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Asymmetrical Brain Activity Discriminates Between Positive and Negative Affective Stimuli in Human Infants

Abstract. *Ten-month-old infants viewed videotape segments of an actress spontaneously generating a happy or sad facial expression. Brain activity was recorded from the left and right frontal and parietal scalp regions. In two studies, infants showed greater activation of the left frontal than of the right frontal area in response to the happy segments. Parietal asymmetry failed to discriminate between the conditions. Differential lateralization of the hemispheres for affective processes seems to be established by 10 months of age.*

Data derived from both clinical and normal adult samples suggest that certain regions of the two cerebral hemispheres are differentially lateralized for the processing of positive and negative emotional stimuli (*1–7*). This claim is based on (i) neuropsychological findings with brain-damaged patients (*1, 2*); (ii) administration of sodium amytal to patients before neurosurgery (*3*); (iii) the study of lateralized signs and the administration of electroconvulsive therapy to patients with affective disorders (*4*); (iv) studies on normal adults using behavioral indices of asymmetric hemispheric processing (*5*); and (v) electrophysiological studies on normal adults in response to emotional stimuli (*6*). Our view underscores the importance of rostral-caudal differences in hemispheric specialization for affect (*8, 9*). We hold that certain regions of the left hemisphere are specialized for the processing of particular positive affective stimuli while corresponding regions of the right are specialized for the processing of particular negative affective stimuli (*8, 9*). Recent electrophysiological evidence suggests that the locus of this asymmetry is the frontal lobes (*6, 10*).

Although few data are available on the neural substrates of affective development during the first year of life, evidence exists on the development of emotional expression during this period (*11–13*). By 7 to 9 months of age, the infant exhibits a range of both positive and negative affects in response to a variety of specific eliciting situations (*11*). By 7 months of age infants can discriminate among a wide range of facial affects (*12, 13*).

To explore the relation between the ontogeny of affective response systems and cerebral asymmetry, we studied electroencephalographic (EEG) asymmetry in 10-month-old infants in response to positive and negative affective stimuli. By 10 months, infants are capable of both discriminating and expressing positive and negative affect and therefore should exhibit the frontal asymmetry observed in adults. We now report evidence of differential frontal activation asymmetries in response to videotaped presentations of happy and sad facial expressions to 10-month-old infants.

In study 1, 18 healthy female infants ($\bar{X}$ age = 308.1 days, standard deviation = 15.01) born to right-handed parents (*14*) were seen, and artifact-free EEG data on ten subjects were obtained (*15*).

The infant sat in her mother's lap facing a 21-inch diagonal video monitor 45 inches away. The EEG was recorded from the left and right frontal and parietal regions (F3, F4, P3, and P4) with all channels referred to a common vertex (Cz) (*16*) and stored on separate channels of FM tape. Both frontal and parietal regions were recorded because we wished to compare asymmetries in two major cortical association regions (*8, 17*).

The positive and negative affective stimuli consisted of a videotape of an actress generating either a happy or sad facial expression (*18*). Half the infants viewed the happy face first and half the sad face. The audio portion was edited out for both segments.

Artifact-free epochs of EEG were filtered (with cut-offs of 48 dB per octave) for activity within the band between 1 and 12 Hz, integrated, and digitized (*19*). Raw data (in microvolt-seconds) and laterality ratio scores [(R − L)/(R + L), EEG activity (1 to 12 Hz)] were the two dependent measures (*20*).

We first compared happy with sad epochs on the frontal laterality ratio score [(F4 − F3)/(F4 + F3)]. Happy epochs elicited greater relative left frontal activation than sad epochs [$F(1, 9) = 5.88$, $P = .039$] (Table 1). Seven subjects showed higher frontal ratio scores during happy versus sad epochs and two showed equal ratio scores. The parietal ratio score failed to discriminate between epochs [$F(1, 7) = 2.52$].

In order to disentangle the separate contributions of the left and right hemispheres to the frontal asymmetry between the conditions, an analysis of variance was computed on the raw EEG data. A significant condition by hemisphere interaction was obtained [$F(1, 9) = 6.20$, $P = .035$] (Table 2). Happy epochs elicited less activity (1 to 12 Hz) in the left than the right frontal region ($P < .05$, Scheffé test). No difference between the hemispheres in the frontal region was obtained in response to the sad stimuli.

