Speed and cerebral correlates of syllable discrimination in infants

Ghislaine Dehaene-Lambertz & Stanislas Dehaene

Institute of Cognitive and Decision Sciences, University of Oregon, Eugene, OR 97405, USA and Laboratoire de Sciences Cognitives et Psycholinguistique, CNRS, INSERM and EHESS, 54 Boulevard Raspail, 75270 Paris cedex 06, France

THE remarkable linguistic abilities of human neonates are well documented[1-5]. Young infants can discriminate phonemes even if they are not used in their native language[2-1], an ability which regresses during the first year of life[4,5]. This ability to discriminate is often studied by repeating a stimulus for several minutes until some behavioural response of the infant habituates, and later examining whether the response recovers when the stimulus is changed[6]. This method, however, does not reveal how fast infants can detect phonetic changes, nor what brain mechanisms are involved. We describe here high-density recordings of event-related potentials in three-month-old infants listening to syllables whose first consonants differed in place of articulation. Two processing stages, corresponding to an increasingly refined analysis of the auditory input, were identified and localised to the temporal lobes. A late frontal response to novelty was also observed. The infant brain recognizes a phonetic change in less than 400 ms.

Sixteen healthy two- to three-month-old infants (average 81 days old, range 63-91), born to monolingual American-English families, were tested. Infants were seated in a carrier affixed to the parent, and their heads were covered with a geodesic sensor net, a very light mesh made of 58 electrodes encased in sponges soaked with a saline solution[7]. Infants faced a loudspeaker placed on top of a TV monitor in a sound-attenuated room. To avoid eye and head movement, a silent video showing attention-grabbing coloured objects was played continuously. The video was not synchronised with the auditory stimuli, thus preventing any visually evoked potentials from appearing after averaging in synchrony with the auditory stream.

On each trial, a sequence of five syllables (/ba/ or /ga/) was presented. In half the trials, one syllable, designated as the standard, was repeated five times (standard trials). In the other half (deviant trials), the standard was repeated only four times, followed by one instance of the other syllable, designated as the deviant. Because repeated and deviant trials were randomly mixed, infants could not predict the nature of the fifth stimulus. Thus any significant difference in event-related potentials (ERPs) to repeated and deviant trials indicated that the two syllables had been discriminated. The onset and topography of the observed differences were used to infer the speed and brain mechanisms of syllable discrimination. The evolution of ERPs with successive repetitions of the standard was also studied. In human adults, reduced ERPs to repetitive sounds as well as novelty-specific responses have been described[8 10].

Each syllable presentation elicited a distinctive waveform characterized by two peaks (Fig. 1). The maturation of these peaks from birth to six months has been previously described", but their functional significance has not been elucidated. The first identifiable event, peak 1, reached its maximum about 220 ms after stimulus onset. Peak 1 amplitude was highest in response to the first syllable of each trial. As soon as the standard was repeated, a significant decrease was observed. Further repetitions of the standard did not lead to further decrease, and there was no recovery to the deviant syllable (Fig. 2). Thus, by 200 ms, the acoustical analysis had not proceeded far enough to separate two syllables that were quite similar in pitch, duration and intensity. The generators of peak 1 appeared insensitive to the subtle acoustical differences that encoded phonetic information. The scalp topography of peak 1 showed synchronous anterior positivities and posterior negativities with a temporal-central inversion plane (Fig. 3), suggesting bilateral generators inside the temporal lobes. We speculate that peak 1 reflected the activation of primary and secondary auditory areas.

The next identifiable event, peak 2, reached its maximum about 390 ms after stimulus onset. Again, the amplitude of peak 2 decreased significantly between the first and the second presentation of the standard, with no further decrease to subsequent

