Research Article

H.M., WORD KNOWLEDGE, AND AGING: Support for a New Theory of Long-Term Retrograde Amnesia

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Abstract—*This study develops a new theory of long-term retrograde amnesia that encompasses episodic and semantic memory, including word knowledge. Under the theory, retrograde amnesia in both normal individuals and hippocampal amnesics reflects transmission deficits caused by aging, nonrecent use of connections, and infrequent use of connections over the life span. However, transmission deficits cause severe and irreversible retrograde amnesia only in amnesics who (unlike normal persons) cannot readily form new connections to replace nonfunctioning ones. The results of this study are consistent with this theory: For low-frequency but not high-frequency words, a famous "hippocampal amnesic" (H.M.) at age 71 performed worse than memory-normal control participants in a lexical decision experiment and a meaning-definition task (e.g., What does* squander *mean?). Also as predicted, H.M.'s lexical decision performance declined dramatically between ages 57 and 71 for low-frequency words, but was age-invariant for high-frequency words.*

Relations between anterograde amnesia (AA) and retrograde amnesia (RA) have puzzled neuropsychologists since Ribot (1882). Whereas all amnesics exhibit AA, that is, postmorbid deficits in representing, retrieving, or consolidating new information, only some amnesics exhibit the poorly understood phenomenon of RA, defined here as long-term deficits in representing or retrieving information acquired years and sometimes decades before the amnesia-causing trauma (see Kapur, 1993, for other types of RA).

Differences between AA and RA have challenged theories under which the hippocampus encodes and stores concepts and events as they unfold in the fleeting present while simultaneously consolidating thousands and perhaps millions of autobiographical details across the life span. Although some degree of AA invariably accompanies RA (but not vice versa), low correlations between the severity of AA and RA have been reported, and unlike AA, RA is often "patchy" (with "islands" of intact memory for salient episodes), is temporally graded (with greater sparing of remote than recent episodes), and varies with the particular amnesic, type of memory, and time since trauma (e.g., Hunkin et al., 1995; Kapur, 1993; Levin et al., 1985; Morris, 1999; Murre, 1996).

In the present article, we develop a new theory of RA that we apply to word knowledge because evaluations of episodic memory in RA research have often involved specific proper names for people, places, and events. We then test predictions of our theory for H.M., an amnesic whose hippocampal systems were bilaterally lesioned in 1953, when he was 27. For many years, researchers believed that H.M.'s extremely severe AA for post-1953 events was his only deficit. However, H.M. exhibited relatively mild RA for famous names at age 44 (Marslen-Wilson & Teuber, 1975) and more severe RA at age 57 (Corkin, 1984). H.M. also exhibited selective deficits in the immediate representation of unfamiliar (but not familiar) sentence concepts at age 41 to 47, and we review these language deficits next because they substantiate the general theoretical framework adopted here.

In an ambiguity-discovery task administered in 1967, H.M. discovered the two meanings of short ambiguous sentences less often than memory-normal control subjects of comparable intelligence (IQ), age, work background, and educational level (MacKay, Stewart, & Burke, 1998). Control subjects born the same year as H.M. and a patient with bilateral frontal lobe damage also outperformed H.M. on this task, indicating that H.M.'s sentence-meaning deficits are related to his particular lesion rather than to cohort effects or general effects of brain damage. An ambiguity-detection task administered in 1973 (Lackner, 1974) also indicated deficits in comprehending ambiguous sentences because H.M. discriminated between ambiguous and unambiguous sentences no better than chance. Other tasks (picture description, sentence reading, description of sentence meanings, and conversational speech) have demonstrated that H.M.'s sentence-meaning deficits are greater for unfamiliar than familiar phrases, also apply to unambiguous sentences and to sentence production, and continue to the present day (see MacKay, Burke, & Stewart, 1998; MacKay & James, 2001).

THE TRANSMISSION-DEFICIT FRAMEWORK AND RA FOR WORDS

Our theory of RA derives from an interactive activation model of memory and language known as node structure theory (NST; MacKay, 1987), which postulates a vast network of interconnected representational units called nodes. Nodes representing word knowledge are organized into a *semantic system* (representing, e.g., word meanings) and a hierarchically organized *phonological system* (representing, e.g., syllables, consonant clusters, and speech sounds). Word retrieval requires three processes in the theory (MacKay, 1990; MacKay & Burke, 1990): binding (to initially form the nodes and connections that represent the word), node activation (to actually retrieve the word), and priming (to prepare the word's nodes for activation).