We performed a second study using the identical stimuli and EEG recording and analysis procedure in order (i) to replicate study 1 and (ii) to restrict EEG data analysis to those epochs during

0036-8075/82/1217-1235$01.00/0

Table 1. Mean (± standard deviation) frontal laterality ratio scores by condition. Higher numbers indicate greater relative left-side activation.

Study	N	Condition		F	P
		Happy	Sad		
1	10	0.021 ± 0.051	−0.001 ± 0.032	5.88	.039
2	14	0.073 ± 0.100	0.032 ± 0.115	7.63	.017

Table 2. Mean (± standard deviation) integrated frontal activity (in microvolt-seconds based on a 5-second epoch).

Study	N	Hemisphere			
		Left		Right	
		Happy	Sad	Happy	Sad
1	10	65.63 ± 16.27	74.90 ± 15.92	67.80 ± 14.11	74.47 ± 14.02
2	14	49.16 ± 13.83	51.54 ± 14.75	56.21 ± 11.35	53.97 ± 10.16

which the infant was fixating on the video monitor.

Twenty female infants (310.13 ± 12.33 days) were selected as in study 1. Artifact-free data were obtained on 14 subjects. A video camera with a close-up lens was positioned behind the video monitor and focused on the infant's face. An observer who was unable to see the video monitor on which the affective stimuli were presented depressed a button whenever the infant was fixating on the monitor. The EEG was analyzed only for artifact-free epochs during which the infant was fixating on the monitor.

Happy epochs again elicited greater relative left frontal activation than sad epochs (Table 1). Eleven subjects showed higher laterality ratio scores during happy versus sad epochs. The parietal ratio score again failed to discriminate between conditions [$F(1, 13) = 2.09$] (*21*).

In the frontal areas, condition and hemisphere interacted significantly [$F(1, 13) = 9.09$, $P = .01$] (Table 2). As in study 1, happy epochs elicited less activity in the left than in the right frontal region ($P < .01$, Scheffé test) (*22*).

The data from both studies demonstrate that infants as young as 10 months of age show greater relative left frontal activation in response to happy than to sad video stimuli. The laterality finding is specific to the frontal region: parietal recordings did not discriminate between conditions. Although the infants' electrocortical response to the happy and sad stimuli differed, the functional significance of this difference is yet to be characterized. Some have suggested that the essential affective difference between the hemispheres is based on approach and avoidance (*23*). Developmental research would suggest that a 10-month-old infant is likely to exhibit both approach and avoidance behaviors in particular situations as a function of the facial expression of its mother (*12*). Thus, the observed frontal activation asymmetry may reflect the differential tendency of the affective stimuli to elicit approach or avoidance behavior.

These data empirically validate one aspect of a more general theory relating the ontogeny of affective experience and expression to the maturation of certain higher cortical functions (*9*). They also extend the findings on affective lateralization in adults and demonstrate such asymmetries in the first year of life.