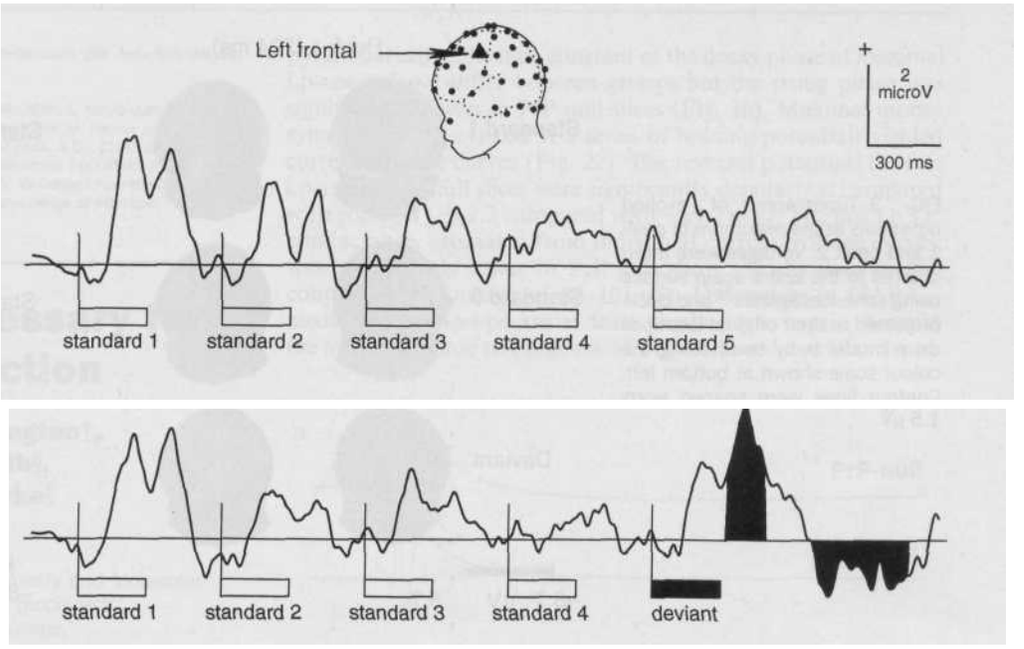

FIG. 1 Grand-averaged ERPs recorded from a left frontal electrode on standard and deviant trials.

METHODS. Two digitized syllables /ba/ and /ga/, naturally produced by a male speaker, were matched for total duration (289 ms), formant transition duration (87 ms), intensity (78 dB) and fundamental frequency (122 Hz). For each infant, one syllable, counterbalanced across subjects, was designated as the standard (S) and the other as the deviant (D). Syllables were presented in groups of five with a stimulus onset asynchrony of 600 ms. On standard trials, the standard was repeated five times (S S S S S) whereas on deviant trials the deviant was introduced in the fifth position (S S S S D). Fifty standard and fifty deviant trials were randomly mixed with an intertrial interval of 3,100 ms. Scalp voltages were recorded from 58 Ag/AgCl electrodes mounted on a Geodesic Sensor Net[7] applied in anatomical reference to the canthomeathal line and referenced to the right mastoid. Two frontal and two infra-orbital electrodes monitored for eye movements. A ground lead was placed on the seventh cervical vertebra. Voltages were amplified (band pass 0.1-50 Hz, 3 dB per octave attenuation, 60 Hz notch filter), digitized at 125 Hz for 4,096 ms (512 samples) starting 200 ms before trial onset, and automatically edited for eye and motion artefacts. Seven infants with less than 25 artefact-free trials were rejected. Data from the remaining 16 infants (average 51.2 trials per infant) were averaged, transformed using the average-reference method, baseline corrected and digitally filtered (band pass 0.5-20 Hz).

