

Inhibition of Return in Newborn Infants

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Inhibition of return is a reduced tendency to orient toward a previously attended spatial location, which, in adults, likely reflects an attentional bias toward novel locations. It is indexed by an increased latency and/or a reduction in the probability of an eye movement to the inhibited location. Previous research had indicated that inhibition of return develops between 3 and 6 months of age. We submitted 32 newborns (M age = 72 hours; $SD = 0.0$) to trials consisting of a pretest phase (a single visual cue shown for 3 s at 30° from fixation), a 2-s interval, and a test phase (two identical stimuli shown simultaneously at 30° for 5 s). We recorded the direction and latency of the first eye movement in both the pretest and test phases as well as the nonnutritive sucking rate. The results showed that eye movements occurred more frequently and with a shorter latency toward the uncued side (i.e., inhibition of return). There was also some indication that inhibition of return occurred only when in the pretest phase the infant shifted gaze toward the cue and nonnutritive sucking rate decreased. It was concluded that inhibition of return is present in the first days after birth.

newborns attention sucking novelty inhibition of return orienting vision

Humans can attend to different regions of visual space either by moving the eyes or by covert orienting; that is, in the absence of eye movements. Covert orienting is demonstrated by the fact that speed of response to a visual stimulus (i.e., the target) depends on the spatial relationship between it and other events in the visual field (i.e., the cues) that attract attention (Posner, 1980; Umiltà, 1988). For example, if the target falls in the same location as the cue (valid trials) more often than in other locations (invalid trials), mean reaction time (RT) to detect the target is faster on valid than on invalid trials.

In contrast, Posner and Cohen (1984) discovered a delayed inhibitory effect that begins about 150 ms after the cue so long as attention

is not maintained at the location of the cue. They named this effect *inhibition of return* to indicate that its function is to decrease the likelihood that attention would return to previously attended locations (Klein, 1988).

Posner and Cohen (1984) generated inhibition of return by presenting a peripheral cue that predicted the likely target location and summoned attention. After this, a stimulus was presented that brought attention back to the center of the display. Finally, the target appeared at either the same location as the cue or at a symmetrical location in the opposite visual hemifield. Maylor (1985; also see Kwak & Egeth, 1992; Possamai, 1986; Tassinari, Aglioti, Chelazzi, Marzi, & Berlucchi, 1987) showed that the spatial relationship between the cue and the target still influenced RT, even under circumstances in which the location of the cue did not predict the likely location of the target, and even if no manipulation was used to summon attention back to the center of the display.

A peripheral, nonpredictive cue, therefore, produces a biphasic effect. If the target occurs shortly after the cue (i.e., up to 150 ms), then RT is faster when the target is in the same location as the cue. This initial facilitation of the cued location is attributed to the summoning of attention by the cue. For longer cue-target intervals (i.e., from 200–300 ms to at least 1500 ms), RT is slower when the target appears in the same location as the cue. This subsequent inhibition of the cued location is attributed to inhibition of return.

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INHIBITION OF RETURN AND EYE MOVEMENTS IN ADULTS

Although inhibition of return is clearly linked to covert shifts of attention elicited by peripheral, nonpredictive cues, it does not occur with all movements of visual attention. In particular, it does not occur with endogenously activated (i.e., voluntary) covert shifts of attention in response to symbolic cues (e.g., an arrow in the center of the display that indicates where to expect the forthcoming target; Posner & Cohen, 1984). However, as shown by Posner, Rafal, Choate, and Vaughan (1985), if an arrow cue induces the subject to make a saccade to an eccentric location, and the eyes are then returned back to the initial fixation point, inhibition of return does occur for subsequent target detection at the location to which the saccade had been made.

Thus, inhibition of return is produced by a cue presented in the periphery of the visual field while the eyes do not move or by endogenous activation of a saccadic eye movement. A common mechanism, related to oculomotor preparation, may be involved in both circumstances. The oculomotor system is activated when a voluntary saccade to a peripheral target is executed; but also, a peripheral cue, which summons attention covertly, primes the oculomotor system to prepare a coordinate saccade (Posner & Cohen, 1980).

