Research Article

DEVELOPMENTS IN LONG-TERM EXPLICIT MEMORY LATE IN THE FIRST YEAR OF LIFE: Behavioral and Electrophysiological Indices

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Abstract—*Coincident with developments in the temporal-cortical explicit memory network, long-term recall abilities are newly emergent late in the first year of human life. We recorded event-related potentials (ERPs) in 9-month-olds as an index of the integrity of the neural substrate underlying a task thought to reflect explicit memory, namely, deferred imitation. ERP measures of recognition memory 1 week after unique laboratory experiences predicted whether and how much infants recalled of the experiences 1 month later. The findings further imply that memory storage and consolidation processes are a major source of variability in long-term recall memory late in the first year of life.*

The ability to recall the past is one that most adults take for granted. Historically, infants (from the Latin *infantia* meaning "inability to speak") were thought to lack this foundational capacity, primarily because they are without language and have no means to convey their recollections (such as through verbal report; see Bauer, 1996, 2002, for discussions). With development of a nonverbal analogue to verbal report, namely, deferred imitation, researchers have found evidence of long-term recall in children well before they can talk. Indeed, it is increasingly apparent that the capacity for recall over long periods of time is emergent by the end of the first year of life. At its emergence, however, the capacity is neither reliable nor robust, as evidenced by marked variability in long-term recall among 9-month-old infants (e.g., Carver & Bauer, 1999). In the present research, we combined behavioral measures of long-term recall with electrophysiological (event-related potential, ERP) measures of brain activity to investigate the source of individual differences in long-term recall late in the first year.

Behavioral evidence of the emergence of long-term recall late in the first year is derived from 9-month-olds' deferred imitation of novel, multistep sequences. Deferred imitation involves using props to produce actions or sequences of actions that, after some delay, infants are invited to imitate. As discussed in Bauer (2002), there are excellent reasons to believe that deferred imitation measures recall memory. First, imitation after exposure to a model long has been accepted as one of the hallmarks of representational capacity (e.g., Piaget, 1952). Second, once children have the linguistic capacity to do so, they talk about events experienced in the context of imitation (e.g., Bauer, Wenner, & Kroupina, 2002). This is strong evidence that the representational format in which the memories are encoded is explicit or declarative, as opposed to implicit or nondeclarative (formats that are inaccessible to language). Third, the paradigm passes the "amnesia test": Adults with temporal lobe amnesia, in whom explicit mnemonic processes are disrupted, are unable to perform an age-appropriate version of the task (McDonough, Mandler, McKee, & Squire, 1995). This suggests that the imitation procedure taps the type of memory that gives rise to recall (see Bauer, in press, for further development of this argument). Ordered recall of multistep sequences is an especially sensitive index of developmental change in explicit memory because correctly ordered reproduction of sequences cannot be accomplished through recognition. Once demonstration of a sequence is complete, information about the order of actions is not perceptually available. To reproduce a sequence in order, temporal information must be retrieved from an event representation, in the absence of ongoing perceptual support. In this requirement, the task is analogous to verbal report (Mandler, 1990).

Among children in the second year of life, recall of the order of events after a delay is reliably observed: Seventy-eight to 100% of 13- to 20-month-olds evidence ordered recall after a 1-month delay; by 20 months of age, almost 70% of children evidence ordered recall after delays as long as 12 months (Bauer, Wenner, Dropik, & Wewerka, 2000). In contrast, although as a group 9-month-olds recall the individual actions of multistep sequences, no more than 50% recall sequences in the correct order after 1 month (Bauer, Wiebe, Waters, & Bangston, 2001; Carver & Bauer, 1999). Even in the 50% of 9-month-olds who evidence long-term ordered recall, the ability is not robust, but depends on multiple experiences of events: With fewer than three exposures, a maximum of only 21% of 9-month-olds evidence ordered recall after 1 month.

