nonspecific effects of retroactive interference and that the effects of such interference differ depending on the degree to which the memory trace has consolidated.

PSYCHOLOGY

The Mathematical Form of Forgetting

A seemingly extraneous issue that may in fact be intimately related to the temporal gradient of retrograde amnesia is the mathematical form of forgetting. A forgetting function is a plot of the amount remembered, R(t), as a function of time since learning, t. Almost everyone has an intuitive feel for what the forgetting function looks like. Everyone knows, for example, that the function will not increase with time but will instead decrease and that it will not be linear but will instead be curvilinear. But what is the mathematical form of this curvilinear function, and what does it imply about the nature of forgetting? If one had to hazard a guess as to the mathematical form of forgetting, a natural choice would be the exponential, $R(t) = ae^{-bt}$, where b and a are parameters analogous to the slope and intercept of a straight line. Many natural processes (e.g., radioactive decay) are exponential in form, and the exponential lends itself to simple mechanistic interpretations. One special property of the exponential is that it implies a constant proportional rate

of decay. If a function drops by 50% in the first hour after learning (e.g., from 80% correct to 40% correct), the fact that it is exponential in form tells you that it will drop another 50% (i.e., from 40% correct to 20% correct) in the next hour. In fact, the function will drop by a factor of 0.5 every hour indefinitely. This constant proportional rate of decay characterizes what are sometimes called memoryless processes, which are processes with properties that don't depend at all on the prior state of the system.

What would a memoryless memory system be? That is, what would the implications be if memories decayed exponentially? The implication might be that memories do not consolidate. The original notion of consolidation held that the future vulnerability of a memory trace depends on how old that trace is—traces that have managed to survive for a time become less vulnerable to interference and thus decay less rapidly than younger traces (Müller & Pilzecker 1900). Thus, if memories consolidate, a forgetting function that drops by 50% in the first hour might drop by only 40% in the next hour (and by a lesser amount each hour after that). The absence of consolidation, by contrast, would be most easily reconciled with a constant proportional rate of decay (which, in turn, would imply exponential forgetting).

Ebbinghaus (1885/1913) reported long ago that forgetting functions are logarithmic in form, whereas Wickelgren (1974) argued that forgetting functions were better described by a power law, such as $R(t) = at^{-b}$. Wixted & Ebbesen (1991, 1997) showed that a variety of empirical forgetting functions are accurately characterized by both the power and logarithmic functions (with a slight edge going to the former) and very poorly described by the exponential. While the power law of forgetting is probably the leading contender (cf. Anderson & Schooler, 1991), White (2001) argued that a modified exponential, the exponential-power function, performs much better than the simple exponential and should be given serious consideration as well (cf. Rubin & Wenzel 1996). From this bewildering array of nonexponential possibilities, one important common denominator has emerged: All of the candidate forgetting functions are characterized by an ever *decreasing* proportional rate of decay, which is a property that forgetting functions might be expected to possess if memories consolidated (i.e., became more resistant to interference) the longer they survived. That forgetting functions possess this property is implied by Jost's (1897) second law, which holds that if two associations are of equal strength but of unequal age, the older association will decay less rapidly than the younger one. In fact, these considerations point to a heretofore unnoticed relationship between Ribot's Law (1881/1882) and Jost's Law. The temporal gradient of retrograde amnesia (i.e., Ribot's Law) implies that memories become more resistant to the effects of *brain damage* as they age. The power law of forgetting and Jost's Law imply (but do not prove) that memories may also become more resistant to the more ordinary effects of *retroactive interference* as they age. From this point of view, Ribot's Law and Jost's Law are simply two sides of the same coin.

Wickelgren (1974) once considered a consolidation-plus-RI explanation for the power law of forgetting, but he ultimately rejected it based on what may be an

incorrect reading of the traditional interference literature. His possible misreading of that literature is not specific to him, and it may account for why psychologists have been so reluctant to embrace the notion that memories consolidate over time. The question of whether a temporal gradient of retroactive interference exists (the kind of gradient that is implied by the power law of forgetting) has been extensively investigated since the turn of the century, but it has never been thoroughly reviewed. What follows is an attempt to sort out that complicated literature.

Temporal Gradient of Retroactive Interference

EARLY STUDIES (1900–1933) A number of studies have attempted to address the question of whether or not the magnitude of retroactive interference differs depending on the temporal point of interpolated learning (i.e., the point during the retention interval when the interference occurs). If memories do need time to consolidate, one might imagine that the effects of subsequent interfering learning would have temporal properties much like the effects of subsequent hippocampal damage. That is, RI should affect younger traces more than it affects older traces. If that is true, and if RI is a significant cause of forgetting, then it would provide a ready explanation as to why forgetting functions are not exponential in form but are instead characterized by an ever-decreasing proportional rate of decay.

The earliest study to address this question was performed by Müller & Pilzecker (1900). An English translation of Müller & Pilzecker's (1900) monograph is unfortunately not available, but Lechner et al. (1999) provide a summary of some of the more important studies performed by these German researchers. In one experiment, subjects studied six pairs of syllables (list A) and then studied a second list (list X) either 17 seconds or 6 minutes later. The results showed that the retention of list A was impaired on a cued recall test 1.5 hours later in the first condition only. Why would list X impair later memory for list A when learned immediately following list A but not when it was learned six minutes after that list? Müller & Pilzecker (1900) argued that physiological processes associated with list learning perseverate for a period of time after learning and that this perseveration serves to consolidate the memory trace. If list X is learned before the consolidation of memory for list A is complete, retroactive interference occurs.

Certain aspects of Müller & Pilzecker's (1900) preferred experimental design warrant close attention. Of particular importance is what the subjects were asked to do during those portions of the retention interval that did not involve studying interfering material. According to Lechner et al. (1999), in Müller & Pilzecker's early experiments, subjects were given reading material to help them suppress any tendency to rehearse the list during retention intervals. However, this practice was later abandoned as evidence for retroactive inhibition emerged, and subjects were merely instructed not to rehearse. It seems clear that Müller & Pilzecker came to believe that mental exertion itself is what interferes with the consolidation process:

> After all this, there is no alternative but to assume that after reading a list of syllables certain physiological processes ... continue with decreasing intensity for a period of time. These processes and their facilitating effects

> on these associations are being weakened to a greater or lesser extent if the experimental subject experiences further mental exertion immediately after reading a list. (pp. 196–197) [translation by Lechner et al. (1999), pp. 81–82]

Thus, a test of the consolidation account amounted to imposing mental exertion at some point during a retention interval that was otherwise characterized by mental quietude. Note how different this idea is from the subsequent notion that came to dominate the interference literature, namely, that an A-B association is disrupted by the subsequent acquisition of an A-C association. The relevance of "cue-overload" effects like this to ordinary forgetting is what Keppel (1968), Ekstrand (1972), and Wickelgren (1977) all came to doubt. By contrast, the importance of mental exertion, which is the kind of RI that Müller & Pilzecker (1900) apparently had in mind, was never fully investigated. One of the main suggestions I make throughout this review is that interference from mental exertion (and its attendant memory formation) is much more relevant to everyday forgetting than interference due to the much more widely studied effects of cue overload.

These considerations help to reconcile the results of two later studies concerned with the temporal gradient of retroactive interference that are usually described as leading to opposite conclusions. One study, by Robinson (1920), presented subjects with a list of 10 three-digit numbers to recall after a 20-minute retention interval. At different points during the retention interval, subjects learned an interfering list of three-digit numbers. Some subjects learned the interfering list shortly after the original list, whereas other groups learned the interfering list later in the retention interval. No temporal gradient of RI was observed (i.e., the degree of RI was the same no matter where the interfering list was presented), which seems inconsistent with the results reported by Müller & Pilzecker (1900) and with a consolidation account in general. However, retention-interval activity during nonstudy periods in this experiment did not involve mental relaxation but instead involved reading newspaper articles from the *Chicago Tribune*. Thus, Robinson (1920) continued to use a procedure (namely, filling the retention interval with reading material) that had been determined by Müller & Pilzecker (1900) to be inappropriate. By design, mental exertion was in effect throughout the retention interval in Robinson's (1920) study, and this may explain why no temporal gradient of RI was observed.

