Making lasting memories: Remembering the significant

James L. McGaugh[1]

Department of Neurobiology and Behavior, Center for the Neurobiology of Learning and Memory, University of California, Irvine, CA 92697-3800

Edited by Francisco J. Ayala, University of California, Irvine, CA, and approved May 9, 2013 (received for review February 15, 2013)

Although forgetting is the common fate of most of our experiences, much evidence indicates that emotional arousal enhances the storage of memories, thus serving to create, selectively, lasting memories of our more important experiences. The neurobiological systems mediating emotional arousal and memory are very closely linked. The adrenal stress hormones epinephrine and corticosterone released by emotional arousal regulate the consolidation of long-term memory. The amygdala plays a critical role in mediating these stress hormone influences. The release of norepinephrine in the amygdala and the activation of noradrenergic receptors are essential for stress hormone-induced memory enhancement. The findings of both animal and human studies provide compelling evidence that stress-induced activation of the amygdala and its interactions with other brain regions involved in processing memory play a critical role in ensuring that emotionally significant experiences are well-remembered. Recent research has determined that some human subjects have highly superior autobiographic memory of their daily experiences and that there are structural differences in the brains of these subjects compared with the brains of subjects who do not have such memory. Understanding of neurobiological bases of such exceptional memory may provide additional insights into the processes underlying the selectivity of memory.

> Our brains, remarkable as they are, could not begin to contain and give equal weight to our every moment of life. (1)

The ability to learn and remember is essential for our survival. Remembering what has happened enables us to predict what is likely to happen and alter our behavior accordingly. As noted by Bernecker (2), "[r]emembering is a fundamental cognitive process, subserving virtually all other important cognitive functions ... Since without memory one couldn't think, some philosophers go as far as to claim that memory is the mark of being human" (ref. 2, p. 1). This latter claim is, of course, off of the mark, because most, if not all, animals display memory of their experiences. However, the many moments of their lives and our lives are not given equal weight in memory: we do not remember equally well all of our experiences. As James (3) commented, "[o]f some [experiences] no memory survives the instance of their passage ... Others ... may be recalled as long as life endures. How can we explain these differences?" (ref. 3, p. 643). There are many possible explanations. Experiences that we attend to are, of course, more likely to be remembered. Some new experiences become lasting, because they fit well with and can be readily processed and integrated with existing memories (4). Additionally, beginning with the pioneering studies of Ebbinghaus (5), we learned that memories are strengthened by repetition or retrieval (6).

Emotional Arousal and Lasting Memory

There is also extensive evidence that experiences that are emotionally arousing are well-remembered (7–10). Experiences of unpleasant occasions, such as an automobile accident, a mugging, or learning about the death of a loved one, are remembered better than those experiences of a routine day (8, 11–18). Memories of pleasant occasions, such as birthdays, holidays, and weddings, are also well-retained. The strength of memories of events varies with the emotional significance of the events. The memories of individuals who were close to San Francisco at the time of the 1989 San Francisco earthquake had better memories of the earthquake months later compared with individuals in Atlanta, Georgia (16). Three years after the terrorist attack on September 11, 2001, individuals who were in downtown Manhattan at the time of the attack had more detailed memories of the attack compared with individuals who were in midtown Manhattan, several miles from the attack (18).

Modulation of Memory Consolidation

These findings clearly support Bacon's (19) assertion that "[m]emory is assisted by anything that makes an impression on a powerful passion, inspiring fear, for example or wonder, shame or joy" (19). However, such evidence provides only the beginnings of an answer to William James' wondering about why some memories are lasting. A more comprehensive answer requires an understanding of the effects of emotional arousal that regulate the strength of memories.

Lasting memories are not created at the time of an experience. There is considerable evidence supporting the hypothesis of Mueller and Pilzecker (20) that experiences initiate neural processes that perseverate and induce, over time, the consolidation of memory. Subsequently, Hebb (21) proposed a dual-trace hypothesis of memory formation. According to this hypothesis, memories are initially based on the reverberation of neural circuits, and long-term memory results from synaptic changes induced by the neural reverberation. Thus, for both the consolidation hypothesis and the dual-trace hypothesis, lasting memory is formed after an experience.

The time-dependent process of memory consolidation, thus, provides an opportunity for conditions occurring after learning (i.e., during the consolidation of memory) to regulate the strength of memory. Studies of the effects of electroconvulsive shock (22) were the first studies to provide experimental evidence supporting the consolidation hypothesis. Electroconvulsive shock treatments impaired memory when administered to rats immediately after training. These findings were replicated and extended in extensive research with rats and mice in experiments using many kinds of treatments that disrupt brain functioning (23, 24). The common finding was that the treatments affected memory when administered shortly after training and were less affective when administered several hours or longer after training. These early findings of retrograde amnesia induced by disrupting brain functioning after learning suggested the possibility that mild stimulation of the brain shortly after an experience might enhance memory (25). The finding of many subsequent studies that memory is enhanced by administration of low doses of CNS stimulants to rats and mice shortly after training but not after a delay provided strong support for this

This paper results from the Arthur M. Sackler Colloquium of the National Academy of Sciences, "In the Light of Evolution VII: The Human Mental Machinery," held January 10–12, 2013, at the Arnold and Mabel Beckman Center of the National Academies of Sciences and Engineering in Irvine, CA. The complete program and audio files of most presentations are available on the NAS Web site at www.nasonline.org/evolution_vii.

Author contributions: J.L.M. wrote the paper.

The author declares no conflict of interest.

This article is a PNAS Direct Submission.

[1]E-mail: jlmcgaug@uci.edu.

implication (25–32). Also, importantly, comparable findings were obtained in studies using human subjects (33).

Endogenous Modulation of Memory Consolidation

The findings of experimentally induced retrograde amnesia and memory enhancement also suggest a hypothesis that might provide an answer to the question of why, as Francis Bacon asserted, memory is assisted by passion. Emotional arousal induces the release of the adrenal stress hormones epinephrine and cortisol (corticosterone in rats). Thus, the levels of the hormones activated by arousing training experiences are increased while memories are undergoing consolidation. Gerard (34) noted that "... as epinephrine is released in vivid emotional experiences, such an intense adventure should be highly memorable" (ref. 34, p. 30). Much subsequent evidence supports this suggestion. As found with stimulant drugs, posttraining administration of epinephrine as well as corticosterone enhances memory for many kinds of training experiences (35–39). Furthermore, adrenoreceptor antagonists (e.g., propranolol) and glucocorticoid receptor antagonists block the effects of emotional arousal and adrenal stress hormones on memory consolidation (40–47). Although most experiments investigating stress hormone influences have used memory of stressful training, such as stress induced by mild footshocks, posttraining administration of stress hormones enhances memory for many kinds of less stressful experiences, including memory for rewards (48).