That pronounced individual differences in the reliability and robustness of long-term ordered recall are seen at 9 months of age is consistent with the time frame of neurobehavioral development. Evidence from developmental neurobiology and cognitive neuroscience converges to suggest that the temporal-cortical network that supports long-term recall begins to reach functional maturity in the latter half of the first year (for reviews, see Carver & Bauer, 2001, and Nelson & Webb, 2002). With brain development should come changes in overt behavior. Moreover, individual differences in the expression of new behavior should be linked with individual differences in underlying neural processes. To test for such relations, we (Carver, Bauer, & Nelson, 2000) conducted a within-subjects examination of ERP and behavioral indices of long-term recognition and long-term recall (respectively) in 9-month-old infants. We found that infants who showed evidence of recognition after 1 week (i.e., differential ERP responses to pictures of previously experienced, or "old," sequences and never-before-experienced, or "new," sequences) also recalled sequences in order after 1 month. In contrast, infants who did not recognize previously experienced sequences after 1 week did not recall temporal order after 1 month. The data thus indicate brain-behavior linkages. They are silent, however, as to the source of individual dif-

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ferences: Did infants who later failed the long-term recognition and recall tests encode the sequences but then forget them before the 1-week recognition test (suggesting storage failure), or did they never encode the sequences (suggesting encoding failure)?

In the present research, by again combining behavioral and ERP indices, we addressed the question of why some 9-month-olds evidence long-term recognition and ordered recall and others do not. We exposed 9-month-old infants to novel two-step sequences. Immediately after exposure, we tested recognition memory using ERPs. One week later, we tested delayed recognition using the same procedure. The immediate and 1-week delayed recognition tests permit evaluation of encoding and storage of event-memory traces, respectively. One month after the second ERP, we tested infants' recall. In addition, we tested relearning of the sequences, as a means of examining the role of retrieval processes.

We expected that if encoding processes are a source of individual differences, then memory failure would be evident as early as the immediate recognition test. Savings in relearning would not be expected because, in effect, no learning occurred. In contrast, if storage processes are a source of individual differences, then infants who did not recall would show evidence of immediate recognition, but failure in the 1-week delayed recognition and 1-month delayed recall tests. Little savings in relearning would be expected because, if memory failure is due to dissolution of the mnemonic trace, there would be little residual on which to build in relearning. Finally, if retrieval processes are a source of individual differences, then infants who evidenced long-term memory failure nevertheless would show robust savings in relearning: With the retrieval burden lessened, evidence of the preserved memory trace would be apparent.

METHOD

Participants

The participants were 57 full-term, normally developing infants with a mean age of 9 months 15 days (n = 29 girls and 28 boys). The participants were treated in accord with the American Psychological Association's guidelines for ethical treatment of human participants. Parents provided informed consent for their infants to participate. All procedures were reviewed and approved in advance by the Institutional Review Board of the University of Minnesota.

Materials

Stimuli for the deferred-imitation task were three novel two-step sequences (see Fig. 1 for an example; additional examples are available from the authors). Stimuli for the recognition tests were digitized pictures depicting Steps 1 and 2 and the end states of the sequences. We tested for possible non-memory-related differences in the sample at the time of enrollment, by having parents complete the MacArthur Communicative Development Inventory for Infants (MCI-I; Fenson et al., 1994), as a measure of language development, and the Infant Behavior Questionnaire (IBQ; Rothbart, 1981), as a measure of temperament characteristics.

Procedure, Data Reduction, and Scoring

Exposure sessions

In a laboratory context, we presented the infants with three novel two-step sequences at each of three sessions (see Table 1; sessions were 1 1/2 days apart; range: 24–96 hr). At Session 1, for each sequence in turn, the infants were first given the props for the sequence so that we had an infant-controlled baseline to assess spontaneous production of target actions. The experimenter then demonstrated the sequence two times, with narration. The infants were not permitted to imitate. At Sessions 2 and 3, the experimenter again demonstrated each sequence two times, for a total of six demonstrations. Again, no imitation was permitted. Rather, imitation was deferred for 1 month, at which time it was used as the measure of delayed recall.

