OBJECT PROCESSING IN THE INFANT BRAIN

Csibra et al. (1) showed a very nice example of induced gamma-band activity (GBA) in the human electroencephalogram (EEG). Increased gamma power was observed in 8-month-old infants when they viewed an illusory Kanizsa square, relative to their response to a control stimulus that did not allow the infants to perceive the illusory square [figure 1 of (1)]. The latency and the general finding of an increase of GBA amplitude when perceiving a figure were in line with the results of a series of studies (2–4) that have used visual stimuli. Some questions arise, however, with respect to the data that were presented and the conclusions that Csibra et al. have drawn from them.

The most surprising finding is the left frontal increase in GBA. In adults, GBA induced by Kanizsa triangles is reported to be most pronounced at occipital electrodes (5), a result consistent with recent functional magnetic resonance imaging (fMRI) findings showing an increased blood oxygen level–dependent (BOLD) response to Kanizsa figures in lateral occipital areas (6). Furthermore, our own study using bistable figures demonstrated a significant increase in GBA at occipital electrodes when the stimulus was switched from a meaningless figure to a meaningful one (7). The Csibra et al. study implies that the increased left frontal activity is based on a peak at electrode F7 only [figure 2 of (1)]. Such focused activity seems rather unusual, especially in view of the high-density electrode array montage. In addition, the GBA suppression in the 8-month-olds and the GBA maximum in the 6-month-olds both lie between the electrodes, owing to the spline interpolation. It seems questionable to interpret these findings as a signature of frontal lobe activity.

Csibra et al. (1) also used event-related potentials (ERPs) as evidence that the 8-month-olds were perceiving the Kanizsa squares. Why, however, should ERPs related to perceiving a figure be maximal at parietal electrodes, when the binding activity is supposed to be located in the frontal lobe? In addition, the neural activity related to perception of the figure would have been around 60 to 100 ms before the binding process. With respect to the ERPs, Csibra et al. stated that only the 8-month-olds show “the pattern characteristic of adult ERPs.” That statement is not entirely correct; in adults, Kanizsa figures have been shown to elicit a larger negativity, with a latency of about 160 ms (5, 8–9). In the 8-month-olds, the effect was a smaller positivity relative to the non-Kanizsa figure, whereas the 6-month-olds showed exactly the opposite pattern (larger positivity for the Kanizsa figures), which Csibra et al. did not discuss. Moreover, it is unclear why for the ERPs the average reference was chosen, rather than the same reference used for the GBA.

A further discrepancy relates to the evoked GBA. The authors showed this evoked activity at electrode sites O1 and O2, and stated that the early phase-locked GBAs were similar to those described in adults. This is only true with respect to the latency of the evoked GBAs. In the study cited by Csibra et al. (5), the amplitude of evoked GBAs has its maximum at central electrode sites (Cz, C4).

Based on the ERPs and evoked GBA findings of the Csibra et al. study, it seems more likely—in line with previous results in adults—that posterior cortical areas were involved in the given task. The question emerges to what extent the Cz reference has reduced the induced GBA amplitude over parieto-occipital areas. In figure 2 of (1), increased GBA is present at left temporal posterior sites, but that was not reported by Csibra et al.; instead, the hypothesis of frontal lobe activity being related to memory processes is based on a peak at one electrode. Why should 8-month-olds keep a mental representation of a Kanizsa square in memory and not the cartoon or the geometric shape, which obviously attracted their attention and was presented for a much longer period? In contrast to the study by Csibra et al., the cited findings with respect to induced GBA at frontal sites (10) included a task that needed storage in working memory to perform it. Although the Csibra et al. report provides an excellent example of using induced GBAs, the suggested links between induced GBAs and ERPs seem highly speculative and also seem to call for a different interpretation. In addition, the focused activity at electrode F7 should be treated with caution.