Amygdala Activation and Memory Modulation

Thus, the experimental evidence provides strong support for the hypothesis that adrenal stress hormones enhance the consolidation of memory of experiences that induce their release. Additionally, the findings provide an initial step to providing an answer to William James' question of why some memories endure. A next essential step requires understanding of how adrenal stress hormones act to influence brain processes involved in memory consolidation. When released into the blood, epinephrine passes poorly, if at all, into the brain (49). Considerable evidence indicates that epinephrine influences on brain function are mediated by activating adrenoreceptors located on the ascending vagus nerve that projects to brainstem nuclei (to the locus coeruleus through the nucleus of the solitary tract) responsible for noradrenergic activation of other brain regions (50, 51). Moreover, direct electrical stimulation of the ascending vagus after learning enhances memory in human subjects as well as rats (52–55). Cortisol passes freely into the brain, where it can activate glucocorticoid receptors throughout the brain.

Several findings suggested the amygdala, a collection of nuclei located in the medial temporal lobe, as a possible critical brain region involved in mediating stress hormone influences on memory consolidation. Findings of several early (56, 57) as well as more recent studies (58, 59) indicated that, in rats, memory is enhanced by brief low-intensity posttraining electrical stimulation of the amygdala. Other early findings indicated that, in rats, β-adrenoreceptor antagonists infused into the amygdala after training impaired memory consolidation and that concurrent infusions of norepinephrine blocked the impairment (60, 61). Other studies reported that systemically administered epinephrine induces the release of norepinephrine in the brain (62) and that epinephrine enhancement of memory consolidation is blocked by intraamygdala infusions of propranolol (63). There is now substantial evidence that norepinephrine or other noradrenergic agonists administered into the amygdala or selectively into the basolateral region of the amygdala (BLA) after training enhances memory for many kinds of training experiences (64–68). Also, posttraining intraamygdala infusions of β-adrenoreceptor antagonists impair memory and block the memory-enhancing effects of both corticosterone and epinephrine administered systemically (63, 69–71). Such findings strongly suggest that glucocorticoid-induced enhancement of memory consolidation requires noradrenergic activation of the amygdala. Noradrenergic activation induced by emotional arousal seems to enable glucocorticoid modulation of memory consolidation (72).

The extensive evidence that memory is influenced by noradrenergic agonists and antagonists infused into the amygdala after training suggests that emotionally arousing training experiences should increase norepinephrine release within the amygdala. The findings of experiments using microdialysis and HPLC to assess norepinephrine release provide strong support for this implication. Footshock training increases the release of norepinephrine within the amygdala (73, 74), and rats that have greater increases in release subsequently display better retention (75). Additionally, several drugs that enhance memory consolidation, including GABAergic and opioid peptidergic antagonists, increase the release of norepinephrine in the amygdala (74, 76).

Amygdala Influences on Other Brain Systems

Decades before initiation of the research discussed above investigating the involvement of the amygdala in memory consolidation, Gerard (34) noted that, "... [because] the amygdala [acts] directly on cortical neurons to alter ... their responsiveness to the discrete impulses that reach the cortex ... these deep nuclei could easily modify the ease and completeness of experience fixation" (ref. 34, p. 30). The amygdala is richly interconnected with other brain regions, including the cortex, known to be involved in processing different aspects of memory. Additionally, there is now considerable evidence supporting the prescient suggestion by Gerard (34) that the amygdala influences memory consolidation through projections to other brain regions (24, 68, 77–81). However, the interactions are not restricted to the cortex.

The findings of many studies using rats indicate that the hippocampus is involved in spatial learning (82–84), whereas the caudate nucleus is involved in the learning of specific cues associated with responses (85–87). Packard et al. (88) found that posttraining activation of the amygdala (using microinfusions of D-amphetamine) enhanced memory for both place learning and cued response learning in a water maze (88). In contrast, hippocampal infusions selectively enhanced spatial memory, and caudate infusions selectively enhanced cued response memory. Posttraining, intra-BLA drug infusions enhance rats' memory of a context as well as the memory of a brief footshock subsequently received in that context (89). Additionally and importantly, McIntyre et al. (90) found that, in rats, noradrenergic activation of the BLA that enhanced memory consolidation increased the expression of activity-regulated cytoskeletal (Arc) protein in the hippocampus. Furthermore, posttraining inactivation of the BLA impairs memory consolidation and decreases hippocampal Arc protein expression. These findings are of interest in view of evidence indicating that Arc is involved in regulating synaptic plasticity and memory consolidation (91). Additionally, electrical stimulation of the BLA enhances the development of hippocampal plasticity as assessed by induction of long-term potentiation (92, 93).

As noted by Gerard (34), the amygdala also projects to the cortex. Electrical stimulation of the BLA activates the cortex, which is indicated by EEG desynchronization (94, 95), and enhances cortical long-term potentiation (96). Additionally, electrical stimulation of the amygdala enhances the development of plasticity in the auditory cortex (97). It is well-established that pairing of a tone stimulus with a reinforcing stimulus (e.g., footshock) alters the representation of the tone in the auditory cortex (98, 99). The frequency-receptive fields shift to the frequency of the tone stimulus, inducing an increased representation of significant sounds. Pairing of a tone with BLA stimulation, which is neither rewarding nor punishing, induces a shift of the auditory tuning curve to that tone of the conditioning tone

frequency. Moreover, the tuning curve continues to shift to the conditioning tone frequency over a period of 45 min after the training (100). Importantly, the learning-induced shifts in responsiveness of the auditory cortex are maintained for several weeks (101).

Emotional Arousal, Adrenal Stress Hormones, and Human Memory

The findings of studies of the influence of arousal on human memory are consistent with the findings of studies using animal subjects: emotional arousal during or after learning enhances long-term memory, and the modulation involves epinephrine and cortisol. However, experiences do not have to be intensely emotional to influence memory strength. Many studies have reported that subjects presented with pictures or words judged to be only mildly emotional, whether positive or negative in effect, subsequently have stronger memories of those stimuli than pictures and words judged not to be emotional (102–106). Viewing of emotionally arousing pictures also enhances memory of a cognitive skill (107).