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10. Recent neuropsychological findings on brain-damaged patients also underscore the importance of the rostral-caudal differences and indicate that the anterior cortical regions are where differential lateralization for certain positive and negative emotions is found (*2*).
11. By 9 months of age, infants show increased wariness and distress to the deep side of the visual cliff [J. J. Campos, S. W. Hiatt, D. Ramsey, C. Henderson, M. Svejda, in *The Development of Affect*, M. Lewis and L. Rosenblum, Eds. (Plenum, New York, 1978)] as well as to stranger's approach in the absence of a primary caretaker [J. J. Campos, R. N. Emde, T. Gaensbauer, C. Henderson, *Dev. Psychol.* **11**, 589 (1975); L. A. Sroufe, *Child Dev.* **48**, 731 (1977)]. Across a variety of cultures and rearing conditions, protest to separation from the primary caretaker increases between 7 and 9 months of age [J. Kagan, *Am. Sci.* **64**, 186 (1976)]. The social smile and expressions of joy and surprise are reliably elicited by 5 months of age in a number of different stimulus situations [S. W. Hiatt, J. J. Campos, R. N. Emde, *Child Dev.* **50**, 1020 (1976)].
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18. Each segment consisted of a 15-second neutral pose followed by 90 seconds of an affective expression. After the neutral pose, the actress spontaneously broke into either smiling and laughter (happy segment) or frowning and crying (sad segment). The affective segments were flagged by an observer who pressed a button when the face changed from a neutral to an emotional expression. We embedded the affective sequence (happy-sad or sad-happy) between identical portions of *Sesame Street* in order to capture the infant's attention. The intersegment interval was 90 seconds.
19. We examined activity between 1 and 12 Hz for three reasons: (i) Most of the activity in infant EEG is below 12 Hz. This frequency range therefore indicates whole-band activity. (ii) Research in adults indicates that suppression of whole-band activity reflects cortical activation [D. B. Lindsley and J. D. Wicke, in *Bioelectric Recording Techniques*, R. Thompson and M. N. Patterson, Eds. (Academic Press, New York, 1974), vol. 1B] since the major portion of the variance in whole-band activity is a function of changes in the alpha band [J. C. Doyle, R. Ornstein, D. Galin, *Psychophysiology* **11**, 567 (1974)]. (iii) Little is known about which specific frequency components of the infant EEG are most responsive to task effects.
20. The laterality ratio score has been commonly used in EEG asymmetry studies [see D. Galin, in *The Neurological Basis of Language Disorders in Children: Methods and Directions for Research*, C. L. Ludlow and M. R. Doran-Quine, Eds. (Government Printing Office, Washington, D.C., 1979)].

21. Experiment 2 produced greater overall frontal activation (Table 2) and greater relative left-frontal asymmetry (Table 1). The only difference between studies 1 and 2 was that the EEG epochs analyzed in the latter were those during which the infant was fixating on the video monitor. The lesser overall activity in study 2 is likely to be a function of the infant's visual attention, which is associated with generalized alpha blocking [for example, O. Creutzfeldt, G. Grunewald, W. Siminova, H. Schmitz, in *Attention in Neurophysiology*, C. R. Evans and T. B. Mulholland, Eds. (Butterworth, London, 1969)]. The greater relative left-frontal asymmetry (across condition) in study 2 may be a function of greater interest displayed during periods of fixation to the video stimulus. The association of interest with approach behavior [C. E. Izard, *Human Emotions* (Plenum, New York, 1977)] and of approach behavior with left-frontal activation have also been proposed (*8, 17*). Differences in the predicted direction in relative frontal asymmetry were obtained irrespective of the differences in overall amplitude between the two studies.

22. The duration of visual fixation to the happy epoch was 48.15 ± 19.61 seconds and to the sad, 62.36 ± 11.46 seconds [$t(13) = 2.34$, $P = .04$]. The longer fixation to the sad stimulus, possibly reflecting greater interest, may have accentuated left-frontal activation during this segment. Despite this difference in visual fixation, asymmetries in frontal activation in the predicted direction were still obtained.
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Substantia Nigra: Site of Anticonvulsant Activity Mediated by γ-Aminobutyric Acid

Abstract. *Localization of the anatomic substrate for anticonvulsant activity mediated by γ-aminobutyric acid (GABA) was examined using intracerebral injections of GABA agonists. Blockade of tonic hindlimb extension in the maximal electroshock test and blockade of tonic and clonic seizures produced by pentylenetetrazole and bicuculline were obtained by elevating GABA in the ventral midbrain tegmentum. Elevation of GABA in forebrain and hindbrain areas had no effect on convulsant activity. Blockade of tonic and clonic seizures was also obtained after microinjections of the direct GABA receptor agonist, muscimol, into the midbrain. The substantia nigra was identified as the critical midbrain site for GABA-mediated anticonvulsant activity. Local injection of GABA agonists into the midbrain provided seizure protection without a widespread augmentation of GABA-mediated activity throughout the brain and without impairing either alertness or motor function. Synapses in the substantia nigra appear to represent an important control mechanism for inhibiting the propagation of generalized convulsions.*

The inhibitory neurotransmitter γ-aminobutyric acid (GABA) has been implicated in both the generation (*1*) and blockade (*2, 3*) of seizure processes. Little is known, however, about anatomic sites at which GABA-dependent transmission might influence seizure propagation (*4*).