In accord with this possibility, Rafal, Calabresi, Brennan, and Sciolto (1989) have shown that the necessary and sufficient condition for inhibition of return is the priming of the oculomotor system. Their subjects had inhibition of return for a target location indicated by a central cue when they were required to program an eye movement to that location irrespective of whether the saccade was later executed or aborted. In other words, inhibition of return was caused by endogenously preparing an eye movement even under circumstances in which there is no peripheral cue and in which no eye movement is actually made. In contrast, no inhibition of return was found, with the same cueing conditions, when no eye movement was ever to be made toward the target. It appears, therefore, that there is a clear relation of inhibition of return to eye movement systems.

Inhibition of return has been linked to the posterior attention system (Posner & Peterson, 1990). This is the portion of the covert visual

attention system that is known to be most closely related to the superior colliculus and surrounding midbrain areas (i.e., the retinotectal visual pathway; also see Johnson, 1990). Evidence that inhibition of return is mediated through the retinotectal pathway derives from observations that inhibition of return (a) is impaired by those midbrain lesions that also impair saccade preparation, and (b) dominates in the temporal hemifield under monocular viewing conditions.

Patients with degenerative diseases of the midbrain which abolish vertical saccadic eye movements show no inhibition of return following covert orienting along the vertical dimension (Posner et al., 1985; Rafal, Posner, Friedman, Inhoff, & Bernstein, 1988). In these patients, horizontal saccades are relatively spared, and they do show inhibition of return following covert orienting along the horizontal dimension. In contrast, other patients with attentional and eye-movement disorders, but without extensive damage to the midbrain (e.g., parietal or Parkinson's patients), show normal inhibition of return along both the horizontal and the vertical dimension.

The retinotectal pathway is known to be asymmetrical (Rafal, Henik, & Smith, 1991; Rafal, Smith, Krantz, Cohen, & Brennan, 1990; Schiller, 1985). Connections from the nasal hemiretina (i.e., from the temporal visual hemifield) go directly to the colliculus of the opposite side, whereas connections from the temporal hemiretina (i.e., from the nasal visual hemifield) have reduced direct input to the ipsilateral colliculus. This asymmetry manifests itself also in normal subjects who possess an intact and fully developed geniculostriate pathway. Under monocular viewing conditions, they show a bias to make saccades toward the temporal hemifield (Posner & Cohen, 1980) and faster covert switches of attention when the cues are presented in the temporal rather than the nasal hemifield (Rafal et al., 1991). In accord with the notion that it is mediated through the retinotectal pathway, inhibition of return is more strongly activated by temporal rather than by nasal cues (Rafal et al., 1989, 1990).

In conclusion, the evidence reviewed here shows that (a) priming of the oculomotor system to prepare a saccade is necessary and sufficient to produce inhibition of return, and (b) inhibition of return depends on the activity of

the retinotectal pathway, which is closely related to eye movements.

INHIBITION OF RETURN IN EARLY INFANCY

Recently, several studies have been concerned with the development of inhibition of return in early infancy (see review in Johnson, *in press*). The onset of inhibition of return is of interest because it may index the transition to a developmental stage in which attention can be directed to a target in the periphery of the visual field. In other words, if one regards inhibition of return as an attentional phenomenon, then its development could be viewed as an indicator of a form of attentional control (Rothbart, Posner, & Boylan, 1990).

The studies concerned with the development of inhibition of return have employed different experimental procedures and can be distinguished based on whether the infant was allowed to make a saccade toward the peripheral cue or rather the eyes did not move from the central fixation point during cue presentation. Another important difference is that whereas some studies have used saccade RT to the target as the dependent measure, others have used the direction of the saccade following target presentation. As will be addressed later, there appears to be converging evidence that the mechanisms underlying inhibition of return develop between 3 and 6 months of age.

Boylan Clohessy, Posner, Rothbart, and Vecera (1991) studied inhibition of return in 3-, 4-, 6-, and 18-month-old infants. Once an infant had fixated a central stimulus, a cue was presented to 30° either left or right of fixation. When the infant had made a saccade to the cue, the peripheral cue was turned off, and the central stimulus was turned on again. When the infant was again oriented to the central stimulus, a target was presented simultaneously on both sides. Infants of 3 months of age showed a preferential orienting toward the same side where the cue had appeared. Infants of 4 months showed no preferential orienting. Infants of 6 and 18 months oriented more often toward the side opposite from that where the cue had appeared. The authors argued that this preferential orienting toward the uncued side was indicative of inhibition of return which develops between 4 and 6 months of age.