The priming and activation processes in NST can be illustrated by referring to Figure 1, which shows connections for the low-frequency (LF) word *squander.* Connections from the lexical node to semantic-system nodes representing, for example, "spend extravagantly" embody the meaning of *squander*, and connections to phonological nodes representing syllables and other hierarchically organized components embody its phonology. A unique feature of NST is that the same lexical node is involved in comprehension, or meaning retrieval (via bottom-up connections from lexical phonology), and production, or phonological retrieval (via top-down connections). For example, in the top-down processes for retrieving the phonology for *squander*, one or more of its meaning nodes (e.g., "spend extravagantly") is normally activated first, which primes (top-down) and enables activation of the *squander* node. Activating *squander* in turn primes (top-down) the syllable nodes representing "skwan" and "der," and so on, until the

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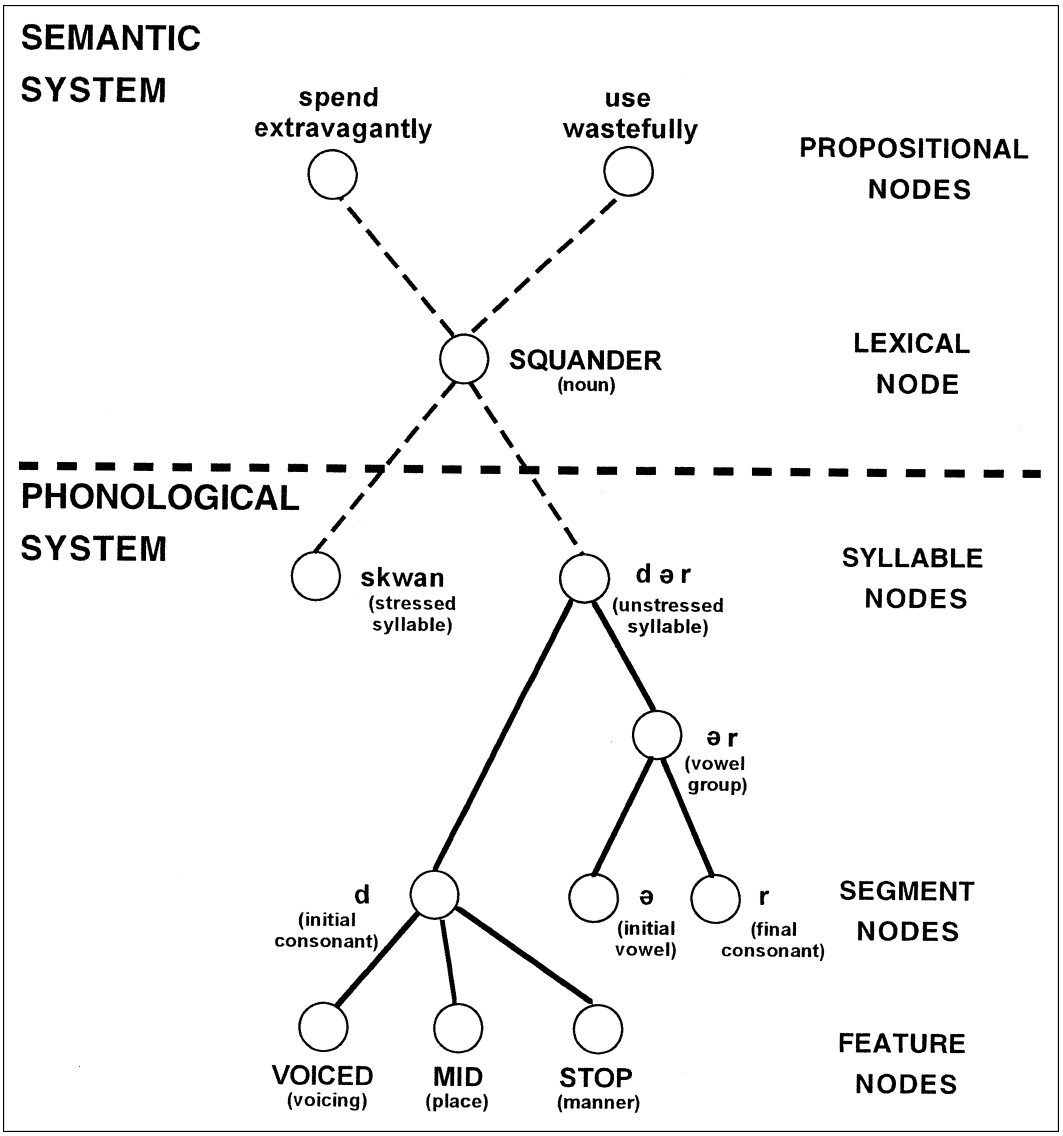

Fig. 1. Sample of nodes representing the word *squander* in the semantic and phonological systems of node structure theory (with many nodes omitted for simplicity). Solid lines represent functional connections. Broken lines represent connections that have transmission deficits and are defunct (permanently nonfunctional) for H.M.

full phonology for *squander* has been activated (retrieved) in sequence (see MacKay, 1987, pp. 39–89, for details).

