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Electrophysiological Indexes of Encoding and Behavioral Indexes of Recall: Examining Relations and Developmental Change Late in the First Year of Life

Patricia J. Bauer and Sandra A. Wiebe
Institute of Child Development
University of Minnesota

Leslie J. Carver
Department of Psychology
University of California–San Diego

Angela F. Lukowski, Jennifer C. Haight, and Jennie M. Waters
Institute of Child Development
University of Minnesota

Charles A. Nelson
Institute of Child Development and Department of Pediatrics
University of Minnesota

Long-term memory undergoes pronounced development in the latter part of the 1st year. This reasearch combines electrophysiological (event-related potential [ERP]) and behavioral (deferred imitation) measures of encoding and recall, respectively, in an examination of age-related changes in and relations between encoding and recall during this time. In a short-term longitudinal study, infants were exposed to different multistep sequences at 9 and at 10 months. In both phases, they were tested for immediate recognition of the events via ERPs (as an index of encoding), and for recall of

Correspondence should be addressed to Patricia J. Bauer, Department of Psychological and Brain Sciences, Duke University, 9 Flowers Drive, Box 90086, Durham, NC 27708–0086. E-mail: patricia.bauer@duke.edu

them 1 month later. At both ages, infants encoded the events; encoding was more robust at 10 months than at 9 months. After the 1-month delay, infants failed to recall the events experienced at 9 months, but evidenced recall of the events experienced at 10 months. In spite of developmental differences in encoding and recall over this period, indexes of encoding at 9 months were correlated with measures of recall of events experienced at 10 months and tested 1 month later.

Traditionally, the capacity for recall of past events was thought to develop relatively late. The assumption held sway largely because infants are unable to participate in the paradigm of choice for memory researchers, namely, verbal report (see Bauer, 2002b, 2002c, for reviews and discussions). In contrast to this perspective, it now is apparent that long before they have the verbal ability to describe the events of their lives, children are able to recall them: Nonverbal measures reveal that by the end of the second year, long-term recall is both reliable and robust (Bauer, 2002a). Indeed, there is increasing evidence that the ability to recall over the long term emerges in the second half of the first year of life, with pronounced developmental changes between 9 and 12 months of age (e.g., Carver & Bauer, 2001). Conceptually, the changes are linked with developments in the neural substrate implicated in encoding, consolidation, storage, and retrieval of declarative memories (e.g., Bauer, 2002c, 2006; Carver & Bauer, 2001; Nelson, 1995, 1997, 2000; Nelson & Webb, 2002). In this research, we combined electrophysiological measures of encoding and behavioral measures of recall in an examination of relations between encoding and recall and of developments therein during this important period of time.

Because infants are unable to provide verbal reports of past events, researchers interested in early developments in declarative memory rely on nonverbal measures. An increasingly common means of assessing developments in long-term recall is via elicited or deferred imitation. In imitation-based tasks, props are used to produce a specific action or sequence of actions that immediately (elicited imitation), after some delay (deferred imitation), or both, infants are permitted to imitate. Arguments that imitation-based techniques provide a nonverbal analogue to verbal report have been developed in detail elsewhere (e.g., Bauer, 1995, 1996, 2002b, 2005b, 2006; Carver & Bauer, 2001; Mandler, 1990; Meltzoff, 1990). Here we provide a brief summary of four of the points in the argument. First, deferred imitation of a model long has been viewed 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 not accessible to language). Third, the paradigm passes the "amnesia test." That is, adults suffering from temporal lobe amnesia 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.

Finally, there is reason to argue that deferred imitation taps recall, rather than recognition. The available props do provide perceptual support for performance. However, all recall is cued, either by an external prompt or by an internal association (Spear, 1978). Moreover, numerous control conditions make clear that the modeled actions and sequences are not discovered by the infants and children through trial and error or problem solving, and that the objects themselves do not suggest or afford the actions to be performed with them (e.g., Bauer, 1992; Meltzoff, 1988, 1995). In the case of multistep sequences, additional evidence that recall processes support performance comes from temporally ordered reproduction of modeled sequences. Once an event sequence is modeled, no perceptual support for the order in which the actions are to be performed remains. To reproduce an ordered sequence, temporal order information must be encoded during presentation of the event and subsequently retrieved from the memory representation, in the absence of ongoing perceptual support. In this, the requirements of imitation-based tasks closely mimic those of verbal recall paradigms (Mandler, 1990).

Using elicited and deferred imitation, researchers have begun to chart the development of long-term recall memory in infancy and very early childhood. As summarized in Bauer (2002a), over the period of 6 months to 20 months, the capacity for recall changes from one that is fragile and temporally limited to one that is reliable and temporally extended. Specifically, at 6 months of age, infants remember single actions after delays of 24 hr (Barr, Dowden, & Hayne, 1996; Collie & Hayne, 1999). No more than 25% of 6-month-olds evidence ordered recall after 24 hr and those who do require multiple experiences of the sequences prior to imposition of the delay (Barr et al., 1996). In contrast, by 20 months of age, almost 70% of children evidence ordered recall of multistep sequences after delays of as long as 12 months (Bauer, Wenner, Dropik, & Wewerka, 2000). Whereas throughout the first 2 years of life, development is gradual and continuous, the period of 9 to 12 months is one of especially marked change in recall ability. For example, infants 9 months of age are able to recall multistep sequences after 1 month, but not after 3 months. Infants only 1 month older are able to recall events after delays as long as 3 months (Carver & Bauer, 2001). In addition, 9-month-olds seem to be dependent on multiple experiences of events to recall them after 1 month (Bauer, Wiebe, Waters, & Bangston, 2001). In contrast, infants aged 11 months (Mandler & McDonough, 1995) and 13 months (Bauer & Hertsgaard, 1993) require only a single experience to support long-term recall.

That developmental change might be especially prominent in the latter part of the first year of life is consistent with the time course of development of the neural substrate that subserves long-term declarative memory. In brief, declarative memory is thought to be dependent on a multicomponent network of neural structures (e.g., Eichenbaum & Cohen, 2001; Markowitsch, 2000; Squire, 1992; Squire,

Knowlton, & Musen, 1993). Formation of a long-term declarative memory trace begins as inputs from multiple neocortical association areas converge on parahippocampal structures (e.g., entorhinal cortex), which serve as the temporary storage sites for the elements of experience. The organizational processing that binds the elements into an integrated memory trace takes place in the hippocampus. The process of organization and consolidation involves neurochemical and neuroanatomical changes that occur over the course of hours to months, during which the memory representation is distributed to or duplicated in cortical areas that are the long-term repositories for declarative memories. Memory traces are vulnerable throughout the process of consolidation: Newly acquired material is lost if consolidation processes are interrupted. Finally, behavioral and neuroimaging evidence implicates prefrontal cortex in retrieval of memories from long-term stores.

Although some components of the temporal–cortical declarative memory network develop early, others undergo a protracted course of postnatal development, with pronounced changes late in the first year and throughout the second year of life. Specifically, as reviewed by Serres (2001), there are a number of indicators that most (but not all) of the medial temporal lobe components of the declarative memory system mature early. However, the balance of the elements in the network, as well as some components of the hippocampal formation itself, develops more slowly. Specifically, the dentate gyrus of the hippocampus (a critical link in the trisynaptic circuit that connects parahippocampal structures to the CA3 and CA1 regions of the hippocampus), the frontal cortex, and the reciprocal connections between the hippocampus and the neocortex all undergo a protracted developmental course (e.g., Bachevalier, Brickson, & Hagger, 1993; Bachevalier & Mishkin, 1994; Benes, 2001; Bourgeois, 2001; Serres, 2001). Indeed, in the dentate gyrus, neurogenesis continues into adulthood (Tanapat, Hastings, & Gould, 2001). The best available evidence suggests that in the human infant, the entire network reaches functional maturity (although certainly not full maturity) late in the first year and over the course of the second year of life, with continued, albeit less dramatic, development for years thereafter (Bauer, 2006; Carver & Bauer, 2001; Nelson, 1995, 1997, 2000; Nelson & Webb, 2002; see Schacter & Moscovitch, 1984, for a similar analysis). This time course coincides with the pronounced behavioral changes observed in the latter part of the first year of life.