Recognition memory test sessions

At the end of Session 3, infants received their first recognition test using ERPs; the second ERP test, at Session 4, was 1 week later (M = 7 days; range: 5–10 days). The stimuli for the recognition tests were digitized pictures depicting the steps and end states of one old sequence (i.e., a sequence to which the infant had been exposed) and one new sequence (i.e., a sequence to which the infant had not been exposed). Different old and new sequences were used at each test (see Table 1). The immediate and 1-week delayed recognition tests permitted evaluation of encoding and storage of event-memory traces, respectively.

ERPs were recorded from 10 scalp locations (Oz, Pz, Cz, Fz, T3, T4, T5, T6, C3, and C4), according to the International 10-20 system (Jasper, 1958). Impedances were less than 10 KΩ. Scalp activity was recorded with Cz as a reference, then rereferenced off-line to linked ears; a ground electrode was placed on the forehead. Electro-ocular (EOG) activity was recorded from bipolar miniature electrodes above and below one eye. Electrical signals were recorded using a Grass Neurodata Acquisition System with Model 12A5 amplifiers (Grass Instruments, Braintree, MA). Electroencephalogram (EEG) and EOG gains were set to 20,000 and 5,000, respectively. The bandpass filters were set at 0.1 and 30 Hz. A 60-Hz notch filter was in place.

Test trials consisted of a 100-ms baseline, followed by presentation of the stimulus for 500 ms (i.e., a photograph of a step or the end state of a sequence appeared for 500 ms); recording continued for 1,200 ms thereafter. Pictures from one old (n = 30 trials) and one new (n = 30 trials) sequence were interspersed. EEG was sampled every 10 ms (100 Hz). The intertrial interval varied randomly between 500 and 1,200 ms.

Data were edited off-line for eyeblink artifacts and then corrected for the influence of eye movement on the EEG (de Haan & Nelson, 1997; Gratton, Coles, & Donchin, 1983). Individual averages and grand means were created for infants with at least 10 artifact-free trials. The average included an equal number of trials for each condition (old sequences, new sequences) for each participant (for infants who recalled temporal order, 14.88 and 16.86 trials at immediate and delayed testing, respectively; for infants who did not recall temporal order, 16.17 and 16.46 trials at immediate and delayed testing, respectively). The data from 20 infants were excluded because the EEG or EOG signals (or both) from both sessions exceeded analog-to-digital limits (i.e., an excessive number of trials contained physiological artifacts). The data from 2 other infants were excluded because the EEG or EOG limits were exceeded at Session 1 and at Session 2 (a) the infant failed to complete the required minimum number of trials (1 infant) or (b) the equipment failed (1 infant). Of the 35 remaining infants, 7 contributed data to the immediate test only, 15 contributed data to the delayed test only, and 13 contributed data to both tests. Reasons for excluding ERP data for one or the other session for these infants included EEG or EOG signals that exceeded limits (21 infants) and failure to complete the required minimum number of trials (1 infant).

Fig. 1. Example two-step sequence: Turn on the light. To reproduce the sequence, infants had to first put a toy car down a vertical compartment of an L-shaped apparatus and then push a rod into the horizontal compartment, thereby causing the car to roll to the end and turn on a light. Note that infants could push the rod before putting the car into the vertical compartment. However, doing so would not cause the light to illuminate.

The groups of infants who contributed no useable ERP data, data to the immediate ERP only, data to the delayed ERP only, and data to both ERPs were drawn from the same population. They did not differ in (a) age at enrollment, (b) vocabulary production or comprehension (as measured by the MCI-I), (c) temperament characteristics (as measured by the IBQ), (d) the delay between ERP tests, (e) the delay between the second ERP test and the recall test, or (f) production of individual target actions or pairs of actions in the target order at delayed recall.

On the basis of prior related research, we focused on the middle-latency (Nc) component of the ERP waveform, which is associated with attentional processes and recognition memory (de Haan & Nelson, 1997; Nelson & Dukette, 1998; Richards, in press). By about the age of 1 year, the latency of the Nc component is approximately 500 ms (Nelson, 1994). Accordingly, following established procedures (de Haan & Nelson, 1997), we defined the middle-latency component as activity between 260 and 870 ms. We calculated (a) the minimum amplitude of the waveform, defined as the greatest deflection from baseline (in microvolts), and (b) latency to Nc, defined as the time, relative to stimulus onset, at which the minimum amplitude occurred.