Within NST, the basic cause of RA is transmission deficits that reduce the rate and amount of priming transmitted across connections throughout the cortex, and increase with aging, as well as nonrecent and infrequent use of information over the life span. For example, transmission deficits can reduce the top-down priming transmitted from *squander* to its syllable nodes, thereby preventing activation-retrieval of its phonology, an instance of phonological RA. Transmission deficits can be illustrated by the mild form of phonological RA known as the tip-of-the-tongue (TOT) phenomenon. People in the TOT state typically know the meaning of a not-recently-used LF word they are trying to retrieve, but cannot retrieve its full phonology, sometimes for many days (Burke, MacKay, Worthley, & Wade, 1991). TOTs are often partial (e.g., the speaker may know the initial sound and number of syllables of the otherwise irretrievable word), and they are known to increase with aging. According to NST, this increase occurs because age-linked transmission deficits temporarily prevent the activation-retrieval of specific phonological units with infrequently and not-recently-used connections. However, TOTs are reversible under NST: Perceiving or producing phonologically related words can prime critical phonological units, enabling the target word to pop into mind (seemingly spontaneously when the speaker is engaged in other activities; James & Burke, 2000).

However, with extreme disuse and old age, transmission deficits can cause particular connections to become defunct (i.e., permanently nonfunctional), and a node with no functional connections is impossible to activate. For example, if the node representing the /skw/ cluster in *squander* has become defunct because of extreme disuse and old age, presenting words such as *squash*, *squint*, and *squall* will no longer acti-

Table 1. *Characteristics of participants*

Participant	Age (years)	Highest level of education completed	Verbal IQ	Performance IQ	Mean IQ
		1997 study			
H.M.	71	High school	107	117	112.00
Control participants					
1	73	High school	115	129	122.00
2	74	High school	114	128	121.00
3	74	High school	110	113	111.50
4	70	High school	115	118	116.50
5	70	High school	117	130	123.50
6	67	High school	120	124	122.00
7	70	High school	107	118	112.50
8	74	High school	115	104	109.50
Average	71.50	High school	114.13	120.50	117.31
SD	2.62		4.02	9.04	5.54
		1983 study[a]			
H.M.	57	High school	97	115	106
Control participants ($n = 4$)	57	High school	Unknown	Unknown	Unknown

[a]From Gabrieli, Cohen, and Corkin (1988).

vate the /skw/ node. Nonetheless, older adults with intact hippocampal binding mechanisms can readily replace a defunct /skw/ node with a new one. When next they encounter /skw/, they simply form new connections to rerepresent /skw/. In contrast, defunct information remains defunct and irretrievable in hippocampal amnesics such as H.M., who cannot readily form new connections either to represent novel information or to rerepresent old information (MacKay & James, 2001).

This explains the loose association between AA and RA: By preventing the rerepresentation of defunct information, AA exacerbates RA. This also explains why RA exhibits temporal gradients. Newly formed connections are fragile and unstable (they lose their connection strength rapidly over time), so that memories immediately preceding the onset of AA quickly tend to become (and remain) defunct in the amnesic. However, remote memories (formed long before the amnesia-causing trauma) are more resistant to RA: Either they are frequently used, with connections that are strong and therefore stable over time, or they are recently used, with weak connections that may have been formed anew via binding mechanisms that were intact at that time. The great variability in how frequently and recently different premorbid memories are used or rehearsed therefore explains the patchy nature of RA, and differences across amnesics in age and time since trauma when tested explain the variable nature of RA across studies.

PARTICIPANTS[1]

Table 1 summarizes background characteristics of the participants in the studies reported here. The control participants in 1997 received $10/hr for participating and were native English speakers (6 female, 2 male) matched as closely as possible with H.M. for highest educational degree (high school), pretrauma employment, age, and IQ. According to recent magnetic resonance imaging data, H.M. has bilateral damage to the cerebellum (due to long-term use of epilepsy-controlling drugs) and hippocampal systems (due to his 1953 operation), but little neocortical damage (Corkin, Amaral, González, Johnson, & Hyman, 1997).

