

zures. Deep cortical pyramids send short axon collaterals in part into terminal fields in cortical layer IV (10) and long corticofugal fibers to VL or VB thalamic neurons. The transmitter released by pyramidal cells is probably glutamate (11), which in high concentration causes swelling of dendrites and swelling or dark cell degeneration of neuronal cell bodies (8, 12). We suggest that swelling of dendrites in the thalamus and dark cell degeneration in both the cortex and thalamus reflect a seizure-induced, excitotoxic process mediated by excessive release of glutamate at synaptic receptors. Such damage to local inhibitory interneurons could release control over neighboring cortical pyramids (13) and disrupt the temporal and spatial containment of the focus. Permanent excitotoxic damage to cortical interneurons could underlie the loss of γ -aminobutyric acid-dependent inhibition found in other models of focal epilepsy (14). Excessive release of glutamate at receptor sites in the thalamus (motor cortex \rightarrow VL; sensory cortex \rightarrow VB) would have excitotoxic consequences for the dendritic structures containing these receptors as well.

Swelling of axon terminals in the cortex cannot be explained by a glutamate-mediated mechanism. A clue to the cause of this morphological change may lie in evidence that thalamocortical axons that project to layer IV are driven both ortho- and antidromically in the course of focal sensorimotor seizures (2). Excessive epileptic firing in these terminals might cause a focal derangement in ionic or metabolic membrane homeostasis that would be manifested as acute swelling.

Our findings support the thesis that sustained seizures can result in neuronal degeneration in the seizure focus and elsewhere within seizure pathways by mechanisms other than anoxia. If an excitotoxic mechanism underlies important components of the neuropathology of experimental seizures, it is possible that brain damage in human epilepsy may in part have a similar basis. If so, the recent discovery of agents that powerfully block both the excitatory and neurotoxic actions of glutamate excitotoxins provides hope for the chemoprophylactic approach to the management of epilepsy-related brain damage (15).

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7. We used a dental drill to make a burr hole (3 mm in diameter) in the dura and applied dental acrylic to build a well on the calvarium surrounding the burr hole. Previous studies with 14 C-labeled penicillin infused into the well have revealed that the convulsant diffuses locally into upper cortical layers immediately beneath the dura coincident with the onset of seizures. A minimal amount diffuses a few millimeters laterally over the surface, but does not penetrate deeper than layer I [R. C. Collins and T. V. Caston, *ibid.* **6**, 117 (1979)].
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10. Pyramidal tract neurons of motor cortex send recurrent collaterals primarily into layers V and VI [P. Landry, A. Labelle, M. Deschenes, *Brain Res.* **191**, 327 (1980); J. P. Donoghue and S. T. Kitai, *J. Comp. Neurol.* **201**, 1 (1981)], but studies in visual cortex suggest that recurrent collaterals in sensory areas have dense projections into layer IV [R. W. Baughman and C. D. Gilbert, *J. Neurosci.* **1**, 427 (1981)].
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Discrimination and Imitation of Facial Expressions by Neonates

Abstract. *Human neonates (average age, 36 hours) discriminated three facial expressions (happy, sad, and surprised) posed by a live model as evidenced by diminished visual fixation on each face over trials and renewed fixations to the presentation of a different face. The expressions posed by the model, unseen by the observer, were guessed at greater than chance accuracy simply by observing the face of the neonate, whose facial movements in the brow, eyes, and mouth regions provided evidence for imitation of the facial expressions.*

Facial expressions of emotions such as happiness, sadness, and surprise have been observed in the very young infant (1) and in several cultures (2). Because of their early appearance and their apparent universality, these basic facial expressions may reflect innate processes (3). We have investigated whether neonates can discriminate and imitate these facial expressions. Projected photographs of facial expressions are discriminated as early as 3 months of age in a visual habituation paradigm (4). The young infant is also physically capable of reproducing these expressions; all but one of the discrete facial muscle actions of adults have been identified in the neonate (5). Although a debate continues on what processes may be involved (6), imitations of facial movements such as lip protrusion, mouth widening, and tongue thrusting have been reported for 12- to 21-day-old babies (7). We now

have evidence for both the discrimination and imitation of facial expressions at an even younger age, shortly after birth.