The procedure employed by Boylan

Clohessy et al. (1991) to study inhibition of return in 12-month-old infants differed from that just described because the unilateral cue was presented very briefly. Actually, it was flashed for 17 ms and was followed 400 ms later by the bilateral target. An eye movement toward the cue was almost never observed, but the inhibition of return produced with this method was similar in magnitude to that found for 6-month-olds with the Boylan Clohessy et al. (1991) paradigm. In the absence of eye movements, one assumes that the cue attracted attention covertly, and the covert shift of attention in turn produced inhibition of return for the subsequent saccade to the target.

Very similar results were obtained for 3- and 6-month-old infants by Hood and Atkinson (1991). They used a 100-ms peripheral cue which was followed either immediately or after an interval of 500 ms, ipsilaterally or contralaterally, by a unilateral target. Because the cue did not cause any eye movements, any effects of the cue on subsequent saccades to the target could be attributed to a covert shift of attention during cue presentation. Six-month-old infants showed faster RTs on no-interval trials, and delayed RTs (i.e., inhibition of return) on 500-ms trials, for saccades when the target appeared on the cued side in comparison with the uncued side. In contrast, 3-month-olds showed no significant effects of the cue in either the no-interval or the 500-ms interval condition. These results were considered to show that the mechanisms underlying inhibition of return (as well as facilitation effects) develop between 3 and 6 months of age.

Hood (*in press*) studied a group of 6-month-olds using a cue which lasted 180 ms and was followed immediately by a single target on either the ipsilateral or the contralateral side. RTs to make a saccade were slower when the target appeared on the same side as the cue (but, see Johnson, *in press*, for a criticism of the procedure which renders it difficult to establish whether the effect was a genuine instance of inhibition of return).

Harman, Posner, and Rothbart (1992) reasoned that the age when inhibition of return can first be demonstrated may depend on the angular distance between the central fixation point and the peripheral cue (Johnson, *in press*). There is reason to believe that this may be the case considering that (a) adults do not show

any inhibition of return under conditions in which hypometric saccades are performed (Rafal et al., 1989), and (b) infants under 4 months commonly show hypometric saccades to distant targets (Aslin, 1981). In the study of Boylan Clohessy et al. (1991), in which inhibition of return was observed from 6 months of age, the cue was shown at 30° eccentricity, and younger infants had to make several hypometric saccades to it. Therefore, inhibition of return may be demonstrated in infants younger than 6 months provided that the cue is shown at an eccentricity such that the target can be reached by a single saccade. In accord with this prediction, Harman et al. (1992) found evidence of inhibition of return in 3-month-old infants when the cue was shown at 10° eccentricity but not when it was shown at 30° eccentricity.

Johnson (in press) tried to establish the age of onset of facilitatory effects and of inhibition of return by studying 2 and 4-month-old infants. When an infant was looking toward the fixation stimulus, the cue was presented 29° to the left or right of it for 100 ms. Then, either 100 or 600 ms later, the bilateral target was simultaneously presented both on the same side as the cue and on the opposite side. RT to make a saccade and direction of the saccade were recorded.

There was no difference in the frequency of saccades toward the cued side between the short and long cued-target interval for 2-month-olds. In contrast, 4-month-olds showed increased orienting toward the cued side on short interval trials and decreased orienting to this side on long interval trials. For RT data, 4-month-olds demonstrated a facilitation effect with the short interval and an inhibition of return with the long interval. No effects were found for 2-month-olds.

The results of Johnson's (in press) study, therefore, showed that both facilitatory effects of covert attention and inhibition of return are present in 4-month-old human infants. Note, however, that in the absence of either eye movements to the cue or of facilitatory effects even with the short cue-stimulus interval, it is difficult to be sure that 2-month-olds noticed the presence of the cue.

THE RETINOTECTAL PATHWAY IN EARLY INFANCY

As mentioned earlier, inhibition of return in adults is regarded as reflecting the residual

functional competence of the retinotectal pathway. This view is based on the observation that, under monocular conditions, there is a bias to make saccades toward the temporal hemifield (Posner & Cohen, 1980), and inhibition of return is more robust in the temporal hemifield (Posner et al., 1985; Rafal et al., 1989).