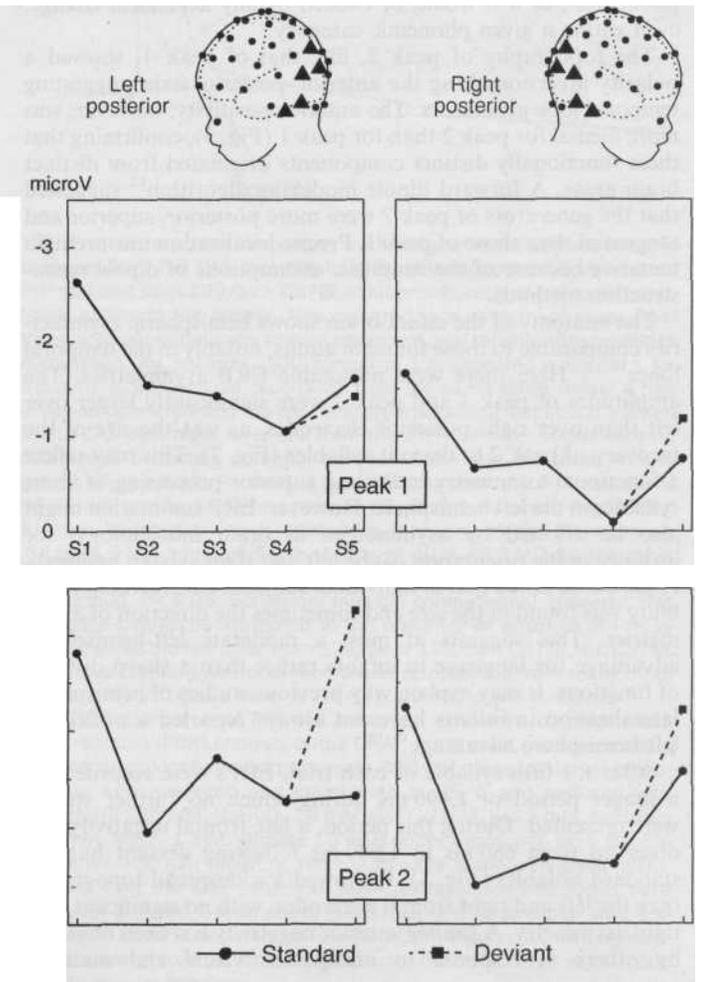

FIG. 2 Evolution of peak 1 and peak 2 voltages from left and right posterior temporal electrodes across the five standards and the deviant syllable. Voltage was averaged over five adjacent electrodes across two 160 ms time windows centred on each peak. A repeated-measures analysis of variance (ANOVA) was done with peak, stimulus number, hemisphere, and condition (standard versus deviant) as factors, with a Greenhouse-Geisser correction for non-sphericity. The decrease between stimulus 1 and stimulus 2 was significant for both peak 1 ($P = 0.0012$) and peak 2 ($P=0.0001$). There was a recovery to the deviant syllable only for peak 2, as measured by the condition x stimulus number interaction (peak 1, $P=0.36$; peak 2, $P=0.021$) and by the main effect of condition on stimulus 5 (peak 1, $P = 0.75$; peak 2, $P = 0.0008$). The latter effect was larger over the left hemisphere than over the right (interaction $P = 0.014$), and both peaks were of higher amplitude over the left hemisphere than over the right (both $Ps< 0.011$). Another ANOVA on five adjacent superior prefrontal electrodes showed similar decrement and discrimination effects, and a non-significant trend towards left lateralization. In addition, a later time window (680-1,080ms) showed a significant main effect of condition ($P = 0.002$), only deviant stimuli eliciting a bilateral frontal negativity (see Fig. 1).