Skaggs (1925) took Müller & Pilzecker's (1900) ideas more seriously and diligently attempted to achieve periods of mental quietude during the "off" times of the retention interval. In fact, Skaggs (1925) went further than Müller & Pilzecker (1900) in that he attempted to ensure that these quiet moments were even free of any rehearsal of the list material. Conceivably, in Müller & Pilzecker's (1900) experiment, interfering material presented a mere 17 seconds after learning might have been especially detrimental because it interfered with spontaneous rehearsal of the list, not because it interfered with an automatic physiological consolidation process. Skaggs (1925) attempted to address this issue and found that it was not easy. As he put it: ". . .it is extremely difficult to secure many 'ideal' rest intervals—ideal in the sense that the subject was mentally passive and indifferent and entirely

away from the original learning material" (pp. 32–33). As such, Skaggs (1925) relied heavily on introspective reports to identify retention intervals characterized by mental quietude, and he came to believe that "Trained subjects are a necessity" (p. 58) because "As subjects become practiced they are better able to take an indifferent and passive attitude during the rest interval" (p. 59). Using practiced subjects, Skaggs (1925) found a temporal gradient of RI in many experiments involving stimulus materials as different as chess positions, words, nonsense syllables, and syllable pairs. In one experiment, for example, subjects learned nonsense syllables and then tried to recall them six minutes later. The interfering material consisted of unrelated mental exertion (namely, solving algebra problems) at different points in an otherwise quiet retention interval. The results generally supported Müller & Pilzecker's (1900) original observation of a temporal gradient of RI, though not all subjects showed the effect.

In other experiments, Skaggs (1925) also confirmed that similarity between the original and interfering material was an important variable, with greater similarity being associated with greater interference. However, no matter how dissimilar the intervening material was, substantial effects of RI were obtained. Conceivably, the interference mechanisms associated with the nonspecific effects of mental exertion differ from those associated with similarity, an argument that was actually made in eloquent fashion by Skaggs (1933) in a little-known comment that appeared in the *Journal of Comparative Psychology*. One process (mental exertion) may reflect an influence of intervening activity on the consolidation of recently formed memory traces. In his words:

> The writer has argued long for the view that there are two factors causing what is now called retroactive inhibitory effects. In one case, a strong mental-neural activity cuts in and disorganizes an on-going mental-neural process, a process of neural inertia. (p. 413)

The second inhibitory process (similarity) may reflect a retrieval phenomenon related to what nowadays might be regarded as cue overload effects. Skaggs (1933) referred to the second process as *reproductive inhibition*, which involved ". . .the establishment of wrong associative tendencies *which operate at the time of recall*" (p. 413, emphasis in original).

This very distinction was considered by Robinson (1920) in an interesting and thoughtful discussion section at the end of his monograph. However, he rejected the possibility that retroactive interference might involve two mechanisms (largely because he failed to find a temporal gradient of RI), and he clearly favored the idea that RI is always due to cue overload (or what he termed "a matter of transfer"). What I am suggesting here is the exact opposite, namely, that ordinary forgetting may not be a cue-overload phenomenon as much as it reflects the nonspecific effects of mental exertion and memory formation.

Why would mental exertion induce forgetting? One possibility, which is bolstered by the psychopharmacological research reviewed below, is that the resources available to consolidate recently formed memory traces are not unlimited. Mental

exertion, such as reading newspaper articles, undoubtedly activates hippocampal circuits as memories associated with that exertion are formed. Even if the intervening study material is not related to the original learning in any obvious way, the new learning draws on a limited pool of resources that may have otherwise been available to consolidate the original learning. As a result, memory for the original material suffers. Note that this way of thinking fits with the findings reported by Jenkins & Dallenbach (1924), whose sleeping subjects presumably avoided mental exertion unrelated to the nonsense syllables that they had studied earlier (cf. Minami & Dallenbach 1946). It also fits with studies investigating the effects of alcohol and benzodiazepines on memory and with studies investigating the role of long-term potentiation in the formation of new memories. Before reviewing those literatures, however, I review additional research on the temporal gradient of retroactive interference that was conducted at a time when cue-overload procedures dominated the study of forgetting.

LATER STUDIES (1933–1974) Quite a few studies performed after 1925 also investigated the question of whether or not a temporal gradient of RI exists. All of these studies relied on the use of similarity or cue overload (not mental exertion per se) to create RI during the retention interval. As such, those studies introduced issues that did not complicate the studies performed by Müller & Pilzecker (1900) and Skaggs (1925). In addition, none of the subsequent studies attempted to control the nature of mental activity that occurred during the off times of the retention interval. It actually would have been impossible to do so because these studies involved retention intervals on the order of days, not minutes. These nontrivial differences between the earlier and later studies might be expected to result in different outcomes. Then again, if the intervening cue-overload learning (e.g., intentionally memorizing a list of paired associates to a criterion of one perfect recall) was significantly more intense than the uncontrolled mental activity during the remainder of the retention interval, and if cue-overload effects do not have temporal properties that are opposite in direction to the effects due to mental exertion itself, then some evidence of the expected temporal gradient might be observed after all. Moreover, the later cue-overload studies have the advantage of investigating the temporal gradient over a much longer time period than the earlier studies did. Recent evidence suggests that memories consolidate over a much longer period of time than the few minutes envisioned by Müller & Pilzecker (1900), so it is worth knowing whether the temporal gradient of RI also extends over a time period that is longer than those employed by earlier researchers.

What did these later cue-overload studies find? Wickelgren (1977) summarized the relevant literature as follows:

> . . .studies of both recall and recognition of AB associations as a function of the delay between AB learning and subsequent AC interfering learning are virtually unanimous in rejecting the hypothesis that greater interference is obtained the shorter the delay between original learning and subsequent interference learning. (Wickelgren 1977, p. 385)

That is, according to Wickelgren, a temporal gradient of RI was not observed, and this is why he rejected the idea that the power law of forgetting arises because memories become less vulnerable to the effects of interference over time. Since this is, so far as I can determine, the standard reading of the relevant literature, it is perhaps not surprising that the field of psychology lost interest in the notion of consolidation. However, a closer look at these studies reveals that several contained a critical design flaw that requires that they be excluded from consideration. Moreover, most of the other studies actually found at least some evidence for a temporal gradient of retroactive interference even though they were not ideally suited to the question.

The flawed studies typically involved an A-B paired-associates learning phase followed by a test of cued recall sometime later (e.g., after a one-week retention interval). An interfering A-C list was learned either immediately after A-B learning or immediately before the recall test. Several studies using a design like this consistently failed to find any evidence that interference was greater when the A-C list was learned immediately after the A-B list was learned (Houston 1967, Howe 1969, McGeoch 1933, McGeoch & Nolen 1933), a result that appears to weigh against a consolidation account. However, whereas an A-C list that is learned shortly after the A-B list might interfere with A-B traces that are incompletely consolidated, an A-C list learned shortly before a recall test should impair retrieval of the A-B traces due to retrieval inhibition (e.g., Anderson et al.1994). That is, strengthening some items associated with a retrieval cue will, for a limited period of time, decrease the likelihood that other items associated with the cue will be recalled (MacLeod & Macrae 2001). Evidence that this is true can be seen in the results of some of the studies considered below. As such, a design like this compares two conditions that ought to be associated with reduced recall (for different reasons) relative to a condition involving an intermediate point of interpolated A-C learning. Skaggs (1933) explicitly warned against this kind of design when commenting on McGeoch & Nolen's (1933) study, but some of the later researchers seem not to have taken notice. In a cue-overload design, the use of more than two points of interpolated learning should reveal an inverted U pattern.