Recall memory test session

Infants' recall and relearning were tested 1 month after the second ERP (M = 28 days; range: 23–37 days). To test recall, the experimenter gave the infants the props for each of six sequences in turn (order was counterbalanced): three old, previously experienced sequences and three new, control sequences. For each sequence, there was an infant-controlled period in which to manipulate the props. Performance on the old sequences provided the measure of delayed recall; performance on the new sequences served as a within-subjects control. The experimenter then demonstrated each sequence once, and allowed the infant to imitate. Performance after the demonstration provided a measure of savings in relearning (i.e., better performance on old relative to new sequences indicates savings).

Sessions were videotaped for later analysis. Agreement between two independent coders was 94% (range: 86–100%, calculated on 26% of the sample). Following established procedures (e.g., Bauer et al., 2000), for both delayed recall and relearning of each sequence, we calculated the number of individual target actions produced (maxi-

Table 1. *Schematic representation of the testing protocol*

	Protocol phase			
Session	Baseline assessment	Modeling	Recognition memory test	Recall memory test
1	Events A, B, C	Events A, B, C	—	—
2	—	Events A, B, C	—	—
3	—	Events A, B, C	Events A, D	—
4	—	—	Events B, E	—
5	—	—	—	Events A, B, C, D, E, F

Note. Unique alpha characters designate unique sequences. In this schematic, Events A, B, and C are "old" (i.e., sequences to which the infant was exposed prior to the delay); Events D, E, and F are "new" (i.e., sequences the infant did not see at any of the exposure sessions). At each recognition test, one old event was paired with one new event. Across infants, each sequence was used as old and new approximately equally often.