STUDY 1A: H.M.'S LEXICAL DECISION PERFORMANCE (1997)

Study 1a used a lexical decision task involving words from *Webster's New Collegiate Dictionary* (1949) that H.M. almost certainly knew before his operation. Because binding processes are irrelevant to retrieving high-frequency (HF) words (MacKay, Stewart, & Burke, 1998), NST predicted no deficits in H.M.'s lexical decision performance for HF words at age 71. However, NST predicted that LF words would resemble pseudowords for H.M.: His connections representing LF words were formed before 1953, and many would have become defunct because of aging, coupled with infrequent and nonrecent use. Moreover, NST predicted that H.M. could not rerepresent defunct LF words because his lesion had destroyed some (but perhaps not all; see MacKay, Burke, & Stewart, 1998) of the binding nodes[2] required to form new cortical connections. NST also predicted deficits for pseudowords: According to this theory, H.M. will often identify pseudowords as words if the

1. Details regarding the participants, materials, procedures, and results of Studies 1 and 2 are available on the Web at http://www.bol.ucla.edu/~mackay/app.htm.

2. The precise location in the brain of various types of binding nodes is an empirical question that NST does not address, although given current information, most binding nodes probably reside in bilateral hippocampus and associated entorhinal, perirhinal, and parahippocampal cortices (e.g., Milner, 1975; O'Keefe & Nadel, 1978). However, it is possible under NST that hippocampal-system damage may diminish the efficacy of binding nodes located outside hippocampal systems.

Table 2. *Mean percentage correct lexical decisions by condition for H.M. and control participants (with* SD*s) in Study 1a*

Participant	Condition			
	High-frequency words ($n = 26$)	Low-frequency words ($n = 26$)	Pseudowords ($n = 42$)	Average ($n = 94$)
H.M.	96	54	71	73
Control participants				
Average	99	87	90	92
SD	2	7	5	3
1	100	92	88	93
2	100	88	86	90
3	96	77	86	86
4	100	88	95	95
5	100	81	98	94
6	100	96	88	94

pseudowords have familiar high-frequency morphological components (e.g., *consultment*: *consult* + *ment*).

Method

The participants in Study 1a were H.M. and memory-normal control participants 1 through 6 (see Table 1). Materials were from MacKay and James (1999): 26 HF words, 26 LF words, and 42 easily pronounceable pseudowords typed in intermixed order in a single column on four pages. Instructions were presented verbally and visually on a prominently displayed card: "Using this pen, circle all and only those stimuli that are real English words."

Results and Discussion

Table 2 shows mean percentage of correct lexical decisions by condition for H.M. and control participants (with standard deviations). H.M. performed well within the range of the control participants for HF words, but not LF words, for which his performance approximated chance responding (50%). The control participants outperformed H.M. by more than 4.7 *SD*s for LF words, and more than 3.8 *SD*s for pseudowords. (*SD* deficits were calculated as percentage correct for control participants minus percentage correct for H.M., divided by the standard deviation of control participants.)

Forgetting, a speed-accuracy trade-off, and perceptual errors cannot explain these results because H.M. had the stimuli before him while responding, had unlimited time to respond, and read (and therefore perceived) the stimuli correctly. Nor were H.M.'s lexical decision deficits due to response bias (overall "yes" responses differed by only 0.67 *SD* for H.M. vs. control participants) or to stimulus length: The control participants outperformed H.M. by 5.5 *SD*s for short stimuli (six letters or fewer; $n = 50$) and by 5.2 *SD*s for long stimuli (seven letters or more; $n = 44$). Nor were H.M.'s lexical decision deficits related to IQ per se: Verbal and overall IQ of the control participants did not correlate reliably with lexical decision accuracy at $p < .096$ for any stimulus category, and the 1 control participant whose IQ almost exactly matched H.M.'s (control participant 3 in Table 1) behaved like other control participants, outperforming H.M. for all but HF words (see Table 2).

Because experimental results indicate that the cerebellum is involved in some cognitive tasks (e.g., Helmuth, Ivry, & Shimizu, 1997; Ivry & Keele, 1989), we ran Study 1a on 3 patients with bilateral lesions restricted to the cerebellum. These patients performed like normal control participants for HF words (100% correct, $SD = 0\%$), LF words (83% correct, $SD = 6\%$), and pseudowords (83% correct, $SD = 16\%$), indicating no cerebellar involvement in this task. Furthermore, the cerebellar patients outperformed H.M. by 4.8 *SD*s for LF words, ruling out H.M.'s bilateral cerebellar damage as the cause of his lexical decision deficits for LF words.