Determining the fit between the timing of neural developments and the timing of behavioral developments is an important step in construction of a neurocognitive model of declarative memory development. Continued progress requires that more specific links be forged, however. In other words, needed are more detailed models of how developmental changes in the underlying substrate for memory relate to changes in the reliability and robustness of recall. The

combination of behavioral and brain imaging techniques has allowed us to begin precisely this type of elaboration for infants in the latter part of the first year of life. For example, in Bauer, Wiebe, Carver, Waters, and Nelson (2003), we investigated whether changes in storage processes (assessed via event-related potentials [ERPs]) that would be expected as a consequence of developments in the medial temporal structures involved in consolidation of memory traces might account for individual differences observed in 9-month-olds' ordered recall (assessed via deferred imitation). Specifically, in two independent samples, we had observed that only roughly 50% of 9-month-olds evidence ordered recall after 1 month (Bauer et al., 2001; Carver & Bauer, 1999). What was not clear was whether the roughly 50% of infants who did not recall the events failed to encode them, or whether they successfully encoded the events, but then experienced storage failure over the delay.

To address this question, in Bauer et al. (2003), we examined 9-month-old infants' immediate recognition (via ERPs, as an index of encoding), delayed recognition (via ERPs, as an index of retention), and behavioral recall. We found that, as evidenced by ERP indexes of recognition memory obtained immediately after exposure to event sequences, the infants successfully encoded the to-be-remembered events. However, 1 week later, as measured by the delayed recognition ERP test, half of the infants had experienced storage failure and no longer evidenced recognition memory. The same infants failed to recall the events 1 month later. Thus, the combination of ERP and behavioral measures permitted implication of storage processes as a source of individual differences in early recall memory development (see also Carver, Bauer, & Nelson, 2000). Behavioral research in which the variance associated with age-related differences in encoding processes was controlled implicates storage processes as a continued source of developmental change in long-term recall throughout the second year of life (e.g., Bauer, 2005a; Howe & Courage, 1997).

By providing a link between developmental changes in the substrate that subserves consolidation and storage processes and age-related decreases in susceptibility to storage failure, the results of Bauer et al. (2003) bring us a step closer to understanding how developmental changes in brain and behavior are related. It is unlikely that changes in storage processes account for all of the variance, however: There also are reasons to speculate that developments in encoding processes contribute to age-related changes in declarative memory late in the first year of life. In the initial stages of encoding, information about a stimulus object or event is maintained in cortical association areas. These areas are implicated by findings that neurons in prefrontal cortex, for example, fire over periods of short delay, and that humans and monkeys with lesions in association areas exhibit deficits in short-term memory (see, for example, Eichenbaum & Cohen, 2001; Fuster, 1997; Markowitsch, 2000, for reviews). The association cortices in general, and pre-

frontal cortex in particular, undergo considerable postnatal development. On the behavior side, there are changes in encoding processes over the course of the first year of life. For example, the number of seconds required to encode a stimulus (as evidenced by the amount of familiarization required to produce a novelty preference) decreases between 3.5 and 6.5 months of age (e.g., Rose, Gottfried, Melloy-Carminar, & Bridger, 1982). We already have seen that relative to 11- and 13-month-olds, 9-month-olds require more opportunities for encoding of event sequences to recall them after a delay (Bauer et al., 2001). Findings such as these implicate encoding processes as a potential source of developmental change in long-term recall late in the first year of life.

In this research, we used ERPs to examine immediate recognition (as a measure of encoding) and deferred imitation (as a measure of recall) to examine long-term declarative memory in infants late in the first year of life. Infants were enrolled in the short-term longitudinal study at 9 months of age, at which time they were exposed to two-step event sequences. Infants' encoding of the sequences was assessed via an immediate ERP test; recall of the sequences was tested 1 month later when the infants were 10 months of age. Within days of the 1-month recall test, the infants were exposed to different two-step event sequences. Infants' encoding of the second set of sequences was tested via an immediate ERP test; recall was tested 1 month later. By comparing encoding and recall of events experienced at 9 months and at 10 months, we were able to evaluate possible age-related differences in encoding and in recall, as well as examine their relation to one another. By investigating the questions in the same sample of infants, we were able to determine whether variability in early encoding processes was related to variability in later recall processes. The 1-month delay between phases of the study was selected both to permit comparison with previous related research (Bauer et al., 2003; Carver et al., 2000) and to ensure sampling during the period of rapid development in declarative memory late in the first year of life.

As in Carver and Bauer (2001), we exposed the infants to novel two-step event sequences at each of two sessions. Immediately after the second exposure, we used ERPs to test infants' recognition of one of the event sequences. As in Bauer et al. (2003) and Carver et al. (2000), recognition would be evidenced by differential ERP responses to photographs depicting events to which the infants had been exposed (i.e., old events) and to photographs depicting events to which the infants had not been exposed (i.e., new events). We used only two exposure sessions, rather than three (as in Bauer et al., 2003), to increase the variability of encoding. Based on Bauer et al. (2003), it seems that after three exposures to events, even 9-month-olds have encoded them. We reasoned that less experience would lead to more variability in initial encoding of the events, thereby permitting examination of relations between variability in encoding and in subsequent recall.

METHOD

Participants

Thirty-one infants participated in the short-term longitudinal study. The average age of the infants at the time of encoding of the first set of test events (Phase 1 events) was 9 months, 14 days (range = 9;02–9;20); their average age at the time of the first recall test was 10 months, 22 days (range = 10;13–11;00). Infants' average age at the time of encoding of the second set of test events (Phase 2 events) was 10 months, 26 days (range = 10;14–11;10); their average age at the time of the second recall test was 12 months, 4 days (range = 11;18–12;19). The sample included 16 girls and 15 boys. All of the infants were full term and experiencing an apparently normal course of development. Infants were recruited from a database of parents who were contacted shortly after their infants' births and who expressed interest in participating in research with their infants. The database is primarily composed of White middle- and upper middle class families. Reflective of this, the sample included 28 White children and 3 children of mixed race, including 1 child of African American and White descent; 1 child of African American, Native American, and White descent; and 1 child of Hispanic, Asian American, Pacific Islander, and White descent. Four other infants were enrolled in the study but did not complete all sessions because of illness, death in the family, or scheduling conflicts. All parents gave informed consent for their infants' participation before the start of the study. Each infant received a small toy or book at each ERP session and following completion of the study. Parents received a pair of movie tickets midway through the study, and a $25.00 gift certificate to a local merchant at the final visit.

Materials

Stimuli included 12 novel, two-step event sequences. For each infant, six events served as test stimuli (three at Phase 1 and three at Phase 2) and the remaining six events served as control stimuli (three at Phase 1 and three at Phase 2). Events were randomized by block such that across infants, each block was used equally often as test and control stimuli at both phases of the study. Each event consisted of two actions performed with unique props. For each event, completion of the two actions in the correct order resulted in an end state. For example, in the event depicted in Figure 1, "Turn on the light," the infant placed a car into a clear covered track. She then pushed a wooden plunger, moving the car to the end of the track, where it depressed a light switch on the base of the track, turning on a light (a complete list of the stimuli is available from Patricia J. Bauer).