Some studies have demonstrated visual preferences, in newborns (Slater, Morison, & Somers, 1988) and in infants at a few weeks postnatally (Atkinson, Hood, Wattam-Bell, Anker, & Tricklebank, 1988), that are likely to be mediated by cortical mechanisms. It would not be correct, therefore, to assume that the visual cortex is completely nonfunctional at birth. However, it has been proposed (Bronson, 1974) that at around 2 months there is the emergence of cortical visual function, whereas earlier visual behavior would have its neural basis in subcortical structures such as the superior colliculus. Johnson (1990; also see Atkinson & Braddick, 1989; Bronson, 1982) has summarized the empirical evidence that at birth, vision depends predominantly on the retinotectal pathway. In his view, the operations of the retinotectal pathway are reflected in the following aspects of the newborn's visually guided behavior.

Under monocular viewing conditions, right from birth, newborns are more likely to orient toward stimuli presented in the temporal hemifield than in the nasal hemifield (Lewis, & Maurer, 1992; Lewis, Maurer, & Blackburn, 1985). Newborns' poorer orienting toward stimuli in the nasal hemifield probably reflects an inability to detect their presence with peripheral vision which causes the development of the nasal hemifield, as measured with static perimetry, to lag behind development of the temporal hemifield (Lewis & Maurer, 1992).

Tracking of slowly moving stimuli is very easily elicited in newborns. This behavior, however, unlike that of older infants and adults, is not smooth, but is saccadic in nature, and eye movements typically fall behind the movement of the stimulus and rarely appear to anticipate its future location (Barten, Birns, & Ronch, 1971). Tracking would be saccadic because the collicular mechanism operates to refoveate the moving stimulus only when it enters the temporal hemifield of one or the other eye. If the stimulus continues to move, this process continues to be engaged.

Newborns show the phenomenon known as

the "externality effect" (Maurer, 1983). That is, they do not appear to attend to small stationary pattern elements within a larger frame or pattern. On the basis of the collicular mechanism, the newborn foveates the largest element in the stimulus array: the frame. However, if the internal elements move, they trigger the collicular mechanism, and the externality effect may be overcome.

The dominant role of the retinotectal pathway in early infancy was confirmed by Atkinson, Hood, Wattam-Bell, and Braddick (1992). They found evidence that in 1-month-olds, visual orienting may depend on a subcortical collicular mechanism operating in isolation, whereas cortical mechanisms may start to operate in 3-month-olds. Results consistent with the view that visual orienting in normal newborns is guided by subcortical neural structures were also reported in a study of two infants lacking one cerebral hemisphere (Braddick, Atkinson, Hood, Harkness, Jackson, & Vargha-Khadem, 1992).

If early vision is predominantly retinotectal in nature, and even in adults, inhibition of return is mediated by the retinotectal pathway, it should be expected that inhibition of return would be very strong in newborns (Rothbart et al., 1990). The results summarized in the foregoing section were, instead, the reverse of this prediction. In fact, inhibition of return was not observed in infants younger than 3 months of age. It must be pointed out, however, that only Johnson (in press) tested infants as young as 2 months, and it is unclear whether in his study the youngest infants oriented to the cue. Newborns were never tested for inhibition of return.

The purpose of this study, therefore, was to determine whether inhibition of return is present in newborns. It was reasoned that inhibition of return might be present at birth, then it might disappear and come back between 3 and 6 months of age. A pattern like this is not without precedent and, in fact, was observed in the development of face recognition. (Note that other sensorimotor behaviors appear to decline after birth; see, e.g., Johnson, 1990; Muir, Clifton, & Clarkson, 1989.) Newborns show a preference for faces that declines, or even disappears, after the first few days of life but reemerges at 2 months (see review in Morton & Johnson, 1991). This has led to the proposal that there are two mechanisms that underlie

face processing (Morton & Johnson, 1991): first, a reflexive tendency, mediated by subcortical structures (e.g., the retinotectal pathway), to orient toward stimuli that resemble faces; this tendency would be present from birth; and second, a cortically mediated process to direct attention toward familiar, face-like stimuli which develops around 2 months of age.