STUDY 1B: AGE-LINKED DECLINES IN H.M.'S LEXICAL DECISION PERFORMANCE

Study 1b compared H.M.'s lexical decision performance at ages 57 (the 1983 data of Gabrieli, Cohen, & Corkin, 1988, Experiment 2a) and 71 (the 1997 data of Study 1a). Word stimuli in these studies were comparable in length, frequency, and difficulty (control participants performed identically in the two experiments: 92% correct, $SD = 3\%$).

We assume that H.M. had no lexical decision deficit for LF words in 1952 (at age 25). However, since his surgery, nodes representing many of H.M.'s LF words have gradually become defunct without the possibility of rerepresentation via formation of new connections (because of H.M.'s lesion). NST therefore predicted a greater-than-normal age-linked decline in H.M.'s lexical decisions for LF words from 1983 through 1997, but no comparable decline for pseudowords (because H.M.'s binding deficits have remained constant since 1953) or for HF words (because frequent use prevents transmission deficits).

Method

Participants were H.M. and his age-matched control participants in 1997 and 1983 (see Table 1). The 1983 stimuli were 90 pseudowords[3] and 90 words (45 HF, 45 LF) that entered English dictionaries at least 3 years before H.M.'s lesion. The 1983 participants pressed keys as quickly as possible to indicate "word" versus "nonword" as each computer-presented stimulus appeared in random order, preceded by a warning signal and the question, "Is the following a real word?"

3. To enable valid comparisons, the present analyses excluded 16 of the 1997 pseudowords that were unlike the 1983 pseudowords.

Table 3. *Mean percentage correct lexical decisions by condition for H.M. and control participants in Study 1b*

Participant	Averaged high- and low-frequency words	High-frequency words	Low-frequency words	Pseudowords
H.M. in 1983	93	Unavailable	Unavailable	88
1983 control participants				
Average	92	Unavailable	Unavailable	94
SD	3			3
H.M. in 1997	73	96	54	85
1997 control participants				
Average	92	99	87	96
SD	3	2	7	3

Note. Data for 1983 are from Gabrieli, Cohen, and Corkin (1988). Data for 1997 are from Study 1a.

Results and Discussion

Because Gabrieli et al. (1988) reported only averaged data, Table 3 presents lexical decision performance averaged for HF and LF words in 1983 and 1997. Performance for control participants was virtually identical at ages 57 and 71, a result consistent with a wide range of data indicating no effects of normal aging on lexical decision accuracy over that age range (Laver & Burke, 1993). However, from 1983 through 1997, the deficit in H.M.'s lexical decision performance increased by only 1.7 *SD*s for pseudowords but by 6.6 *SD*s for words. These age-linked effects are shown in Figure 2 (two graphs on the left) as *SD* deficits.

Although separate 1983 scores for HF versus LF words were unavailable, H.M.'s averaged score for HF and LF words (93% correct) enabled us to calculate his lowest possible 1983 score for LF words (86%) from his highest possible 1983 score for HF words (100%). Therefore, H.M. performed at most only 4% better for HF words in 1983 (100% correct) than 1997 (96% correct), a difference of only 1.5 *SD*s relative to control participants, whereas H.M. performed at least 32% better for LF words in 1983 (86% correct) than 1997 (54% correct), a 5.4-*SD* difference relative to control participants (see the graphs in the right half of Fig. 2). These findings indicate that H.M.'s decline from 1983 to 1997 was almost completely attributable to LF words, as NST would predict.

These age-linked effects are consistent with longitudinal data on H.M.'s ability to produce HF versus LF words (MacKay & James, 1999), and shed further light on H.M.'s 1983 deficits on verbal fluency tests, the token test of language comprehension, and the reporter's test of language production (reported in Corkin, 1984). These 1983 deficits puzzled MacKay, Burke, and Stewart (1998), who argued that lexical-level processes tapped by these tests were largely intact in 1967 through 1973. However, the present data confirm that H.M. had lexical-level deficits for LF words in 1983, and resolve the puzzle as to their possible causes: an abrupt and early-onset factor (his 1953 lesion) and a progressive but mainly late-onset factor (effects of cognitive aging).