At each ERP recognition memory test, infants saw digitized still photographs of one of the test events (which infants had seen previously) and one of the control events (which infants had not seen previously). There were three photographs of

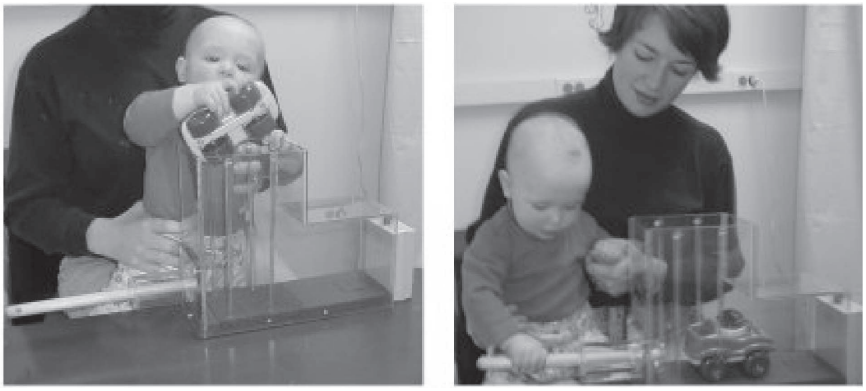

FIGURE 1 Example two-step sequence. An example two-step test sequence is "Turn on the light." To reproduce the sequence, infants first had to put a toy car down a vertical compartment of an "L"-shaped apparatus, and then push a rod into the horizontal compartment, thereby causing the car to roll to the 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. From "Developments in Long-Term Explicit Memory Late in the First Year of Life: Behavioral and Electrophysiological Indices," by P. J. Bauer et al., 2003, *Psychological Science, 14,* p. 632. Copyright 2003 by Blackwell. Reprinted with permission.

each event: (a) the props used in the event, (b) a woman's hand completing the first step of the event, and (c) a woman's hand completing the second step of the event. Each event was used as a test event and as a control event equally often.

Procedure

Exposure sessions. As reflected in Table 1, in each phase, at each of two sessions, the infants were exposed to three novel two-step event sequences.[1] Each exposure session began with a brief warm-up period during which the experimenter engaged the infant in play with a commercially available infant toy (e.g., musical stacking rings). Infants sat on their parents' laps or on the testing table. The warm-up period was followed by exposure to three test sequences. During the first exposure session (i.e., Session 1 for Phase 1, and Session 4 for

[1]In actuality, in each phase of the protocol, the infants were exposed to the event sequences at each of three sessions. In each phase, the third exposure took place during a session held 1 week after the immediate ERP test. Thus, the immediate ERP data were derived after two exposures to the sequences, whereas the 1-month delayed recall data were derived after three exposures to the sequences. The protocol was designed to afford variability in the immediate ERP data, yet provide a sufficient number of exposures to the events to support long-term recall. At the session held 1 week after the immediate ERP, before the third exposure to the sequences, the infants received another ERP test (to examine 1-week delayed recognition); different old and new sequences were included in each ERP test. The data on delayed recognition will be included in a later report.

TABLE 1
Schematic Diagram of the Test Protocol

	Protocol Phase			
Phase	*Baseline Assessment*	*Modeling*	*ERP Recognition Test*	*Behavioral Recall Test*
Phase 1				
Session 1	Events A, B, C	Events A, B, C	—	—
Session 2	—	Events A, B, C	Events A and D	—
Session 3	—	—	—	Events A, B, C and D, E, F
Phase 2				
Session 4	Events G, H, I	Events G, H, I	—	—
Session 5	—	Events G, H, I	Events G and J	—
Session 6	—	—	—	Events G, H, I and J, K, L

Note. Unique alpha characters designate unique sequences. In this schematic, for Phase 1, Events A, B, and C are "old" (i.e., sequences to which the infant was exposed prior to the delay); and Events D, E, and F are "new" (i.e., sequences the infant did not see at any of the exposure sessions). For Phase 2, Events G, H, and I are "old"; and Events J, K, and L are "new." Across infants, events were counterbalanced such that each was used approximately equally often in Phase 1 and in Phase 2, and as an old and new event. At each recognition test, one old event was paired with one new event; different old and new events were represented in each recognition test. Use of events for recognition testing was completely counterbalanced. ERP = event-related potential.

Phase 2), for each sequence in turn, infants were provided with the props used to produce the sequence and allowed to explore them for a baseline assessment period lasting approximately 1.5 min. Performance during the baseline period allowed us to assess the spontaneous level of production of the target actions and sequences. The experimenter then took back the props and modeled the sequence twice, labeling the event ("This is how I turn on the light") and narrating the steps as they were performed ("Put in the car. Push the stick."). Infants were not permitted to imitate the sequences. The second exposure session took place within 4 days of the first session (Session 2 for Phase 1: *M* delay between visits = 1.77 days, range = 1–4 days; Session 5 for Phase 2: *M* delay between visits = 1.73 days, = range 1–3 days). During the second exposure session, the three events were presented in a different random order. Infants were not permitted to touch or manipulate the props at any point.

Recognition memory testing. Following the second exposure to the three test events (see Table 1; at Session 2 for Phase 1 and Session 5 for Phase 2), infants' recognition memory was tested using ERPs. Because of the time necessary to apply electrodes, there was a brief delay between exposure to the sequences and the beginning of the ERP test (Phase 1: *M* delay = 30 min, range = 20–36

min; Phase 2: *M* delay = 24 min, range = 15–35 min). ERPs were recorded at 25 scalp locations (Fz, Cz, Pz, AF3, AF4, F3, F4, F7, F8, FC5, FC6, C3, C4, CP1, CP2, CP5, CP6, P3, P4, T3, T4, T5, T6, O1, O2), placed according to the international 10–20 system of electrode placements (Jasper, 1958). Electrodes were sewn into a nylon cap fastened under the infant's chin with a Velcro chinstrap. Electrodes were filled with a conductive gel and a mildly abrasive cream. Impedances were kept below 10 kΩ, and were generally less than 5 kΩ. Scalp activity was referenced to Cz during data collection, and rereferenced to an average reference offline. Electroocular activity was recorded from bipolar miniature electrodes placed in a transverse position above and below the infant's right eye. All electrical signals were recorded using a Grass Neurodata Acquisition System with Model 12A5 amplifiers. Electroencephalogram (EEG) gain was set to 20,000 and electrooculogram (EOG) gain was set to 5,000. Bandpass filters were set at 0.1 and 30 Hz. A 60-Hz notch filter was in place.

Each infant was tested individually while seated on the parent's lap facing a computer monitor approximately 75 cm away. The monitor was embedded in a black screen that blocked the infant's view of the remainder of the room. The screen contained small holes through which an observer could watch the infant, and, when necessary, redirect the infant's attention to the screen. Each trial consisted of a 100-msec baseline followed by presentation of the stimulus for 500 msec. EEG recording continued for an additional 1,200 msec. EEG was sampled every 10 msec (100 Hz) throughout each trial. This was followed by an intertrial interval that varied randomly between 500 and 1,200 msec. The observer controlled presentation of stimuli with a button box, and pressed a button when the infant looked away, signaling the computer to repeat the trial. Brain activity was not recorded when the infant was not looking at the screen. Up to 100 trials were presented, including three photographs of one event to which the infant had been exposed (old event) and three photographs of one event to which the infant had not been exposed (new event), each presented repeatedly. The photographs of old and new events were interspersed with one another in random order. If the infant became upset during testing, the ERP test was terminated. As a result, most infants completed fewer than 100 trials (Phase 1: *M* = 57.9 trials, range = 21–100 trials; Phase 2: *M* = 61.1 trials, range = 6–100 trials).

