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Chunking and Consolidation: A Theoretical Synthesis of Semantic Networks, Configuring in Conditioning, S–R Versus Cognitive Learning, Normal Forgetting, the Amnesic Syndrome, and the Hippocampal Arousal System

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Horizontal versus vertical associative memory concepts are defined. Vertical associative memory involves chunking: the specification of new (previously free) nodes to represent combinations of old (bound) nodes. Chunking is the basis of semantic memory, configuring in conditioning, and cognitive (as opposed to stimulus–response) learning. The cortex has the capacity for chunking, but the hippocampal (limbic) arousal system plays a critical role in this chunking process by differentially priming (partially activating) free, as opposed to bound, neurons. Binding a neuron produces negatively accelerated repression of its connections to the hippocampal arousal system, consolidating the memory by protecting the newly bound neuron from diffuse hippocampal input and thus retarding forgetting. Disruption of the hippocampal arousal system produces the amnesic syndrome of an inability to do new chunking (cognitive learning)—anterograde amnesia—and an inability to retrieve recently specified chunks—retrograde amnesia.

With a time period of development spanning over two thousand years of human history, the doctrine of the association of ideas is to this day the dominant theory of the mind. I imagine there were always protesters who claimed that the mind was too complex to be encompassed by such associationism. However, until recently, there was no comparably elegant and general alternative theory. Now there is such a theory, though as is so often the case, the new theory is more of an extension than a repudiation of classical associationism.

The basic idea derives from the concept of chunking. Chunking was originally formulated by G. Miller (1956), but subsequently chunking has been implicated in a wide variety of phenomena in cognitive psychology. Chunking probably means different things to different people. In this article it stands for a learning process by which a set of nodes representing constituents (components, attributes, features) of a whole comes to be associated to a new node that, thereby, represents the whole chunk (Estes, 1972, 1975; Johnson, 1970, 1972; LaBerge, 1973, 1976; Wickelgren, 1969). Chunking has been proposed as the basis of both concept learning (associating attributes to a chunk node representing the concept; Wickelgren, 1969) and propositional learning (associating concepts to a chunk node representing the proposition; Anderson & Bower, 1973; Wickelgren, 1976b, 1976c) in semantic memory. Semantic memory models typically label different types of associations (links) between nodes to differentiate the types of relations between concepts (Anderson & Bower, 1973; Quillian, 1968; Rumelhart, Lindsay, & Norman, 1972), but this is known to be logically unnecessary (Anderson & Bower, 1973, pp. 88–90). Fur-

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thermore, there is some evidence that associations between concepts in a proposition are not differentiated with respect to function within the proposition (Anderson, 1975, 1976 [pp. 278–283]). Unlabelled associations are assumed in this article.

Horizontal and Vertical Associations in Human Cognitive Learning

The basic ideas regarding this distinction have been described previously (Anderson & Bower, 1973; Estes, 1972; Johnson, 1972; Wickelgren, 1969, 1976b, 1976c, 1977a, 1977b [pp. 18–22, 243–247, 251]), but only I am to be considered responsible for the following specific formulation. The fundamental difference between horizontal and vertical associations concerns whether new nodes are added to memory as a result of the learning process. In horizontal associative memory, the set of idea nodes is fixed, and contiguous activation of a set of nodes strengthens the associations among all pairs of nodes in the activated set so that the subsequent activation of any one node will often be sufficient to reactivate the entire set (Hebb, 1949). In vertical associative memory, nodes can be partitioned into two large subsets: (a) bound nodes, which have some strong input and output associations specifying what each node represents (what turns them on and what they turn on), and (b) free nodes, which have a multiplicity of weak input–output associations to a large number of free and bound nodes. In learning (chunking), a set of simultaneously activated bound nodes must have its associations strengthened (in both upward and downward directions) to some previously unspecified free node that, by the chunking learning process, becomes bound to represent that entire set of constituent nodes. In a horizontal associative memory, complex ideas are represented by a strongly associated set of elementary ideas, whereas in a vertical associative memory, the complex idea is represented by a single node that has strong associations to and from its constituent elementary ideas. Vertical associative memories are at least locally hierarchical in their associative network structure, but horizontal associative memories are not. The encoding of propositions in terms of constituent concepts, in both types of associative memory, is illustrated in Figure 1.

The fundamental limitation of a horizontal associative memory is in its extreme susceptibility to associative interference. For example, in the horizontal associative network in Figure 1, the incorrect propositions, *fish live in air* and *birds live in water,* are as strongly encoded as the correct propositions, *fish live in water* and *birds live in air.* In the vertical associative network, such interference is avoided by binding each concept constituent to a new chunk node representing the entire proposition. In this way, vertical associative learning reduces the associative interference problems that arise when the same concepts are involved in many propositions. Moreover, chunking permits complex ideas to be embedded as unitary constituents of even more complex ideas, as the embedding of concepts in propositions and simple propositions in more complex propositions.

Human Amnesia

Amnesia is the loss of memory for reasons other than those that normally produce forgetting. There are several different types of amnesia, but the most frequent type (or at least the most frequently described type) is the so-called *amnesic syndrome.* The amnesic syndrome has two components: retrograde amnesia (RA) and anterograde amnesia (AA). Retrograde amnesia refers to the selective loss of memory for events that occurred before the onset of the amnesic agent. Anterograde amnesia refers to a loss of memory for events that occurred after the amnesic agent. Many different amnesic agents can produce the amnesic syndrome, including concussion, electroconvulsive shock, focal electrical stimulation of certain regions of the brain (in the region of the hippocampus and temporal lobe), epileptic seizures (in the temporal-hippocampal area), lesions in the hippocampal region, some drugs (especially those that initiate seizures in the hippocampus), impaired blood or oxygen supply to the hippocampus, certain infections (encephalitis, tuberculous meningitis), and Korsakoff psychosis, which results from prolonged heavy

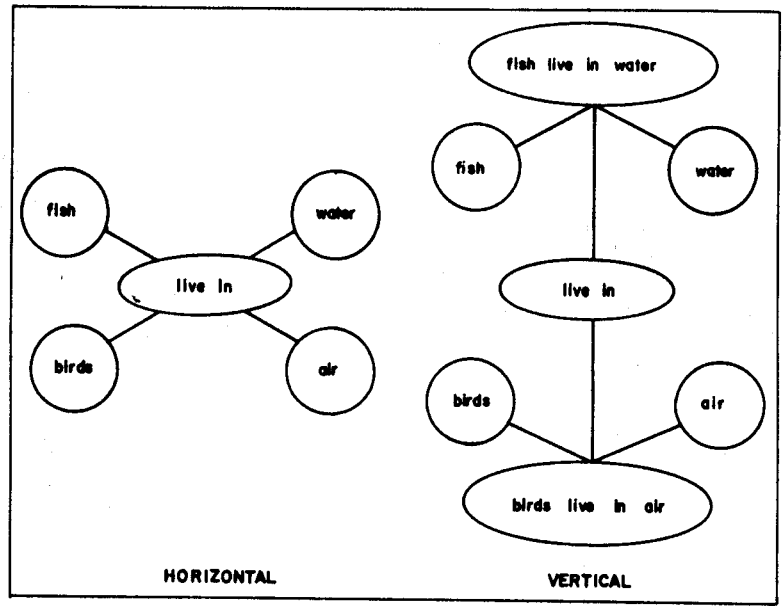

Figure 1. Encoding of the propositions *fish live in water* and *birds live in air* in terms of concept constituents in both horizontal and vertical associative memories.

alcohol consumption. Very likely, there are some differences in the memory disorders produced by these different agents, but there appears to be a substantial common component, which is called the amnesic syndrome (Barbizet, 1970; Victor, 1969).