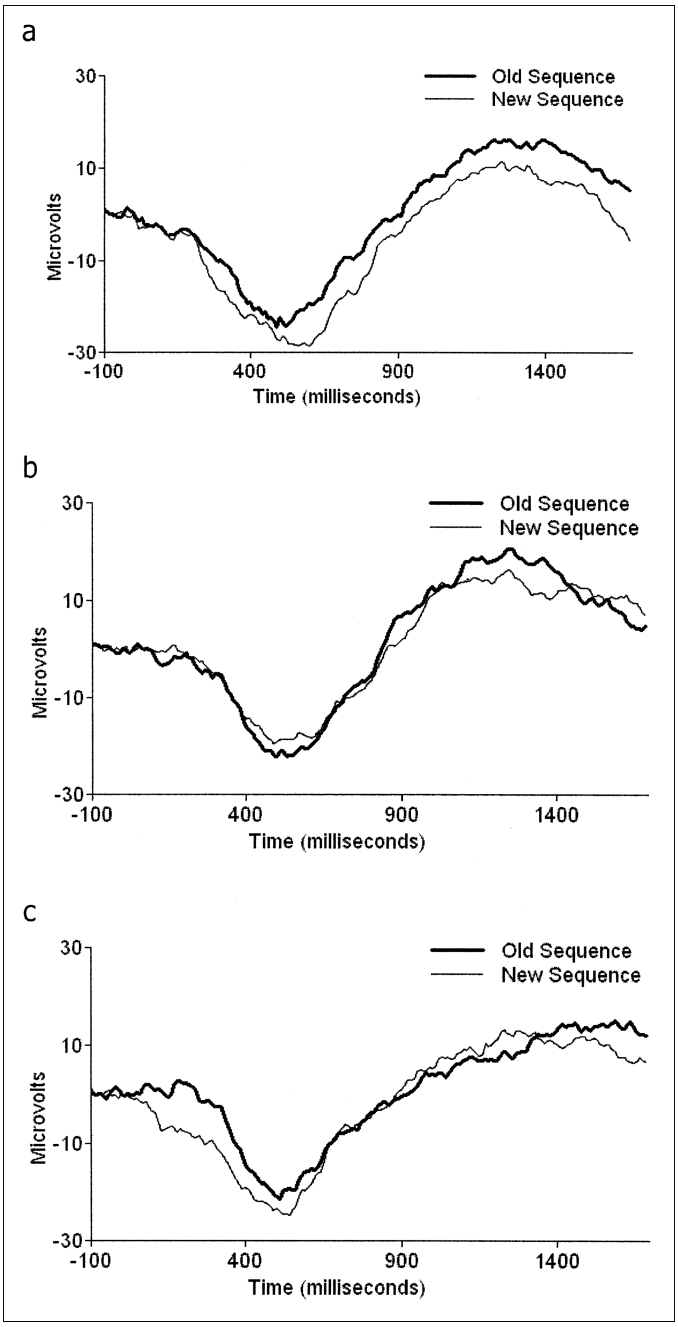

Fig. 2. Event-related potential (ERP) waveforms at electrode site Fz at the immediate ERP (a), at the 1-week delayed ERP for infants who later failed to show ordered recall (b), and at the 1-week delayed ERP for infants who later evidenced ordered recall (c).

mum = 2.0 per sequence) and the number of pairs of actions produced in the target order (maximum = 1.0 per sequence).

RESULTS

Of the 57 infants, 26 (46%) reproduced at least one sequence in the correct temporal order after 1 month (i.e., on one or more of the old sequences, they produced Steps 1 and 2, in correct order); 31 (54%) of the infants did not. This distribution is a replication of the results in prior research (Bauer et al., 2001; Carver & Bauer, 1999). Because whether infants evidenced ordered recall is the outcome of interest, in subsequent analyses, recall status (i.e., evidenced ordered recall vs. did not evidence ordered recall) was used as a grouping variable.

Among infants who reproduced at least one full sequence in correct temporal order, performance was better on old sequences than on new sequences, thereby indicating recall, $F(1, 25) = 20.05$, $p < .0001$, for individual target actions (Ms = 1.28 and 0.94, SDs = 0.69 and 0.62, for old and new, respectively) and $F(1, 25) = 27.00$, $p < .0001$, for ordered pairs of actions (Ms = 0.36 and 0.14, SDs = 0.48 and 0.34, for old and new, respectively). In contrast, among infants who did not recall at least one ordered sequence, production of individual target actions did not differ for old and new sequences (Ms = 0.73 and 0.68, SDs = 0.57 and 0.49, respectively). By definition, this group produced 0 ordered pairs of actions.

Given previous research in which memory-related ERP components were centrally and frontally distributed (Carver et al., 2000), we examined the midline leads Fz and Cz for evidence of immediate and delayed recognition. Analysis of the immediate ERP data indicated that both groups of infants (i.e., ordered-recall group, $n = 8$, vs. no-ordered-recall group, $n = 12$) encoded the events. There was a main effect of condition, indicating greater negative amplitude of the middle-latency (Nc) component to pictures of the new sequences than to pictures of the old sequences, $F(1, 18) = 4.40$, $p = .05$ (see Fig. 2a). There was no suggestion of an effect of group. That both groups recognized the events immediately after exposure suggests that encoding processes are not the source of individual differences in 9-month-olds' long-term recall.

At the 1-week delayed recognition test, amplitude of the Nc did not differ as a function of whether the infants were viewing pictures from new or old sequences. However, analysis of the latency of the Nc revealed a significant Condition × Group interaction, $F(1, 26) = 5.65$, $p < .03$. For infants who did not recall the order of any of the sequences ($n = 13$), there was no effect of condition ($p > .35$; see Fig. 2b and Fig. 3). Thus, there was no evidence of discrimination. In light of apparently successful encoding of the stimuli (as reflected in the immediate ERP), lack of evidence of long-term recognition in these infants implies storage failure.