Recall memory testing. One month after the final exposure session (see Table 1; Session 3 for Phase 1 and Session 6 for Phase 2), infants returned to the laboratory for a recall memory test (Phase 1: *M* delay = 29.7 days, range = 20–46 days; Phase 2: *M* delay = 24.1 days, range = 15–35 days). Infants were tested in the same room by the same experimenter as in the first exposure session. Infants were tested on the three events to which they had been exposed (one of which had been used in ERP testing), as well as three new events (one of which had been used in ERP testing), which served as a within-subjects control (see Table 1). For each of the six

events in turn, the experimenter placed the props on the table in front of the infant and provided a general verbal label (e.g., "You can use this stuff to turn on the light"). The infant was then allowed to manipulate the props for approximately 1.5 min. Infants' behavior during this phase provided the measure of delayed recall (in the case of old events, with new events serving as within-subjects control stimuli). The experimenter then took back the props and demonstrated the event twice, labeling the event and narrating the steps; then the infant received the props for another 1.5 min in an assessment of relearning (in the case of memory stimuli) or immediate imitation (in the case of control stimuli). This procedure was repeated for each of the six events in turn. (Data from the relearning phase are not included in this article, but are available from Patricia J. Bauer.) Phase 1 ended after delayed recall testing for the events experienced at 9 months of age (Session 3). Infants returned to the laboratory several days later for their first exposure to the Phase 2 event sequences (Session 4).

Data Reduction

Behavioral data. Due to malfunction of recording equipment, the behavioral data of 6 infants were unavailable for coding. Trained behavioral coders (all three of whom were naive to the hypotheses of the study) viewed videotapes of the remaining 25 infants and noted infants' production of the actions that made up each event sequence (maximum = 2.0), and the order in which they were produced (maximum = 1.0). Each individual coded approximately one third of the infants. To determine the reliability of coding, 7 of the infants (28% of the sample) were independently recoded by a different one of the three coders. Across the phases of the study, reliability of coding was 84% (range = 78–91%); Phase 1 reliability was 82% (range = 74–94%) and Phase 2 reliability was 86% (range = 72–95%). Although lower than the estimates of reliability typically observed in research with older children (which average 90% and higher; e.g., Bauer et al., 2000), these values are comparable to those obtained in previous research with infants in the first year of life (e.g., Bauer et al., 2001; Carver & Bauer, 2001). The mean levels of performance across the three events in each condition (old events, new events) were used in all analyses.

Electrophysiological data. The EEG data were rereferenced offline to an average reference. Our choice of the average reference was motivated by several factors. Ideally, the reference should be a neutral site reflecting no activity related to neural processing. In practice, such a site does not exist on the human body (Dien, 1998; Geselowitz, 1998; Junghöfer, Elbert, Tucker, & Braun, 1999). Many studies of infant ERPs, including our own previous work, have used linked mastoids as a reference. However, signal recorded from the mastoids is influenced by brain activity at nearby brain regions, such as the temporal lobes. Reference based

on linked mastoids is known to attenuate the amplitude of components at nearby sites (Dien, 1998). Perhaps because of our choice of reference, in our previous work, we have observed effects at the midline leads, with little activity related to memory apparent at the temporal leads (Bauer et al., 2003; Carver et al., 2000).

In this study, to expand the range of sites over which activity might be observed, we selected an average reference constructed from 24 scalp leads (25 scalp locations minus the collection reference electrode Cz). In at least one published study with adults using 19 channels, an average reference has been compared to a linked ears reference and the component of interest (P300) showed similar latency and wave shape (Onofrj, Fulgente, Thomas, Locatelli, & Comi, 1995). Because in this research we used more channels with infants (who have smaller heads), we were afforded even better coverage. Even with this technique, however, we cannot draw conclusions about the localization of neural sources for ERP components, due to insufficient spatial coverage (e.g., Junghöfer et al., 1999; Nunez, 1990). Also, because different references will result in different absolute amplitudes of ERP components, there are limits on the extent to which we are able to compare the findings of this research with those of previous studies in which different reference techniques were used. Nevertheless, we are able to make qualitative comparisons, because ERP waveforms and topographic maps constructed from different references display similar features (Geselowitz, 1998; Onofrj et al., 1995).

In constructing the grand means, data were excluded if the EEG signal exceeded analog to digital values in any 50-msec window, or if the EOG signal exceeded 250 microvolts in any 250-msec window. Individual averages were obtained for each infant, with the constraint that an equal number of trials were included for each condition. If 10 or more trials were included in the averages for an infant, the average waveform was visually inspected to exclude data contaminated by motion artifact.

For Phase 1, data from 11 infants were deemed interpretable. Data from the other 20 infants were excluded from further ERP analysis (a) due to equipment failure ($n = 8$ infants), (b) because one or more electrodes was not reading or was offscale throughout testing ($n = 5$ infants), (c) because of blink or movement artifact ($n = 6$ infants), or (d) because the infant was too fussy to complete the required number of trials ($n = 1$ infant). For infants included in the analyses, the average number of trials completed was 68.6 (range = 43–100 trials).

For Phase 2, 14 infants provided interpretable ERP data. Data from the remaining 17 infants were excluded from further ERP analysis because (a) of equipment failure ($n = 4$ infants), (b) one or more electrodes was not reading or was offscale throughout testing ($n = 5$ infants), (c) of blink or movement artifact ($n = 7$ infants), or (d) the infant was too fussy to complete the required number of trials ($n = 1$ infant). For infants included in the analyses, the average number of trials completed was 73.9 (range = 36–100 trials). Seven infants provided interpretable ERP data at both the first and second phases of the study.

For the two ERP sessions, grand means were constructed by averaging infants' ERP waveforms separately for photographs of old and new events. Two windows of interest were identified: (a) a middle-latency window from 260 msec to 700 msec from stimulus onset, and (b) a long-latency window from 900 msec to 1,500 msec. Infants' middle-latency ERP components have been found to relate to cognitive processes including attention and memory (e.g., Bauer et al., 2003; de Haan & Nelson, 1997; Nelson & Dukette, 1998; Richards, 2003). Long-latency slow wave activity has been shown to relate to memory (e.g., Carver et al., 2000). Dependent measures for analyses were computed for each infant within the windows of interest. As in previous related research (Bauer et al., 2003; Carver et al., 2000), in the middle-latency window, we analyzed the peak amplitude of deflection from baseline and the latency to peak amplitude. For slow wave activity, the dependent measure of interest was an area score integrating area under the curve relative to baseline.

RESULTS

The results are presented in three sections. First, we present the results of analyses of the electrophysiological (ERP) tests of immediate recognition memory. Second, we present the results of analyses of the infants' behavioral memory performance after the 1-month delays. In the third section we present the results of analyses in which we examined relations between ERP measures of recognition and behavioral measures of recall, both within and across phases.

Electrophysiological Tests of Recognition Memory

In previous related research (Bauer et al., 2003; Carver et al., 2000), we concentrated analyses on the midline leads Fz, Cz, and Pz. The rationale was that, when using linked ear reference, little activity was apparent at leads T3, T4, T5, T6 (Bauer et al., 2003; Carver et al., 2000), C3, and C4 (Bauer et al., 2003). In more recent research using average reference techniques, however, there is evidence of differential brain activity at posterior-lateral leads in response to photographs of familiar and novel objects (toys; e.g., Snyder, 2002; Snyder & Nelson, 2001). Because in this research we used average reference, in addition to the midline leads, we also analyzed the posterior-lateral leads T5, T6, O1, O2, P3, and P4. Specifically, for the midline leads, we conducted 2 (phase: Phase 1, Phase 2) × 2 (condition: old events, new events) × 3 (lead: Fz, Cz, Pz) mixed analyses of variance (ANOVAs) with repeated measures on condition and lead (although 7 infants contributed data to both phases, because most infants contributed data to one but not both phases, we treated phase as a between-subject variable). For the posterior-lateral leads, we conducted parallel analyses involving six levels of lead (T5, T6, O1, O2, P3, and P4). Based on de Haan and Nelson (1997), we expected that the com-

ponents would have different polarities at the anterior and posterior leads: At the anterior leads, we expected negative deflection in the middle-latency window and a positive slow wave; at the posterior leads, we expected positive deflection in the middle-latency window and negative deflection in the long-latency window.[2]