STUDY 2: H.M.'S SEMANTIC RA (1998)

In Study 2, H.M. attempted to define the 39 words and 12 pseudowords that he categorized as words in Study 1a. NST predicted that H.M. would accurately define HF words (e.g., *payment*) but not LF words (e.g., *squander*): Because of transmission deficits he acquired between ages 26 and 71, visual presentation of an LF word such as *squander* would transmit so little bottom-up priming to its meaning nodes (see Fig. 1) as to render activation-retrieval of what *squander* means impossible, a case of semantic RA. Study 2 also ex-

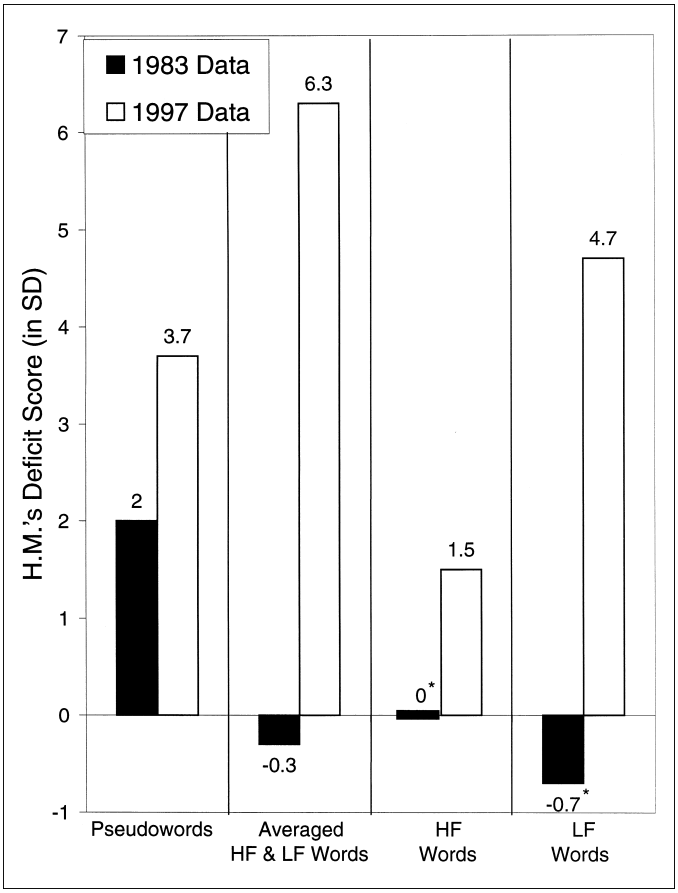

Fig. 2. H.M.'s lexical decision deficits (difference relative to control participants in standard deviations) in 1983 (Gabrieli, Cohen, & Corkin, 1988) versus 1997 (Study 1a) for pseudowords, high-frequency (HF) and low-frequency (LF) words averaged together, HF words, and LF words. Deficits for which data were unavailable (H.M.'s worst possible performance for LF words and best possible performance for HF words in 1983; see the text) are marked with asterisks.

Table 4. *Example stimuli (in italics) and typical responses of H.M. in Study 2*

High-frequency words	
1. *Forget.*	Well, to lose your memory, in a way. (correct)
2. *Noise*.	Sound, loud sound, it can be. (correct)
3. *Marine*.	And it could be, uh, a sssoldier that's a sailor at the same time, and it could be, uh, water things. (correct)
4. *Payment*.	Payment is what you make if you owe something. (correct)
5. *Worry.*	Worry is to, uh, fret over something. (correct)
Low-frequency words	
6. *Gulp*.	Gulp is, uh, when, uh, you swallow. (correct)
7. *Squander.*	And squander is, uh . . . to take things as one's own, other persons' things.
8. *Lentil*.	That's a combination word, in a way, from lent and til. [So what would that mean put together?] Well, area and time of.
9. *Primp*.	And then primp, and that could be, uh, meaning, something extraordinary that you enjoy.
10. *Chameleon*.	You think of, uh, a jewel. A red jewel. Yeah. From France.
Pseudowords	
11. *Billitow.*	Hm . . . I can't figure out what that is exactly. Amount of money that you do owe.
12. *Quintity.*	[Yeah. Do you know what that means?] No.
13. *Friendlihood.*	And friendlihood is, uh, a bunch of people liking themselves.
14. *Retrend.*	And retread, that could be the tire on a car, retread, or something that's redone. [This one's actually different.] Retrend. [Yes.] And, the way of doing something different, the way something is done different, two ways.
15. *Unmelt*.	Melt is, unmelt, rather. That is something to stay s- uh stiff or . . . not, uh, melted in any way.
16. *Reversement*.	Amusement. [Read it again.] Rearmusement is uh . . . [Read it one more time.] Reversement. That is to, take the . . . what it is, can be, should be.