Several studies did include an intermediate point of interpolated learning, and these studies are more relevant to the question of interest. Sisson (1939) conducted one such study in which subjects first studied a ten-word list and then completed a free recall test 48 hours later. Different groups of subjects learned an interfering list of ten words (which were synonyms of the words in the original list) 0, 24, or 48 hours after the presentation of the first list (the latter occurring just prior to the recall test). Figure 1 reproduces the results of this study, and it is clear that a highly significant inverted U was obtained. One interpretation of this result (which is, of course, not the only interpretation) is that interfering material studied shortly after original learning permanently interfered with consolidation of the original list, whereas interfering material studied shortly before recall temporarily interfered with retrieval of the original list. The least interference occurred at the intermediate retention interval, perhaps because memories had been given a chance to consolidate and competition at retrieval was relatively

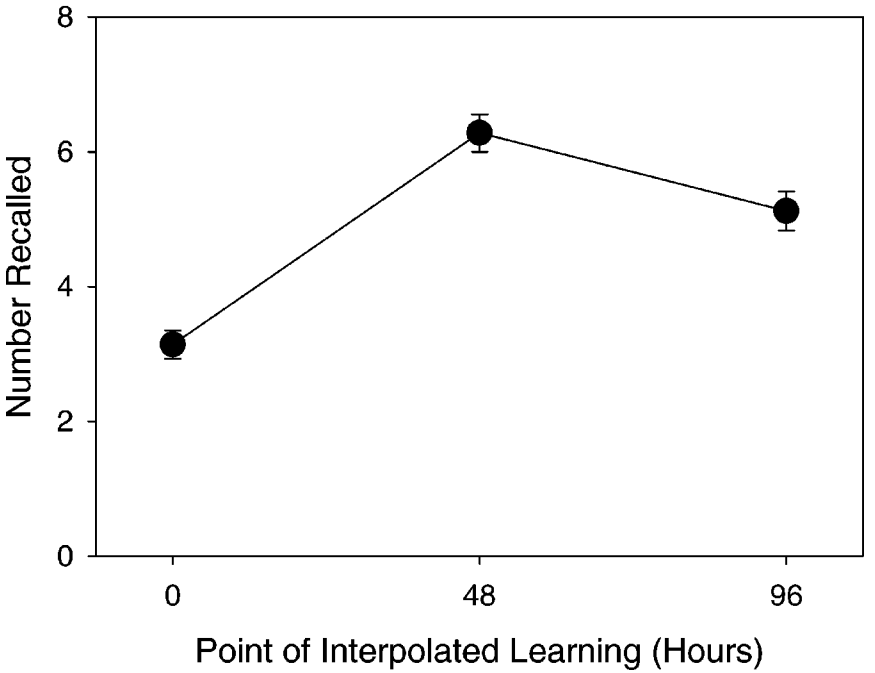

Figure 1 Number of items recalled from the original list after a 96-hour retention interval. An interfering list was learned immediately after the original list, 48 hours after the original list, or immediately prior to the retention test. The data are taken from Sisson (1939).

low due to the 24-hour gap between interfering learning and recall of the original list.

Postman & Alper (1946) conducted a more traditional verbal learning study in which subjects learned a list of ten paired associates (the A-B list) to a criterion of one perfect recall followed by a cued recall test 16 days later. Different groups of subjects learned an A-C interfering list at one of nine points during that retention interval (0, 1, 2, 4, 8, 12, 14, 15, or 16 days after A-B learning). A problem with this study is that there were only seven subjects per condition, so the data were predictably variable (and apparently uninterpretable). However, some indication of what was found can be gleaned by combining groups into short (0, 1, 2), medium (4, 8, 12), and long (14, 15, 16) conditions in order to create larger sample sizes. Figure 2 shows the results when the data are collapsed in that manner, and what emerges, once again, is the anticipated inverted U. This study obviously does not offer strong evidence in favor of the reality of that pattern, but the results certainly cannot be taken to weigh against the idea (even though they often are).

Archer & Underwood (1951) conducted another study involving an intermediate point of interpolated learning. In that study, subjects learned a list of ten paired associates (the A-B list) followed by a cued recall test 48 hours later. Different

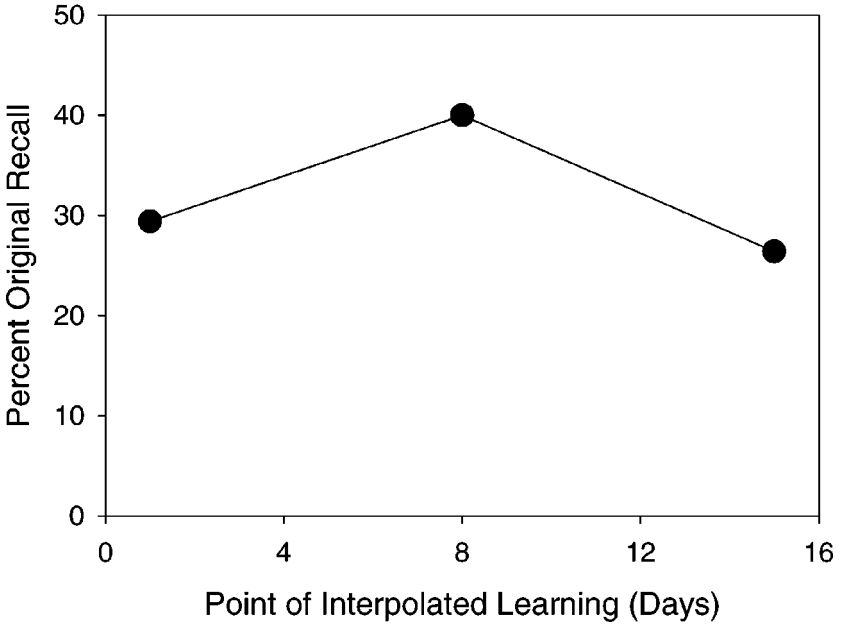

Figure 2 Percentage of items recalled from the original A-B list after a 16-day retention interval. Different groups of subjects learned an interfering A-C list at one of nine different points in the retention interval. The data have been averaged over sets of three adjacent groups to decrease the extreme variability that would otherwise be apparent. The data are taken from Postman & Alper (1946).

groups of subjects learned an A-C interfering list immediately following the A-B list, 24 hours after the A-B list, or 48 hours after the A-B list (just prior to the recall test). The degree of A-C learning was also varied across three levels, and this manipulation was crossed with the point of interpolated learning manipulation, yielding nine groups in all (three levels of A-C learning by three points of interpolated learning). The A-B list was learned to a criterion of one perfect recall, whereas the interfering A-C list was learned to a criterion of 60% correct (low), 100% correct (medium), or 100% correct plus five additional presentations of the A-C list (high). The question of interest was whether an inverted U pattern was observed. Such a pattern might not be expected in the low degree of A-C learning condition because that condition may not have entailed a degree of mental activity and memory formation much beyond what would have occurred anyway. The condition most likely to result in the expected inverted U is the high degree of A-C learning condition. Figure 3 shows the results of this experiment. No inverted U is evident in the low and medium conditions, but the inverted U pattern is clearly evident in the high degree of learning condition. Statistical power was quite low in that condition because there were only ten subjects per group. Still, a quadratic trend analysis performed (by me) on the data from this condition reveals a marginally significant effect, $F(1,74) = 2.83$, $p < 0.10$.