Midline leads. ERP waveforms at the midline leads are represented in Figure 2. Analyses of activity in the middle-latency window provided evidence of differential processing of old and new stimuli, as well as suggestions of age-related differences in processing. In both cases, the evidence is from effects that approached but did not reach the conventional level of statistical significance. Because the effects are consistent both with prior, related research and with other significant effects observed at the posterior-lateral leads (discussed in the next section), we present them nonetheless. The analyses indicated a trend toward longer latency to minimum amplitude in the middle-latency window to photographs of the old events ($M = 505.04$, $SD = 132.16$) relative to the new events ($M = 474.35$, $SD = 129.47$), $F(1, 23) = 2.71$, $p < .12$. Analysis of the Condition × Lead interaction, $F(2, 46) = 2.81$, $p = .07$, revealed a significant effect at Pz, $F(1, 23) = 8.02$, $p < .01$ (Ms = 535.00 and 447.20, SDs = 140.74 and 150.17, for old and new events, respectively). The main effect is parallel to the results of Bauer et al. (2003), in which we observed a correlation between latency to peak amplitude to old events after a 1-week delay and 9-month-olds' recall 1 month later: Infants who had longer latencies to peak amplitude had higher levels of delayed recall. Longer latencies may be reflective of cognitive processes associated with reintegration of memory traces that are relatively fragile, either because of delay (as in Bauer et al., 2003) or because of a small number of exposures (as in this research).

There also was suggestive evidence of differential processing of the stimuli by the infants at 9 and 10 months of age. On the variable of minimum amplitude, the interaction of Phase × Condition approached significance, $F(1, 23) = 2.63$, $p < .12$. Consistent with the results of Bauer et al. (2003), at 9 months of age, minimum amplitude in the middle-latency window tended to be greater to the new events relative to the old events (Ms = –9.24 and –7.42, SDs = 8.48 and 6.65, respectively). In contrast, at 10 months of age, negative amplitude tended to be greater to the old relative to the new events (Ms = –10.17 and –7.86, SDs = 10.43 and 10.07, respectively). Although lead was not involved in the interaction, inspection of Figure 2 makes clear that the trend was localized at Cz. A similar change in the direction of effects is apparent at the posterior-lateral leads, discussed next. These effects, al-

[2]Negative amplitude at anterior midline electrode sites at approximately 400 msec to 800 msec after stimulus onset is an identifiable component known as the Nc (e.g., de Haan & Nelson, 1997). However, both because the component has not previously been identified at posterior-lateral electrode sites, and because at those sites we expected the deflection from baseline to be positive, rather than negative, in this research we do not label activity in the middle-latency window as an Nc, per se.

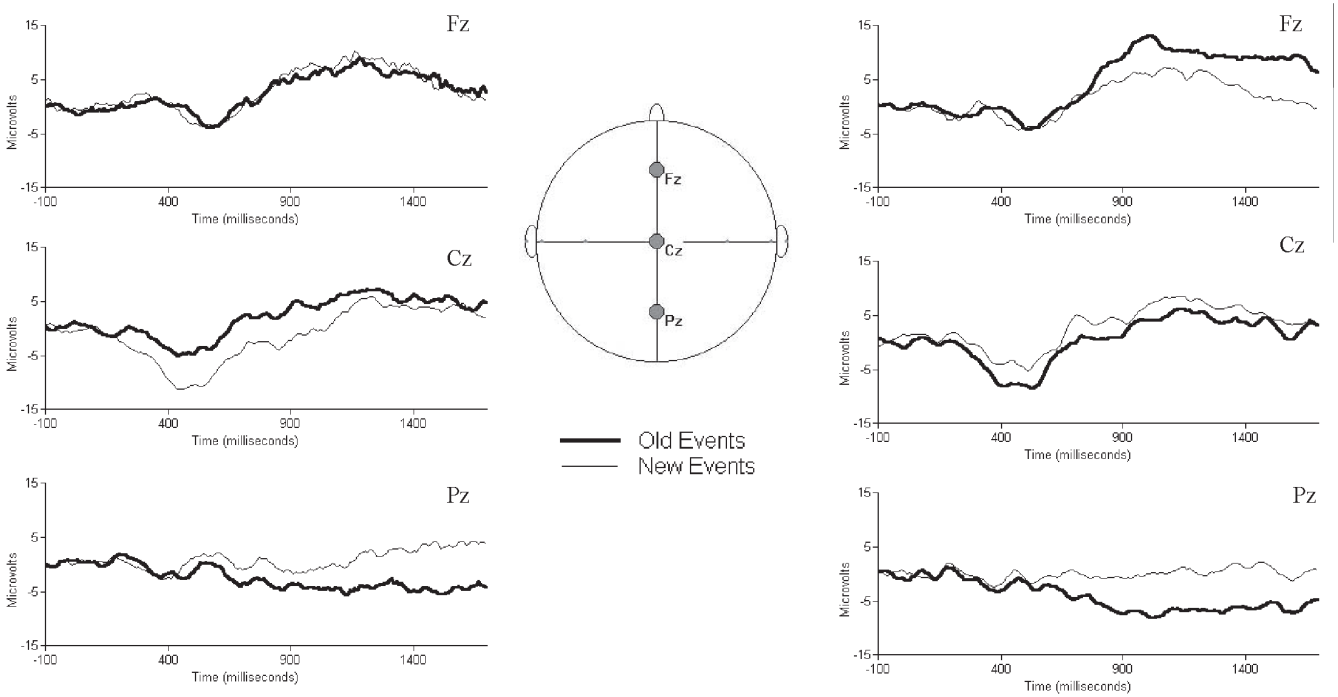

FIGURE 2 ERP waveforms for the midline leads Fz, Cz, and Pz. Waveforms for Phase 1, when the infants were 9 months of age, are on the left, and waveforms for Phase 2, when infants were 10 months of age, are on the right.

though not robust, nevertheless suggest that the infants had encoded the to-be-remembered events, and that there are age-related differences in encoding processes at 9 relative to 10 months of age.

Analysis of the long-latency window revealed no suggestions of differential response as a function of phase or condition.

Posterior-lateral leads. ERP waveforms at the posterior-lateral leads are represented in Figure 3. Analyses of activity in the middle-latency window revealed no evidence of differential processing of old and new stimuli. Neither was there evidence of differential processing by 9- and 10-month-old infants. Analysis of activity in the long-latency window revealed evidence of both effects, however.

