

# Cortical processing of visual motion in young infants

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## Abstract

High-density EEG was used to investigate the cortical processing of a rotating visual pattern in 2-, 3-, and 5-month-old infants and in adults. Motion induced ERP in the parietal and the temporal–occipital border regions (OT) was elicited at all ages. The ERP was discernable in the 2-month-olds, significant and unilateral in the 3-month-olds and significantly bilateral in the 5-month-olds and adults. The motion induced ERP in the primary visual area was absent in the 2-month-olds and later than in the OT area for the 3-month-olds indicating that information to OT may be supplied by the V1 bypass at these ages. The results are in agreement with behavioural and psychophysical data in infants.

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## 1. Introduction

Visual motion elicits activation in a complex and widespread neural network (Sunaert, Van Hecke, Marchal, & Orban, 1999). One area, the MT+ complex, is considered to have a key-role in this process (Zeki, 2004). It is activated by visual motion (Barton et al., 1996; Born & Bradley, 2005; Gruber, Muller, Keil, & Elbert, 1999; Probst, Plendl, Paulus, Wist, & Scherg, 1993; Sunaert et al., 1999; Tootell et al., 1995; Uusitalo, Virsu, Salenius, Näsänen, & Hari, 1997; Zeki, 1991), processes perceived motion direction, and is crucial for the control of smooth pursuit eye movements (Komatsu & Wurtz, 1988; Newsome, Wurtz, & Komatsu, 1988; O'Driscoll et al., 1998). Patients with brain lesions that include the MT area have impaired motion perception (McLeod, Heywood, Driver, & Zihl, 1989; Schenk & Zihl, 1997; Zeki, 2004) and cannot perform smooth pursuit eye movements (Schoenfeld, Heinze, & Woldorff, 2002).

In adults, the signal input to the MT complex is realized by two parallel visual pathways: one that propagates from

lateral geniculate nucleus (LGN) to V1, V2 and finally to V5, the primary visual pathway, and one that projects to the MT+/V5 via superior colliculus (SC) and pulvinar (Buchner et al., 1997; Callaway, 2005; Ffytche, Guy, & Zeki, 1995; Schneider & Kastner, 2005; Schoenfeld et al., 2002) or via LGN (Sincich, Park, Wohlgenuth, & Horton, 2004). The pathway via SC is suggested to be a phylogenetic old pathway, functioning for non-conscious fear (Morris, Öhman, & Dolan, 1999) and fast moving stimuli (Buchner et al., 1997; Ffytche et al., 1995). Interestingly, this short latency pathway has been suggested to dominate the immature visual motion processing in newborn infants (Atkinson, 2000; Dubowitz, Mushin, De Vries, & Arden, 1986; Snyder, Hata, Brann, & Mills, 1990). Martin et al. (1999), using functional MRI to study brain activation in young infants, found responses in subcortical structures when presenting flickering light to them. They concluded that the visual pathway for motion via SC is functioning in the neonate.

In addition to the activation of cells sensitive to coherent motion, (Sunaert et al., 1999), visual motion also activates cells sensitive to the temporal correlation of the stimuli, that is, flicker (Bach & Ullrich, 1994; Tootell et al., 1995; Spileers, Mangelschots, Maes, & Orban, 1996).

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The response to flickering light is present at birth (Vitova & Hrbek, 1970) and the sensitivity develops during infancy (Apkarian, 1993; Fiorentini & Trimarchi, 1992; Regal, 1980). In adults, several experiments with visual evoked potential, VEP, (Göpfert, Müller, & Simon, 1990; Kuba & Kubova, 1992; Kubová, Kuba, Spekreijse, & Blakemore, 1995; Schlykova, van Dijk, & Ehrenstein, 1993) as an endpoint have shown that the response to flickering light strongly depends on adaptation (Herrmann, 2001; Kuba, Kubova, Kremláček, & Langrova, 2007; Maurer & Bach, 2003; Schlykova et al., 1993), and the choice of pattern parameters is critical if a genuine motion response will be induced. However, the distribution of cells sensitive to flicker is different from the distribution of motion sensitive cells. Earlier fMRI studies on adults have shown that the flicker response to visual motion is maximal in V1 while the motion-specific response is less prominent or even insignificant at this location (Sunaert et al., 1999). Sunaert et al. (1999) found that the flicker response continues to be strong in the ventral pathway but diminishes rapidly in the dorsal pathway. For instance, the response to flicker in the MT+ area was only 20–50% of the response in V1. In fact, the spatially different distributions of cells sensitive to coherent motion and to flicker give indications of the degree to which visual motion activates these two different kinds of cells in young infants.