In support of the view that emotional arousal modulates memory consolidation, several studies have reported that inducing arousal after subjects learn material enhances memory tested after a retention interval of 1 d or longer (102, 103, 105, 108, 109). In one study, subjects learned a word list and then watched an emotionally arousing pleasant (comedy) or unpleasant (surgery) brief video either immediately or after delays of up to 45 min. When viewed within 30 min, both the pleasant and unpleasant postlearning videos enhanced memory as assessed 1 wk later (108). Furthermore, the effect of postlearning arousal is not restricted to laboratory experiments. College students who watched an arousing video clip after a lecture compared with students who did not watch the clip performed significantly better on a midterm examination 2 wk later (110).

There is also extensive evidence that arousal influences on memory consolidation involve both epinephrine and cortisol. Administration of the adrenoreceptor antagonist propranolol before subjects' viewing of a series of pictures accompanied by an emotionally arousing story blocked the enhancing effects of emotional arousal on memory assessed as 1 wk later (111). Administration of epinephrine or cold pressor stress (induced by holding an arm in ice water), which induces the release of epinephrine and cortisol, immediately after presentation of emotionally arousing pictures enhances subjects' memory of the pictures (112, 113). Furthermore, Hupbach and Fieman (114) reported that arousal induced by exposure to cold pressor stress after a memory retrieval test increased salivary cortisol and enhanced memory of the test material when tested several days later.

Other studies have reported evidence that adrenergic activation selectively influences memory for emotionally arousing stimulation (115). Cold pressor stress induced after listening to neutral and emotional words selectively enhanced memory of the emotional words on a test the next day. Furthermore, levels of cortisol and salivary α-amylase, a biomarker for noradrenergic activity, assessed immediately after the cold pressor stress correlated highly with subsequent memory performance (116). Additionally, Segal and Cahill (117) found that levels of salivary α-amylase assessed shortly after subjects viewed a series of emotional and neutral pictures correlated significantly and selectively with memory of the emotional pictures on a 1-wk retention test (117). Salivary α-amylase measured after exposure to emotionally arousing pictures also correlated highly with subsequent memory assessed by successful discrimination of pictures seen from other similar pictures (i.e., pattern separation) (118). Such discrimination is known to involve the hippocampus (119).

Findings of human studies provide additional evidence that emotional arousal influences on memory involve activation of the amygdala. In an initial study using PET imaging, Cahill et al. (120) found that amygdala activation induced by watching emotionally arousing films correlated highly with memory of the films as tested 3 wk later. Subsequent studies using PET imaging reported similar findings (121, 122). Furthermore, studies using functional MRI imaging found that the relationship between amygdala activity during learning and subsequent memory varied directly with the intensity of emotional arousal and that the valence—positive or negative—is not critical (123–125).

Imaging studies have also provided evidence, consistent with evidence obtained with animal studies, that emotional arousal influences on consolidation of long-term memory involve interactions of the amygdala with other brain regions, including the hippocampus, during learning (126–132). Findings of human brain imaging studies using functional MRI provide additional evidence that emotional arousal influences on memory involve noradrenergic activation of the amygdala. Propranolol blocks amygdala activation induced by emotionally arousing stimuli as well as subsequent memory of the stimuli (133–136). Furthermore, administration of either the adrenergic drug yohimbine or hydrocortisone enhanced amygdala and hippocampus activation as well as memory as tested 1 wk later (137).

The findings of several studies suggest that intense or excessive activation of this noradrenergic system may contribute to the development of posttraumatic stress disorder (PTSD) (138). Propranolol administered to traumatized patients within several hours after a traumatic experience expressed fewer physiological signs of PTSD when tested 1 mo later (139, 140). Additionally, a study of the incidence of PTSD in wounded military personnel reported that patients given morphine within hours after the injury expressed fewer signs of PTSD when examined months after the experience (141). Because opiates inhibit the release of norepinephrine (142) a morphine-induced reduction in noradrenergic activation shortly after the trauma may have attenuated the development of PTSD.

Exceptional Human Memory

The findings summarized above provide the beginnings of understanding why, as William James wondered, some memories endure. Moreover, he suggested that the fact that many, perhaps most, memories are fleeting is adaptive. There is usually no need for memory of every detail of our daily experiences. As James (3) commented, "[s]election is the very keel on which our mental ship is built. If we remembered everything, we should, on most occasions be as ill off as if we remembered nothing" (ref. 3, p. 680). The fictional character in Borges' (143) short story, "Funes the Memorious," illustrated James' observation. After he was thrown from a horse, Funes expressed an extraordinary ability to learn and remember. He "... remembered not only every leaf of every tree of every wood, but also every one of the times that he had perceived ... it" (143). Also, he claimed to have "... more memories than all mankind has had..." (143). However, he also admitted, as James had anticipated, that his memory was like a garbage heap. Borges, thus, agreed with William James in stressing the importance of forgetting.

Luria (144) subsequently documented the now well-known case of a subject referred to as S, who had extraordinarily strong memory ability resembling the ability of Funes. Luria concluded that S's memory capacity and durability were unlimited. Also, he asked, "[h]ow had he come by this capacity for indelible memory traces?" (ref. 144, p. 61). Although another individual was subsequently determined to have comparable memory ability (145), Luria's question remains unanswered. It should also be noted that S's phenomenal memory seemed to be of little help to him in his daily personal life.

A small percentage of autistic individuals are capable of highly exceptional but restricted memory abilities. Calendar calculation is one of the most commonly reported abilities. Some autistic

individuals can readily state the day of the week for any specified date over a range of centuries, despite an inability to remember how to do simple addition and subtraction (146, 147). Such complex memory-based ability, like the ability of subject S, remains unexplained.