METHOD

Subjects

They were 32 normal, healthy, fullterm newborns (00 males and 00 females) from the maternity ward of the Pediatric Clinic of the University of Padua. They were tested within the first 3 days after birth (M age = 72 hours, range = 24–96 hours). Informed consent was obtained from the parents. The experiments took place during the hour preceding the infants' scheduled feeding time.

Apparatus

An infant sat on the experimenter's lap, and the head was held lightly in a cushioned headrest in order to maintain a midline position. The eyes were roughly aligned with the edge of a 90°-angle screen at a distance of about 30 cm from it. Visual stimulation was provided by three bulbs (12 volts) that subtended 6.5° of visual angle. The center bulb was red and was placed in the edge of the 90°-angle screen. The two peripheral bulbs were white and were placed 30° (inner border) to the left or right of the center bulb. When turned on, the three bulbs blinked at a rate of 300 ms on and 300 ms off.

The ambient light of the cubicle where the experiments took place was on so that the experimenter could see the infant's eyes on a monitor by means of a video camera mounted behind the display screen and aimed at an opening in it. The screen was medium gray in color. Plain white curtains were drawn on both sides of the chair where the experimenter was seated, with the infant in her lap, obscuring irrelevant stimuli.

A pacifier was inserted into the infant's mouth and non-nutritive sucking was recorded by a pressure transducer that was interfaced with an Apple IIE microcomputer through an A/C card. Rate of sucking was recorded during the pretest and test phases of each trial as well as for 10 s prior to the beginning of the trial (i.e., the baseline). The microcomputer also controlled the sequence of stimulus presentation on the screen.

Procedure

When the infant was judged to be looking at the flashing center bulb (i.e., the fixation stimulus), the experimenter pressed a key. This turned off the fixation stimulus and initiated presentation of the cue stimulus (i.e., one of the peripheral flashing bulbs) which remained on for 3 s. Whether the cue appeared to the left or to the right of fixation was determined by a pseudorandom sequence with the constraint that there should be an equal number of left and right presentations. When the 3-s presentation of the unilateral cue ended, the cue was turned off, and the flashing fixation stimulus was turned on again for 2 s. For sake of brevity, this will be referred to as the *pretest phase*.

At the end of the pretest phase, the test phase began. The fixation stimulus was turned off, and both flashing peripheral targets were simultaneously presented. The bilateral stimulation lasted 5 s after which period the test phase ended.

Videotapes of infant's eye movements throughout the experiment were subsequently analyzed. In both the pretest and test phases, only those trials were considered for which the coders (two of the experimenters and one student who acted independently from one another) agreed on the direction of the saccade and on the frame in which the saccade had started. RT was defined as the time between the onset of a peripheral stimulus (i.e., either the cue or the target) and the time at which the eyes first started to move. It was measured frame by frame from the video recorded tapes to the nearest 33 ms.

The average nonnutritive sucking rate was recorded prior to the pretest phase (i.e., baseline rate), during the pretest phase, and during the test phase.

We intended to submit each infant to 12 trials. However, a number of trials (106, overall) could not be run because of a change of state just before the pretest phase (i.e., the infant became too fussy, too drowsy, or cried). Of the remaining 245 trials (4–12 for each infant), 138 were lost because (a) the coders could not agree on the direction of the saccade in the pretest or in the test phase ($n = 11$); (b) during the test phase, the infant did not move the eyes ($n = 59$); (c) the infant failed to reorient to the center stimulus after the pretest phase ($n = 44$); or (d) the infant changed state during either the pretest or the test phase ($n = 24$).

In total, there were 107 scorable trials to which each infant contributed at least 2 trials. On 36 of these trials, however, RT was not measured because the coders could not determine with certainty in which frame the movement had started. On 37 trials, nonnutritive sucking rate could not be measured because of apparatus failures. Overall, data on saccade direction were obtained for 28 infants, and data on saccade latency were obtained for 20 infants.

RESULTS

In what follows, it is important to keep in mind that in the pretest phase, infants could orient toward the cue, but they could also fail to shift gaze or move the eyes away from the cue. Of course, inhibition of return was indexed by an eye movement in the test phase toward the side that had not been cued in the pretest phase.