In pure cases of the amnesic syndrome, patients have no detectable deficits in perception, motor responses, and thinking (e.g., Milner, 1966). Intelligence test scores and short-term memory test scores may be completely normal, as is the ability to retrieve long-term memories established long before the onset of the amnesic agent (Milner, 1966). What is impaired is the ability to learn, store, or retrieve new long-term memories (AA) and the storage or retrieval of memories learned recently before the amnesic agent (RA). Most RA is of very brief duration, a matter of seconds or minutes prior to the onset of transient amnesic agents, but sometimes the RA is such that the patient loses memory for days, weeks, months, or even years of his or her life prior to the amnesic agent (Russell, 1959, 1971; Russell & Nathan, 1946; Seltzer & Benson, 1974; Squire, Slater, & Chace, 1975). Long RAs generally shrink over a recovery period, with the oldest memories being recovered first (Gottlieb & Wilson, 1965; Russell, 1959, 1971; Russell & Nathan, 1946). It is important to note that the memories that are impaired in RA are not the weakest or the strongest memories but the most recent memories, independent of strength (Russell, 1959, 1971; Russell & Nathan, 1946; Seltzer & Benson, 1974; Squire et al., 1975). Recovery from RA follows the same pattern, with the oldest memories being recovered first.

Co-occurrence of RA and AA

Retrograde and anterograde amnesia are closely linked following the rule of *no RA without AA* (Barbizet, 1970; Evans, 1966; Fisher & Adams, 1964; Ignelzi & Squire, 1976; Russell, 1959, 1971; Russell & Nathan, 1946; Shuttleworth & Morris, 1966; Victor, 1969). The rule of *no RA without AA* means that one can order cases of the amnesic syndrome as follows: (a) least severe cases with short-duration AA and little or no RA, (b) more severe cases with moderate-duration AA and initially moderately long RA, with the RA shrinking as the AA clears up, and (c) long-duration AA and long-duration RA, with

eventual recovery possibly from both RA and AA or only from RA or no recovery at all.

With respect to the close relation between RA and AA, it is important to note that it is quite possible to have serious and persistent AA (deficits in new long-term learning) without much of a persisting RA for the period prior to the onset of the amnesic agent. Such cases of AA without (much) persistent RA are cases in which the hippocampus or related structures have been permanently damaged and RA does accompany the AA at the time of the onset of the destruction of the hippocampal (limbic) system. When the system is temporarily disrupted, there appears to be a tight correlation in recovery from both RA and AA (Ignelzi & Squire, 1976), but when the system is destroyed, patients appear eventually to recover from most of the RA but not the AA (e.g., Marslen-Wilson & Teuber, 1975; Milner, 1966; Milner, Corkin, & Teuber, 1968; Squire & Slater, 1978; Teuber & Milner, 1968). *No RA without AA* does not mean that RA and AA always occur together as a package; it means that RA always implies AA, but AA does not always imply RA. The AA (memorizing defect) is more common than RA. It is easier to disrupt the ability to learn new things than it is to disrupt already established memories. The longer the memory has been established, the harder it is to disrupt it (law of recency). So the rule is, New learning is the easiest to disrupt, recent learning is next, and old learning is hardest.

Learning, Storage, or Retrieval Deficit?

There has been some interest in whether RA and AA are deficits in learning, storage (impaired consolidation or accelerated forgetting), or retrieval. By definition, RA cannot be considered to be a learning deficit, but the other two possibilities for RA are logically open. A general retrieval deficit is incompatible with the temporally selective character of the memory loss (law of recency). A recency-dependent retrieval deficit is attractive for that portion of the RA for which memory is eventually recovered. However, although patients usually recover from RA (except for the last few seconds prior to the amnesic agent) and this recovery from RA is often (but not always) accompanied by recovery of the ability to learn new things (recovery from AA), memory for the events that occurred during the period of the amnesic syndrome does not improve after recovery from RA and AA. Such events were attended to and processed in short-term memory at the time of their occurrence, but apparently there was a deficit in acquiring long-term memory traces for these events.

Thus, although retrieval-deficit explanations of the amnesic syndrome are currently very popular, they have severe logical problems in accounting for the most basic facts regarding both RA and AA. If what is fouled up in amnesia is the retrieval process, how is it that we can retrieve old memories with little or no impairment? If what is fouled up is our ability to retrieve recently established memories, why do amnesic patients recover the memories for events occurring prior to the onset of the amnesic agent but do not recover memories for events occurring after the onset of the amnesic agent during the period of AA? I think two conclusions are clear: (a) Whereas RA is, in some sense, by definition, a "retrieval deficit" (since patients usually recover from RA), AA is clearly not a retrieval deficit. (b) Whatever the common factor is underlying RA and AA, it is not to be found in the retrieval process. When we recover from RA, we recover our ability to retrieve memories established before the amnesic agent (oldest memories recovered first); when we recover from AA, we recover the ability to learn new things, but we do not recover memories for events occurring after the amnesic agent during the period of the AA.

Perhaps AA is a storage deficit due to slower consolidation or faster forgetting; however, the evidence appears to be against this hypothesis as well. Storage deficits should be revealed by faster forgetting (whether due to less adequate consolidation or more potent forces producing trace loss). Since amnesic patients are frequently capable of some learning (albeit much slower than normal), we can study forgetting during the period of AA. Some studies support the hypothesis of at least slightly faster forgetting

in amnesics in both short-term retention for seconds to tens of seconds (Cermak, Butters, & Goodglass, 1971) and long-term retention for minutes to months (Warrington & Weiskrantz, 1968; Weikrantz & Warrington, 1970). However, in these studies the differences in long-term forgetting rate were minor in comparison with the large difference in initial learning, which suggests that the primary deficit in AA is in learning (acquisition) and that any retention (storage) deficit is clearly secondary.

The Cermak et al. (1971) study is not definitive because the amnesics could not be assumed to have the same degree of learning as controls merely because immediate retention scores (0-sec delay) were nearly perfect for both groups. Zero-delay conditions may be confounded by unknown amounts of iconic or echoic memory, and also near-perfect scores are insensitive to differences in overlearning, which can be very large. When this zero-delay point is eliminated from the Cermak et al. data, the results no longer clearly support the hypothesis of faster short-term forgetting in amnesics. Finally, there are several studies that have found equivalent forgetting rates in amnesics and controls in both short-term retention (Baddeley & Warrington, 1970; Murray & Hitchcock, 1973; Wickelgren, 1968) and long-term retention (Huppert & Piercy, 1976; Wickelgren, 1974). Faster forgetting does not appear to be the primary deficit in AA; the primary deficit in AA is impaired long-term learning (Huppert & Piercy, 1977; Wickelgren, 1974, 1977b [pp. 325–329]).

Since RA must necessarily be considered either a frequently reversible storage deficit or a retrieval deficit, not a learning deficit, and since AA clearly appears to be a learning deficit, the common factor underlying these two components of the amnesic syndrome is not to be found in any learning-storage-retrieval stage analysis. We must look to other analyses of the memory process.

AA Is a Chunking Deficit

Another line of research directed at the nature of the AA deficit has proven to be very illuminating. This research line was begun by Corkin (1968), who discovered that the famous amnesic patient H.M. of Scoville and Milner (1957) could establish new long-term memory for motor skills, such as rotary pursuit, bimanual tracking, and mirror drawing, with apparently normal retention over periods of several days. The exact pattern of memory and memory loss was fascinating. All higher level cognitive memory was absent. For example, H.M. would swear he had never performed the task before but would show nearly normal improvement in, say, time-on-target in rotary pursuit as a result of his training on prior days. Moreover, the nature of H.M.'s improvement in rotary pursuit was consistent with the same cognitive versus noncognitive distinction. Normal subjects acquire an image of the wavy line in rotary pursuit that allows them to anticipate its movement and thus get off target less frequently. H.M. showed no such improvement. He got off target just as often, but he got back on target faster and faster with practice. Thus, H.M. was impaired on the more cognitive component of the task but was capable of partially compensating for this by improving his previously learned perceptual-motor skill of moving to a target.