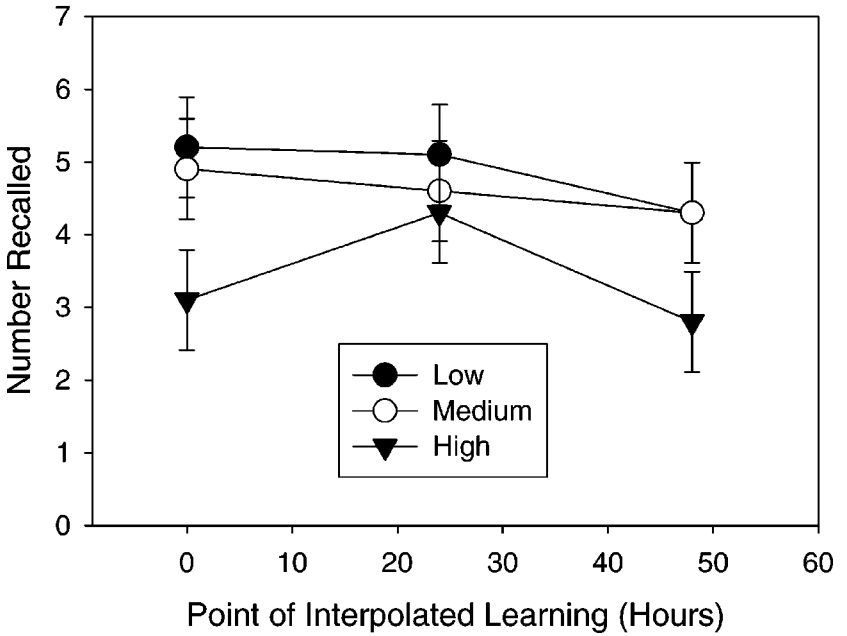

Figure 3 Number of items recalled from the original A-B list after a 48-hour retention interval. Different groups of subjects learned an interfering A-C list at one of three different points in the retention interval, and the degree of A-C learning was varied over three levels (for a total of nine groups in all). The data are taken from Archer & Underwood (1951).

Another study often cited as weighing against the notion of a temporal gradient of retroactive interference was performed by Newton & Wickens (1956). This study was essentially a replication of the medium degree of A-C learning condition from Archer & Underwood (1951), except that it involved twice as many subjects. Using a standard analysis of variance, they found no statistically significant effect of the temporal variable, but their results are reproduced in Figure 4. Once again, a clear inverted U is evident, and a higher-power quadratic trend analysis performed on these data (again, by me) yielded a significant result,[1] $F(1,54) = 5.91$, $p < 0.05$. Other findings reported by Newton & Wickens (1956), it should be acknowledged,

[1]Newton & Wickens (1956) did not provide the mean square error term for their Experiment 1, a value that is needed to perform a quadratic trend analysis. However, they did provide mean square error terms for their Experiments 2 and 3. The latter experiments were very similar to the first (same number of subjects, same number of lists, same number of learning trials, etc.) except they involved an A-B, C-D design instead of an A-B, A-C design. The mean square error terms for those two experiments were 3.52 and 3.25, respectively, and I used the average of those two values to perform the quadratic trend analysis for the data from Experiment 1.

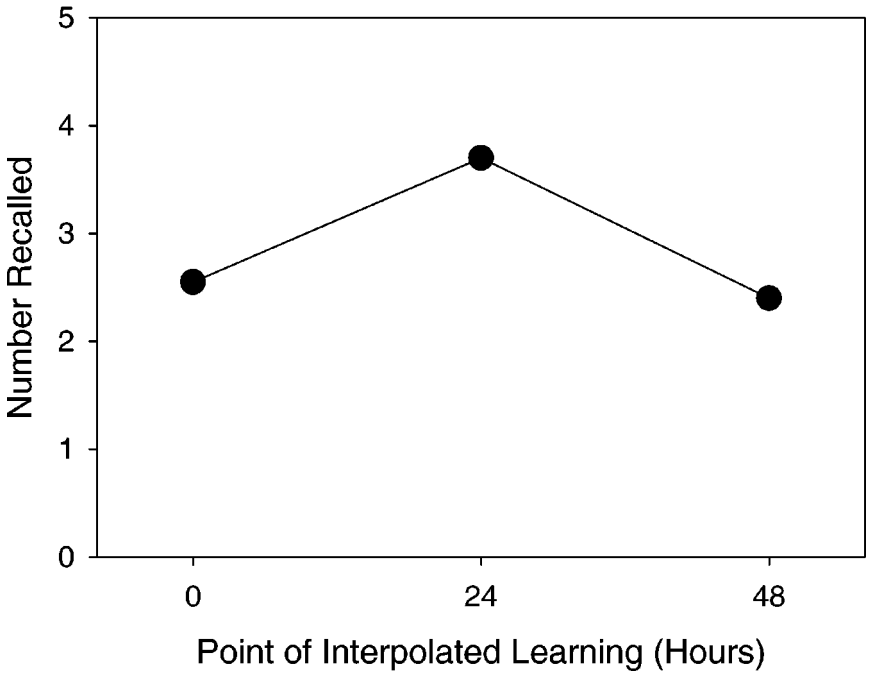

Figure 4 Number of items recalled from the original A-B list after a 48-hour retention interval. Different groups of subjects learned an interfering A-C list at one of three different points in the retention interval. The data are taken from Newton & Wickens (1956).

are hard to reconcile with any theory. In addition to the A-B, A-C study just described, they reported two studies involving an A-B, C-D design (with the C-D learning occurring at three points during the 48-hour retention interval). The results of those studies indicated that interference increased monotonically as the delay between A-B and C-D learning increased. This result is not predicted by a consolidation account or any other account based on interference theory, so its implications are hard to fathom.

The last two studies that investigated the temporal gradient of RI were published in 1974. Both involved cue-overload procedures and relatively short retention intervals. Wickelgren (1974) briefly described an experiment that he performed to address this issue. That experiment involved a continuous associative recognition procedure in which subjects were presented with a long series of paired-associate words to learn. Occasionally, a test pair was presented and subjects were asked to indicate whether the pair was intact (consisting of two words that had appeared together earlier in the series) or rearranged (consisting of two words that had appeared earlier in the series as part of different pairs). Because the entire retention interval between study and test was filled with the intentional learning of

intervening word pairs, the temporal point of interpolated interference due to memory formation itself was not actually manipulated. Instead, memory formation was in effect throughout. What was manipulated was the temporal point of interpolated interference due to cue overload. Thus, if an A-B word pair had been studied earlier in the series, an interfering A-C pair was presented either early or late in the retention interval. Compared to a control condition, performance was impaired by the presentation of an A-C pair, but the degree of impairment was the same whether the A-C pair appeared early or late in the retention interval (i.e., no temporal gradient was observed).

Wickelgren's (1974) experiment involved a retention interval filled with the intentional formation of new memories, and memory formation itself may be the kind of interference that degrades previously encoded memories (more so for young, unconsolidated traces than for older, more consolidated traces). Cue overload effects are almost certainly a retrieval phenomenon, and consolidation theory does not speak to the question of whether cue overload effects vary depending on the temporal point of interpolated interference. Thus, from this perspective, Wickelgren's (1974) findings should not have been taken as evidence against the temporal gradient of RI.