For infants who demonstrated ordered recall 1 month later ($n = 15$), latency to peak amplitude was shorter for new sequences than for old sequences, $F(1, 14) = 6.61$, $p < .03$ (see Fig. 2c and Fig. 3).[1] We interpret the longer latency to peak amplitude for the old sequences as indicative of reintegration processes associated with long-term recognition (Brainerd, Reyna, Howe, & Kingma, 1990). The results illustrated in Figure 4a are consistent with this suggestion: For infants who demonstrated ordered recall, latency to peak amplitude was longer at the delayed than at the immediate recognition test.[2] This effect was not

1. The effect is differentially apparent in Figures 2 and 3 because of differences in the way the values used to create the figures were calculated. The waveforms in Figure 2 are for descriptive purposes. They were created by averaging amplitude across infants for each 10-ms sampling. The values entered into the statistical analyses were the averages of individual infants' minimum amplitudes within the time window. Figure 3 reflects the latency to minimum amplitude for each group of infants.

2. The effect could not be evaluated statistically because there was no appropriate error term, given that some observations were between- and some within-subjects. However, for infants who evidenced ordered recall, the error bars for the latency to peak Nc at immediate and delayed testing did not overlap; this suggests that if the effect had been analyzed, it would have been statistically reliable.

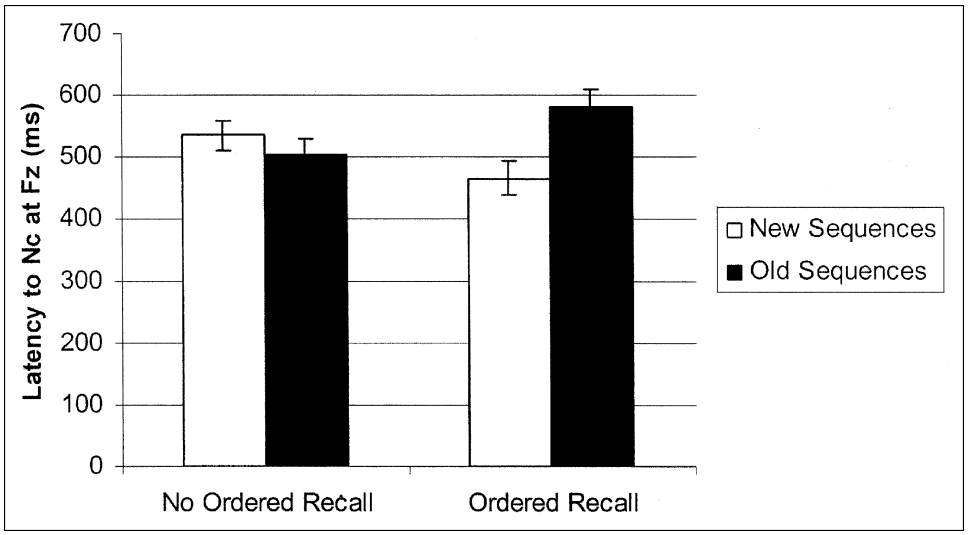

Fig. 3. Latencies to peak Nc at electrode site Fz at the 1-week delayed recognition test. The graph shows latencies in response to pictures from new and old sequences for infants who did not demonstrate ordered recall after 1 month (left; difference not significant) and infants who demonstrated ordered recall after 1 month (right; difference significant, $p < .03$).

observed for infants who did not evidence long-term ordered recall, or for new sequences for either group (Fig. 4b). Across groups, the size of the difference in latency to peak amplitude in response to pictures from old and new sequences predicted the number of sequences recalled in order after 1 month: The sizes of the differences at Fz and Cz predicted 28% and 21% of the variance in level of long-term ordered recall, respectively (rs = .53 and .46, ps $< .02$). The correlations between the difference scores and the infants' performance on new, control sequences were not reliable (ps $> .10$). Thus, ERP responses after 1 week did not predict general performance on the imitation task. Rather, they predicted infants' levels of long-term ordered recall.

Across groups, there was evidence of savings in relearning (i.e., better performance on old than new sequences), $F(1, 55) = 5.14$, $p < .03$, and $F(1, 55) = 3.91$, $p = .05$, for individual target actions and ordered pairs of actions, respectively. However, there were main effects of group, Fs(1, 55) = 9.18 and 6.87, ps $< .02$, for individual target actions and ordered pairs of actions, respectively, indicating that the savings effect was larger for infants who evidenced ordered recall than for infants who did not. That savings in relearning was observed even among infants who did not evidence ordered recall implies that storage failure was not complete and thus that retrieval processes make some contribution to individual differences in 9-month-olds' long-term ordered recall. The smaller size of the savings effect among infants who did not evidence long-term ordered recall implies that the memory traces that were available to them for retrieval were less well preserved than the memory traces of their peers who did evidence long-term ordered recall.