Subsequent research clearly establishes this pattern: Amnesics are often able to further strengthen already existing associations but are unable to acquire totally new cognitive memories. Warrington and Weiskrantz (1968, 1970) found better recognition by amnesics of more fragmented versions of familiar words and pictures over repeated learning trials, with their improvement being only slightly inferior to that of normals. Brooks and Baddeley (1976) found a substantial level of learning and apparently normal long-term retention in the repeated assembly of the same jigsaw puzzle. For a time, it seemed that one could characterize the amnesic deficit in terms of levels of processing, with amnesics being impaired in high-level semantic memory but not in lower level perceptual and motor memory. There may well be some merit to the hypothesis that human amnesics can acquire totally new lower level noncognitive traces, such as learned food aversions and other simple classically and instrumentally conditioned responses (though this has,

surprisingly enough, not yet been clearly established).

However, what is clear is that human amnesics are deficient in new cognitive learning and not deficient in further strengthening of previously established traces, regardless of level. Even clearly semantic-level associations, such as bowl–plate or army–soldier, can be further strengthened by amnesics, provided they are already familiar associations, but unfamiliar paired associates cannot be learned by amnesics (Winocur & Weiskrantz, 1976). What these findings suggest is that amnesics are deficient in the ability to do new chunking. Amnesics can strengthen already existing associations (probably including highly prepared noncognitive horizontal associations at subcortical levels), but they cannot establish new vertical associations to connect sets of items by means of intervening chunk nodes. Along these lines, Baddeley and Warrington (1973) demonstrated that although amnesics improve their free-recall scores when words are clustered on the basis of phonemic similarity or previously learned taxonomic categories, they gain no advantage from instructions to cluster words by means of mnemonic imagery, in sharp contrast to normal subjects. The chunking-deficiency hypothesis also explains the Warrington and Weiskrantz (1974) and Winocur and Weiskrantz (1976) finding that human amnesics are much more susceptible to associative interference than are normals, since, as was explained in a previous section, one of the principal advantages of learning by chunking is the reduction of associative interference.

If AA is a chunking (learning) deficit, what is RA, and why does AA always accompany RA? Their co-occurrence cries out for an explanation in terms of a common mechanism, but in the entire history of past research on the amnesic syndrome, only one even remotely precise and plausible hypothesis has been put forward as regards a common mechanism, by Weiskrantz (1966). Weiskrantz assumed that the noise level of competing traces in retrieval was raised relative to the correct trace in the amnesic syndrome. Long-term memory traces were assumed to increase in strength over time (as a result of consolidation). Hence the oldest traces could still be discriminated above the increased background noise level, but more recently established traces could not. Besides being a bit vague about what the "noise level" of competing traces might be and why it should increase when the hippocampus and related structures are disrupted, this theory has a couple of other flaws. First, it assumes that trace strength is increasing with age, and this is contradicted by all the studies showing long-term forgetting. This flaw can be easily corrected by saying that it is a second trace property (resistance or fragility), not strength, that is increasing monotonically with age. The theory of RA and AA advanced in this article makes just this assumption. Second, Weiskrantz's (1966) theory is a pure retrieval-deficit explanation, and, as such, it fails to explain why most RA is temporary but all AA is permanent.

The present article offers a precise and accurate explanation of the amnesic syndrome in both humans and animals that is much more than just an ad hoc explanation of amnesia. It is an explanation that derives from a general theory of the integrated neural mechanisms of both chunking and consolidation. Before we get to this, however, there is relevant evidence concerning conditioning in lower animals and its disturbance by hippocampal disruption which must be discussed.

Horizontal and Vertical Associations in Conditioning: Configuring and S–R Versus Cognitive Learning

The ideas in this section depend heavily on findings and ideas expressed earlier by Bitterman (1969, 1975), Razran (1971), Hirsh (1974), and Wickelgren (1977b, pp. 81–83, 104–108), so I briefly recapitulate the old news and get on to the new news as quickly as possible. A great phylogenetic advance in the evolution of learning apparently occurs somewhere between fish and amphibians, on the one hand, and birds and mammals, on the other. Presumably because of the greater development of the highest levels of the brain in the tectum and cerebral cortex, respectively, birds and mammals are able to demonstrate the phenomenon of

configuring in classical and instrumental conditioning, but fish and amphibians cannot configure (Razran, 1971, pp. 207–221). Configuring is the conditioning of a response to a compound stimulus (A and B) in the absence of this conditioned response to the components. In birds and mammals, configuring can be established either by differential contrasting (e.g., A and B are followed by the unconditioned stimulus [US], but A alone and B alone are not followed by the US) or simply by prolonging conditioning for hundreds of trials. After tens of trials, the conditioned response will be elicited by A alone and B alone as well as by the AB compound; but after hundreds of trials, the response to the components disappears, and only the response to the compound remains. The most plausible explanation is that birds and mammals possess the capacity to acquire new chunk nodes representing the compound stimulus, so the chunk node can have associations that its constituents do not.

Organisms that can chunk (birds and mammals) can form chunk nodes to represent propositional expectations—in situation S, response R will produce consequence K. This appears to be the most straightforward way to formulate what Tolman (1948) meant by cognitive expectancy learning. To explain actual choice behavior, a decision mechanism is also required to map a family of SR_i–K_i expectancies onto a choice of response R_i. A simple psychological-level decision rule is to assume an incentive value associated with all consequences K_i and to choose the response R_i that produces the K_i with the highest value. Different drive states are associated with different assignments of incentive value to events K_i. It is not hard to generate a possible physiological mechanism to implement such a decision rule in the nervous system, and it is trivial to implement such decision rules on a digital computer. There is no reason today to claim that cognitive theories of conditioning leave the animal "lost in thought in the middle of maze unable to move." Once this impediment to cognitive theories of learning is removed, it is clear that for the rat (and probably all birds and mammals), Tolman was right concerning what is learned, namely SR–K expectancies, not S–R associations (Wickelgren, 1977b, pp. 104–107).

However, the S–R reinforcement theory of Thorndike (1898) and Hull (1943), in some ways, acquires even richer significance by the precise formulation of the cognitive theory, because it becomes clear just how powerful and interesting a theory it is. The S–R theory does not require the formation of new chunk nodes and vertical associations. It is strictly a horizontal associative theory, and it is probably limited to a restricted range of highly prepared potential associations because of the susceptibility of horizontal associative memories to associative interference.

The role assigned to reinforcement by Thorndike's (1898) law of effect is equally fascinating. In one sense, reinforcement is *more* critical in the S–R theory because only motivationally significant consequences, K, substantially strengthen associations between Ss and Rs that have been recently and contiguously activated. This restriction focuses the limited S–R learning capacity on the motivationally most important S–R sequences and, therefore, reduces susceptibility to interference. Accordingly, in a horizontal associative S–R theory one expects no learning in the absence of motivationally significant reinforcers (i.e., no latent learning), and the demonstration of latent learning in rats constitutes one of the principal supports for the vertical-associative cognitive theory of learning in such animals (G. Kimble, 1961, pp. 226–234). By contrast, in his exhaustive review of learning and instinctive behavior in animals, Thorpe (1963, p. 106) concluded, "In the 'lower' vertebrates, and 'higher' invertebrates, the perceptual world is preponderatingly instinctively determined, and latent learning, if it occurs, probably plays only a relatively small part." The few highly specific instances of latent learning below birds and mammals which Thorpe does cite are likely explicable on a highly prepared instinctive basis rather than on a general cognitive expectancy-learning basis.

In another sense, reinforcement is *less* important in the S–R theory than in the cognitive theory because it is not coded as a part of what is learned in the S–R theory. From this property, one derives the conflicting pre-

dictions concerning *reinforcement contrast* which Bitterman (1969, 1975) has shown to sharply distinguish the instrumental conditioning of birds and mammals from that of fish and amphibians. All vertebrates apparently show magnitude-of-reinforcement effects on the learning–performance curve, such that greater reward produces better performance of the reinforced response. In higher vertebrates, this is typically a performance difference—the animals have learned equally well that performance of the response leads to a reward, but the expected magnitude of the reward also has an influence on their decision. When the magnitude of reward is switched, higher vertebrates immediately demonstrate that they recognize the change by switching their performance level on the next trial (G. Kimble, 1961, pp. 122–123). Because higher vertebrates have explicitly encoded the reinforcer as a part of what was learned, they can be "surprised" by a deviation from their expectation and change their performance abruptly.