In this study, a series of three facial expressions (happy, sad, and surprised) were modeled by an adult for 74 neonates (mean age, 36 hours) (8). The model held the neonate upright with the newborn's head supported in one hand and torso in the other hand. The two faces were separated by approximately 10 inches. The neonate's visual fixations on the adult's face and the neonate's facial movement patterns were recorded by an observer who stood behind the model in order to see the infant's face but remained unaware of the expression being modeled. Split-screen videotaping of the neonates' and model's faces provided checks on the reliability of coding by observer and face presentation by model (9).

To sustain alertness and to elicit the

neonate's visual fixations on the model's face, the model provided vestibular stimulation (two deep knee bends) and auditory stimulation (two tongue clicks) prior to each trial. The model then fixed a happy, sad, or surprised expression on her face. Three series of trials or one series for each face were presented in a counter-balanced Greco-Latin-square order to control for state change effects. Face 1 was sustained in a fixed position until the infant looked away from the model's face, at which time the model re-elicited the neonate's visual fixation with vestibular and auditory stimulation. Face 1 trials were repeated until the neonate looked at that face for less than 2 seconds. Face 2 and face 3 trials were

then presented according to the same procedure (Fig. 1).

For each trial, the observer coded on a paper grid (i) total time per trial; (ii) predominant target and pattern of neonatal visual fixation per trial on the model's eyes, mouth, or alternately on the eyes and mouth; (iii) the presence of specific mouth movements of the neonate, including widening of the lips (as in a happy face), tight and somewhat protruded lips (pouting or sad face), wide opening of the mouth (as in a surprise face), or tongue protrusion; (iv) presence or absence of eye widening (as in a surprise face); (v) presence of relaxed or furrowed brow (as in happy or sad face, respectively); and (vi) observer's guess

as to which expression was being modeled.

Because we used a trials-to-criterion procedure yielding a different number of trials per expression per infant (range, 4 to 15; mean, 5.8), the number of trials during which these movements occurred was converted to the proportion of the total number of trials presented for each facial expression. For the same reason, the trials were divided in thirds (early, middle, and late) for analyses of the visual habituation data. Repeated measures analyses of variance were then conducted with the order of trials (3) as a between-subjects measure and the facial expressions modeled (3) as a repeated measure. There were no significant effects of order of trials or type of facial expression in the habituation-dishabituation. Visual fixations significantly decreased from the middle to late trials [mean decrease, 11.9 seconds, $F(2, 142) = 5.49$, $P < .005$] and significantly increased from the late trials of the facial expression to the early trials of the subsequent expression [mean, 8.1 seconds, $F(2, 142) = 5.81$, $P < .005$]. Thus, the visual habituation and dishabituation of the facial stimuli suggest that neonates can discriminate at least these three basic facial expressions (10).

The neonate visually fixated the mouth region and alternately looked at the mouth and eye regions for a greater proportion of the trials than the eye region, irrespective of the facial expression being modeled (11). The neonate's alternating fixations on the mouth and the eye region occurred during a greater proportion of the surprise expression trials than for the other facial expressions [$F(2, 142) = 4.74$, $P < .01$]. Fixations on the mouth region occurred for a greater proportion of trials during happy and sad than surprised expressions [$F(2, 142) = 3.26$, $P < .05$]. The model's surprise expression featured salient eye and mouth positions (both widened), whereas the happy and sad expressions were characterized primarily by mouth positions—widening of the lips for the happy expression and tightened, protruding lips for the sad expression (Fig. 1). These differential visual fixation patterns suggest that the neonate can perceive distinctive features of these facial expressions: of the mouth in happy and sad faces, and of both the mouth and eyes in the surprise expression.