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REFERENCES

TECHNICAL COMMENTS

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Csibra et al. (1) reported that 8-month-old infants showed EEG responses in the gamma frequency range (around 40 Hz) in response to illusory Kanizsa figures, whereas 6-month-old infants did not. The ERPs showed a significant enhancement of the positivity around 136 ms for Kanizsa figures compared with the response for the non-Kanizsa figures in the older infants, but the opposite effect was seen in the younger ones. It was argued that the ERP effect in the 8-month-old infants indicates the processing of illusory contours and resembles the pattern seen in adults in a similar experiment by Herrmann et al. (2). That is not completely correct, however, even after taking into account the observation that infant ERP components are usually delayed in latency (3).

Although the ERPs of the two studies do look similar, the differences between Kanizsa figures and non-Kanizsa figures occur in different components of ERP. Csibra et al. (1) reported a significant difference for the P1 component (135 ms), which has its counterpart around 90 ms in adults. Herrmann et al. (2), however, reported a significant difference for the N1 (160 ms), which occurs around 200 ms in infants. If the P1 were the critical variable, then infants should be able to detect illusory figures earlier than adults, a notion that is both counterintuitive and that contrasts with results of other studies that show that infants take longer to perform such processing than do adults (4). In addition, it is surprising that the effect reverses between 6 months and 8 months of age: whereas the young infants exhibit larger responses to Kanizsa squares, the older infants show enhanced responses to non-Kanizsa figures. Csibra et al. did not discuss that anomaly.

Based on our adult ERP and ERF (event-related field) data—which do not reveal a discrimination of illusory Kanizsa figures from non-Kanizsa figures in the P1 time interval (2, 5)—we propose a different interpretation. We argue that illusory contours cannot modulate the amplitude of the P1, but attending to a visual stimulus can enhance the visual P1 response (6). In addition, illusory figures are processed differently when attention is directed at their processing (5). Because, in the study by Csibra et al., the two black-and-white figures were presented with-
in sequences of attractive colorful pictures, they “popped out” from that context and were processed more attentively. This interpretation is strongly supported by the similarity between the infants’ late positive ERP component (figure 3A of (J)) and the target P3 component of ERPs from young adults (Fig. 1). For the older infants, who already have a memory representation of Kanizsa squares (or at least of squares), the nonfigural non-Kanizsa stimuli pop out more strongly, because the color pictures also represent figurative images. Therefore, the non-Kanizsa square evokes a larger P1, which is in line with attention being able to modulate P1 amplitude.

Csibra et al. investigated within age groups how stimuli affect P1 amplitude. When the differences are examined between age groups for each type of stimulus, it seems as if the P1 in response to Kanizsa figures does not change between young and old infants. The P1 in response to non-Kanizsa stimuli, however, is larger in old infants than in young infants. Indeed, there is no clear P1 response to non-Kanizsa stimuli in younger infants, which suggests that they might not be processed as coherent figures at all.

For these reasons, and because it has previously been shown that attention modulates gamma responses (5, 7), it cannot be excluded that attention and processing of illusory contours interact in the generation of gamma activity in the infant brain.

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Response: Both of these comments stress differences between the findings of our report (J) on gamma-band activation in human infants and those of similar studies in adults. Such differences are indeed important, but we do not think that they warrant the reinterpretation that the commentators attempt to apply to our results.
Specifically, we have not yet observed this effect in infants (7) in a paradigm that did show the effect in adults (8).

Both comments worry about our claim that the ERPs of the 8-month-olds showed a pattern characteristic of adult ERPs, because it occurred as a smaller positivity rather than an enhanced negativity. It can be difficult to determine whether an ERP effect is a modulation of an identifiable component (such as P1 or N1) or an additional shift of electrical activity. Such “additive components” are not unprecedented in ERP research [e.g., the N400 (9)]. There is no simple way to find the ERP components in infants that correspond to components in adults, and we think that a negative voltage shift in the same latency range is as good a match as one based on enumeration of peaks.

Finally, we agree completely that we have to be cautious about the interpretation of our findings. Indeed, at the end of our paper, we stated that “more studies are needed to determine whether the frontal activation is directly related to the binding process or reflects further attentional processing on the object ‘bound’ elsewhere in the infant brain.” The comments by Müller and by Herrmann and Friederici have helped to identify aspects of our study that merit further research, but they do not bring into question the main finding of an association between object perception and binding-related gamma oscillation in infants.

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