Recent research has identified a few human subjects who have a remarkable memory ability referred to as highly superior autobiographical memory (HSAM) (148, 149). The first subject identified to have this kind of memory (originally referred to as AJ and now known as Jill Price) wrote: "I am thirty-four years old and . . . have had this unbelievable ability to recall my past . . . I can take a date, between 1974 and today, and tell you what day it falls on, what I was doing that day and if anything of great importance occurred on that day I can describe that to you as well" (148). Extensive testing confirmed her claims. She was remarkably accurate in recalling the experiences of most of the days of her life beginning at about the age of 11 y. Her extensive diary entries confirmed her memories of events that occurred on specific days. Her memory of significant public events is equally superior. Also, importantly, she does not do calendar calculation: unlike autistic savants, she cannot readily provide the day for dates when she was very young or future dates. After the publication of the paper by Parker et al. (148), testing of many dozens of subjects who claimed to have strong autobiographical memory yielded several dozen subjects who surpassed age- and sex-matched controls in remembering the days and dates of personal and public events as well as details for each event (149). All responses were verified by checking available personal and public records. In contrast and perhaps surprisingly, HSAM subjects did not generally excel in learning and remembering information as assessed by laboratory tests (e.g., learning pairs of words and series of digits). Their exceptional memory ability seems to be restricted to experiences of daily life as reflected in episodic remembering. Their memory is not like the memories of Borges' character Funes the Memorious, Luria's subject S, or autistic savants. They are also not like the memory experts, who have learned specific mnemonic tricks enabling the learning of specific kinds of information (150).

MRI scans revealed that several brain regions of HSAM subjects differed from those regions of controls. Several brain regions differed in size and shape (e.g., putamen and caudate) as well as coherence of fiber tracts (e.g., uncinate faciculus) as assessed by diffusion tensor imaging. These results are, of course, only correlational and do not provide critical evidence that these anatomical differences are the bases of or contribute in some way to HSAM. However, it is worth noting that several of the brain regions found to be structurally different in HSAM and control subjects have been implicated in previous studies of autobiographical memory (151–153).

Unusual Memory: Fleeting and Lasting

Studies of unusual memory have significantly influenced memory research as well as our understanding of the neural systems underlying memory. The clinical findings of Ribot (154) that brain damage impairs most recent memories, sparing older ones, were the first to reveal that lasting memories are consolidated slowly over time. The findings of seminal studies of the patient HM (155, 156) forced the novel conclusion that different forms of memory are enabled by different brain systems (86, 157, 158), and thus, they significantly altered research investigating brain systems and memory. The novel finding that some human subjects create highly lasting memories of episodes of their daily experiences as well as memories of significant public events may, ultimately, lead to findings that provide new understanding of how our brains retain and retrieve memories.

Studies have not, as yet, investigated whether the strong memory of HSAM subjects involves experience-induced activation of stress hormones and activation of the amygdala. It may be that the modulatory systems of HSAM subjects are more highly activated by experiences or more sensitive to modestly arousing experiences.

However, HSAM subjects do not remember in precise detail all of their experiences. Like the rest of us, they remember best the more significant events of daily life. Their memories are not like the memories of Funes the Memorious. However, HSAM subjects differ from the rest of us in that they can retain their episodic memories for decades. If lasting memory is important for survival, why is it that so few individuals have this kind of long-lasting memory? It might be that these subjects' memory systems are genetically programmed to retain acquired information. Although the evidence, to date, indicates that none of the HSAM subjects have relatives who have strong memory, additional research is needed to determine whether this ability may have a genetic basis.

We might also wonder whether this ability might have been more common and more commonly used in centuries past. After all, the inventions of the printing press, computers, and cell phones in recent centuries have made it less necessary for us to create lasting records of our experiences. It is said that, before writing was available to keep records of important events, such as a wedding or granting of land, a child was selected to observe an event and then thrown into a river so that the child would subsequently have a lifelong memory of the event. As noted above, for most of us, "[o]ur brains, remarkable as they are, [can] not begin to contain and give equal weight to our every moment of life" (1). Selectively remembering our more important experiences seems to be the best strategy. It is what we generally do, thanks to the modulating influences of emotional arousal on lasting memory.

ACKNOWLEDGMENTS. Research was supported by National Institute of Mental Health Public Health Service Grant MH12526 and Gerard Family Trust.

1. Glore J (1987) *Personal Arts* (SCR Theatre, Costa Mesa, CA), Vol 2.
2. Bernecker S (2010) *Memory* (Oxford Univ Press, Oxford).
3. James W (1890) *Principles of Psychology* (Henry Holt and Company, New York).
4. Craik FIM, Lockhart RS (1972) Levels of processing: A framework for memory research. *J Verbal Learn and Verbal Behav* 11:671–684.
5. Ebbinghaus H (1885) *Uber das Gedachtnis* (Drucker and Humblat, Leipzig, Germany).
6. Roediger HL, 3rd, Butler AC (2011) The critical role of retrieval practice in long-term retention. *Trends Cogn Sci* 15(1):20–27.
7. Brown R, Kulik J (1977) Flashbulb memories. *Cognition* 5(1):73–99.
8. Conway MA (1995) *Flashbulb Memories* (Erlbaum, Brighton, England).
9. McGaugh JL (2003) *Memory and Emotion: The Making of Lasting Memories* (Columbia Univ Press, New York).
10. Reisberg D, Hertel P (2003) *Memory and Emotion* (Oxford Univ Press, New York).
11. Stratton GM (1919) Retroactive hypermnesia and other emotional effects on memory. *Psychol Rev* 26(6):474–486.
12. Pillemer DB (1984) Flashbulb memories of the assassination attempt on President Reagan. *Cognition* 16(1):63–80.
13. Bohannon JN, 3rd (1988) Flashbulb memories for the space shuttle disaster: A tale of two theories. *Cognition* 29(2):179–196.
14. Christianson S-A (1992) *Handbook of Emotion and Memory: Current Research and Theory* (Lawrence Erlbaum Associates, Hillsdale, NJ).
15. Conway MA, et al. (1994) The formation of flashbulb memories. *Mem Cognit* 22(3): 326–343.
16. Neisser U, et al. (1996) Remembering the earthquake: Direct experience vs. hearing the news. *Memory* 4(4):337–357.
17. Schmolck H, Buffalo EA, Squire LR (2000) Memory distortions develop over time: Recollections of the O.J. Simpson trial verdict after 15 and 32 months. *Psychol Sci* 11(1):39–45.
18. Sharot T, Martorella EA, Delgado MR, Phelps EA (2007) How personal experience modulates the neural circuitry of memories of September 11. *Proc Natl Acad Sci USA* 104(1):389–394.
19. Bacon F (2000) *The New Organon*, eds Jardine L, Silverthorne M (Cambridge Univ Press, Cambridge, United Kingdom).
20. Mueller GE, Pilzecker A (1900) Experimentelle Beitrage zur Lehre vom Gedächtniss. *Z Psychol* 1(1):1–288.