Lower vertebrates, however, show an entirely different pattern. The small-reward group requires many learning trials to reach the appropriate higher level of performance after being switched to the large reward, and the group switched from large to small reward does not reduce its performance of the operant at all, even after many trials with the smaller reward (Bitterman, 1969, 1975)! Although lower vertebrates will extinguish responses when shifted to zero reward, a small reward is sufficient to maintain a conditioned response at a higher level than it could have attained without prior training with the large reward. Lower vertebrates appear to learn S–R associations, just as Thorndike (1898) asserted, with larger reinforcers "stamping-in" a stronger association between S and R. This gives such animals a strong "urge" to perform response R in situation S, but with no expectation concerning the consequences of that response. Having no expectation of any particular consequence K, they cannot be "surprised" by a change in the reward and cannot, therefore, show reinforcement contrast effects.

Animal Amnesia

The discovery of the dramatic memory deficits produced by hippocampal lesions in humans unleashed a torrent of research on the effects of hippocampal lesions in subhuman mammals (see reviews by Hirsh, 1974; Isaacson, 1974). This research is still regarded by many to be disappointing and puzzling because, it is alleged, the animal hippocampal syndrome is very different from the human hippocampal syndrome. Human bilateral hippocampals were said to have a complete inability to acquire new long-term memories, whereas similar lesions in rats and monkeys frequently left them with considerable capacity for long-term learning, including no impairment at all in many simple conditioning tasks (Hirsh, 1974; Isaacson, 1974). There probably are some differences in the effects of hippocampal lesions in different species, but the view that the human and animal hippocampal amnesic syndromes are fundamentally different is quite incorrect. The results of both human and animal studies are in complete agreement when viewed from the perspective of the present theory. According to this theory, hippocampal lesions (or disruptions of the hippocampus by various agents) produce an inability to chunk (form new vertical associations) in the cortex, in all mammals (so far as is known), but hippocampal lesions do not prevent further strengthening of existing associations in the cortex, or subcortical horizontal associative learning. Stated another way, hippocampal lesions block new cognitive expectancy learning, but they do not block the strengthening of existing expectancies or new subcortical S–R associative learning. Evidence supporting this conclusion for human amnesia was described in a prior section and for animal amnesia is described in the present section.

Recency-Dependent RA and Co-occurring AA

As in human beings, many different amnesic agents seem to evoke the same basic amnesic syndrome of AA and recency-dependent RA: electroconvulsive shock (ECS), various drugs, and specific electrical, chemi-

cal, or surgical disruption of the hippocampus and related structures (Gold & King, 1974; Hirsh, 1974; Isaacson, 1974; Kesner & Wilburn, 1974; R. Miller & Springer, 1973; Squire, 1975; Weiskrantz, 1966). Besides this property of co-occurring RA and AA, RA in subhuman mammals is also recency-dependent, just as in human beings. Recently acquired memories are more susceptible to RA than are older memories, and, just as in humans, the range of time over which retrograde amnesic effects can be obtained is highly variable, from seconds to at least a couple of weeks, depending upon the nature and the strength of the amnesic agent (Deutsch, 1971; Squire, 1975; Weiskrantz, 1966; Wiener, 1970).

Recovery From RA

Furthermore, as in humans, there is frequently (but not always) recovery from RA and AA but generally not recovery of memory for the events occurring during the period of the AA (Flexner & Flexner, 1968; Gold & King, 1974; Lewis, 1969; Lewis, Miller, & Misanin, 1968; R. Miller & Kraus, 1977; R. Miller & Springer, 1973; Zinkin & Miller, 1967). As in humans, hippocampal lesions produce permanent AA (though there is considerable debate concerning the similarity of the human and animal AA—discussed in a subsequent section) and a small RA (Bohdanecka, Bohdanecky, & Jarvik, 1967; Glick & Greenstein, 1973; Hirsh, 1974; Isaacson, 1974; Weiskrantz, 1966). Although truly spontaneous recovery from RA with the passage of time has only occasionally been observed in animals, most of the physiological recovery agents (e.g., stimulants such as amphetamines and corticosteroids, or saline injections into the hippocampus) and psychological recovery agents (stimuli involved in the unavailable learning experience) are more effective in producing recovery from RA, the more time that has elapsed since the offset of the amnesic agent (DeVietti & Bucy, 1975; DeVietti & Hopfer, 1974).

The effectiveness of cue reminders in animals for recovery from RA suggests a reexamination of the clinical reports of spontaneous recovery from RA in humans. Coexisting with these clinical reports of spontaneous recovery of the oldest memories, are clinical reports that memories recover by spreading out from so-called "islands of memory" in the period of time subject to the RA (Zangwill, 1964). Islands of preserved memory constitute exceptions to the recency rule that the lost memories are the most recent memories. An attractive, but unproved, resolution of this matter may be that although older memories are indeed less susceptible to RA and easier to recover, cuing from external stimuli or internal recall of preserved memories is the force producing recovery in both humans and lower mammals because of the massive amount of linguistic and other cuing to which amnesic humans are subjected from their own memories and from the memories of concerned relatives and friends. That is to say, much or all so-called "spontaneous" recovery may really be uncontrolled cue-induced recovery. Considering how many cue reminders human amnesics are given, there is more merit in viewing recovery from RA in humans, as in lower mammals, to be cue induced rather than spontaneous.

Delayed Onset of RA

A rather interesting property of RA in subhuman mammals that appears not to have been investigated in humans is delayed onset of RA. Several animal studies with various amnesic agents (hippocampal electrical stimulation, ECS, and drugs such as cycloheximide and Metrazol) have showed that RA may not be at a maximum immediately after the administration of the amnesic agent but instead may develop over a period of tens of minutes or hours after the administration (Berman & Kesner, 1976; Hughes, Barrett, & Ray, 1970; R. Miller & Springer, 1971; Palfai & Kurtz, 1973; Squire & Barondes, 1972). The late Hans-Lukas Teuber told me he thought delayed onset of RA might also occur in humans, but to my knowledge there has been no scientific study of this.

RA for Reactivated Memories?

Consider a memory that was acquired long enough before the onset of some amnesic agent to be preserved from the RA effect of that agent. Would such an older memory become susceptible to RA by being reactivated (retrieved, put into the active state) shortly before the onset of the amnesic agent? If so, then the law of recency for RA refers not to the time since original learning of a trace but to the time since its latest activation. Some studies of the amnesic syndrome have demonstrated such RA for reactivated memories in subhuman mammals (DeVietti & Kirkpatrick, 1977; Misanin, Miller, & Lewis, 1968), but the only study of this in humans undergoing electroshock treatments (Squire, Slater, & Chace, 1976) obtained no such effect. Retrograde amnesia for reactivated memories is a very important theoretical issue on which too little research has been done to draw a definite conclusion regarding its existence or explanation.

Is RA a Storage or a Retrieval Deficit?

Because only recently learned (or possibly recently activated) memories are susceptible to RA, an explanation of RA as being due to a general retrieval deficit is untenable. Because substantial recovery from RA is possible, explanation of RA as being due to a permanent storage deficit (i.e., complete *destruction* of poorly consolidated traces) is untenable. The most plausible explanation is that RA is a reversible storage deficit that selectively affects more recently learned (or activated) traces to make them temporarily unretrievable. Since the deficit is selective for recent traces, the deficit is probably due to an alteration in the state of these recent memory traces. However, it is logically possible (but unlikely) that the retrieval process is altered in such a way as to selectively miss recently learned (or activated) traces. This is a resolvable empirical question. If one can specifically cue a subject to produce recovery of a "younger" memory trace A while an older memory trace B remains unavailable, then even a recency-specific retrieval deficit is ruled out. No one seems to have presented such evidence (except Zangwill's, 1964, clinical evidence that recovery from RA is due to the expansion of islands), but given the specificity of the "reminder" cues that are used to produce recovery from RA, my guess would certainly be that recovery would not be general for all recent memories but highly selective for the memories related to the reminder cue.