DISCUSSION

The present research is consistent with prior investigations indicating substantial individual variability in long-term recall at 9 months of age (Bauer et al., 2001; Carver & Bauer, 1999). It also represents a replication of prior research in which recognition after 1 week, as evidenced by ERP responses, was associated with recall after 1 month (Carver et al., 2000). Although the effect was observed in the amplitude of the Nc in our previous study and in the latency of the component in the present study, both measures are indicative of utilization of cognitive processing resources (Nelson & Monk, 2001), and thus, the findings from the two studies can be considered converging.

The present research also extends the literature in three substantial and significant ways. First, it is informative regarding the locus of mnemonic failure in the subgroup of 9-month-olds who do not demonstrate ordered recall. These infants encoded the sequences (as evidenced by their immediate ERP responses), but then apparently failed to effectively consolidate them for long-term storage (as evidenced by their 1-week delayed ERP and 1-month delayed recall responses); even after reexposure, the memory traces of these infants were not as robust as those of their peers who evidenced long-term ordered recall. Although this conclusion is by necessity drawn across paradigms (recognition and recall), the pattern is compelling nonetheless. Second, the present research is the first in which the recall memory of individual infants has been predicted by their recognition memory 1 month earlier. That is, on the basis of ERP responses 1 week after exposure to test sequences, we predicted the number of sequences successfully recalled 1 month later. This finding is especially noteworthy given that we obtained ERP responses to still photographs of individual, unordered sequence steps and the predicted outcome was successful deferred imitation of ordered sequences using three-dimensional props. The relation between ERP responses after 1 week and ordered recall 1 month later may mean that it is possible to identify an ERP "signature" that indexes the integrity of 9-month-old infants' long-term recall memory systems.

Third, the present research supports specific predictions about neural developments that contribute to developmental change in long-term recall memory late in the first year of life. The findings suggest that integration and consolidation processes are a major source of developmental change. Integration and consolidation processes are presumed