21. Hebb DO (1949) *The Organization of Behavior* (Wiley, New York).
22. Duncan CP (1949) The retroactive effect of electroshock on learning. *J Comp Physiol Psychol* 42(1):32–44.
23. McGaugh JL, Herz MJ (1972) *Memory Consolidation* (Albion Publishing Company, San Francisco).
24. McGaugh JL (2000) Memory—a century of consolidation. *Science* 287(5451): 248–251.
25. McGaugh JL (1966) Time-dependent processes in memory storage. *Science* 153(3742): 1351–1358.
26. Breen RA, McGAUGH JL (1961) Facilitation of maze learning with posttrial injections of picrotoxin. *J Comp Physiol Psychol* 54:498–501.
27. Westbrook WH, McGaugh JL (1964) Drug facilitation of latent learning. *Psychopharmacology (Berl)* 5:440–446.
28. McGaugh JL, Petrinovich LF (1965) Effects of drugs on learning and memory. *Int Rev Neurobiol* 8:139–196.
29. Krivanek J, McGaugh JL (1968) Effects of pentylenetetrazol on memory storage in mice. *Psychopharmacology (Berl)* 12(4):303–321.
30. McGaugh JL (1968) Drug facilitation of memory and learning. *Psychopharmacology: A Review of Progress*, PHS Publ. No. 1836:891-904, ed Efron DH (US Government Printing Office, Washington, DC).
31. McGaugh JL (1973) Drug facilitation of learning and memory. *Annu Rev Pharmacol* 13:229–241.
32. McGaugh JL, Roozendaal B (2009) Drug enhancement of memory consolidation: Historical perspective and neurobiological implications. *Psychopharmacology (Berl)* 202(1–3):3–14.
33. Soetens E, D'Hooge R, Hueting JE (1993) Amphetamine enhances human-memory consolidation. *Neurosci Lett* 161(1):9–12.
34. Gerard RW (1961) The fixation of experience. *Brain Mechanisms and Learning*, eds Fessard A, Gerard RW, Konorski J (Thomas, Springfield, IL), pp 21–35.
35. Micheau J, Destrade C, Soumireu-Mourat B (1984) Time-dependent effects of post-training intrahippocampal injections of corticosterone on retenion of appetitive learning tasks in mice. *Eur J Pharmacol* 106:39–46.
36. Sandi C, Rose SPR (1994) Corticosterone enhances long-term retention in one-day-old chicks trained in a weak passive avoidance learning paradigm. *Brain Res* 647(1): 106–112.
37. Zorawski M, Killcross S (2002) Posttraining glucocorticoid receptor agonist enhances memory in appetitive and aversive Pavlovian discrete-cue conditioning paradigms. *Neurobiol Learn Mem* 78(2):458–464.
38. Roozendaal B, et al. (2006a) Basolateral amygdala noradrenergic activity mediates corticosterone-induced enhancement of auditory fear conditioning. *Neurobiol Learn Mem* 86(3):249–255.
39. Berlau DJ, McGaugh JL (2006) Enhancement of extinction memory consolidation: The role of the noradrenergic and GABAergic systems within the basolateral amygdala. *Neurobiol Learn Mem* 86(2):123–132.
40. Gold PE, Van Buskirk RB (1975) Facilitation of time-dependent memory processes with posttrial epinephrine injections. *Behav Biol* 13(2):145–153.
41. Gold PE, McGaugh JL (1977) Hormones and memory. *Neuropeptide Influences on the Brain and Behavior*, eds Miller LH, Sandman CA, Kastin AJ (Raven, New York), pp 127–143.
42. McGaugh JL (1983) Hormonal influences on memory. *Annu Rev Psychol* 34:297–323.
43. McGaugh JL, Gold PE (1989) Hormonal modulation of memory. *Psychoendocrinology*, eds Brush RB, Levine S (Academic, New York), pp 305–339.
44. Roozendaal B (2000) 1999 Curt P. Richter award. Glucocorticoids and the regulation of memory consolidation. *Psychoneuroendocrinology* 25(3):213–238.
45. Roozendaal B, McGaugh JL (2011) Memory modulation. *Behav Neurosci* 125(6): 797–824.
46. Krugers HJ, Zhou M, Joels M, Kindt M (2011) Regulation of excitatory synapses and fearful memories by stress hormones. *Front Behav Neurosci* 5(2011):62.
47. Parfitt GM, Barbosa AK, Campos RC, Koth AP, Barros DM (2012) Moderate stress enhances memory persistence: Are adrenergic mechanisms involved? *Behav Neurosci* 126(5):729–734.
48. Dornelles A, et al. (2007) Adrenergic enhancement of consolidation of object recognition memory. *Neurobiol Learn Mem* 88(1):137–142.
49. Arai T, Watanabe T, Nagaro T, Matsuo S (1981) Blood-brain barrier impairment after cardiac resuscitation. *Crit Care Med* 9(6):444–448.
50. Miyashita T, Williams CL (2006) Epinephrine administration increases neural impulses propagated along the vagus nerve: Role of peripheral beta-adrenergic receptors. *Neurobiol Learn Mem* 85(2):116–124.
51. McIntyre CK, McGaugh JL, Williams CL (2012) Interacting brain systems modulate memory consolidation. *Neurosci Biobehav Rev* 36(7):1750–1762.
52. Clark KB, et al. (1998) Posttraining electrical stimulation of vagal afferents with concomitant vagal efferent inactivation enhances memory storage processes in the rat. *Neurobiol Learn Mem* 70(3):364–373.
53. Clark KB, Naritoku DK, Smith DC, Browning RA, Jensen RA (1999) Enhanced recognition memory following vagus nerve stimulation in human subjects. *Nat Neurosci* 2(1):94–98.
54. Clayton EC, Williams CL (2000) Adrenergic activation of the nucleus tractus solitarius potentiates amygdala norepinephrine release and enhances retention performance in emotionally arousing and spatial memory tasks. *Behav Brain Res* 112(1-2):151–158.
55. Hassert DL, Miyashita T, Williams CL (2004) The effects of peripheral vagal nerve stimulation at a memory-modulating intensity on norepinephrine output in the basolateral amygdala. *Behav Neurosci* 118(1):79–88.
56. Gold PE, Hankins L, Edwards RM, Chester J, McGaugh JL (1975) Memory interference and facilitation with posttrial amygdala stimulation: Effect on memory varies with footshock level. *Brain Res* 86(3):509–513.