Nevertheless, regardless of how the above issue is resolved, the recency law for RA shows that there is some second property of a memory trace (in addition to trace strength), call it trace fragility, which is changing over time since learning (or reactivation). The more time that has elapsed since learning (or reactivation), the lower the fragility and the less the susceptibility to disruption by amnesic agents (and also the slower the rate of normal forgetting). Whether RA is a reversible storage deficit or a reversible retrieval deficit, the recency law of RA demonstrates that memory traces undergo consolidation during the storage phase that follows activation (in learning or possibly retrieval). This conclusion appears to be necessary regardless of whether RA proves to be a storage deficit or a retrieval deficit. Either way, the selective disruption of recent memories supports a consolidation process that progressively reduces the susceptibility of memory traces to disruption (Ribot, 1896; Russell, 1959; Squire et al., 1975; Wickelgren, 1972, 1974).

AA Is a Chunking Deficit

We observed in humans that the AA of the amnesic syndrome does not apply equally to all types of learning. Rather, the learning that appears primarily to be disrupted is higher cognitive learning, which I hypothesize to require strengthening associations to new chunk nodes. If animal AA is also a chunking deficit, then we would expect deficits in cognitive expectancy learning, not S–R learning. This should primarily disrupt more complex learning tasks, learning in the presence of interfering associations, learning without reinforcement (latent learning), and reinforcement contrast effects. This is exactly

the pattern of the results for AA in animals.[1]

Hippocampal animals are deficient in learning complex mazes (D. Kimble, 1963; Kveim, Setekliev, & Kaada, 1964; Leaton, 1969). Chunking is presumed to occur in complex maze learning to form a cognitive map composed of what-leads-to-what (propositional) expectancy nodes. Such animals are also deficient in learning conditional discriminations (D. Kimble, 1963) and in time-sequence learning, such as alternation (Franchina & Brown, 1970; Gross, Chorover, & Cohen, 1956; Means, Leander, & Isaacson, 1971; see review by Hirsh, 1974). Hippocampal animals are not deficient in simple T-maze or other single-stage, noncontingent discrimination learning tasks (Douglas, 1967; D. Kimble, 1968) in which simple S–R learning is adequate. Stimulus–response learning is adequate for such simple choice learning tasks because there is little similarity between the learning task and other choice situations. Hence, associative interference is minimal, and the chunking process is not needed to master the task.

In simple choice learning tasks that maximize interference between present learning and similar prior learning, animals with hippocampal lesions do show deficits. Hippocampals are deficient in extinction of previously learned responses (Douglas, 1967; Douglas & Pribam, 1966; Hirsh, 1974; D. Kimble, 1968; D. Kimble & Kimble, 1970). They are deficient in discrimination reversal learning, although acquisition of the initial discrimination was not retarded (Douglas, 1967; Hirsh, 1974; D. Kimble, 1968). Finally, they are slower in learning a passive avoidance response to an object they had previously learned to approach, though once again the initial approach behavior was learned as fast as by normals. Without previously established approach tendencies, passive avoidance learning is not generally deficient in hippocampals (Douglas, 1967; Hirsh, 1974; D. Kimble, 1968). According to the present hypothesis, animals with a hippocampus can form new chunk nodes in the cortex to represent something equivalent to the compound concept "situation A at this later time." This new compound chunk node can then be associated to the newly appropriate response, avoiding some of the interference caused by the prior association of a different response to the "situation A" node. Hippocampals cannot form such new contextually dependent chunk nodes representing compound concepts and are subject to greater associative interference from prior similar learning.

According to the hypothesis that the hippocampus plays some essential role in cognitive learning by the cortex, animals with hippocampal lesions should be deficient in latent learning by exploration in the absence of primary reinforcers and appropriate drives, and they are (Hirsh, 1974). Because a horizontal associative memory (memory without the chunking capacity to form new nodes) is so much more limited by interference in the number of associations that can be simultaneously maintained, S–R learning is limited to situations involving strong incentives (primary reinforcers in the presence of appropriate drives). Hippocampal mammals appear to revert to learning by means of the S–R reinforcement mechanism and no longer demonstrate higher cognitive learning in the absence of primary incentives.

Finally, mammals with hippocampal lesions behave like fish in reinforcement-contrast paradigms; that is, they do not immediately run faster when the amount of reinforcement is increased or slower when it is decreased (Franchina & Brown, 1971; Hirsh, 1974). Like fish, hippocampal mammals appear to have stamped-in an S–R association as a result of prior reinforcement, but without any encoding of the expected reinforcement.

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[1] The arguments and the evidence described in this section are based largely on an excellent review by Hirsh (1974). Hirsh proposed a contextual-retrieval function for the hippocampus, which makes the hippocampal-amnesic syndrome a retrieval deficit for more complex (contextually contingent) information. For the reasons presented earlier in this article, I think retrieval-deficit explanations of either RA or AA are implausible. However, Hirsh's arguments transfer essentially perfectly to the hypothesis that AA is a chunking (learning) deficit for complex information.

Neural Theory of Chunking and Consolidation

A major theoretical problem is to specify a mechanism by which the hippocampus (more generally, some system involving the hippocampus and neighboring nuclei or fibers of passage) can control chunking in the cortex. Because of the close association between RA and AA in the amnesic syndrome and the recency-dependent character of RA, we have the clue that the hippocampal chunking (learning) mechanism must have a close connection to the consolidation (storage) process. We also want the proposed mechanism to be simple and neurally plausible. What follows is one idea for such a mechanism.

A psychological node representing an idea (chunk, concept, image, proposition, etc.) may be neurally encoded by an activation gradient defined over all the neurons in the cortex. If we assume that degree of activation (rate of firing) is the only property of a neuron that is involved in this neural encoding system (a major assumption), then the conflict between specific-neuron theories and more holistic (distributed, holographic) theories of encoding comes down to the following: When we think of an idea, is the subset of neurons that are activated above (or below) baseline (spontaneous firing rate) a very small or a very large subset of all the neurons in the cortex? Regardless of where along this continuum the truth lies, creating a new chunk node to represent A and B probably requires strengthening the associations (chains of synaptic connections) from some of the neurons that are strongly activated in the code for A and the code for B to the neurons that are strongly activated in the code for the *A and B* chunk. The following theory describes a possible mechanism by which the hippocampal system might control this part of chunking. The theory assumes that nodes are encoded by small sets of neurons (specific-neuron encoding), though some parts of the theory might be adaptable to a holistic, distributed encoding theory.

Cortical neurons are assumed to be partitioned into two sets: free and bound. Bound neurons already have strong input and output connections (associations or synapses) to other neurons, derived from some combination of genetic bias and prior learning (including prior chunking). A bound neuron already represents some idea (feature, segment, concept, or proposition); a free neuron does not, but it can come to represent a new idea (combination of previously bound neurons) by means of the chunking process. Previously, I specified an axonal *growth* process as a mechanism by which two or more constituent neurons could come to specify a new chunk neuron (Wickelgren, 1969). However, a growth process is implausible on a number of counts, not the least of which is the apparent ability of new long-term memory traces to be acquired (learned and retrievable, though not fully consolidated) in a matter of seconds or milliseconds. Here I propose that the mechanism of chunking is a potentiation of preexisting connections. This selective strengthening hypothesis assumes that all free neurons are genetically connected in a weak manner to a large number of other neurons (both free and already bound) in the cortex (perhaps on the order of 4×10^4 neurons—which is an estimate of the average number of synapses/neuron in the human cortex, according to Cragg, 1975). There are approximately 2×10^{10} neurons in the human cerebral cortex (Pakkenberg, 1966). Thus, for any pair of bound neurons to become vertically associated by the chunking process, there need be specification of *very* few intervening chunk interneurons (perhaps as few as one chunk interneuron, since $|4 \times 10^4| \times |4 \times 10^4| \doteq 2 \times 10^{10}$).