We undertook a series of studies (i) to establish whether drug-induced elevation of GABA in a selected region of the brain confers protection from generalized motor seizures, (ii) to evaluate the anticonvulsant activity of a direct-acting GABA receptor agonist applied locally into selected brain regions, and (iii) to identify a specific nucleus in which GABA-mediated synapses may control seizure activity. We found that protection from seizures induced by maximal electroshock or by convulsant drugs was obtained as a result of GABA elevation or direct stimulation of GABA receptors within a restricted region of the ventral midbrain tegmentum, the substantia nigra.

We first microinjected γ-vinyl-GABA (GVG) into several brain regions; GVG is an irreversible catalytic inhibitor of GABA transaminase, previously shown to have anticonvulsant effects (*3, 5*). The drug was applied intracerebrally through a 26-gauge cannula positioned stereotaxically into ether-anesthetized male Sprague-Dawley rats.

The injection sites are illustrated schematically on a parasagittal section of rat brain (Fig. 1B). Anticonvulsant activity was assessed with the maximal electroshock seizure test. The seizures consist of a sequential tonic flexion and extension of the fore- and hindlimbs; blockade of the tonic hindlimb extension is the customary index of anticonvulsant activity (*6*). The animals were tested at 6 hours and at 24 hours after GVG microinjection. At 6 hours, significant anticonvulsant activity was obtained only from injections placed in the midbrain tegmentum; this effect was still present at 24 hours (Fig. 1C).

By measuring the increase in GABA content of a number of different brain areas, we assessed the effective diffusion of GVG for each of the groups receiving microinjections (Fig. 1A). All injections produced marked increases in GABA in the immediate vicinity of the injection site. At 6 hours after GVG injection, areas sampled within a 2- to 3-mm radius of the injection site exhibited the greatest increases in GABA content (*7*). The re-

Table 1. Seizure protection after bilateral microinjection of GVG and muscimol into ventral midbrain tegmentum. The values for maximum electroshock seizure (MES) represent the duration of tonic hindlimb extension in seconds. The values for PTZ and bicuculline are derived from a scale that we use to rate the severity of behavioral seizures (*23*). The doses of chemoconvulsants used (PTZ, 40 mg/kg, intravenously; bicuculline, 0.3 mg/kg, intravenously) produced severe, explosive clonic seizures in all controls; tonic forelimb extension was observed in at least 80 percent of these animals. Column headings indicate time between microinjection and seizure test. Rats weighing 300 to 350 g were used in the chemoconvulsant tests. PTZ (Knoll) was diluted in saline and injected intravenously in a volume of 0.1 to 0.15 ml/100 g. Bicuculline was dissolved in a small volume of concentrated HCl and diluted with saline; the *p*H was adjusted to 5.8 with NaOH, and the solution was kept on ice to minimize loss of activity (*24*). The values are means ± standard errors for four to eight rats per group.

Microinjection	Seizure severity score						
	MES			PTZ			Bicuculline, 6 hours
	2.5 hours	5 hours	8 hours	2.5 hours	5 hours	8 hours	
Saline	4.8 ± 0.2	6.5 ± 0.7	6.1 ± 0.5	2.3 ± 0.2	2.6 ± 0.2	2.8 ± 0.3	2.1 ± 0.3
GVG, 5 μg		0.6 ± 0.6*†			1.2 ± 0.4*†		0.5 ± 0.2
Muscimol							
25 ng	1.8 ± 0.6*	4.8 ± 2.1		1.0 ± 0.2*	1.3 ± 0.9		
50 ng	0.0 ± 0.0*	3.8 ± 1.5*	6.0 ± 0.7			2.6 ± 0.4	
75 ng		0.7 ± 0.7*					

*Significantly different from saline-injected controls ($P < .05$; Student's *t*-test). †Measured at 6 hours after injection.

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