Note. Correct definitions are labeled "correct"; incorrect definitions are unlabeled. Experimenter prompts appear in brackets. The full set of stimuli is available on the Web at http://www.bol.ucla.edu/~mackay/app.htm.

amined whether H.M. defines LF words and pseudowords in similar ways, which would be consistent with the hypothesis that some LF words resemble pseudowords for H.M.

Method

H.M. saw the stimuli on index cards with (approximately) the following verbal and written instructions: "Read aloud and then define these stimuli, which you said were words in an earlier study." Control participants 2, 3, 7, and 8 (see Table 1) saw these same stimuli but only read aloud and defined ones they considered words (because, unlike H.M., control participants generally refused to define nonwords in pilot studies).

Results and Discussion

Quantitative effects

Table 4 provides typical examples of H.M.'s definitions, scored as correct or incorrect. With correct-versus-incorrect scoring, H.M. defined HF words slightly more accurately than control participants (92% vs. 90%, $SD = 5\%$), but defined LF words 3.8 *SD*s less accurately than control participants (29% vs. 75%, $SD = 12\%$). With relaxed procedures that scored partially accurate definitions as correct, H.M. defined LF words 3.3 *SD*s less accurately than control participants (36% vs. 75%, $SD = 12\%$). These deficits were not due to reading errors, as H.M. performed identically for correctly read ($n = 34$) and misread ($n = 5$) stimuli, and 3% worse (39% errors) in another study 12 months later when he defined the same stimuli after the experimenter read them aloud. Rather, these deficits indicate semantic RA as predicted by NST: H.M. misdefined LF words that he almost certainly understood at age 26 because his propositional nodes for LF words were defunct as a result of sporadic and nonrecent use, coupled with aging.[4]

Qualitative effects

Incorrect definitions differed in conviction and content for H.M. versus control participants. H.M. seemed confident of his definitions, even when quite wrong, whereas control participants often expressed uncertainty or offered tentative definitions (e.g., "*Labyrinth*. It may have something to do with the bible . . . . I'm not sure what it is."). Also, H.M. produced misderivations and malaprops virtually never seen in control participants. Misderivations were definitions based on

4. A separate test on December 12, 1999, replicated the present aging effects for LF words (resembling those in Studies 1 and 2) that H.M. definitely knew and used appropriately in earlier biographical interviews (Marslen-Wilson, 1970). (Details of this test are also available at the Web site referred to in footnote 1.)

erroneous morphological analyses, as when H.M. defined *lentil*: "a combination word, in a way, from *lent* and *til* . . . . [meaning] area and time of" (incorrect because *lent* and *til* neither mean "area and time of" nor are morphological components of *lentil*). Malaprops were erroneous definitions that fit some other word better than the target word (e.g., H.M. defined *chameleon* as the phonologically similar *carnelian*: "a red jewel . . . from France").

Finally, H.M.'s definitions for pseudowords resembled his malaprops and misderivations for LF words (see Table 4). For example, much as in his *carnelian*-for-*chameleon* malaprop, H.M. equated phonological and semantic similarity when he defined the pseudoword *pediodical* as "about the same thing as *periodical*." Similarly, H.M. misapplied derivational processes appropriate for LF words such as *unbend* in defining the pseudoword *unmelt* as "something to stay . . . stiff or . . . not melted." Such parallels suggest that LF words and pseudowords were equally meaningless for H.M.

GENERAL DISCUSSION

Our main results support NST predictions. According to NST, H.M.'s definitions and lexical decisions for HF words are intact for two reasons: Hippocampal binding is unnecessary for activating HF words and their meanings, and H.M.'s connections for HF words are strong and functional because of recent and frequent use. However, H.M.'s deficits for LF words reflect transmission deficits associated with normal aging, nonrecent use, and infrequent use over his lifetime. These transmission deficits have caused H.M.'s representations for LF words to deteriorate and become defunct. Moreover, unlike memory-normal individuals, H.M. lacks the binding nodes required to efficiently establish new cortical connections for rerepresenting defunct LF words. Defunct phonological connections for LF words therefore caused H.M.'s deficits in discriminating LF words from pseudowords (Studies 1a and 1b), and defunct connections to proposition nodes representing the meanings of LF words caused H.M.'s deficits in defining LF words (Study 2; see Fig. 1). Finally, H.M. often indicated that pseudowords were words because he lacks the higher-level lexical and proposition nodes that distinguish pseudowords from LF words, and based his decisions on familiarity with the pseudowords' lower-level components, such as syllables and morphemes.