How do weak associations become stronger? We can do no better than to make the standard neural contiguity conditioning assumption required for strengthening both already existing vertical and horizontal associations, namely, temporally contiguous activation of two neurons strengthens the synapses connecting them (e.g., Hebb, 1949). Three further points need to be made to extend the standard neural contiguity conditioning mechanism, to handle the specification (binding) of free neurons in chunking.

First, when two (or more) bound neurons are to be activated, there will be, necessarily, one maximally activated chain of free neurons connecting them. By lateral inhibition, the maximally activated chain (presumably, the most direct connection, in general) can suppress all other free neurons outside that chain, so the maximally activated chain of free neurons (perhaps as few as one intervening neuron) comes, by contiguity conditioning, to represent the combination of the previously bound neurons that initially activated it. It is clear that the maximally activated chain of free neurons should be the most direct (shortest) chain that connects the two (or more) constituent bound neurons and also that there should be connections both from the bound neurons "upward" to the free chunk neuron(s) and "downward" from the chunk neuron to all constituent neurons.

Second, previously bound neurons generally have strong associations to other bound neurons. How could the free neurons in a chain of weak associations compete successfully by the lateral inhibition process with previously bound neurons that are strongly associated to the constituent neurons to be chunked? I propose that the hippocampal (limbic) system operates at this point in the chunking process. The hippocampal system is assumed to be connected in a diffuse excitatory manner to all of the free neurons in the cortex and not at all connected, or connected in an inhibitory manner, to the bound cortical neurons. In chunking (vertical associative learning), the hippocampal arousal system differentially primes the free neurons to ensure that it will be some small set (chain) of *free* neurons that is maximally activated by the constituent neurons to be chunked, not some already *bound* neurons. In retrieval of previously stored information (which occurs in all perception, recognition memory, and recall), a different arousal system (the reticular activating system?) might do just the opposite—prime the set of bound neurons at the expense of the free neurons, or just prime all neurons equally, which would favor bound neurons because of their strong existing associations. Routtenberg (1968) described a mountain of evidence to support the hypothesis of just these two arousal systems. Routtenberg's hypotheses concerning the functions of these two arousal systems are somewhat different from the present hypothesis, but they are, in a general way, strikingly consistent with it.

Third, since the chunking process is continually converting cortical free neurons into cortical bound neurons, the hippocampal arousal system must have its excitatory synapses to cortical neurons repressed by the strengthening of cortical–cortical synapses in chunking. Inhibitory synapses (from the hippocampal system or elsewhere) to cortical neurons might remain unaffected, converting net excitatory hippocampal input to free neurons into net inhibitory input for bound neurons. The strengthening of one set of "genetically preferred" cortical–cortical synapses to a neuron, by the chunking process, represses (functionally disconnects) the neuron from excitatory hippocampal input and thus "protects" that neuron from being involved in new chunking, which could cause interference with the prior learning.

Far from being an exotic assumption, there is considerable evidence for such synaptic repression of previously strong synapses by genetically preferred synapses (Mark, 1974; Wall, 1977). The mechanism of synaptic repression may be similar to that which occurs at the motor end plate of the neuromuscular junction when motor neurons have grown back to make synaptic contact after being cut. Cutting the motor nerve to a muscle (denervation) produces a heightened sensitivity to the neurotransmitter over a large portion of the muscle surface (denervation supersensitivity). When the motor nerve regenerates and makes contact with the muscle, sensitivity is gradually reduced at portions of the muscle surface that are not in synaptic contact with motor neurons. Neural regeneration experiments have also demonstrated preferential affinities between particular nerves and particular muscles, such that initially certain neurons make functional synaptic contacts with a muscle and later other (genetically preferred) neurons make contacts and repress sensitivity at the previous (less preferred) contact sites (see Mark,

1974, for a review). Thus, it is quite reasonable to guess that synaptic repression may play an important role in permitting the hippocampal arousal system to restrict its excitatory activation to free neurons.[2]

Synaptic repression does not occur immediately. It takes days or weeks. Perhaps it is never complete but is only approaching a limit. As Russell (1959), Weiskrantz (1966), and Wickelgren (1972, 1974, 1976a) have argued previously from a variety of evidence, including normal forgetting functions and the distribution of RAs of various longevities, consolidation of long-term memory is also never complete but is only going on at a continually diminishing rate the older the memory trace. Thus, it is entirely reasonable to note that the repression of hippocampal input to newly bound cortical neurons is essentially a consolidation process, since it serves to protect previously bound neurons from being involved in new chunking. This accounts for the negatively accelerated forgetting function, as progressive repression of the hippocampal arousal input serves increasingly to protect the newly specified chunk neurons from nonspecific hippocampal activation and the degradative effects of such nonspecific input on specific learned associations.

Besides accounting for the continuous deceleration in forgetting rate over time and the progressive reduction in susceptibility to RA (recency law for RA), the theory also accounts for the chunking deficit of AA and the correlation of RA and AA. According to this theory, when the hippocampus is lesioned or disrupted in its functioning, which cortical neurons should be most affected? The free neurons, of course, because they are the ones still maximally connected to the hippocampal arousal system. (The inhibitory input to cortical neurons need not derive from the hippocampus and may even be generated internally by "tonic" lateral inhibitory interaction of cortical neurons.) Next most strongly affected by hippocampal lesion or dysfunction would be recently specified bound neurons, with greater effect the more recently the neuron has been bound by the chunking (learning) process. This is exactly the pattern of deficiency in the amnesic syndrome: New chunking is disrupted (AA), according to the present theory, either because the hippocampal system is not playing its essential role in the chunking process or because its prior abnormal functioning has in some way temporarily disrupted the cortical neurons to which it is still connected (free neurons that are used in new chunking). The latter explanation is essential to explain the (usually reversible) recency-dependent RA, so it is clearly the more parsimonious explanation of AA, but the former explanation presumably underlies the permanent AA of humans and animals with hippocampal lesions.

In any case, the theory explains why RA and AA tend to occur together—because both the cortical neurons needed for new chunking and those recently specified by the chunking process are still functionally connected to the hippocampal system. Thus, lesions, electrical stimulation, or chemical disruption of the hippocampal system will tend first and foremost to produce AA because free neurons are the most strongly connected to the hippocampus. If the hippocampal disruption is more severe, recently specified cortical neurons will be disrupted and RA will be observed, with less RA the older the memory trace. Except for Weiskrantz's (1966) theory, which I have argued previously in this article must be incorrect, this is the only theory that accounts for the basic facts of RA and AA with a common mechanism. Moreover, the theory provides an integration of a wide variety of phenomena, including chunking in verbal learning and semantic memory, configuring in conditioning, cognitive versus S–R learning in animals, normal forgetting functions, retrograde

[2] I suppose this is as good a time as any to note that when I talk of hippocampal-cortical synapses, I do not necessarily imply that there is a direct connection of hippocampal neurons to cortical neurons, though such direct connections have recently been reported (Rosene & Van Hoesen, 1977). It could also be an indirect connection. I use the term *hippocampal arousal system* in much the same way Routtenberg (1968) uses the term *limbic arousal system*, to refer to a system that may include more than just the hippocampus.

and anterograde amnesia in humans and animals, cortical synaptic connectivity, the hippocampal (limbic) arousal system, and synaptic repression.

References

Anderson, J. R. Item-specific and relation-specific interference in sentence memory. *Journal of Experimental Psychology: Human Learning and Memory*, 1975, *104*, 249–260.

Anderson, J. R. *Language, memory, and thought*. Hillsdale, N.J.: Erlbaum, 1976.

Anderson, J. R., & Bower, G. H. *Human associative memory*. Washington, D.C.: V. H. Winston, 1973.

Baddeley, A. D., & Warrington, E. K. Amnesia and the distinction between long- and short-term memory. *Journal of Verbal Learning and Verbal Behavior*, 1970, *9*, 176–189.

Baddeley, A. D., & Warrington, E. K. Memory coding and amnesia. *Neuropsychologia*, 1973, *11*, 159–165.