The present study asked when cortical processing of visual motion develops in human infants and how the different parts of the visual cortex are activated. There is yet no brain imaging study that has answered these questions. The reason is that methods like PET, MEG and MRI are not generally accessible to a non clinical group of infants. Information about when the processing of visual motion begins to involve the cerebral cortex comes from behavioural studies and studies using VEP (Hamer & Norcia, 1994; Mason, Braddick, & Wattam-Bell, 2003; Wattam-Bell, 1991, 1992). For example, Braddick, Birtles, Wattam-Bell, and Atkinson (2005) studied motion direction sensitivity in young infants with VEP and concluded that between 5 and 18 weeks of age the response becomes progressively stronger. Considering that human infants younger than 6–8 weeks of age do not discriminate motion direction, and do not smoothly pursue small moving objects is another indication that the MT complex is not processing coherent motion before that age. Between 6 and 14 weeks of age infant's ability to discriminate motion direction (Atkinson, 2000; Braddick et al., 2005; Wattam-Bell, 1991), and to smoothly pursue moving objects (Aslin, 1981; Rosander & von Hofsten, 2002; von Hofsten & Rosander, 1997), improves rapidly. In a study of pattern motion integration in 2 to 5 month old infants, Dobkins, Fine, Hsueh, and Vitten (2004) concluded that at 2 months of age, cortical mechanisms process global coherent motion.

Questions related to how cortical processing of visual motion gets established, and especially how it differentially activates cells sensitive to the spatio-temporal

(coherent motion) and temporal correlation (flicker) of the stimuli, can be made by analysis of the emerging spatio-temporal distributions of cortical activation over age. We used high-density EEG (EGI 128 Geodesic sensor net) in an ERP design to identify patterns of neural activity in 2-, 3- and 5-month-old infants and an adult group, when they watched stationary and rotating patterns of simple elements. The analyses were focused on changes occurring in the occipital–temporal border, the occipital and parietal regions as these are the ones activated by visual motion in adults. The way these cortical areas become increasingly involved with age provide information of how the visual pathways develop. The relationship between the activations of V1 and MT+, for instance, gives an indication of the degree to which visual motion activates flicker sensitive cells and cells sensitive to coherent motion. Furthermore, the relative timing of the activations of MT+ and V1 gives an indication of the origins of the input to these areas. For example, if the short latency visual pathway via the SC is functioning in the youngest infant groups, moving stimuli can be expected to activate MT+ before or without activation in the primary visual area.

Another set of questions relates to hemispheric asymmetries in the processing of visual motion. Such asymmetries have earlier been observed in adults and children for motion VEPs (Hollants-Gilhuijs, De Munck, Kubova, van Royen, & Spekreijse, 2000). O'Driscoll et al. (1998) found left-side response with PET in the temporal–occipital order area during smooth pursuit. Furthermore, in a study of attention to motion Pavlova, Birbaumer, and Sokolov (2006) found left hemisphere MEG response in the parieto–occipital region. Uusitalo et al. (1997) measured cortical responses to rotational stimuli in adults using MEG. In some of their subjects the responses to motion were only detected unilaterally.

## 2. Materials and methods

### 2.1. Subjects

Adult subjects and parents of the participating infants were informed about the experiment upon arrival at the lab. A written consensus was signed in accordance with the Helsinki Declaration. The experiment was approved by the Ethics committee at Uppsala University. A total of 52 infants and 12 adults participated. There were 18 full-term infants aged 6–9 weeks (“2-months”), 16 infants aged 9.5–14 weeks (“3-months”) and 18 infants aged 20–23 weeks old (“5-months”). They were healthy and had no visual problems. The adults were 25–30 years old and had normal vision. All parents and all adult subjects were right-handed.

### 2.2. Stimuli

The stimuli were designed in E-prime (Psychology Software Tools Inc., 2002). This program also synchronized the stimulus monitor with the EEG measurements. The stimuli consisted of an inner and an outer set of simple geometric figures positioned at the corners of two concentric pentagons on a static background grid (Fig. 1). The colour of the figure elements was the same for a specific stimulus but varied between them (Table 1). The elements of the inner pentagon were 14–17 mm in diameter and were posi-

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