The main analyses were based on statistical tests performed on the average performance of each infant. However, considering that the rationale of this study rested on the fact that the infant shifted gaze to the cue stimulus (i.e., the crucial event in the pretest phase was a movement from fixation to the cue), the data were also analyzed separately by taking into account the infant's behavior in the pretest phase. This was done by using statistical tests for independent observations. Because each infant contributed roughly the same amount of

trials, treating trials as independent observations is not entirely inappropriate and might provide interesting additional information.

Main Analyses

Data on saccade direction were obtained for 28 infants. In the test phase, for 18 infants, trials indicating inhibition of return were more numerous than trials without inhibition of return. For 6 infants the reverse occurred, whereas for the remaining 4 infants, inhibitions were as numerous as noninhibitions. A sign test showed that this difference was significant ($p < .011$).

The average number of inhibitions of return was 1.67, whereas the average number of noninhibitions was 0.70. An analysis of variance (ANOVA) with one within-subjects factor showed that this difference was also significant, $F(1, 26) = 11.84, p < .005$. The same results were obtained with a second ANOVA performed on arc-sine transformed mean percentages (69% inhibitions vs. 31% noninhibitions), $F(1, 26) = 6.97, p < .025$.

Not enough RT data could be obtained for 8 infants. Of the remaining 20, only 3 produced both inhibitions and noninhibitions. Although there were clearly too few to allow a meaningful statistical analysis, it is perhaps worth noting that RT was faster for inhibitions than for noninhibitions (1,800 vs. 1,950 ms). Fourteen infants produced only inhibitions, and 3 produced only noninhibitions ($p < .006$, with a binomial test). Mean RT was faster for the infants that produced inhibitions than for those that produced noninhibitions (1,439 vs. 3,183 ms), $t(15) = 2.71, p < .025$, with a test for independent observations.

A further analysis showed that for the 16 infants for whom sufficient data were obtained, nonnutritive sucking rate decreased in the pretest phase with respect to the baseline (0.580 vs. 0.989 suck/s), $t(15) = 3.40, p < .01$, with a test for nonindependent observations.

Eye Movements Toward the Cue

In the pretest phase, there was a movement of the eyes from the fixation stimulus to the cue on 61 trials (produced by 28 subjects). It is important to note that on 55 trials, a single saccade was enough for the eyes to reach the cue.

These shifts of gaze in the pretest phase had

an average RT of 1,715 ms, an average movement time of 369 ms, and were accompanied by a significant decrease in nonnutritive sucking rate with respect to the baseline (0.541 vs. 0.823 suck/s), $t(30) = 3.31, p < .005$.

In the test phase, the infants oriented toward the uncued side (i.e., inhibitions of return) on 43 trials, whereas on 18 trials they oriented toward the cued side (i.e., noninhibitions). This difference was significant with a binomial test ($p < .05$). RT could be measured for 40 trials and was faster in the case of eye movements toward the uncued side than toward the cued side (1,331 vs. 2,325 ms), $t(38) = 2.47, p < .025$.

Eye Movements Away From the Cue

In the pretest phase, infants oriented away from the cue on 30 trials (produced by 17 subjects). Average RT was 1,752 ms, and nonnutritive sucking rate did not significantly change with respect to the baseline (0.900 vs. 0.962 suck/s).

In the test phase, there were no significant differences in either frequency of saccades or response latency between the uncued side (14 trials; M RT = 1,314 ms on 7 trials) and the cued side (16 trials; M RT = 1,525 ms on 8 trials).

No Eye Movements

In the pretest phase, on 12 trials (produced by 12 subjects), infants did not shift gaze and, thus, did not produce any RTs. Average nonnutritive sucking rate did not significantly change with respect to the baseline (0.975 vs. 0.993 suck/s).

Also in this case, in the test phase there were no significant differences in either frequency of saccades and RTs between the uncued side (9 trials; M RT = 2,286 ms on 8 trials) and the cued side (7 trials; M RT = 2,014 ms on 6 trials).

DISCUSSION

In the pretest phase, the unilateral cue stimulus was meant to summon the infant's attention and to elicit a coordinate shift of gaze. This, in turn, should have produced inhibition of return in the test phase. That is, in the test phase, when the bilateral target stimulus was presented, the infant was expected to show a tendency to orient preferentially toward the uncued side. It was also predicted that inhibition of return

would manifest itself through faster RTs to orient toward the uncued side.