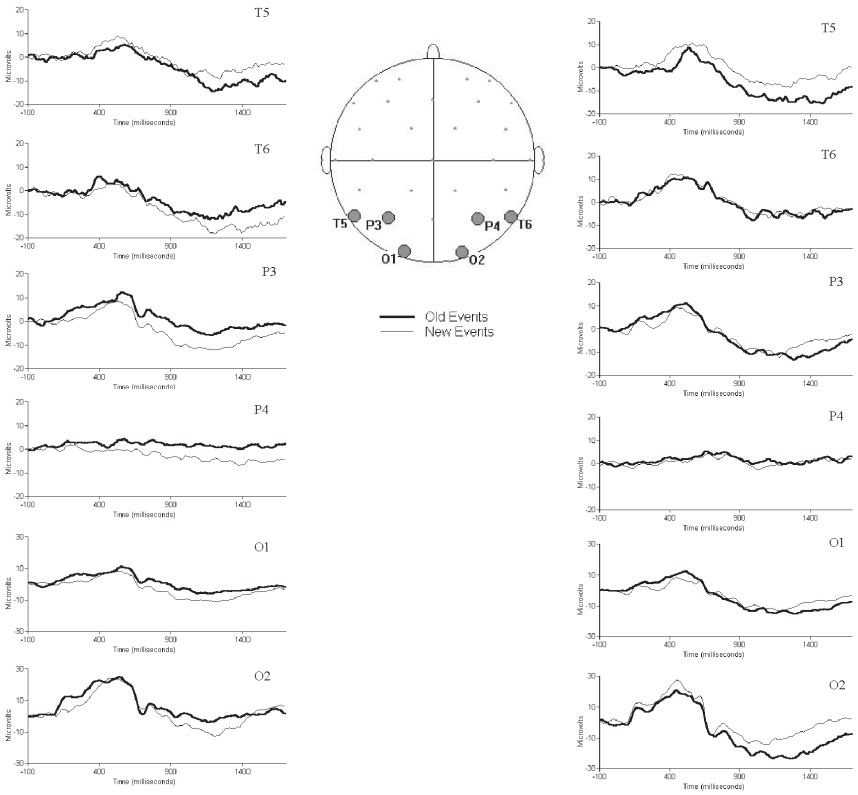

FIGURE 3 ERP waveforms for the posterior-lateral leads T5, T6, P3, P4, O1, and O2. Waveforms for Phase 1, when the infants were 9 months of age, are on the left, and waveforms for Phase 2, when infants were 10 months of age, are on the right.

Analysis of the area scores revealed a significant Phase × Condition interaction, $F(1, 23) = 8.54$, $p < .01$. Separate analyses for each phase revealed evidence of differential processing of the old and new stimuli at both ages, $F(1, 10) = 4.41$, $p = .06$, at 9 months; and $F(1, 13) = 4.19$, $p = .06$, at 10 months. At 9 months of age, area scores were greater for new events than for old events (*M*s = –5299.17 and –2612.00, *SD*s = 7573.88 and 6300.88, respectively). At 10 months of age, the infants showed the opposite pattern: Area scores were greater for old events than for new events (*M*s = –6209.95 and –3832.87, *SD*s = 8459.91 and 9137.48, respectively). The effects are parallel to the trends observed in analysis of minimum amplitude at the midline leads (reported in the preceding section). Moreover, the effects cannot be attributed to a small number of infants. At 9 months of age, 7 infants had nominally greater area scores for new than for old events, whereas only 3 had nominally greater area scores for old than for new events (binomial $p < .05$; 1 infant's area scores were within 10 microvolts of each other, which we treated as a tie). At 10 months of age, 11 of the 14 infants had nominally greater area scores for old than for new events (binomial, $p < .01$). Neither can the effects be attributed to the fact that the 10-month-olds had experienced ERP testing before, at 9 months: In a separate group of 17 infants tested for the first time at 10 months of age (i.e., cross-sectional control infants drawn from the same source and representing the same population as the infants in the longitudinal sample), the effect of condition, although not statistically reliable ($p = .26$), nevertheless was in the same direction as that for the infants in the longitudinal sample. These effects suggest that the infants had encoded the to-be-remembered events, and that there are age-related differences in encoding processes at 9 relative to 10 months of age.

Additional analysis of the Phase × Condition interaction revealed that the differences in processing of the stimuli at 9 and 10 months of age were specific to the old events. As reflected in Figure 4, on the new events, the area scores of the infants at 9 and 10 months of age did not differ ($F < 1.00$). In contrast, on the old events, infants' area scores at 9 months of age were significantly smaller than their area scores at 10 months of age, $F(1, 23) = 5.72$, $p < .03$. This indicates a more robust response to events encoded at 10 months of age relative to events encoded at 9 months of age. Finally, there was a main effect of lead, $F(5, 115) = 2.54$, $p < .05$. Across phases and conditions, the area scores at leads O1 and O2 differed from those at lead P4 (Tukey, $p < .05$). Because the effect is not informative with regard to developmental changes or differences in processing of old and new events, we make no attempt to interpret it.

Behavioral Tests of Recall Memory

Given evidence that the infants had encoded the to-be-remembered event sequences, and of differences in 9- and 10-month-old infants' recognition responses, was there evidence of long-term recall, and of age-related differences therein? One

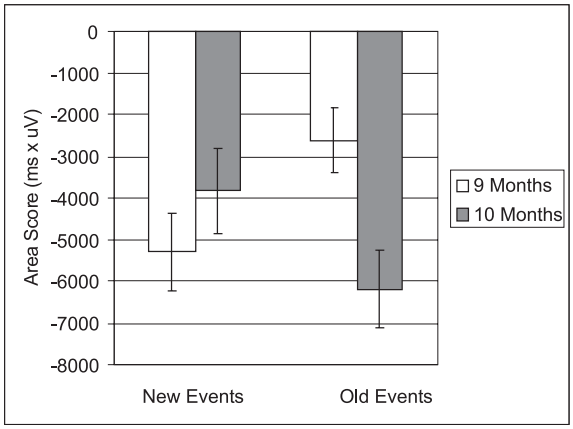

FIGURE 4 Area scores for the posterior-lateral leads (T5, T6, P3, P4, O1, and O2) as a function of phase (events encoded at 9 months and events encoded at 10 months) and condition (new events and old events).

way to address this question would be to compare the infants' spontaneous levels of production of the target actions and sequences during the baseline periods of each phase with their levels of performance after the delays. Such analyses would not, however, account for maturational changes over the 1-month delays. A stronger test of recall is afforded by comparison of performance on the old events with that on the new, control events. Accordingly, we conducted 2 (phase: Phase 1, Phase 2) × 2 (condition: old events, new events) within-subjects ANOVAs on the number of individual target actions produced and the number of pairs of actions produced in the target order (these analyses could be conducted within subjects because all infants contributed behavioral data to both phases; as noted earlier, because of failure of videotaping equipment, behavioral data were available for only 25 of the 31 infants in the sample).

Long-term recall of the sequences was affected by the infants' ages at the time of encoding of the events, as evidenced by interactions of Phase × Condition for both dependent measures, $F(1, 22) = 10.91$, $p < .004$, for individual target actions produced; $F(1, 22) = 7.14$, $p < .02$, for pairs of actions produced in the target order. As reflected in Figure 5, whereas the infants showed no evidence of recall after 1 month for the events to which they had been exposed at 9 months of age (i.e., levels of performance on old and new events did not differ, for either dependent measure, Fs < 1.00), they remembered the events to which they had been exposed at 10 months of age (i.e., infants performed a greater number of target actions and a greater number of pairs of actions in the target order on old relative to new events), Fs(1, 22) = 27.19 and 12.70, ps < .002, respectively. Thus, as expected, based on the results of Carver and Bauer (2001),

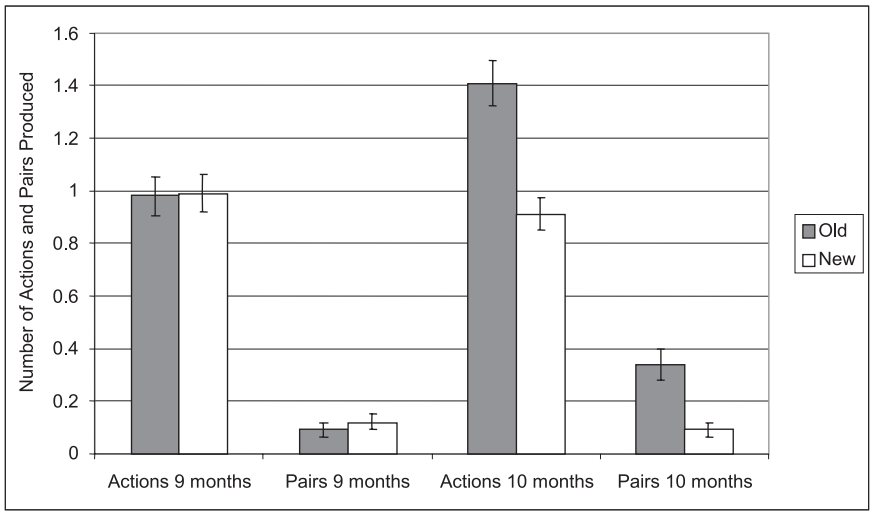

FIGURE 5 Mean number of actions (maximum = 2.0) and pairs of actions (maximum = 1.0) produced as a function of phase (events exposed at 9 months and tested at 10 months and events exposed at 10 months and tested at 12 months) and condition (old events and new events).

for example, there were pronounced age-related differences in recall behavior across the period of 9 to 10 months of age.