57. McGaugh JL, Gold PE (1976) Modulation of memory by electrical stimulation of the brain. *Neural Mechanisms of Learning and Memory*, eds Rosenzweig MR, Bennett EL (MIT Press, Cambridge, MA), pp 549–560.
58. Bergado JA, Rojas Y, Capdevila V, González O, Almaguer-Melian W (2006) Stimulation of the basolateral amygdala improves the acquisition of a motor skill. *Restor Neurol Neurosci* 24(2):115–121.
59. Bass DI, Partain KN, Manns JR (2012) Event-specific enhancement of memory via brief electrical stimulation to the basolateral complex of the amygdala in rats. *Behav Neurosci* 126(1):204–208.
60. Kesner RP, Ellis ME (1983) Memory consolidation: Brain region and neurotransmitter specificity. *Neurosci Lett* 39(3):295–300.
61. Gallagher M, Kapp BS, Pascoe JP, Rapp PR (1981) A neuropharmacology of amygdaloid systems which contribute to learning and memory. *The Amygdaloid Complex*, ed Ben-Ari Y (Elsevier, Amsterdam), pp 343–354.
62. Gold PE, van Buskirk R (1978) Posttraining brain norepinephrine concentrations: Correlation with retention performance of avoidance training and with peripheral epinephrine modulation of memory processing. *Behav Biol* 23(4):509–520.
63. Liang KC, Juler RG, McGaugh JL (1986) Modulating effects of posttraining epinephrine on memory: Involvement of the amygdala noradrenergic system. *Brain Res* 368(1):125–133.
64. Hatfield T, McGaugh JL (1999) Norepinephrine infused into the basolateral amygdala posttraining enhances retention in a spatial water maze task. *Neurobiol Learn Mem* 71(2):232–239.
65. Huff NC, Wright-Hardesty KJ, Higgins EA, Matus-Amat P, Rudy JW (2005) Context pre-exposure obscures amygdala modulation of contextual-fear conditioning. *Learn Mem* 12(5):456–460.
66. LaLumiere RT, Buen T-V, McGaugh JL (2003) Post-training intra-basolateral amygdala infusions of norepinephrine enhance consolidation of memory for contextual fear conditioning. *J Neurosci* 23(17):6754–6758.
67. Roozendaal B, Castello NA, Vedana G, Barsegyan A, McGaugh JL (2008) Noradrenergic activation of the basolateral amygdala modulates consolidation of object recognition memory. *Neurobiol Learn Mem* 90(3):576–579.
68. McGaugh JL (2004) The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annu Rev Neurosci* 27:1–28.
69. Quirarte GL, Roozendaal B, McGaugh JL (1997) Glucocorticoid enhancement of memory storage involves noradrenergic activation in the basolateral amygdala. *Proc Natl Acad Sci USA* 94(25):14048–14053.
70. Roozendaal B, Quirarte GL, McGaugh JL (2002) Glucocorticoids interact with the basolateral amygdala β-adrenoceptor—cAMP/cAMP/PKA system in influencing memory consolidation. *Eur J Neurosci* 15(3):553–560.
71. Roozendaal B, Okuda S, Van der Zee EA, McGaugh JL (2006) Glucocorticoid enhancement of memory requires arousal-induced noradrenergic activation in the basolateral amygdala. *Proc Natl Acad Sci USA* 103(17):6741–6746.
72. Okuda S, Roozendaal B, McGaugh JL (2004) Glucocorticoid effects on object recognition memory require training-associated emotional arousal. *Proc Natl Acad Sci USA* 101(3):853–858.
73. Galvez R, Mesches MH, McGaugh JL (1996) Norepinephrine release in the amygdala in response to footshock stimulation. *Neurobiol Learn Mem* 66(3):253–257.
74. Quirarte GL, Galvez R, Roozendaal B, McGaugh JL (1998) Norepinephrine release in the amygdala in response to footshock and opioid peptidergic drugs. *Brain Res* 808(2):134–140.
75. McIntyre CK, Hatfield T, McGaugh JL (2002) Amygdala norepinephrine levels after training predict inhibitory avoidance retention performance in rats. *Eur J Neurosci* 16(7):1223–1226.
76. Hatfield T, Spanis C, McGaugh JL (1999) Response of amygdalar norepinephrine to footshock and GABAergic drugs using in vivo microdialysis and HPLC. *Brain Res* 835(2):340–345.
77. McGaugh JL (2002) Memory consolidation and the amygdala: A systems perspective. *Trends Neurosci* 25(9):456–461.
78. Stefanik MT, et al. (2013) Optogenetic inhibition of cocaine seeking in rats. *Addict Biol* 18(1):50–53.
79. Paré D, Collins DR, Pelletier JG (2002) Amygdala oscillations and the consolidation of emotional memories. *Trends Cogn Sci* 6(7):306–314.
80. Pelletier JG, Likhtik E, Filali M, Paré D (2005) Lasting increases in basolateral amygdala activity after emotional arousal: Implications for facilitated consolidation of emotional memories. *Learn Mem* 12(2):96–102.
81. Popescu AT, Popa D, Paré D (2009) Coherent gamma oscillations couple the amygdala and striatum during learning. *Nat Neurosci* 12(6):801–807.
82. O'Keefe J, Dostrovsky J (1971) The hippocampus as a spatial map. Preliminary evidence from unit activity in the freely-moving rat. *Brain Res* 34(1):171–175.
83. Olton DS, Becker JT, Handelmann GE (1979) Hippocampus, space, and memory. *Behav Brain Sci* 2:313–365.
84. Eichenbaum H, Stewart C, Morris RGM (1990) Hippocampal representation in place learning. *J Neurosci* 10(11):3531–3542.
85. Packard MG, White NM (1991) Dissociation of hippocampus and caudate nucleus memory systems by posttraining intracerebral injection of dopamine agonists. *Behav Neurosci* 105(2):295–306.
86. Packard MG, McGaugh JL (1996) Inactivation of hippocampus or caudate nucleus with lidocaine differentially affects expression of place and response learning. *Neurobiol Learn Mem* 65(1):65–72.
87. Packard MG, Goodman J (2012) Emotional arousal and multiple memory systems in the mammalian brain. *Front Behav Neurosci* 6(2012):14.
88. Packard MG, Cahill L, McGaugh JL (1994) Amygdala modulation of hippocampal-dependent and caudate nucleus-dependent memory processes. *Proc Natl Acad Sci USA* 91(18):8477–8481.