Figure 2 depicts the proportion of trials on which differential mouth movements were observed during the different face trials. Because of the problem posed by different baseline frequencies, the distributions of each behavior were ana-

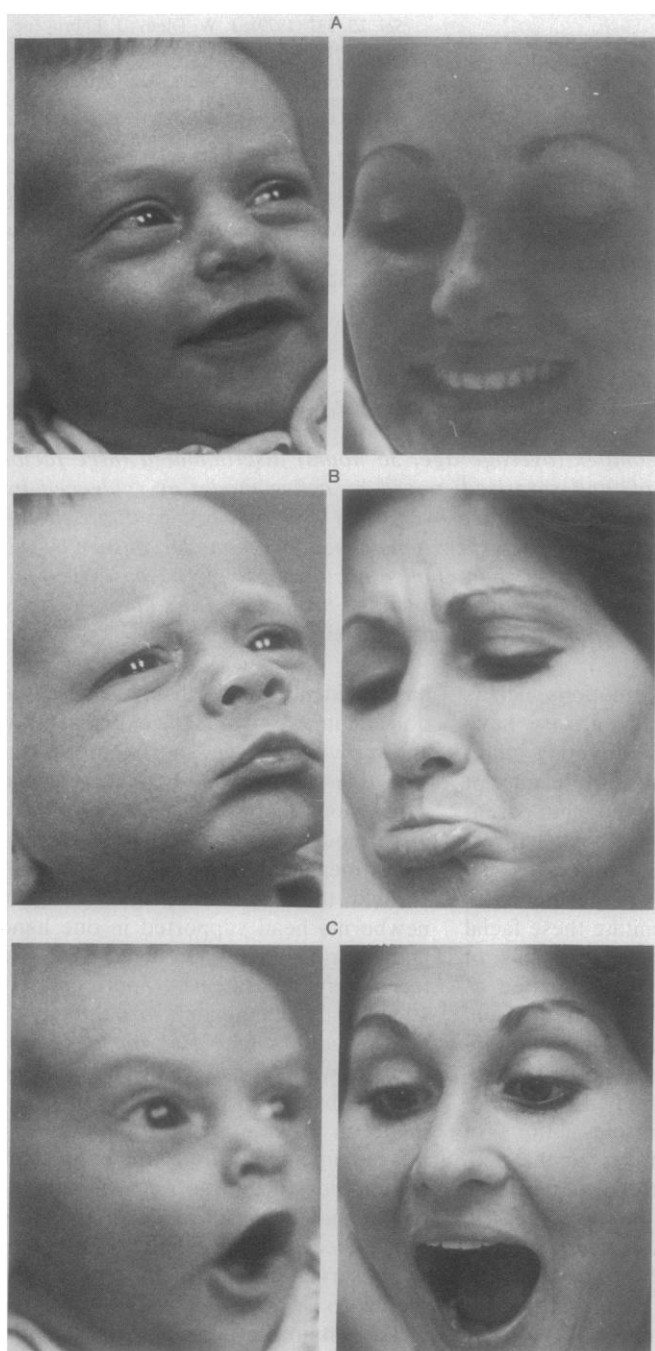


Fig. 1. Sample photographs of a model's happy, sad, and surprised expressions and an infant's corresponding expressions.

lyzed separately across the different expressions modeled (7). There were no differences in the proportion of trials that tongue protrusion occurred as a function of different facial expressions ($P > .05$). However, widened eyes and wide mouth opening occurred for a greater proportion of surprise than other face trials [$F(2, 142) = 3.97, P < .05$ and $11.49, P < .001$, respectively]. Lip widening occurred more frequently during happy face trials [$F(2, 142) = 3.41, P < .05$], and tightened-mouth-protruding-lips [$F(2, 142) = 3.41, P < .05$] and furrowed brow [$F(2, 142) = 10.16, P < .001$] occurred more frequently during sad expression trials. Comparison of the occurrence of these facial movements across the expressions for which those movements would not be expected (omitting the imitative expression movements corresponding to the modeled expressions) yielded no significant differences. This finding provides additional support for the notion that the neonate's facial movements that simulate those of the model are attributable to imitation. An analysis of early, middle, and late trials data for these expressions revealed that a greater proportion of these expressions occurred during the middle trials [$F(2, 142) = 5.29, P < .01$], suggesting that these were not arousal responses or fixed action patterns (12).

Analyses of the observer's guesses included only those data for each subject's first series of trials. The chance probability of correctly guessing the facial expression would be 33 percent. The surprise facial expressions were correctly guessed 76 percent of the trials, at a significantly greater than chance level ($\chi^2_{(24)} = 65.23, P < .001$). Surprise expressions were correctly guessed more often than happy (58 percent) or sad expressions (59 percent). However, the happy ($\chi^2_{(23)} = 35.76, P < .05$) and sad expressions ($\chi^2_{(24)} = 38.41, P < .05$) were also correctly guessed. The surprise expression featured two salient features in two regions (eyes and mouth). When the infant reproduces both of these features, the probability of an accurate guess by the observer was increased.