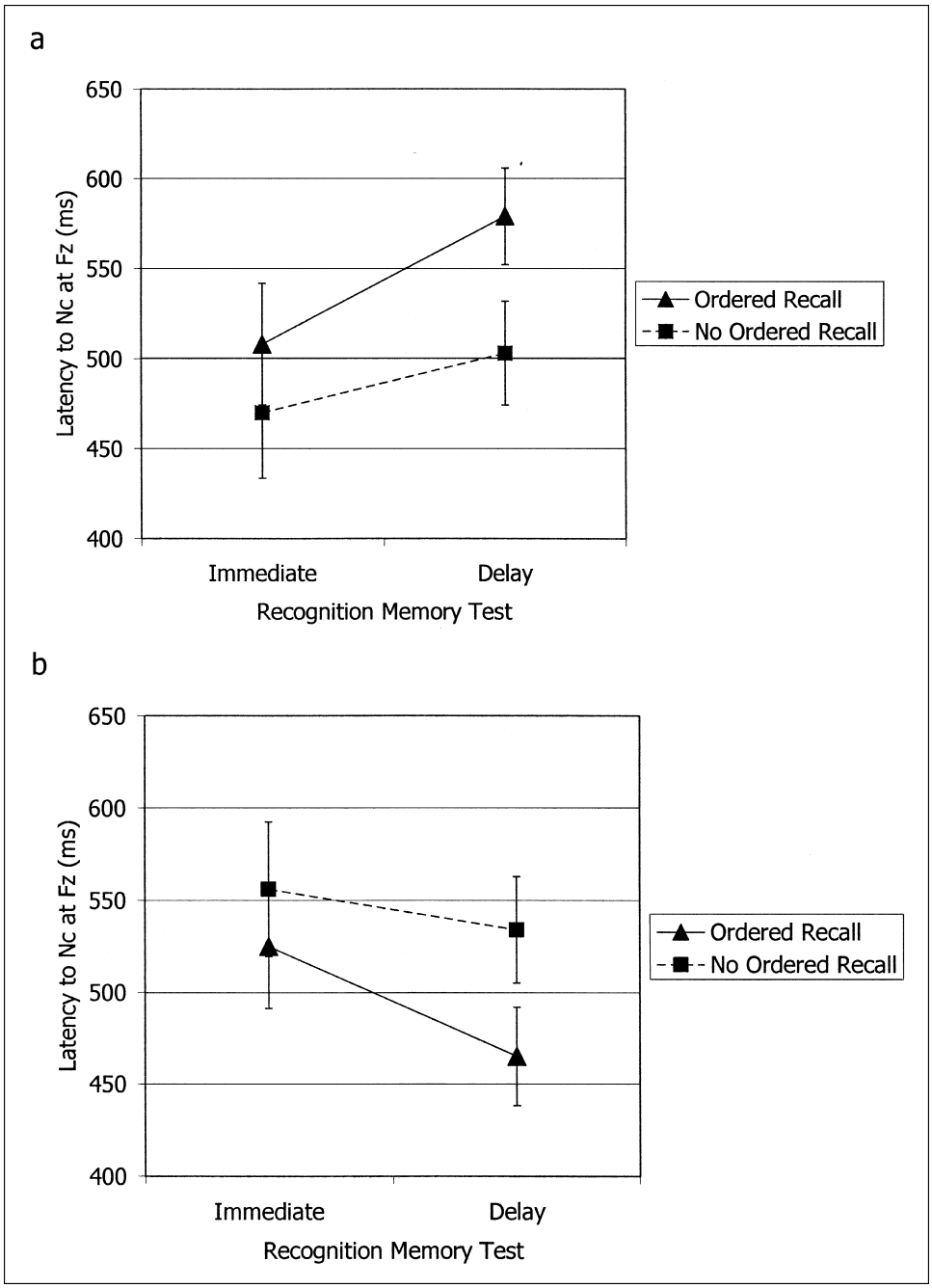

Fig. 4. Latencies to peak Nc at electrode site Fz. The graph shows latencies in response to pictures from old sequences (a) and new sequences (b) at immediate and delayed recognition testing, separately for infants who did and did not demonstrate ordered recall after 1 month.

to be carried out by the hippocampus (e.g., Squire, Knowlton, & Musen, 1993). It is thought that inputs from multiple neocortical association areas converge on parahippocampal structures (e.g., entorhinal cortex), where, without further processing, they are maintained temporarily and only as isolated elements (e.g., Eichenbaum & Cohen, 2001). To be consolidated for long-term storage, information must pass from the entorhinal cortex to the hippocampal formation. Although there are direct projections from entorhinal cortex to the CA3 region of the hippocampus, in the adult organism the major pathway is through the dentate gyrus. The dentate gyrus is hypothesized to be especially important to memory for temporal-order information (Lisman, 1999). This analysis implies that the developmental status of the dentate gyrus may place a lower limit on the capacity for long-term explicit memory in general (see Nelson, 1995) and temporally ordered recall in particular (see Bauer, Burch, & Kleinknecht, 2002). Consistent with this suggestion, the time course of development of the den-

tate gyrus of the hippocampus coincides with the time course of age-related changes in long-term recall: Whereas much of the hippocampal formation matures early, the dentate gyrus undergoes a more protracted course of development with significant changes taking place near the end of the first year (Serres, 2001).

In conclusion, in this research we combined behavioral and ERP measures to examine developments in long-term explicit memory late in the first year of life. The behavioral measures revealed individual differences in long-term recall among 9-month-olds. The ERP measures suggest that a significant source of the individual differences is the processes by which memories are consolidated for long-term storage and subsequent retrieval. Indeed, electrophysiological indices of the strength of the memory trace after 1 week predicted the level of success of ordered recall 1 month later. We suggest that the pattern of findings can be accounted for by a neuro-developmental model that implicates in particular relatively late developments in the dentate gyrus of the hippocampus.

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