These predictions were borne out by the main analyses which was carried out on the average performance of each infant for either direction or latency of the saccade.

It is also interesting to note that the analyses performed on single trials, assumed to be independent observations (admittedly, a questionable assumption), showed that trials indicating inhibition of return were more frequent and produced faster RTs only when an eye movement toward the cue occurred in the pretest phase. On these trials, a decrease of nonnutritive sucking rate was observed also which confirms that the shift of gaze toward the cue in the pretest phase was a component of a phasic orienting reaction (Mendelson, 1979). In contrast, no signs of inhibition of return were found on those trials in which, during the pretest phase, the nonnutritive sucking rate did not decrease and the eyes did not move or moved away from the cue indicating that the infant's attention was not attracted by the cue.

In conclusion, the results of this study clearly suggest that inhibition of return can be observed in newborns aged 24 to 96 hours, provided the cue proves effective in attracting attention and thus producing an orienting reaction. Also, preliminary results of a study currently in progress (Valenza, Simion, & Umiltà, in preparation) confirmed the presence of inhibition of return in newborns of this age even though the experimental conditions were markedly different: they included monocular viewing, infant-control procedure, patterned stimuli, and 15° eccentricity.

The presence of inhibition of return in newborns is exactly what should be predicted assuming that inhibition of return is mediated by the retinotectal pathway (Rafal et al., 1989), and that at birth vision is predominantly, perhaps exclusively, mediated by the retinotectal pathway (Atkinson et al., 1992; Braddick et al., 1992; Bronson, 1974, 1982; Johnson, 1990, in press).

Of course, at this point, the question arises of why previous studies failed to show inhibition of return at 2 to 3 months of age, even though it was present a few months later (Boylan Clohessy et al., 1991; Hood, in press; Hood & Atkinson, 1991; Johnson, in press).

An interesting possibility is that, as happens for other newborn sensorimotor behaviors which possibly are mediated by the retinotectal pathway (Johnson, 1990; Morton & Johnson, 1991; Muir et al., 1989), inhibition of return declines after birth to reemerge later in life.

The best known instance of such a biphasic developmental pattern is processing of face-like stimuli (see review in Morton & Johnson, 1991). Newborn infants, as early as 10 min after birth, track slowly moving face-like stimulus patterns further than they track various patterns with "scrambled" face features. A number of observations suggest that this preferential tracking of face-like patterns may be primarily mediated by the retinotectal pathway.

The face-preference effect is found in newborns when using tasks in which the infant is required to track a moving stimulus but not in tasks that utilize habituation or infant-control procedures. It then declines, or disappears, in the second month of life. The face-preference effect reemerges around 2 months of age at which time it can be observed when using habituation and infant-control procedures.

Morton and Johnson (1991) have explained the biphasic developmental pattern of face-preference effects on the basis of two independent mechanisms. At birth, the preferential tracking of face-like patterns would depend on the activity of the already matured retinotectal pathway. In contrast, at 2 months, cortical structures would begin to control the infant's preferential responses to faces.

The temptation is to extend this interpretation to the biphasic developmental pattern of inhibition of return. There is, however, an obvious problem with this interpretation. Inhibition of return seems to be mediated by the retinotectal pathway even in adults as suggested by the nasal-temporal differences reported by Rafal et al. (1989, 1990). Therefore, it is difficult to think of two independent mechanisms that alternate in mediating inhibition of return at different stages of development.

A less interesting possibility is that inhibition of return is always present, but previous studies could not find it before 3 to 6 months of age because of inappropriate experimental conditions.

In this study, inhibition of return was almost always preceded, in the pretest phase, by single

saccades directly aimed at the cue. That the infant moved the eyes directly to the cue is confirmed by the comparatively fast movement time. Note that in our study, average movement time was 369 ms, whereas in the study of Boylan Clohessy et al. (1991, Figure 1), 3-month-olds had an average movement time of about 1,200 ms. Apparently, our experimental conditions were such that eye movements directed to the cue were favored. With different experimental conditions, trials in which hypometric saccades had occurred in the pretest phase would certainly have been included. Considering that hypometric saccades do not cause inhibition of return in adults (see Experiment 6 of Rafal et al., 1989), it is clear that the inclusion of trials with hypometric saccades to the cue in the pretest phase might have obscured inhibition of return in the test phase.