Finally, a study performed by Landauer (1974) found clear evidence of a temporal gradient. Subjects in this experiment studied a continuous series of syllable-digit paired associates (such as CEM-2). Critical pairs (i.e., pairs that would be tested for memory at some later point) were followed by interfering items after varying delays, with filler items being easy, nonattention-demanding, nonconfusable word-digit pairs. Thus, the background task during the retention interval did not involve complete mental quietude, but the mental demands were reduced relative to the demands associated with learning the interfering material. A retention test conducted 20 minutes later revealed that memory for the critical items was worse when the difficult interfering items appeared shortly after the critical items compared to when the interfering items were delayed. In a second experiment, Landauer (1974) found a temporal gradient of RI even when more demanding material was presented throughout the retention interval. Given the results of Wickelgren's (1974) study and earlier research by Robinson (1920), who also used a filled retention interval, one might have expected to see the absence of a temporal gradient in this case, but one was found anyway.

SUMMARY The main point of this section is that when interference consists of mental exertion imposed on an otherwise quiet retention interval, a temporal gradient of retroactive interference is reliably observed. Similar effects are observed when traditional A-B, A-C designs are used, but the additional complications introduced by the use of a cue-overload procedure have obscured that fact for many years. The results point to a theory of forgetting according to which the processes associated with the formation of new memories retroactively interfere with previously formed memories that are still undergoing the process of consolidation. The temporal gradient of RI can be hard to detect because, as Skaggs (1925) noted long

ago, achieving mental quietude is not easy, and mental quietude may be essential to deactivate hippocampal circuits that, when active, interfere with the consolidation of prior memories.

For similar reasons, imaging studies that have attempted to detect hippocampal activation during a retrieval task have often failed (Martin 1999). What makes that activity hard to find, perhaps, is that the hippocampus may be active during retrieval tasks as well as during baseline comparison tasks that do not nominally involve encoding and retrieval. As Martin (1999) observed, much evidence is consistent with the idea that the medial temporal lobe ". . .is automatically engaged whenever an event is experienced" (p. 62). If the hippocampus is active both when the subject is given a retrieval task and when the subject is left to his her own devices, then detecting a contrast in activity between a test condition and baseline condition will be difficult indeed. The use of trained subjects may help to achieve the mental quietude necessary to reduce hippocampal activity, but the psychopharmacological methods reviewed below may do so in a more efficient way.

PSYCHOPHARMACOLOGY

The Curious Phenomenon of Retrograde Facilitation

As indicated above, damage to the medial temporal lobes induces anterograde amnesia (the inability to form new memories) as well as temporally graded retrograde amnesia (an impairment of recently formed memories). Anterograde amnesia can be induced by methods other than hippocampal damage, and some of these methods actually result in retrograde *facilitation*. That is, recently formed memories are retained better than they otherwise would have been even though new memories cannot easily be formed. A consideration of the conditions associated with this phenomenon reinforces the view that memories consolidate over time and that much of what we forget is lost because of retroactive interference arising from ordinary mental exertion and consequent memory formation acting on partially consolidated memory traces. The argument to be advanced below is that certain agents (such as alcohol and benzodiazepines) close the hippocampus to new input, thereby inducing anterograde amnesia, without compromising its ability to consolidate previously formed memories. Because new input is prevented, recently formed (and, therefore, incompletely consolidated) memories are protected from the retroactive interference that they would otherwise encounter. As such, these drugs act in the same way that sleep does even though the individual remains conscious. By contrast, hippocampal lesions both prevent new input (resulting in anterograde amnesia) and terminate the ongoing consolidation of recently formed memories (resulting in retrograde amnesia as well).

ALCOHOL The anterograde amnesic effects of alcohol consumed prior to the learning of new material have been well established (Lister et al. 1987). The extreme version of this effect is alcoholic "blackout," which involves a complete

loss of memory for events occurring while the individual was conscious but extremely intoxicated. It is generally accepted that blackouts are not the result of state-dependent learning but instead reflect a failure to encode or consolidate new information (Lisman 1974). In spite of its effects on the formation of new memories, alcohol intoxication generally does not affect one's ability to retrieve old memories (Birnbaum et al. 1978).

Whereas alcohol consumption induces a certain degree of anterograde amnesia for material studied under the influence of the drug, many studies have reported that it actually results in improved memory for material studied just prior to consumption (Bruce & Pihl 1997; Lamberty et al. 1990; Mann et al. 1984; Parker et al. 1980, 1981). This phenomenon is referred to as retrograde facilitation or retrograde enhancement, and its existence makes alcohol-induced amnesia quite unlike the amnesia produced by damage to the medial temporal lobes.

Theories advanced to explain this curious phenomenon include reduced retroactive interference versus a direct enhancement of the consolidation process. Efforts to differentiate these possibilities have proven to be inconclusive (e.g., Hewitt et al. 1996), so a choice between them will probably depend on the identification of the specific physiological mechanism responsible for the observed effects. As described in more detail below, recent evidence suggests one very plausible candidate, namely, the effects of alcohol on long-term potentiation. For the moment, though, it is worth pointing out that parsimony also favors the interference interpretation. That is, given the interference interpretation, the amnesic effects of alcohol can explain the retrograde facilitation of previously formed memories without having to postulate an additional effect of the drug on the consolidation process itself. By contrast, the idea that alcohol directly enhances the consolidation process seems less parsimonious and, in some ways, more problematic. Substances that enhance the consolidation process directly would be expected to result in both anterograde facilitation and retrograde facilitation, not anterograde *amnesia* and retrograde facilitation (which is what alcohol does). One such substance that is widely assumed to directly enhance the consolidation process is glucose, and, as might be expected, it does result in both anterograde and retrograde facilitation (Manning et al. 1992).

The simplest view, therefore, is that alcohol facilitates recently established memories precisely because it prevents the formation of new memories that would otherwise cause retroactive interference (Mueller et al. 1983). Although it has not been specifically shown, it stands to reason that drinking alcohol does not protect memories that are years old (and fully consolidated). Instead, it is the recently formed memories that differentially benefit because, theoretically, those are the ones most vulnerable to the effects of RI. This conclusion is the same one that emerged from a review of studies concerned with the temporal gradient of retroactive interference. Those studies were concerned with the effects of *introducing* RI, and they revealed that more recently formed memories are affected to a greater extent than older memories. The alcohol studies cited above are concerned with

the effects of *subtracting* RI, and the parallel result obtains (namely, newly formed memories are enhanced to a greater extent than older memories).

BENZODIAZEPINES Retrograde facilitation has also been observed with another class of amnesia-inducing drug, namely, benzodiazepines. The basic experimental paradigm is the same as that used with alcohol. Subjects typically study one list of words prior to drug administration and then study another list following drug administration. Memory for both lists is tested sometime later (usually while the subject is still under the influence of the drug), and performance is compared to that of a placebo control group. Typically, the drug group exhibits impaired recall for the list learned under the influence of the drug (thereby confirming its amnesia-inducing properties) and enhanced recall for the list learned prior to taking the drug (Coenen & Van Luijtelaar 1997, Fillmore et al. 2001, Hinrichs et al. 1984, Weingartner et al. 1995). Explanations for the retrograde enhancement effect once again include reduced interference, enhanced consolidation, or enhanced retrieval.

Coenen & Van Luijtelaar (1997) argued that the effects of benzodiazepines on memory were analogous to the beneficial effects of sleep reported by Jenkins & Dallenbach (1924). In both cases, information learned prior to sedation is remembered better than it otherwise would have been because retroactive interference is reduced due to the reduced rate of information uptake while sedated (or asleep). Note that this explanation is entirely in line with the idea that ordinary forgetting is a retroactive effect of subsequent memory formation that accompanies ordinary mental activity. If mental activity is reduced by sleep or if memory formation associated with mental activity is reduced by alcohol or a benzodiazepine drug, prior memories are protected from the effects of retroactive interference. And the memories that are protected are those that were recently formed and have not yet had a chance to consolidate.