89. Malin EL, McGaugh JL (2006) Differential involvement of the hippocampus, anterior cingulate cortex, and basolateral amygdala in memory for context and footshock. *Proc Natl Acad Sci USA* 103(6):1959–1963.
90. McIntyre CK, et al. (2005) Memory-influencing intra-basolateral amygdala drug infusions modulate expression of Arc protein in the hippocampus. *Proc Natl Acad Sci USA* 102(30):10718–10723.
91. Guzowski JF, et al. (2000) Inhibition of activity-dependent arc protein expression in the rat hippocampus impairs the maintenance of long-term potentiation and the consolidation of long-term memory. *J Neurosci* 20(11):3993–4001.
92. Ikegaya Y, Saito H, Abe K (1995) High-frequency stimulation of the basolateral amygdala facilitates the induction of long-term potentiation in the dentate gyrus in vivo. *Neurosci Res* 22(2):203–207.
93. Akirav I, Richter-Levin G (1999) Biphasic modulation of hippocampal plasticity by behavioral stress and basolateral amygdala stimulation in the rat. *J Neurosci* 19(23): 10530–10535.
94. Dringenberg HC, Vanderwolf CH (1996) Cholinergic activation of the electrocorticogram: An amygdaloid activating system. *Exp Brain Res* 108(2):285–296.
95. Dringenberg HC, Saber AJ, Cahill L (2001) Enhanced frontal cortex activation in rats by convergent amygdaloid and noxious sensory signals. *Neuroreport* 12(11):2395–2398.
96. Dringenberg HC, Kuo MC, Tomaszek S (2004) Stabilization of thalamo-cortical long-term potentiation by the amygdala: Cholinergic and transcription-dependent mechanisms. *Eur J Neurosci* 20(2):557–565.
97. Chavez CM, McGaugh JL, Weinberger NM (2012) Amygdala strengthening of cortical memory representations. *The Amygdala—A Discrete Multitasking Manager*, ed Ferry B (In Tech, Rijeka, Croatia).
98. Weinberger NM (2004) Specific long-term memory traces in primary auditory cortex. *Nat Rev Neurosci* 5(4):279–290.
99. Weinberger NM (2007) Associative representational plasticity in the auditory cortex: A synthesis of two disciplines. *Learn Mem* 14(1–2):1–16.
100. Chavez CM, McGaugh JL, Weinberger NM (2009) The basolateral amygdala modulates specific sensory memory representations in the cerebral cortex. *Neurobiol Learn Mem* 91(4):382–392.
101. Chavez CM, McGaugh JL, Weinberger NM (2013) Activation of the basolateral amygdala induces long-term enhancement of specific memory representations in the cerebral cortex. *Neurobiol Learn Mem* 101:8–18.
102. Anderson AK, Yamaguchi Y, Grabski W, Lacka D (2006) Emotional memories are not all created equal: Evidence for selective memory enhancement. *Learn Mem* 13(6): 711–718.
103. Liu DLJ, Graham S, Zorawski M (2008) Enhanced selective memory consolidation following post-learning pleasant and aversive arousal. *Neurobiol Learn Mem* 89(1):36–46.
104. Henckens MJAG, Hermans EJ, Pu Z, Joëls M, Fernández G (2009) Stressed memories: How acute stress affects memory formation in humans. *J Neurosci* 29(32): 10111–10119.
105. Nielson KA, Lorber W (2009) Enhanced post-learning memory consolidation is influenced by arousal predisposition and emotion regulation but not by stimulus valence or arousal. *Neurobiol Learn Mem* 92(1):70–79.
106. Kensinger EA, Addis DR, Atapattu RK (2011) Amygdala activity at encoding corresponds with memory vividness and with memory for select episodic details. *Neuropsychologia* 49(4):663–673.
107. Steidl S, Razik F, Anderson AK (2011) Emotion enhanced retention of cognitive skill learning. *Emotion* 11(1):12–19.
108. Nielson KA, Powless M (2007) Positive and negative sources of emotional arousal enhance long-term word-list retention when induced as long as 30 min after learning. *Neurobiol Learn Mem* 88(1):40–47.
109. Nielson KA, Radtke RC, Jensen RA (1996) Arousal-induced modulation of memory storage processes in humans. *Neurobiol Learn Mem* 66(2):133–142.
110. Nielson KA, Arentsen TJ (2012) Memory modulation in the classroom: Selective enhancement of college examination performance by arousal induced after lecture. *Neurobiol Learn Mem* 98(1):12–16.
111. Cahill L, Prins B, Weber M, McGaugh JL (1994) β-adrenergic activation and memory for emotional events. *Nature* 371(6499):702–704.
112. Cahill L, Alkire MT (2003) Epinephrine enhancement of human memory consolidation: Interaction with arousal at encoding. *Neurobiol Learn Mem* 79(2):194–198.
113. Cahill L, Gorski L, Le K (2003) Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learn Mem* 10(4):270–274.
114. Hupbach A, Fieman R (2012) Moderate stress enhances immediate and delayed retrieval of educationally relevant material in healthy young men. *Behav Neurosci* 126(6):819–825.
115. Maheu FS, Joober R, Beaulieu S, Lupien SJ (2004) Differential effects of adrenergic and corticosteroid hormonal systems on human short- and long-term declarative memory for emotionally arousing material. *Behav Neurosci* 118(2):420–428.
116. Smeets T, Otgaar H, Candel I, Wolf OT (2008) True or false? Memory is differentially affected by stress-induced cortisol elevations and sympathetic activity at consolidation and retrieval. *Psychoneuroendocrinology* 33(10):1378–1386.
117. Segal SK, Cahill L (2009) Endogenous noradrenergic activation and memory for emotional material in men and women. *Psychoneuroendocrinology* 34(9):1263–1271.
118. Segal SK, Stark SM, Kattan D, Stark CE, Yassa MA (2012) Norepinephrine-mediated emotional arousal facilitates subsequent pattern separation. *Neurobiol Learn Mem* 97(4):465–469.
119. Yassa MA, Stark CE (2011) Pattern separation in the hippocampus. *Trends Neurosci* 34(10):515–525.
120. Cahill L, et al. (1996) Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proc Natl Acad Sci USA* 93(15):8016–8021.
121. Hamann SB, Ely TD, Grafton ST, Kilts CD (1999) Amygdala activity related to enhanced memory for pleasant and aversive stimuli. *Nat Neurosci* 2(3):289–293.
