Pointing errors in immediate and delayed conditions in unilateral optic ataxia

P. REVOL, Y. ROSSETTI, A. VIGHETTO, G. RODE, D. BOISSON and L. PISELLA

1 Service de rééducation neurologique, Hôpital H Gabrielle, Route de Vourles, BP 57, F-69565 Saint Genis-Laval cedex, France
2 Espace et Action, Inserm U534, 16 avenue du doyen Lépine, CP 13, F-69676 Lyon, France
3 Service Neurologique, Hôpital Neuro-cardiologique, 59 Bd Pinel, F-69003 Lyon, France
4 Institut Fédératif des Neurosciences, Bâtiment B13, Hôpital Neuro-cardiologique, 59, boulevard Pinel, F-69394 Lyon cedex 03, France

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Abstract—The present paper provides an analysis of the pointing errors of a patient with unilateral optic ataxia (O.K.) following right hemispheric damage, revealing the type of errors related to the use of the contralesional hand and/or to the reaching of targets located in the contralesional visual field. In addition, comparison between immediate and delayed pantomime pointing allow testing of whether pointing deficits of this patient are specific to real-time visuo-motor control and, subsequently, whether delay could improve his pointing performance. The results show different patterns in the four hand–field combinations. The following conclusion can be drawn from the results of the delayed condition. In the case of patient O. K., the delay reduced the pointing variability for both hands in the left visual field but not in the right visual field. However, the pointing biases did not improve accordingly. As in healthy subjects, target locations tended to be coded in memory with a bias directed toward the fixation point. These results are discussed and contrasted with respect to those previously obtained in the literature in patients with bilateral optic ataxia.

Keywords: Delay; pointing movement; optic ataxia; human; parietal cortex; visual guidance.

1. INTRODUCTION

Visual localization of the goal is the first stage in the process of performing accurate goal directed movements. Numerous studies in animal and man have shown that visually guided movements imply sensorimotor transformations which convert the retinal visual information in egocentric motor coordinates useful for the organization of movement in space (centred on trunk, shoulder, head or arm:

*To whom correspondence should be addressed. E-mail: pisella@lyon.inserm.fr
This visuo-motor processing is made within a neural network including the posterior parietal cortex (PPC) and the premotor cortex, among other brain areas (Jeannerod et al., 1995; Burnod et al., 1999; Battaglia-Mayer and Caminiti, 2002). All of these brain regions are part of the ‘dorsal’ stream of the visual cortical system (see Goodale and Milner, 1992; Milner and Goodale, 1995; Rossetti et al., 2000).

When a lesion occurs in the PPC, patients show a characteristic inaccuracy when they reach or grasp visual objects (Balint, 1909; Holmes, 1918). Balint (1909) suggested that these deficits were due to a visuo-motor disconnection that he called ‘optic ataxia’, whereas Holmes (1918) proposed that these same deficits rather result from an impairment in spatial perception that he called ‘visual disorientation’. Following studies (Garcin et al., 1967; Ratcliff and Davies-Jones, 1972; Vighetto, 1980; Vighetto and Perenin, 1981; Perenin and Vighetto, 1988; Jeannerod et al., 1994) tended to confirm that these visuo-motor deficits can occur without perceptual disorder, rather supporting the definition of Balint (1909). Despite demonstrating an exaggerated grip aperture compared to healthy subjects, these patients are not impaired in object recognition (Perenin and Vighetto, 1988; Goodale et al., 1991; Jeannerod et al., 1994), and can even correctly match the object’s dimension with their finger grip when they do not perform the reaching movement (Goodale and Milner, 1992; Milner and Goodale, 1995). Interpretation of optic ataxia as a specific visuo-motor disorder was also reinforced by the careful study of reaching behaviour by Perenin and Vighetto (1988). First, the testing of auditory-guided movements showed that optic ataxia appears to be a modality-specific reaching impairment. Second, although verbal discrimination of dot position was impaired in some patients, a direct causal link between these subtle deficits in visual space perception and the gross misreaching errors was excluded by the authors. Finally, patients showed reaching errors related to both a visual field effect and to a hand effect. This combination of sensory and motor influences, as well as the localization of the underlying lesion in-between the visual and motor areas, supported the notion that the deficit lies at the visuo-motor interface rather than at the sensory or motor level.

Superimposition of the lesions of 6 left brain-damaged and 2 right brain-damaged patients with optic ataxia using CT scans (Perenin and Vighetto, 1988) revealed a symmetrical converging region including the superior parietal lobule, in and above the intraparietal sulcus, and sparing the human inferior parietal lobule. Recent functional imaging studies confirmed the involvement of this region in distal and proximal visuo-motor guidance during grasping (Grafton et al., 1996; Faillenot et al., 1997; Binkofski et al., 1998; Culham and Kanwisher, 2001) and pointing (Grafton et al., 1992; Kertzman et al., 1997; Desmurget et al., 2001) movements, respectively. Pointing errors associated with optic ataxia have recently been analyzed in two patients with fairly symmetrical bilateral parietal lesions (Milner and Dijkerman, 2001; Milner et al., 2003). Analyzing pointing performances in patient A. T., Milner et al. (1999) noted that the absolute errors increased with tar-