Although reduced retroactive interference seems to be the most parsimonious account of retrograde facilitation (cf. Hinrichs et al. 1984), Weingartner et al. (1995) performed the only direct test of this hypothesis, and they arrived at a different conclusion. Their study is worth considering in some detail because it shows just how different the implications of a study can appear to be depending on whether one views them through the lens of traditional interference theory or in terms of the alternative nonspecific view of interference proposed here. Weingartner et al. (1995) presented subjects with a list of 12 words presented at a rate of one word per second ten minutes prior to the administration of triazolam (a benzodiazepine) or placebo. Ninety minutes after drug or placebo administration, a second, interfering, list of 12 words was presented at the slower rate of one word every five seconds. Twenty minutes later (nearly two hours after drug or placebo), a recognition test was administered that consisted of the 24 words from the two study lists as well as 12 new words. Memory for the predrug list was enhanced relative to the placebo control group (i.e., retrograde facilitation was observed), but, somewhat surprisingly, memory for the postdrug list was not significantly impaired

(i.e., an amnesic effect for these words was not observed). Why the typical amnesic effect was not obtained for the postdrug words is not clear, but the fact that those words were successfully encoded seemed to provide an unexpected opportunity to test the interference account. Specifically, according to a standard interference account, because the interfering postdrug words were learned well, the presumed interfering force was still in play. As such, no retrograde enhancement should have been observed. Because the typical enhancement of memory for predrug words occurred anyway, it seemed to follow that the interference explanation must be incorrect.

This interpretation makes sense in light of traditional interference theory, according to which interference arises mostly from the subsequent study of similar materials. If the only material that would retroactively interfere with the predrug list consists of words similar to the ones that appeared on that list, then the retrograde enhancement effect observed in this study is hard to explain on the basis of reduced retroactive interference. However, when considered in light of a theory that attributes forgetting to the retroactive effects of mental exertion and memory formation in general, the results do not weigh against an interference interpretation at all. In fact, they support that view.

Subjects in this study were tested nearly two hours after drug administration, and during much of that time they almost surely encoded much less information than they otherwise would have. True, they did manage to encode 12 words during one minute of that two-hour period to the same extent as in the placebo condition, but other evidence reported by Weingartner et al. (1995) clearly demonstrates that the rate of memory formation was greatly reduced for much of the rest of the postdrug period. While still under the influence of the drug, for example, these same subjects studied 21 sentences shortly after taking the recognition test, and their subsequent recall for words in those sentences was markedly impaired. Thus, the drug did induce a state of amnesia (thereby reducing the demands placed on the hippocampus) even though, for one 60-second period, subjects managed to encode a list as well as they ordinarily would have in the absence of the drug.

Similar considerations also apply to an earlier article by Parker et al. (1981), in which they argued that the facilitative effects of alcohol on prior memories are probably not due to interference reduction because, in that study, "...no other formal task was administered to subjects in the intoxicated state, [so] there was no task-related source of interference which alcohol could reduce" (p. 91). However, according to the view espoused here, the interference that matters most is not necessarily task related—it is the interference that accompanies ordinary mental activity and the memory formation associated with that activity (which was undoubtedly reduced by alcohol).

Temporal Properties of Retrograde Facilitation

To summarize, sleep, alcohol, and benzodiazepines all result in retrograde enhancement of memory, and, theoretically, they all do so for the same reason:

The reduced rate of memory formation protects recently formed memories from interference, interference that would otherwise arise because of the demands placed on a limited-resource hippocampal system. Although it has not been shown that a temporal gradient exists for the retrograde enhancement effects of alcohol and benzodiazepines, such a gradient almost surely exists. Whereas a glass of wine might help you to retain a recently encoded prewine conversation, it seems unlikely to enhance all prior memories dating back to childhood to a similar degree. The same arguments apply to the effects of sleep, but in this case the relevant experiments demonstrating the temporal gradient have actually been performed.

As described in Ekstrand (1972), Heine (1914) exposed subjects to nonsense syllables either immediately or two to three hours prior to sleep. A savings test conducted after a retention interval of 24 hours revealed better retention for the group that went to sleep immediately. Ekstrand (1972) reported a similar experiment involving memory for paired-associate words following a 24-hour retention interval in which subjects slept either during the eight hours that followed list presentation or during the eight hours that preceded the recall test. In the immediate sleep condition, 81% of the items were recalled; in the delayed sleep condition, only 66% were recalled. The enhanced performance in the immediate sleep condition presumably arises because, in that condition, memories were protected from interference during the time period when they are the most vulnerable (i.e., shortly after the memories were formed). Note how similar these studies are to the interference studies conducted long ago by Skaggs (1925). Whereas Skaggs manipulated the point of interpolation of mental exertion on a background of mental quietude and found a temporal gradient of interference (with more recent memories being differentially impaired), Heine (1914) and Ekstrand (1972) manipulated the point of interpolation of mental quietude (i.e., sleep) on a background of ordinary mental exertion and found a temporal gradient of retroactive facilitation (with more recent memories being differentially facilitated). The studies were actually conceptually identical (e.g., the immediate sleep condition could be construed as a delayed interference condition), and the results were the same.

NEUROSCIENCE

As already indicated, the standard view in neuroscience holds that new memories that have not yet had a chance to consolidate are more vulnerable to the effects of hippocampal damage than older memories are. As I argued above and as Müller & Pilzecker (1900) argued long ago, new memories are also more vulnerable to the effects of retroactive interference than older memories. This is why, according to my argument, new memories are actually enhanced when subjects subsequently take an amnesia-inducing drug or fall into a state of sleep.

The prevailing view of how memories are initially formed is that the process involves a rapidly formed and relatively long-lasting increase in the probability that

postsynaptic neurons in the hippocampus will fire in response to neurotransmitters released from presynaptic neurons. The laboratory analog of this theoretical memory mechanism is long-term potentiation (LTP). LTP is a long-lasting enhancement of synaptic transmission in response to brief, high-frequency stimulation of presynaptic neurons. This artificially induced increase in synaptic efficacy typically lasts only a few days or weeks (but see Abraham et al. 2002, for a case of very long-lasting LTP), so it presumably does not represent the way in which memories are permanently coded. Still, LTP is readily induced in hippocampal neurons, and it is the leading candidate for modeling the neural basis of initial memory formation (Martin et al. 2000). Moreover, the nonspecific retroactive interference story described above plays out almost exactly at the level of LTP, thereby grounding that account with a specific mechanism.

Amnestic Drugs, Sleep, and LTP

Alcohol and benzodiazepines both block the induction of LTP in the hippocampus (Del Cerro et al. 1992, Evans & Viola-McCabe 1996, Givens & McMahon 1995, Roberto et al. 2002, Sinclair & Lo 1986). Moreover, it has also been shown that alcohol does not impair the maintenance of hippocampal LTP induced one hour prior to drug administration (Givens & McMahon 1995). The same is presumably true of benzodiazepines, but this has yet to be empirically demonstrated. Blocking the induction of LTP without impairing the maintenance of previously established LTP is a close neural analog of the effects of alcohol on memory that were reviewed in the previous section. That is, while under the influence of the drug (during which time the induction of hippocampal LTP is impaired), memory formation is impaired by those drugs. At the same time, memories formed prior to drug intake, like LTP effects formed prior to drug intake, are not impaired. In fact, the prior memories are actually enhanced for reasons that will become clear when the effects of LTP induction on previously established LTP are considered in more detail below.

Note that non–rapid eye movement (non-REM) sleep also seems to block the induction of hippocampal LTP (Jones Leonard et al. 1987) without disrupting the maintenance of previously induced LTP (Bramham & Srebo 1989). These experiments, which were performed on sleeping rats, showed that while LTP can be induced during REM sleep (possibly accounting for the fact that we can sometimes remember our dreams), it cannot be induced during non-REM sleep (possibly accounting for the fact that we cannot remember any mental activity that takes place during that stage of sleep). Whereas REM sleep is associated with salient visual imagery (i.e., dreams), non-REM sleep is associated with a considerable amount of mental activity as well (e.g., Pivik & Foulkes 1968). However, memories of that mental activity are rarely formed (i.e., we are completely amnesic for what we think about during non-REM sleep). As a result, during non-REM sleep, prior memories are protected from interference that might otherwise occur.