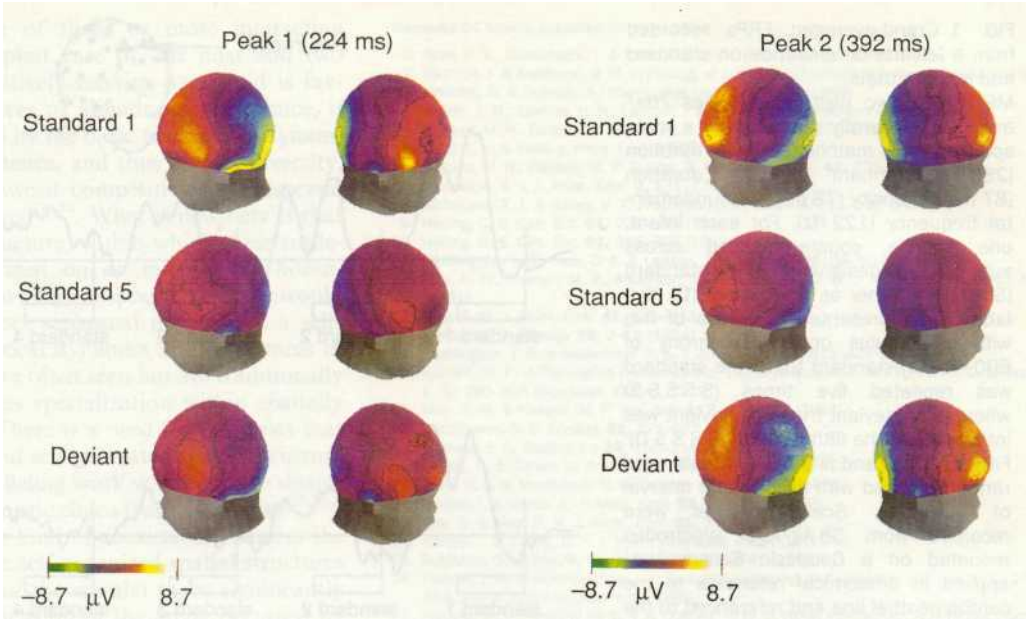

FIG. 3 Topography of evoked potentials at the maximum of peak 1 and peak 2. Voltages were interpolated to the entire scalp surface using spherical splines[25] and back-projected to their original locations on a model baby head using the colour scale shown at bottom left. Contour lines were spaced every 1.5 microV.

repetitions. However, when the deviant syllable was introduced, peak 2 recovered to at least its original level (Fig. 2). Thus, contrary to peak 1, the decrease of peak 2 with repeated stimuli was syllable-specific, and a change in the initial consonant was sufficient to elicit a complete recovery. The neural generators of peak 2 were clearly sensitive to phonetic information, indicating that a single instance of a novel syllable could be recognized in less than 400 ms by the infant brain. Further studies should verify if peak 2 recovery is driven by a categorical perception of phonemes[1], or if it would be elicited by any acoustical change, even within a given phonemic category.

The topography of peak 2, like that of peak 1, showed a polarity inversion along the anterior-posterior axis, suggesting temporal lobe generators. The anterior positivity, however, was more medial for peak 2 than for peak 1 (Fig. 3), confirming that these functionally distinct components originated from distinct brain areas. A forward dipole modelling algorithm[12] suggested that the generators of peak 2 were more posterior, superior and tangential than those of peak 1. Precise localization must remain tentative because of the simplistic assumptions of dipole reconstruction methods.

The anatomy of the infant brain shows hemispheric asymmetries comparable to those found in adults, notably in the temporal lobes[13 15]. Here there were noticeable ERP asymmetries. The amplitudes of peak 1 and peak 2 were significantly larger over left than over right posterior electrodes, as was the size of the recovery of peak 2 to deviant syllables (Fig. 2). This may reflect a functional asymmetry indicating superior processing of short syllables in the left hemisphere. However, ERP summation might also be affected by asymmetries in brain morphology, for instance in the orientation of the left and right sylvian fissures[15]. It should be noted that in individual subjects, considerable variability was found in the size and sometimes the direction of asymmetries. This suggests at most a moderate left-hemispheric advantage for language in infants rather than a sharp division of functions. It may explain why previous studies of hemispheric lateralization in infants have not always reported a noticeable left-hemisphere advantage[16 19].

After the fifth syllable of each trial, ERPs were recorded for a longer period of 1496 ms during which no further stimuli were presented. During this period, a late frontal negativity was observed from 680 ms to 1080 ms following deviant but not standard syllables (Fig. 1). It showed a widespread topography over the left and right frontal electrodes, with no significant left-right asymmetry. A similar anterior negativity has been observed by others in response to unexpected visual and auditory stimuli[20 23]. This suggests that the relevant parameter for eliciting this component is the novelty of the stimuli regardless of their input modality. Thus, two- to three-month-old infants may already possess a supramodal anterior network for novelty detection, perhaps involving arousal and attention-orienting processes[23-24], and which can be activated in less than one second.

The fact that young infants can discriminate phonemes and react to novelty was long known from behavioural methods. High-density ERPs, however, go beyond the simple listing of infants' abilities by permitting the decomposition of complex capacities into a series of processing steps, whose duration and brain implementation can be estimated. The method, although still lacking in spatial resolution, should provide new windows into the brain mechanisms and the fine temporal sequence of cognitive development processes.

Received 23 March; accepted 1 June 1994.