Relations Between Electrophysiological and Behavioral Measures

To determine whether differences in encoding, as measured by differential ERP responses to photographs from old and new events, were related to differences in long-term recall, as measured by deferred imitation, following procedures established in Bauer et al. (2003), we calculated difference scores for peak amplitude (minimum and maximum amplitude for anterior and posterior-lateral leads, respectively), latency to peak amplitude, and area: Infants with larger difference scores showed more robust differentiation of photographs from old and new events. We calculated correlations between the difference scores and the dependent measures from deferred imitation; namely, the number of individual target actions produced and the number of pairs of actions produced in the target order. We calculated the correlations within Phase 1, within Phase 2, and across phases. In Phase 1, immediate ERP responses obtained when the infants were 9 months of age were not related to their delayed recall performance 1 month later. The lack of effect is perhaps not surprising, given that there was little evidence of recall of events to which the infants were exposed at the age of 9 months.

In Phase 2, for events to which the infants were exposed at the age of 10 months, at midline lead Cz, the difference in latency to peak amplitude in response to photographs of the old and the new events was correlated with the number of individual target actions the infants recalled 1 month later, $r(11) = .59$, $p < .05$. This effect is reminiscent of the finding in Bauer et al. (2003) that the difference in latency to peak amplitude at Cz after a 1-week delay accounted for significant variance in 9-month-old infants' ordered recall after a 1-month delay. In both cases, longer latencies were associated with better recall memory. As in Bauer et al. (2003), we suggest that this effect is reflective of cognitive processes associated with reintegration of memory traces. In this research, production of the individual target actions of the events after the 1-month delay also was related to differences in the area scores at posterior-lateral leads T5 and T6: rs(11) = .56, $p < .05$, and .53, $p < .10$, respectively. Finally, at posterior-lateral lead T6, differences in peak amplitude and in area scores were correlated with the number of pairs of actions the infants produced after the 1-month delay: rs(11) = .59, $p < .05$, and .75, $p < .01$, respectively. Thus, infants who showed larger differences in their responses to photographs of the old and new events at the time of encoding had higher levels of recall of the individual actions and the temporal order of actions of the events 1 month later.

There also were cross-lagged correlations from ERP responses in Phase 1, when the infants were 9 months of age, to behavioral responses in Phase 2, when the infants were an average of 12 months of age. Specifically, the difference in area scores at midline lead Cz at the first immediate recognition test (when the infants were 9 months of age) was correlated with the number of pairs of actions produced in the target order in the second delayed recall test (when the infants were 12 months of age), $r(9) = .66$, $p < .05$. A parallel although nonsignificant effect was observed for the measure of the number of target actions produced, $r(9) = .50$, $p = .11$; the same trend was observed at midline lead Pz, $r(9) = .50$, $p = .11$. Trend-level correlations also were observed between the difference in peak amplitude to photographs of old and new events at 9 months at midline lead Pz and the number of individual target actions produced at 12 months, $r(9) = .50$, $p = .11$, and between the difference in peak amplitude at midline lead Fz and the number of ordered pairs of actions produced at 12 months, $r(9) = .50$, $p = .12$. The difference in latency to peak amplitude in response to the photographs of the old and new events at the immediate recognition test at 9 months of age at midline lead Cz also was correlated with the number of pairs of actions produced in the target order at 12 months of age, $r(9) = .56$, $p < .10$. Although most of the cross-lagged correlations failed to reach the conventional level of statistical significance, they are striking nonetheless, given that they span a period of almost 3 months, during which substantial and significant developmental changes are apparent in both recognition and recall.

DISCUSSION

This research provided evidence of differences in encoding processes and in long-term recall behavior by infants at 9 months of age and at 10 months of age. Whereas at both ages, the infants encoded the two-step event sequences to which they were exposed, the evidence of encoding was different at the two points in time. At 9 months, in the middle-latency window, the infants evidenced greater negative amplitude to photographs of new relative to old events; in the long-latency window, area scores were greater for new than for old events. At 10 months, the opposite pattern was observed: The infants evidenced greater negative amplitude and greater area scores for old relative to new events. Infants' recall behavior also changed over the space of 1 month. The infants did not evidence 1-month delayed recall for events experienced at 9 months of age, but they recalled both the individual target actions and the temporal order of actions of the events experienced at 10 months of age. In spite of the differences in both encoding and recall at 9 and 10 months, indexes of encoding at 9 months were correlated with measures of recall of events experienced at 10 months and tested 1 month later.

There are numerous suggestions that the differences in encoding and recall behavior over the 1-month space of time between Phase 1 and Phase 2 of the research are indicative of developmental differences as opposed to chance fluctuations. First, the sample was longitudinal, making it unlikely that the differences are due to sampling error. Second, at each age, the patterns were not confined to a few outliers, but were observed in a significant number of infants. Third, the pattern at 9 months of age, although not statistically significant in this sample, is a replication of that observed with an independent sample of 9-month-olds in Bauer et al. (2003). Fourth, the direction of effect in the long-latency window in the longitudinal sample at 10 months of age also was observed in an independent cross-sectional control sample of 10-month-olds, and thus cannot be attributed to effects of repeated ERP testing. Finally, encoding behavior in Phase 1 of this study was correlated with recall behavior in Phase 2. Although certainly the best test of the robustness of a developmental pattern is replication of it, this list should inspire confidence that the age-related changes are "real."

What is the nature of the developmental differences observed at 9 months and 10 months of age? Consistent with prior, related research (Bauer et al., 2003), this study contains evidence of developments in storage processes over the 1-month period. Whereas at both ages the infants encoded information about the event sequences (see the next paragraph for discussion of the nature of the information encoded), and at the older age they maintained their memories of the events over the 1-month delay, at the younger age, forgetting was apparent after 1 month. In previous studies, we have observed 9-month-olds to recall both the individual actions and the temporal order of event sequences after a 1-month delay (e.g., Carver &

Bauer, 2001). One possible explanation for the lower performance of the 9-month-olds in this study is interference from the ERP test itself. In ERP testing, the steps of old events were presented in random order, interspersed with photographs of new events. In previous studies (i.e., Bauer et al., 2003; Carver et al., 2000), we exposed infants to the sequences three times before ERP testing. In this research, the infants had only two exposures prior to the recognition test. For 9-month-olds, memory representations based on three experiences are more robust than representations based on only two experiences (Bauer et al., 2001). In this research, even though the infants had encoded the sequences, because memory representations are susceptible to interference throughout the period of consolidation, the scrambled ERP procedure may have interfered with what were relatively fragile memory representations. That the potentially disruptive ERP procedure did not interfere with consolidation and storage when the infants experienced precisely the same protocol at 10 months is further testament to the significance of developments in consolidation and storage processes over this space of time. Indeed, elsewhere we have speculated that in the face of a stronger memory representation, exposure to photographs of events in the context of ERP testing actually may facilitate long-term recall (Bauer et al., 2001).