In light of these considerations, one might imagine that sleep characterized mainly by non-REM sleep (during which LTP cannot be induced and memories

cannot be formed) would result in greater retrograde facilitation than sleep characterized mainly by REM sleep (during which LTP can be induced and memories can be formed). An alternative view that was once entertained by the field is that REM sleep is critical to memory formation because it serves to directly enhance the consolidation process (e.g., Fishbein 1996). Ekstrand and colleagues (1972, Yaroush et al. 1971) performed an experiment that was designed to distinguish between these possibilities. These researchers capitalized on the observation that most REM sleep occurs in the second half of the night, whereas most non-REM sleep occurs in the first half. Some subjects in this experiment learned a list, went to sleep immediately, and were awakened four hours later for a test of recall. Others slept for four hours, were awakened to learn a list, slept for another four hours, and then took a recall test. The control (i.e., awake) subjects learned a list during the day and were tested for recall four hours later. The subjects all learned the initial list to the same degree, but the results showed that four hours of mostly non-REM sleep facilitated delayed recall relative to the other two conditions, which did not differ from each other (i.e., REM sleep did not facilitate memory).

These results have been replicated in studies by Plihal & Born (1997, 1999). Curiously enough, these researchers also confirmed earlier work by Karni et al. (1994) showing that REM sleep *does* facilitate the retention of nonhippocampus-dependent procedural memories. Why that might be is somewhat mysterious, but with regard to hippocampus-dependent declarative memories it seems clear that a period of non-REM sleep (during which time the induction of hippocampal LTP is inhibited) has a greater facilitative effect on memory than a similar period of REM sleep. This result fits with the observation that many antidepressant medications, which greatly reduce REM sleep, do not seem to cause memory problems (Vertes & Eastman 2000). In fact, one might even predict that such medications would enhance memory compared to placebo controls over a retention interval that involved a night of sleep (but this prediction has not been tested).

Although the inhibition of LTP induction during non-REM sleep may serve to protect prior memory traces from interference when they are in a fragile physiological state, recent work by McNaughton and his colleagues raises an interesting additional possibility, namely, that when the demands placed on it are reduced, the hippocampus is freed up to actively coordinate memory trace formation in the neocortex. This type of consolidation is conceptually distinct from simply enhancing the physiological stability of the trace. Wilson & McNaughton (1994) found that hippocampal place cells that fired together during a waking experience in rats tended to be *reactivated* together during slow-wave (non-REM) sleep. Hoffman & McNaughton (2002) further showed that coordinated firing activity between different areas of the neocortex is also replayed during quiet wakefulness in macaques. Such reactivation may be a process that emerges whenever the demands placed on the hippocampus are minimized. Indeed, the conditions that appear to protect a fragile memory trace (conditions that are elaborated in the next section) may be a lot like the conditions that set the occasion for the reactivation of previous

neural firing patterns (e.g., non-REM sleep, mental quietude). Whether or not this is generally true remains to be determined.

Induction of New LTP Interferes with Previously Induced LTP

Although alcohol, benzodiazepines, and non-REM sleep all block the induction of LTP in the hippocampus, a much more efficient and selective way to inhibit hippocampal LTP is to use an N-methyl-D-aspartate (NMDA) receptor antagonist, such as AP5 or CPP. Morris (1989) showed that AP5 not only prevents the induction of LTP, it also impairs the learning of hippocampus-dependent tasks (i.e., tasks that animals with hippocampal lesions cannot learn). Thus, like alcohol, benzodiazepines, and non-REM sleep, NMDA antagonists inhibit hippocampal LTP and create anterograde amnesia for hippocampus-dependent tasks.

Several of the LTP studies reviewed below rely on NMDA antagonists to investigate the mechanism of retroactive interference. As with psychological studies concerned with the temporal gradient of RI, all of the LTP studies reviewed below involve an initial phase of original learning and a subsequent phase of interfering learning. In some cases, the original learning consists of animals actually learning a behavioral task (e.g., the Morris water maze task), but in other cases the original learning is "virtual" in that it consists of the artificial induction of hippocampal LTP. The virtual learning studies are concerned with the effect of interference on the maintenance of LTP rather than on the maintenance (i.e., retention) of previously formed memories. The same distinction (actual versus virtual) applies to the interfering task. That is, in some cases, interference consists of exposing the animal to an actual behavioral task (e.g., exploring a novel environment), but in other cases it consists of the artificial induction of LTP. Whether interfering learning is actual or virtual, the induction of interfering LTP can be blocked by the use of NMDA antagonists. This should prevent new interfering learning from occurring and should protect original learning (be it actual learning or virtual learning) from impairment that would otherwise occur.

Izquierdo et al. (1999) conducted an animal learning study that was an exact analogue of retroactive interference studies conducted by experimental psychologists. They first trained rats on a task called one-trial step-down inhibitory avoidance (task 1) and subsequently exposed them to a novel environment (task 2) that would serve to interfere with memory for the first task. The avoidance task involves placing the animal on a platform and then delivering a brief shock when it steps down onto a metal grid. Latency to step down from the platform on subsequent test trials is the measure of memory for the training trial (long latency implies good memory). Prior work has shown this to be a hippocampus-dependent task, so it is widely used to investigate declarative memory processes. Exposure to the novel environment (task 2) involved placing the animal in an open field with a pink floor adorned with black-lined squares. After exposure to the task-1 learning trial, the animals were exposed to task 2 either one hour or six hours later, and memory for task 1 was assessed after a 24-hour retention interval. Note that the design of

this study is conceptually identical to studies concerned with the temporal point of unrelated interpolated learning, such as those performed by Skaggs (1925). Moreover, a temporal gradient of RI was observed: 24-hour memory of the avoidance task was impaired only when the seemingly unrelated interfering task was presented one hour after learning. This presumably occurred because the memories had not yet had a chance to consolidate when the interfering learning took place. After six hours, the memories were more fully consolidated, so exposure to the novel environment had less of an interfering effect.

Thus, once again, we find evidence for a temporal gradient of retroactive interference. Such a result would be surprising if the traditional reading of the related psychology literature were correct. According to that reading, the temporal point of interpolated (i.e., interfering) learning does not affect the degree of retroactive interference (e.g., Wickelgren 1977). According to my revised reading of that literature, however, it clearly does. Results like those reported by Izquierdo et al. (1999) serve to reinforce that conclusion.

Of particular interest is whether the induction of LTP associated with exposure to the novel environment is responsible for the observed temporally graded retroactive interference. To investigate this, Izquierdo et al. (1999) administered an NMDA antagonist directly into the hippocampus of some of the rats prior to their exposure to the novel environment (which occurred one hour after avoidance learning). The NMDA antagonist prevents the induction of LTP that might be associated with exposure to a novel environment and reduces learning about that environment. With the induction of LTP thus prevented, no retroactive interference effects were observed. That is, memory for the avoidance task was unimpaired by subsequent exposure to the novel environment.

A conceptually similar result was reported by Brun et al. (2001). They showed that memory for a submerged platform in the Morris water maze task was impaired by the subsequent induction of hippocampal LTP by means of high-frequency stimulation delivered through implanted electrodes. In other words, memory for original learning was impaired by virtual interference learning consisting of the induction of LTP. However, if an NMDA receptor antagonist was infused into the hippocampus prior to delivering the high-frequency stimulus (thereby preventing the induction of potentially interfering LTP), no memory impairment was observed. Thus, whether interfering learning was actual or virtual, preventing the induction of LTP during the interference phase of the experiment spared the original learning.