H.M.'s exaggerated age-linked declines for words but not pseudowords, and for LF words but not HF words, also support NST predictions. H.M.'s lexical decisions for pseudowords showed relatively little age-linked decline from 1983 through 1997 because the lower-level components (e.g., syllables, morphemes) that caused H.M. to identify them as words are used frequently and are therefore less prone to age-linked deterioration than higher-level components; that is, by occurring in many words, lower-level components are used more frequently than higher-level components unique to a particular word (e.g., lexical nodes). However, H.M. showed exaggerated age-linked declines for LF words because his binding deficits prevent rerepresentation of connections for LF words that have become defunct as a result of aging and infrequent and nonrecent use. Because memory-normal control participants can readily form new connections to rerepresent defunct information, their aging effects are less dramatic. NST therefore predicts continued deterioration in H.M.'s representations for LF words relative to age-matched control participants because cognitive aging is a progressive factor that H.M. cannot offset via formation of new connections.

Implications of H.M.'s Word Knowledge Deficits for Other Theoretical Frameworks

Distributed-memory theories resembling NST

Other distributed-memory theories (e.g., Carpenter & Grossberg, 1993; Grafman & Weingartner, 1996; McClelland, 1985; Metcalfe, Cottrell, & Mencl, 1992; Wickelgren, 1979) can explain hippocampal AA, but none can explain the present data without postulating interactions between aging and hippocampal AA.

Other (selected) theories of RA

Generalized damage to retrieval mechanisms is a frequently postulated cause of RA (e.g., Kopelman, Stanhope, & Kingsley, 1999) that cannot explain the selective nature of H.M.'s RA (only for LF words and only at age 71). Neither can the present data be explained by retrieval-deficit theories that propose frontal lobe damage causes RA by disrupting the organization of retrieval processes (Kopelman et al.): H.M. has no known frontal damage (Corkin et al., 1997). Under another theory, progressive anterograde deficits preceding detection and diagnosis of Alzheimer's and Korsakoff's amnesia cause RA (e.g., Albert, Butters, & Levin, 1979). However, progressive anterograde deficits cannot explain the present data because H.M.'s sudden-onset AA has remained unchanged since 1953.

Working memory capacity

That H.M.'s reduced working memory capacity caused the word-comprehension deficits reported here is unlikely because a deficit in a general factor such as working memory capacity predicts across-the-board deficits, not selective deficits for LF (but not HF) words.

Stages-of-processing framework

The present results contradict the widely accepted stages-of-processing framework that originally predicted a pure memory deficit and no language deficits for H.M. (see MacKay, Burke, & Stewart, 1998). Because comprehension and storage constitute independent and serially ordered stages in stages-of-processing theories, this framework even implicates H.M.'s word-comprehension deficits as a likely cause of his word-memory deficits. However, before discarding the many previous studies that tested H.M.'s memory using LF words, it may be worthwhile to reconsider two distributed-memory points: that no distinct dividing line separates comprehension and memory for verbal materials, and that H.M.'s binding deficits simultaneously contribute to his list-memory and word-comprehension deficits (MacKay, Stewart, & Burke, 1998).

Limitations and Caveats

One caveat concerns the scope restrictions of our theory. NST does not apply to AA caused by diffuse cortical damage, or to information acquired shortly before the amnesia-causing trauma, or to RA stemming from transient amnesic attacks (Kapur, 1993). Relations between RA and AA are also specific rather than general under NST, so that phonological RA predicts problems in acquiring new word forms, but not necessarily in acquiring new episodic and semantic representations.

Another caveat concerns H.M.'s hospitalization in the 1980s. Following this hospitalization, watching television may have come to dominate H.M.'s everyday language processing (Gabrieli et al., 1988), magnifying his deficits by reducing exposure to LF words.

One last caveat concerns relations between binding nodes and H.M.'s lesion. Within NST, thousands of binding nodes of different types are specialized for conjoining different classes of never-previously-linked units during normal comprehension, production, and acquisition of language (at all ages; MacKay, 1990). Binding nodes relevant to the present results combine phonological units into, for example, syllables, and semantic units into, for example, propositions. However, if posterior hippocampal systems contain binding nodes, we cannot be certain how many or which binding nodes have been spared in H.M. Nor can we be certain that hippocampal systems are the exclusive locus for phonological and propositional binding nodes (although Study 1a ruled out the cerebellum).

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