Barbizet, J. *Human memory and its pathology*. San Francisco: Freeman, 1970.

Berman, R. F., & Kesner, R. P. Posttrial hippocampal, amygdaloid, and lateral hypothalamic electrical stimulation: Effect on short- and long-term memory of an appetitive experience. *Journal of Comparative and Physiological Psychology*, 1976, *90*, 260–267.

Bitterman, M. E. Thorndike and the problem of animal intelligence. *American Psychologist*, 1969, *24*, 444–453.

Bitterman, M. E. The comparative analysis of learning. *Science*, 1975, *188*, 699–709.

Bohdanecka, M., Bohdanecky, Z., & Jarvik, M. E. Amnesic effects of small bilateral brain puncture in the mouse. *Science*, 1967, *157*, 334–336.

Brooks, D. N., & Baddeley, A. D. What can amnesic patients learn? *Neuropsychologia*, 1976, *14*, 111–122.

Cermak, L. S., Butters, N., & Goodglass, H. The extent of memory loss in Korsakoff patients. *Neuropsychologia*, 1971, *9*, 307–315.

Corkin, S. Acquisition of motor skill after bilateral medial temporal-lobe excision. *Neuropsychologia*, 1968, *6*, 255–265.

Cragg, B. G. The density of synapses and neurons in normal, mentally defective, and ageing human brains. *Brain*, 1975, *98*, 81–90.

Deutsch, J. A. The cholinergic synapse and the site of memory. *Science*, 1971, *174*, 788–794.

DeVietti, T. L., & Bucy, C. E. Recovery of memory after reminder: Evidence for two forms of retrieval deficit induced by ECS. *Psysiological Psychology*, 1975, *3*, 19–25.

DeVietti, T. L., & Hopfer, T. M. Complete amnesia induced by ECS and complete recovery of memory following reinstatement treatment. *Physiology and Behavior*, 1974, *12*, 599–603.

DeVietti, T. L., & Kirkpatrick, B. R. Stimulation of specific regions of brain in rats modifies retention for newly acquired and old habits. *Journal of Comparative and Physiological Psychology*, 1977, *91*, 662–673.

Douglas, R. J. The hippocampus and behavior. *Psychological Bulletin*, 1967, *67*, 416–442.

Douglas, R. J., & Pribram, K. H. Learning and limbic lesions. *Neuropsychologia*, 1966, *4*, 197–221.

Estes, W. K. An associative basis for coding and organization in memory. In A. W. Melton & E. Martin (Ed.), *Coding processes in human memory*. New York: Wiley, 1972.

Estes, W. K. Memory, perception, and decision in letter identification. In R. L. Solso (Ed.), *Information processing and cognition: The Loyola Symposium*. Hillsdale, N.J.: Erlbaum, 1975.

Evans, J. H. Transient loss of memory, an organic mental syndrome. *Brain*, 1966, *89*, 539–548.

Fisher, C. M., & Adams, R. D. Transient global amnesia. *Acta Neurologica Scandinavica, Suppl. 9*, 1964, *40*, 1–83.

Flexner, L. B., & Flexner, J. B. Intracerebral saline: Effect on memory of trained mice treated with puromycin. *Science*, 1968, *159*, 330–331.

Franchina, J. J., & Brown, T. S. Response patterning and extinction in rats with hippocampal lesion. *Journal of Comparative Physiology and Psychology*, 1970, *70*, 66–72.

Franchina, J. J., & Brown, T. S. Reward magnitude shift effects in rats with hippocampal lesions. *Journal of Comparative Physiology and Psychology*, 1971, *76*, 365–370.

Glick, S. D., & Greenstein, S. Comparative learning and memory deficits following hippocampal and caudate lesions in mice. *Journal of Comparative Physiology and Psychology*, 1973, *82*, 188–194.

Gold, P. E., & King, R. A. Retrograde amnesia: Storage failure versus retrieval failure. *Psychological Review*, 1974, *81*, 465–469.

Gottlieb, G. G., & Wilson, I. Cerebral dominance: Temporary disruption of verbal memory by unilateral electroconvulsive shock treatment. *Journal of Comparative Physiology and Psychology*, 1965, *60*, 368–372.

Gross, C. G., Chorover, S. L., & Cohen, S. M. Caudate, cortical, hippocampal, and dorsal thalamic lesion in rats: Alternation and Hebb-Williams maze performance. *Neuropsychologia*, 1956, *3*, 53–68.

Hebb, D. O. *The organization of behavior*. New York: Wiley, 1949.

Hirsh, R. The hippocampus and contextual retrieval of information from memory: A theory. *Behavioral Biology*, 1974, *12*, 421–444.

Hughes, R. A., Barrett, R. J., & Ray, O. S. Retrograde amnesia in rats increases as a function of ECS-test interval and ECS intensity. *Physiology and Behavior*, 1970, *5*, 27–30.

Hull, C. L. *Principles of behavior*. New York: Appleton-Century, 1943.

Huppert, F. A., & Piercy, M. Recognition memory in amnesic patients: Effect of temporal context and familiarity of material. *Cortex*, 1976, *12*, 3–20.

Huppert, F. A., & Piercy, M. Recognition memory in amnesic patients: A defect of acquisition? *Neuropsychologia*, 1977, *15*, 643–652.

Ignelzi, R. J., & Squire, L. R. Recovery from anterograde and retrograde amnesia following percutaneous drainage of a cystic craniopharyngioma. *Journal of Neurology, Neurosurgery, and Psychiatry*, 1976, *39*, 1231–1236.

Isaacson, R. L. *The limbic system*. New York: Plenum Press, 1974.

Johnson, N. F. The role of chunking and organization in the process of recall. In G. H. Bower (Ed.), *The psychology of learning and motivation* (Vol. 4). New York: Academic Press, 1970.

Johnson, N. F. Organization and the concept of memory code. In A. W. Melton & E. Martin (Eds.), *Coding processes in human memory*. New York: Wiley, 1972.

Kesner, R. P., & Wilburn, M. W. A review of electrical stimulation of the brain in context of learning and retention. *Behavioral Biology*, 1974, *10*, 259–293.

Kimble, D. P. The effects of bilateral hippocampal lesions in rats. *Journal of Comparative Physiology and Psychology*, 1963, *56*, 273–283.

Kimble, D. P. Hippocampus and internal inhibition. *Psychological Bulletin*, 1968, *70*, 285–295.

Kimble, D. P., & Kimble, R. J. The effect of hippocampal lesion on extinction and "hypothesis" behavior in rats. *Psychological Behavior*, 1970, *5*, 735–738.

Kimble, G. A. *Hilgard & Marquis' conditioning & learning* (2nd ed.). New York: Appleton-Century-Crofts, 1961.

Kveim, O., Setekliev, J., & Kaada, B. R. Differential effects of hippocampal lesions on maze and passive avoidance learning in rats. *Experimental Neurology*, 1964, *9*, 59–72.

LaBerge, D. Attention and the measurement of perceptual learning. *Memory & Cognition*, 1973, *1*, 268–276.

LaBerge, D. Perceptual learning and attention. In W. K. Estes (Ed.), *Handbook of learning and cognitive processes: Attention and memory* (*Vol. 4*). Hillsdale, N.J.: Erlbaum, 1976.

Leaton, R. N. Effect of the introduction of blind alleys on maze performance of rats with hippocampal lesions. *Journal of Comparative Physiology and Psychology*, 1969, *67*, 104–109.

Lewis, D. J. Sources of experimental amnesia. *Psychological Review*, 1969, *76*, 461–472.

Lewis, D. J., Miller, R. R., & Misanin, J. R. Recovery of memory following amnesia. *Nature*, 1968, *220*, 704–705.

Mark, R. *Memory and nerve cell connections*. Oxford, England: Clarendon Press, 1974.

Marslen-Wilson, W. K., & Teuber, H. L. Memory for remote events in anterograde amnesia: Recognition of public figures from news-photographs. *Neuropsychologia*, 1975, *13*, 343–364

Means, L. W., Leander, J. D., & Isaacson, R. L. The effect of hippocampectomy on alternation behavior and response to novelty. *Physiology and Behavior*, 1971, *6*, 17–22.