This problem is exacerbated with younger infants who are known to produce many hypometric saccades (Johnson, 1990). Note that this interpretation is supported by the finding that inhibition of return could be observed in 3-month-olds if the cue is moved from 30° to 10° eccentricity (Harman et al., 1992). At 10° eccentricity, hypometric saccades are much less numerous than at 30°.

It is also worth reiterating that in this study, nonnutritive sucking slowed down in the pretest phase only on those trials that then produced inhibition of return in the test phase. This is further evidence that only on these trials was the pretest phase characterized by a true orienting reaction to a salient cue stimulus (Mendelson, 1979).

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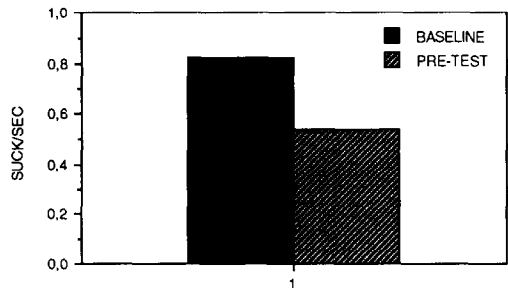
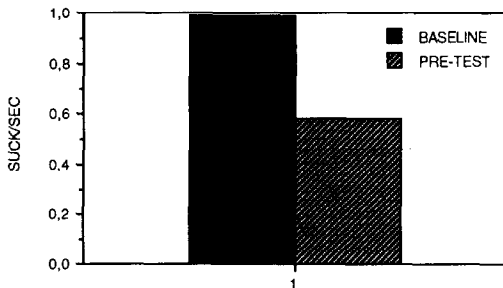
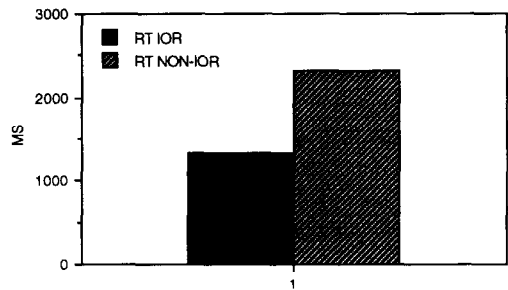
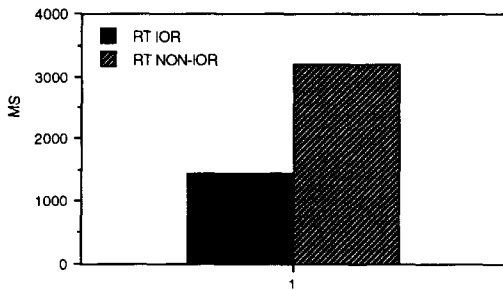
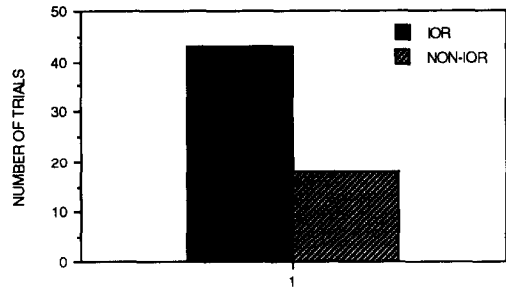
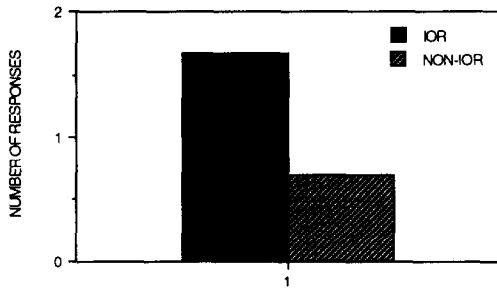


Figure 1. Upper panel: Average number of inhibitions (IOR) and noninhibitions (NON-IOR) of return. Middle panel: Mean reaction time (RT) in ms for saccade responses that indicated inhibitions and noninhibitions of return. Lower panel: Average number of sucks/second during the baseline and the pretest phases.

Figure 2. Panels are positioned as in Figure 1. Here, however, the data are only from those trials in which an eye movement toward the cue occurred during the pretest phase.

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