122. Hamann SB, Ely TD, Hoffman JM, Kilts CD (2002) Ecstasy and agony: Activation of the human amygdala in positive and negative emotion. *Psychol Sci* 13(2):135–141.
123. Canli T, Zhao Z, Brewer J, Gabrieli JD, Cahill L (2000) Event-related activation in the human amygdala associates with later memory for individual emotional experience. *J Neurosci* 20(19):RC99.
124. Canli T, Desmond JE, Zhao Z, Gabrieli JD (2002) Sex differences in the neural basis of emotional memories. *Proc Natl Acad Sci USA* 99(16):10789–10794.
125. Kensinger EA, Corkin S (2004) Two routes to emotional memory: Distinct neural processes for valence and arousal. *Proc Natl Acad Sci USA* 101(9):3310–3315.
126. Kilpatrick L, Cahill L (2003) Modulation of memory consolidation for olfactory learning by reversible inactivation of the basolateral amygdala. *Behav Neurosci* 117(1):184–188.
127. Dolcos F, LaBar KS, Cabeza R (2004) Interaction between the amygdala and the medial temporal lobe memory system predicts better memory for emotional events. *Neuron* 42(5):855–863.
128. LaBar KS, Cabeza R (2006) Cognitive neuroscience of emotional memory. *Nat Rev Neurosci* 7(1):54–64.
129. Kensinger EA, Krendl AC, Corkin S (2006) Memories of an emotional and a non-emotional event: Effects of aging and delay interval. *Exp Aging Res* 32(1):23–45.
130. Ritchey M, Dolcos F, Cabeza R (2008) Role of amygdala connectivity in the persistence of emotional memories over time: An event-related FMRI investigation. *Cereb Cortex* 18(11):2494–2504.
131. Ritchey M, LaBar KS, Cabeza R (2011) Level of processing modulates the neural correlates of emotional memory formation. *J Cogn Neurosci* 23(4):757–771.
132. Schwarze U, Bingel U, Sommer T (2012) Event-related nociceptive arousal enhances memory consolidation for neutral scenes. *J Neurosci* 32(4):1481–1487.
133. van Stegeren AH, et al. (2005) Noradrenaline mediates amygdala activation in men and women during encoding of emotional material. *Neuroimage* 24(3):898–909.
134. van Stegeren A, Rohleder N, Everaerd W, Wolf OT (2006) Salivary alpha amylase as marker for adrenergic activity during stress: Effect of betablockade. *Psychoneuroendocrinology* 31(1):137–141.
135. van Stegeren AH, et al. (2007) Endogenous cortisol level interacts with noradrenergic activation in the human amygdala. *Neurobiol Learn Mem* 87(1):57–66.
136. Strange BA, Dolan RJ (2004) β-adrenergic modulation of emotional memory-evoked human amygdala and hippocampal responses. *Proc Natl Acad Sci USA* 101(31): 11454–11458.
137. van Stegeren AH, Roozendaal B, Kindt M, Wolf OT, Joëls M (2010) Interacting noradrenergic and corticosteroid systems shift human brain activation patterns during encoding. *Neurobiol Learn Mem* 93(1):56–65.
138. Pitman RK (2006) Secondary pharmacological prevention of PTSD: Therapeutic implications of a translational model. *PTSD: Brain Mechanisms and Clinical Implications*, eds Kato N, Kawata M, Pitman RK (Springer, Berlin), pp 281–296.
139. Pitman RK, et al. (2002) Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biol Psychiatry* 51(2):189–192.
140. Vaiva G, et al. (2003) Immediate treatment with propranolol decreases post-traumatic stress disorder two months after trauma. *Biol Psychiatry* 54(9):947–949.
141. Holbrook TL, Galarneau MR, Dye JL, Quinn K, Dougherty AL (2010) Morphine use after combat injury in Iraq and post-traumatic stress disorder. *N Engl J Med* 362(2):110–117.
142. McGaugh JL, Introini-Collison IB, Nagahara AH (1988) Memory-enhancing effects of posttraining naloxone: Involvement of β-noradrenergic influences in the amygdaloid complex. *Brain Res* 446(1):37–49.
143. Borges JL (1944) Funes el memorioso. *Ficciones*, ed Kerrigan A, trans Bonner A (1962) (Grove Press, New York).
144. Luria AR (1968) *The Mind of a Mnemonist; A Little Book about a Vast Memory* (Harvard Univ Press, Cambridge, MA).
145. Hunt E, Love T (1972) How good can memory be?. *Coding Processes in Human Memory*, eds Melton AW, Martin E (Winston-Wiley, Washington, DC).
146. Hurst LC, Mulhall DJ (1988) Another calendar savant. *Br J Psychiatry* 152:274–277.
147. Heavey L, Pring L, Hermelin B (1999) A date to remember: The nature of memory in savant calendrical calculators. *Psychol Med* 29(1):145–160.
148. Parker ES, Cahill L, McGaugh JL (2006) A case of unusual autobiographical remembering. *Neurocase* 12(1):35–49.
149. LePort AKR, et al. (2012) A behavioral and neuroanatomical investigation of highly superior autobiographical memory. *Neurobiol Learn Mem* 98(1):78–92.
150. Wilding J, Valentine E (1997) *Superior Memory* (Psychology Press, East Sussex, United Kingdom).
151. Kapur N, Ellison D, Smith MP, McLellan DL, Burrows EH (1992) Focal retrograde amnesia following bilateral temporal lobe pathology. A neuropsychological and magnetic resonance study. *Brain* 115(Pt 1):73–85.
152. Levine B, et al. (1998) Episodic memory and the self in a case of isolated retrograde amnesia. *Brain* 121(Pt 10):1951–1973.
153. Steinvorth S, Corkin S, Halgren E (2006) Ecphory of autobiographical memories: An fMRI study of recent and remote memory retrieval. *Neuroimage* 30(1):285–298.
154. Ribot R (1882) *Diseases of Memory* (Appleton, New York).
155. Scoville WB, Milner B (1957) Loss of recent memory after bilateral hippocampal lesions. *J Neurol Neurosurg Psychiatry* 20(1):11–21.
156. Milner B (1972) Disorders of learning and memory after temporal lobe lesions in man. *Clin Neurosurg* 19:421–446.
157. Mishkin M, Malamut B, Bachevalier J (1984) Memories and habits: Two neural systems. *Neurobiology of Learning and Memory*, eds Lynch G, McGaugh JL, Weinberger NM (Guilford, New York), pp 65–77.
158. White NM, McDonald RJ (2002) Multiple parallel memory systems in the brain of the rat. *Neurobiol Learn Mem* 77(2):125–184.