Miller, G. A. The magical number seven, plus or minus two: Some limits on our capacity for processing information. *Psychological Review*, 1956, *63*, 81–97.

Miller, R. R., & Kraus, J. N. Somatic and autonomic indexes of recovery from electroconvulsive shock-induced amnesia in rats. *Journal of Comparative Physiology and Psychology*, 1977, *91*, 434–442.

Miller, R. R., & Springer, A. D. Temporal course of amnesia in rats after electroconvulsive shock. *Physiology and Behavior*, 1971, *6*, 229–233.

Miller, R. R., & Springer, A. D. Amnesia, consolidation, and retrieval. *Psychological Review*, 1973, *80*, 69–79.

Milner, B. Amnesia following operation on the temporal lobes. In C. W. M. Whitty & O. L. Zangwill (Eds.), *Amnesia*. London: Butterworths, 1966.

Milner, B., Corkin, S., & Teuber, H. L. Further analysis of the hippocampal amnesic syndrome: 14-year follow-up study of H. M. *Neuropsychologia*, 1968, *6*, 215–234.

Misanin, J. R., Miller, R. R., & Lewis, D. J. Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace. *Science*, 1968, *160*, 554–555.

Murray, D. J., & Hitchcock, C. H. The nature of the memory deficit in Korsakoff's psychosis. *Canadian Journal of Psychology*, 1973, *27*, 414–421.

Pakkenberg, H. The number of nerve cells in the cerebral cortex of man. *Journal of Comparative Neurology*, 1966, *128*, 17–20.

Palfai, T., & Kurtz, P. Time-dependent effects of Metrazol on memory. *Pharmacology Biochemistry and Behavior*, 1973, *1*, 55–59.

Quillian, M. R. Semantic memory. In M. L. Minsky (Ed.), *Semantic information processing*. Cambridge, Mass.: M.I.T. Press, 1968.

Razran, G. *Mind in evolution*. Boston: Houghton Mifflin, 1971.

Ribot, T. H. *Diseases of memory: An essay in positive psychology*. New York: Appleton, 1896.

Rosene, D. L., & Van Hoesen, G. W. Hippocampal efferents reach widespread areas of cerebral cortex and amygdala in the rhesus monkey. *Science*, 1977, *198*, 315–317.

Routtenberg, A. The two-arousal hypothesis: Reticular formation and limbic system. *Psychological Review*, 1968, *75*, 51–80.

Rumelhart, D. E., Lindsay, P. H., & Norman, D. A. A process model for long-term memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory*. New York: Academic Press, 1972.

Russell, W. R. *Brain, memory, learning*. London: Oxford University Press, 1959.

Russell, W. R. *The traumatic amnesias*. London: Oxford University Press, 1971.

Russell, W. R., & Nathan, P. W. Traumatic amnesia. *Brain*, 1946, *69*, 280–300.

Scoville, W. B., & Milner, B. Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry,* 1957, *20,* 11–21.

Seltzer, B., & Benson, D. F. The temporal pattern of retrograde amnesia in Korsakoff's disease. *Neurology,* 1974, *24,* 527–530.

Shuttleworth, E. C., & Morris, C. E. The transient global amnesia syndrome. *Archives of Neurology,* 1966, *15,* 515–520.

Squire, L. R. Short-term memory as a biological entity. In D. Deutsch & J. A. Deutsch (Eds.), *Short-term memory.* New York: Academic Press, 1975.

Squire, L. R., & Barondes, S. H. Variable decay of memory and its recovery in cycloheximide-treated mice. *Proceedings of the National Academy of Sciences of the United States of America,* 1972, *69,* 1416–1420.

Squire, L. R., & Slater, P. C. Anterograde and retrograde memory impairment in chronic amnesia. *Neuropsychologia,* 1978, in press.

Squire, L. R., Slater, P. C., & Chace, P. M. Retrograde amnesia: Temporal gradient in very long term memory following electroconvulsive therapy. *Science,* 1975, *187,* 77–79.

Squire, L. R., Slater, P. C., & Chace, P. M. Reactivation of recent or remote memory before electroconvulsive therapy does not produce retrograde amnesia. *Behavioral Biology,* 1976, *18,* 335–343.

Teuber, H. L., & Milner, B. Persistent anterograde amnesia after stab wound of the basal brain. *Neuropsychologia,* 1968, *6,* 267–282.

Thorndike, E. L. Animal intelligence: An experimental study of the associative processes in animals. *Psychological Review, Monograph Supplements,* 1898, *2* (No. 8).

Thorpe, W. H. *Learning and instinct in animals* (2nd ed.). Cambridge, Mass.: Harvard University Press, 1963.

Tolman, E. C. Cognitive maps in rats and men. *Psychological Review,* 1948, *55,* 189–208.

Victor, M. The amnesic syndrome and its anatomical basis. *Canadian Medical Association Journal,* 1969, *100,* 1115–1125.

Wall, P. D. The presence of ineffective synapses and the circumstances which unmask them. *Philosophical Transactions of the Royal Society,* 1977, *278,* 361–372.

Warrington, E. K., & Weiskrantz, L. A study of learning and retention in amnesic patients. *Neuropsychologica,* 1968, *6,* 283–291.

Warrington, E. K., & Weiskrantz, L. Amnesic syndrome: Consolidation or retrieval? *Nature,* 1970, *228,* 628–630.

Warrington, E. K., & Weiskrantz, L. The effect of prior learning on subsequent retention in amnesic patients. *Neuropsychologica,* 1974, *12,* 419–428.

Weiskrantz, L. Experimental studies of amnesia. In C. W. M. Whitty & O. L. Zangwill (Eds.), *Amnesia.* London: Butterworths, 1966.

Weiskrantz, L., & Warrington, E. K. A study of forgetting in amnesic patients. *Neuropsychologia,* 1970, *8,* 281–288.

Wiener, N. I. Electroconvulsive shock induced impairment and enhancement of a learned escape response. *Physiology and Behavior,* 1970, *5,* 971–974.

Wickelgren, W. A. Sparing of short-term memory in an amnesic patient: Implications for strength theory or memory. *Neuropsychologia,* 1968, *6,* 235–244.

Wickelgren, W. A. Learned specification of concept neurons. *Bulletin of Mathematical Biophysics,* 1969, *31,* 123–142.

Wickelgren, W. A. Trace resistance and the decay of long-term memory. *Journal of Mathematical Psychology,* 1972, *9,* 418–455.

Wickelgren, W. A. Single-trace fragility theory of memory dynamics. *Memory & Cognition,* 1974, *2,* 775–780.

Wickelgren, W. A. Memory storage dynamics. In W. K. Estes (Ed.), *Handbook of learning and cognitive processes (Vol. 4).* Potomac, Md.: Erlbaum, 1976. (a)

Wickelgren, W. A. Network strength theory of storage and retrieval dynamics. *Psychological Review,* 1976, *83,* 466–478. (b)

Wickelgren, W. A. Subproblems of semantic memory: A review of "Human Associative Memory" by J. R. Anderson & G. H. Bower. *Journal of Mathematical Psychology,* 1976, *13,* 243–268. (c)

Wickelgren, W. A. Concept neurons: A proposed developmental study. *Bulletin of the Psychonomic Society,* 1977, *10,* 232–234. (a)

Wickelgren, W. A. *Learning and memory.* Englewood Cliffs, N.J.: Prentice-Hall, 1977. (b)

Winocur, G., & Weiskrantz, L. An investigation of paired-associate learning in amnesic patients. *Neuropsychologia,* 1976, *14,* 97-110.

Zangwill, O. L. Psychological studies in amnesic states. *Proceedings of the Third World Congress of Psychiatry,* 1964, *3,* 219.

Zinkin, S., & Miller, A. J. Recovery of memory after amnesia induced by electroconvulsive shock. *Science,* 1967, *155,* 102–103.

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