This study also implicates encoding processes as a source of developmental change between 9 and 10 months of age. Whereas at both ages, the infants encoded the events to which they were exposed, they apparently did so in different ways, as evidenced by different patterns of ERP responses to old and new events. The differences were seen across leads, with trends at the midline leads and significant effects at the posterior-lateral leads. The differences also were seen across measures, namely, in peak amplitude in the middle-latency window at the midline leads and in the slow wave in the long-latency window at the posterior-lateral leads. One possibility is that the age-related differences had to do with what the infants encoded about the events. For example, at 10 months of age, the infants may have encoded the individual actions of the events and the temporal order of the actions. In contrast, at 9 months, they may have encoded only the features of the objects used in the events. Although this possibility fits both the recognition and the recall data (i.e., at 9 months, the infants recognized still photographs of the props used in the events, yet after the 1-month delay they recalled neither the actions of the sequences nor their temporal order), we do not deem it likely. Over 24 hr, 9-month-olds recall both the individual actions and the temporal order of two-step event sequences to which they have been exposed only once (Wiebe, 2003). There is every reason to believe then that in this research, the infants encoded temporally ordered sequences of action.

Rather than to differences in what was encoded about the events, we attribute the different patterns at 9 and 10 months of age to differences in the relative distribution of processing resources devoted to the familiar and novel stimuli. That at 9 months of age, peak amplitude in the middle-latency window at the midline leads

tended to be larger to the new events relative to the old events, indicates that the infants were devoting greater processing resources to the new stimuli. The pattern they exhibited is reminiscent of that observed among 6-month-old infants engaged in the difficult task of differentiating pictures of their mothers from pictures of women of similar appearance (de Haan & Nelson, 1997, Experiment 3). In contrast, at 10 months of age, the infants in this research exhibited a pattern similar to that observed among de Haan and Nelson's (1997) 6-month-olds engaged in the relatively easy task of differentiating pictures of their mothers from pictures of women of dissimilar appearance: They devoted more processing resources to the more familiar stimuli (de Haan & Nelson, 1997, Experiment 1). If the comparison across these studies is reasonable, then we may conclude that in this research, the task of differentiating between photographs of old and new events was more challenging for infants when they were 9 months of age, relative to when they were 10 months of age. Consistent with this suggestion, activity in the long-latency window was more robust to events encoded at 10 months than to events encoded at 9 months. The difference in activity was seen only on the old events: Long-latency activity did not differ for photographs of new stimuli at 9 and 10 months. Although we cannot be certain based on these data alone, it seems likely that the differences in encoding were the first step in a cascade that resulted in differences in consolidation and storage, and ultimately, in long-term recall.

That the differences in encoding processes at 9 and 10 months of age have implications for long-term recall is suggested by findings of relations between the measures. At 9 months of age, we did not observe correlations between measures of encoding and measures of recall 1 month later. This likely was a result of the low level of delayed recall of the events encoded at 9 months (and in the case of temporally ordered recall, associated low variability in performance). At 10 months of age, differences in latency to peak amplitude for old and new events, and differences in area scores for old and new events, accounted for as much as 35% and 31% of the variance, respectively, in the infants' recall of the individual target actions of the events 1 month later. The latency effect is reminiscent of the relation in Bauer et al. (2003) between latency to peak amplitude after a 1-week delay and recall after 1 month among 9-month-olds. In both cases, longer latencies may be reflective of cognitive processes associated with reintegration of memory traces (see Brainerd, Reyna, Howe, & Kingma, 1990, for discussion of reintegration processes). Also at 10 months of age, differences in peak amplitude and in area scores accounted for 35% and 56% of the variance, respectively, in ordered recall 1 month later. Thus, more robust encoding of the events at 10 months was related to higher levels of recall over a 1-month delay.

We also observed correlations between Phase 1 ERP measures and Phase 2 behavior. The size of the difference in slow wave activity associated with the old and new events at 9 months of age accounted for 44% of the variance in recall of the temporal order of events experienced at 10 months and recalled 1 month later. Al-

though the effects only approached significance, parallel trends were observed in explanation of the variance in infants' recall of the individual target actions of the events experienced at 10 months (with 25% of variance accounted for). Thus, infants who evidenced more robust patterns of recognition at 9 months of age had higher levels of recall for events experienced at 10 months of age. In addition, a trend-level correlation once again implicated reintegration processes as important for long-term recall: The difference in latency to peak amplitude at 9 months of age accounted for 31% of the variance in ordered recall of events experienced at 10 months of age. The findings of cross-lagged correlations are striking given that they were observed between ERP responses obtained after two exposures to events at 9 months of age and recall after three exposures, to different events, almost 3 months later. The findings imply substantial continuity in declarative memory processes by late in the first year of life. We end discussion of the correlational findings with a note of caution, however, both because we calculated a number of correlations (within Phase 1, within Phase 2, across phases; the full correlation matrices are available on request from Patricia J. Bauer), only some of which were significant, and because many of the correlations discussed approached, but did not reach, the conventional level of statistical significance.

It is important to note that age-related differences in encoding and storage processes do not end at 1 year of age. On the contrary, they are readily apparent throughout the second year of life. For example, Howe and Courage (1997) found that, relative to 15-month-olds, 12-month-olds required more trials to learn multistep event sequences to a criterion. In turn, 15-month-olds were slower to learn the to-be-remembered material, relative to 18-month-olds. When the variance in encoding processes is controlled, age-related differences in storage processes become obvious. In Bauer (2005a), for instance, we observed that for children exposed to event sequences at 13 months of age, storage failure was virtually complete after 3 months. In contrast, for children who experienced events at 20 months of age, storage failure was not apparent until the delay had stretched to 6 months, and was not complete even after a 9-month delay (see also Howe & Courage, 1997). Findings such as these make clear that even though there are rapid developments in basic mnemonic processes in the latter part of the first year of life, age-related changes continue throughout the second year, presumably in conjunction with neurodevelopmental changes in the structures implicated in long-term declarative memory.

The final contribution of this research is methodological. In previous related research, we have recorded ERP responses from a small number of electrodes, and used linked ears as the reference. In this research, we recorded from 25 electrodes, and used an average reference. Likely as a result, this was the first study in which we observed differential brain activity at posterior-lateral leads in response to photographs of old and new events (Snyder, 2002, and Snyder & Nelson, 2001, reported similar findings in response to pictures of familiar and novel toys). Also in a

departure from previous research, in this study, effects at the midline leads were not especially pronounced. One possibility is that the midline effects were smaller because of the use of the average reference. We deem this possibility unlikely, however, given the similarity in the morphology, if not the amplitude, of the effects observed at the midline leads relative to those observed in, for example, Bauer et al. (2003). A more interesting and we believe, more likely, possibility is that the effects at the midline leads were attenuated, relative to those in prior research, because in this research, the ERP indexes were obtained after only two exposures to the events, as opposed to after three exposures as in the previous research (Bauer et al., 2003; Carver et al., 2000). As discussed earlier, based on behavioral data, we have reason to believe that infants' memory representations are less robust after two relative to three exposures to events (Bauer et al., 2001). Because this is the first study in which we have examined posterior-lateral leads, we have no basis on which to evaluate the possibility that there, too, effects may have been larger, had infants had more exposures to the to-be-remembered events. The behavioral data, coupled with the ERP data in this research, point to the importance of additional investigation into encoding processes late in the first year of life.

In conclusion, in this research we observed rapid changes in encoding processes and recall performance in the latter part of the first year of life. Within the space of 1 month, infants revealed different patterns of response to old and new events at encoding. Perhaps as a result, they exhibited pronounced differences in the robustness of recall after delays of 1 month. The age-related changes in mnemonic performance likely are related to neurodevelopmental changes in association and medial temporal cortices, as well as the hippocampus proper, taking place in the same space of time. By implicating encoding processes as one major source of developmental change in this period, this study takes us a step closer to understanding how changes in brain relate to changes in behavior.

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