These results immediately suggest a neurophysiological mechanism for temporally graded retroactive interference. Specifically, the induction of LTP during interfering learning impairs recently established LTP associated with original learning, even if the tasks are unrelated. This hypothesis was tested directly by Xu et al. (1998). This experiment was much like the one by Izquierdo et al. (1999) described above except that instead of using an actual task 1, these researchers used a virtual task 1 by artificially inducing hippocampal LTP in freely behaving rats. Once again, they did so by delivering trains of high-frequency stimulation through electrodes implanted into the rats' brains. Exposure to a novel environment (task 2)

one hour later completely reversed the previously induced LTP. However, if exposure to the novel environment was delayed for 24 hours after induction of LTP, no effect of that exposure on LTP was observed. Thus, a temporal gradient was observed yet again, and it suggests that recently established LTP is more vulnerable to the disruptive effects of subsequent interference than more remotely established LTP is (presumably because the latter has had time to consolidate). A conceptually similar study performed by Abraham et al. (2002) involved a much more prolonged interference phase and showed that, under such conditions, the LTP temporal gradient can be observed over a period of weeks (instead of hours, as in Xu et al. 1998). In this study, LTP was induced in the hippocampus of rats, and the animals were then housed in their typical "stimulus-poor" home cage environments for two weeks. In this low-interference environment, LTP decayed very gradually. Over the next week, some of these animals were exposed to a complex environment (involving a larger cage, multiple objects, and other animals) for 14 hours per day. Exposure to this environment for several days resulted in complete reversal of the previously induced LTP, whereas LTP in the control animals continued its very gradual decay. By contrast, when exposure to a complex environment was postponed until 90 days after the induction of LTP, no measurable interfering effect was observed.

The next logical experiment was recently reported by Villarreal et al. (2002). Hippocampal LTP was again induced via implanted electrodes (i.e., a virtual learning procedure was used), and the magnitude of LTP was assessed for the next nine days. Some rats received an NMDA receptor antagonist one hour after LTP induction (a treatment that should prevent the further induction of LTP), whereas control rats received a water vehicle. No explicit retroactively interfering task was arranged, so any interference that occurred was presumably due to the normal events in the life of a laboratory rat or to other routine aspects of the experimental procedure (e.g., daily injections of water). The results revealed that LTP decayed back to baseline for the control rats over the next seven days but remained elevated for the experimental subjects. When the NMDA antagonist was no longer administered (after day seven), LTP in the experimental rats also decayed quickly. These results again suggest that previously established LTP falls prey to the interfering effects of subsequently induced LTP.

In an exact analog of the alcohol-induced retrograde enhancement studies reviewed earlier, Villarreal et al. (2002) also trained rats on an eight-arm radial maze and then administered an NMDA receptor antagonist or water vehicle to different subgroups of rats over the next five days. The rats receiving the NMDA antagonist exhibited a retrograde enhancement effect when their memories were tested after six days of treatment. Note how similar this pattern is to the retrograde enhancement effects observed with alcohol and benzodiazepines. Like NMDA receptor antagonists, alcohol and benzodiazepines block the induction of LTP (and induce anterograde amnesia) without compromising previously established LTP. Indeed, these substances protect previously established LTP from the interfering effects of subsequently induced LTP, which may be why retrograde enhancement is observed.

Whereas retrograde enhancement is seen in humans when memory is blocked by alcohol for a matter of hours, in rats the phenomenon may require a much more prolonged phase of memory blockade (perhaps because the rate of interfering memory formation is so low when no interfering task is explicitly arranged).

To summarize, whether original learning is actual or virtual, subsequent interfering learning (whether actual or virtual) creates retroactive interference. The interfering effect is less pronounced the longer the delay between original and interfering learning is (pointing to a role for consolidation), and the effect of interfering learning can be abolished by preventing the induction of LTP using an NMDA antagonist (pointing to the induction of LTP as the source of RI).

CONCLUSION

My attempt to articulate a coherent theory of forgetting was prompted by dissatisfaction with what I derisively referred to as the "atheoretical laundry-list" account of forgetting that pervades the field today. The alternative I propose is that the hippocampus plays an important role in consolidating newly formed memory traces (this is actually the standard view in neuroscience) and that ordinary mental exertion and memory formation interfere with that process, perhaps by drawing on a limited pool of hippocampal resources. The interfering mental activity need not be related to the originally learned material; the formation of memories per se (which, theoretically, involves the induction of hippocampal LTP) disrupts the consolidation of recently formed memories (which, theoretically, involves disrupting the persistence of previously established LTP). Findings from the traditional interference literature, the psychopharmacology literature, and the neuroscience literature converge on this way of thinking.

It is important to emphasize that this theory does not imply that new memories fully overwrite immediately preceding memories. As Morris (1998) observed when commenting on the impressive memory abilities of food-caching birds, "A memory system that could recall only the last item cached, wiping out memory of earlier items, would be unhelpful" (p. 835). But a memory system that creates new memories even at the expense of partially degrading other memories would still be helpful, and that may be the very kind of memory system we have. What the exact variables are that govern the degree to which prior memories are degraded is not known, but one obvious possibility is that the greater and more variable the new learning is, the greater the interfering effect will be. Entering a novel situation that involves unfamiliar activities, strange sights, and unusual sounds may elicit the most hippocampal activity (e.g., Martin 1999, Tulving et al. 1994) and, therefore, the greatest rate of new memory formation. As such, that may be the kind of situation that maximally interferes with the consolidation of previous memories. Indeed, as indicated above, exposure to a novel environment is a potent interfering force in studies involving rats (and that interference decreases the longer ago the original memory was formed).

Throughout this chapter, I have relied on the traditional notion of consolidation according to which new memories are clear but fragile and old ones are faded but robust. Some new evidence suggests that this traditional model may be in need of revision. Nader et al. (2000), for example, reported evidence in support of an old idea (Misanin et al. 1968) that it is recently *activated* memories that are vulnerable to the effects of interference (even if those activated memories had once been consolidated). If this intriguing finding holds up, then the theory advanced here would necessarily apply to recently activated memories instead of just recently formed memories. Dudai (2004) provides a detailed review of the evidence pertaining to the recently revived notion of reconsolidation.

Finally, by articulating this new theory of forgetting, I do not mean to imply that the other elements of the laundry list of factors that might contribute to forgetting are irrelevant. They might very well be relevant to ordinary forgetting even though this has not yet been convincingly established. One such variable that was long ago rejected by experimental psychologists is natural decay. In arguing against decay theory, McGeoch (1932) observed, "No one has ever published experimental evidence that synaptic junctions decrease in intimacy, or anything else, when one forgets" (p. 368). But times have changed in that regard. Bailey & Chen (1989) showed that synaptic varicosities of sensory neurons in aplysia (a simple model system for human neurophysiology) increase in number from approximately 1200 to almost 3000 following a sensitization learning procedure. The number drops off to about 1500 over the next three weeks, which parallels a decrease in the behavioral magnitude of sensitization over the same time period. While this loss might reflect the retroactively interfering forces of (undetected) subsequent learning of some kind, it could instead reflect the natural sequelae of a biologically active neuronal system. And while the sea slug is a comparatively simple system, it is not hard to imagine that similar events unfold in the human brain.

Thus, my point is not that multiple factors are not involved in the process of forgetting. But just because multiple factors are relevant to the story of forgetting, that doesn't mean the field should restrict itself merely to enumerating the possible contributing factors, as it has for some time now. The voluminous body of research on forgetting that has accumulated over the last century tells a much more interesting story than that.

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