1. Eimas, P. D., Siqueland, E. R., Jusczyk, P. W. & Vigorito, J. Science 171, 303-306 (1971).
2. Best, C. T., McRoberts, G. W. & Nomathemba, M. S. *J. exp. Psych, hum. Percept. Perform.* 13, 345-360 (1988).
3. Eimas, P. D., Miller, J. L. & Jusczyk, P. W. in *Categorical Perception* (ed. Hamad, S.) 161-188 (Cambridge Univ. Press, New York, 1987).
4. Werker, J. F. & Tees, R. C. *Infant Behav. Dev.* 7, 49-63 (1984).
5. Kuhl, P. K., Williams, K. A., Lacerda, F., Stevens, K. N. & Lindblom, B. Science 255, 606-608 (1992).
6. Jusczyk, P. W. in Measurement *of Audition and Vision in the First Year of Postnatal Life: A Methodological Overview* (eds Gottlieb, G. & Krasnegor, N. A.) 195-222, (Ablex, Norwood, NJ, 1985).
7. Tucker D. *Electroenceph. din. Neurophysiol.* 87, 154-163 (1993).
8. Woods, D. L. & Elmasian, R. Electraencep/i. *din. Neurophysiol.* 66, 447^t59 (1986).
9. Naatanen, R. & Gaillard, A. W. K. in Tutorials in Event-related *Potential Research: Endogenous Component* (eds Gaillard, A. W. K. & Ritter, W.) 119-142 (North-Holland, Amsterdam, 1983).
10. Naatanen, R. Behav. Brain Sci. 13, 201-288 (1990).
11. Novak, G. P., Kurtzberg, D., Kreuzer, J. A. & Vaughan, H. G. Jr Electroencepn. *din. Neurophysiol.* 73, 295-305 (1989).
12. Scherg, M. & Berg, P. *BESA: Brain Electrical Source Analysis* Handbook (Max-Planck Institute for Psychiatry, Munich, 1990).
13. Witelson, S. F. & Pallie, W. Brain 96, 641-646 (1973).
14. Chi, J. G., Dolling, E. C. & Gilles, F. H. Ann. *Neurol.* 1, 86-93 (1977).
15. LeMay, M. in *Cerebral Dominance* (ed Geschwind, N. & Galaburda, A. M.) 26-42 (Harvard Univ. Press, Cambridge, MA, 1984).
16. Bertoncini, J. *et al. Brain Lang.* 37, 591-605 (1989).
17. Vargha-Khadem, F. & Corballis, M. Brain *Lang. 8,* 1-9 (1979).
18. Best, C. T., Hoffman, H. & Glanville, B. B. Percep. Psychophys. 31, 75-85 (1982).
19. Molfese, D. L. & Burger-Judisch, L. M. in *Cerebral Laterality: Theory and Research* (ed. Kitterle, F. L.) 71-102 (Erlbaum, Hillsdale, NJ, 1991).
20. Courchesne, E. in *Tutorials in Event-related Potential Research: Endogenous Component* (eds Gaillard, A. W. K. & Ritter, W.) 329-344 (North-Holland, Amsterdam, 1983).
21. Kurtzberg, D., Stapells, D. R. & Wallace, I. F. in *Identification of Infants with Developmental Disabilities* (eds Vietze, P. M. & Vaughan, H. G.) 160-180 (Allyn & Bacon, New York, 1988).
22. Mills, D. L., Coffey-Corina, S. A. & Neville, H. J. *J. Cogn. Neurosd.* 5, 317-334 (1993).
23. Nelson, C. A. & deRegnier, R. Devi *Neuropsychol.* 8, 119-134 (1992).
24. Rothbart, M. K., Posner, M. I. & Boylan, A. in Trie Development *of Attention: Research and Theory led.* Enns, J. T.) 47-66 (Elsevier North-Holland, Amsterdam 1990).

25. Perrin, F., Pernier, J., Bertrand, D. & Echallier, J. F. *Electroenceph. ctin. Neurophysiol.* 72, 184-187 (1989).

ACKNOWLEDGEMENTS. We thank P. Compton, E. Gorr, D. Houston, L McDougall, M. Murias, D. Perdue, T. Sumera and L. Thomas-Thrapp for technical help and M. Posner and D. Tucker for discussions. This study was supported in part by INSERM (G.D., S.D.), CNRS (S.D.) the NSF (S.D.), the Fyssen Foundation (G.D.) and the Societe de Medecine Perinatale (G.O.) and by grants from the W.H. Keck Foundation and by the James S. McDonnell Foundation and Pew Memorial Trust support for the Center for the Cognitive Neuroscience of Attention.