These results suggest that the neonate is capable of discriminating at least these three different facial expressions. Ensuring

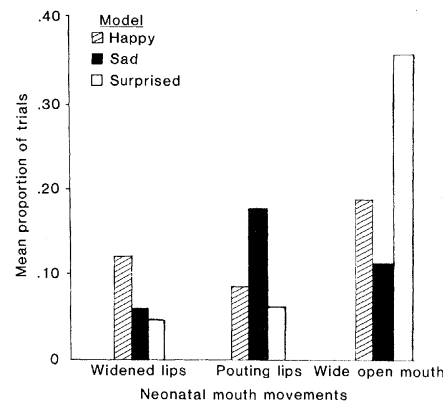


Fig. 2. Mean proportion of trials during which different neonatal mouth movements occurred as a function of facial expression modeled. Mouth movements include widened lips (as in a happy expression), pouting lips (sad), and wide open mouth (surprised). Proportions do not total 100 because these discrete movements occurred predominantly during the middle trials (approximately 39 percent of the trials).

ing that the infant was alert and using a trials-to-criterion habituation procedure so that each infant could process the information at his own pace may have facilitated the demonstration of facial discriminations.

The imitative expressions provide support for Meltzoff and Moore's data (7) on imitative gestures by 12- to 21-day-old infants. They suggested three potential underlying mechanisms: shaping of the response by the model, an innate releasing mechanism, and the neonate's capacity to integrate visual and proprioceptive information. Consistent with their conclusions, the videotapes of the model's behaviors suggest that shaping or reinforcing the neonate's responses did not occur. Although "fixing" a face is not easy and may produce some muscle movement, there were no discernible movements on the model's face. That these imitations might be based on an innate releasing mechanism or fixed action pattern is also unlikely (13) given the organization and lack of stereotypy of the infants' differential responses to these three different facial expressions. Instead, we favor the view of Meltzoff and Moore (7) that there is an innate ability to compare the sensory information of a visually perceived expression (as evidenced in this study by their ability

ty to discriminate the facial expressions) with the proprioceptive feedback of the movements involved in matching that expression (as manifested by their differential responses to the facial expressions).

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8. All infants were full term (mean, 39.4 weeks), of normal birth weight (mean, 3296 g), and experienced an uncomplicated vaginal delivery with a minimum of obstetric medication and no general anesthesia. Two different adults served as models.
9. Reliability figures are based on the tapes of 12 infants and calculated by Kappa [J. J. Bartko and W. T. Carpenter, *J. Nerv. Ment. Dis.* **163**, 307 (1976)]; total time per trial (.97); predominant pattern visual fixation (.91); mouth position (.82); eye position (.86); brow position (.83); and guess (.76).
10. Mean (\pm standard deviation) fixation times (in seconds): early trials, 12.4 ± 9.7 ; middle trials, 16.2 ± 11.2 ; and late trials, 4.3 ± 2.9 .
11. Mean proportion of trials with fixations on eyes: .13 (happy model), .15 (sad), and .23 (surprise); on mouth: .32 (happy), .40 (sad), and .23 (surprise); and on both eyes and mouth: .23 (happy), .29 (sad), and .45 (surprise).
12. As Meltzoff and Moore (7) have suggested, if each infant's response to one expression is compared with his response to another similar expression demonstrated by the same adult at the same distance from the infant and under the same conditions, and if differential imitation occurs, it cannot be attributed to a mere arousal of activity by a human face.
13. S. W. Jacobson's data [*Child Dev.* **50**, 425 (1979)] suggest that a fixed action pattern mechanism may explain the matching behavior demonstrated by Meltzoff and Moore (7). Although A. P. Burd and A. E. Milewski (paper presented at the biennial meeting of the Society for Research in Child Development, Boston, 2 to 5 April 1981) were unable to replicate Jacobson, this failure may be due to subtle differences in procedure relating to infant state or position of the eliciting stimulus.

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