Coulthard, Parton, and Husain (2006) recently published an elaborated and noteworthy review on deficits of motor control in patients with spatial neglect. Based on their survey and the presentation of two single cases with optic ataxia, they presented detailed anatomical conclusions on the neural implementation of action control in humans that challenged recent behavioural and anatomical findings.

Their line of reasoning was based on the discoveries of temporal and spatial motor deficits in stroke patients suffering from spatial neglect. Several studies claimed that such patients take more time to initiate a new movement and/or demonstrate longer movement times when these movements are directed towards the side contralateral of the brain lesion (Heilman, Bowers, Coslett, Whelan, & Watson, 1985; Husain, Mattingley, Rorden, Kennard, & Driver, 2000; Mattingley, Bradshaw, & Phillips, 1992; Mattingley, Husain, Rorden, Kennard, & Driver, 1998). Other studies revealed spatial inaccuracies. Patients with spatial neglect either demonstrated a terminal pointing error or an increased curvature or variability of trajectories (Goodale, Milner, Jakobson, & Carey, 1990; Harvey, Milner, & Roberts, 1994; Jackson, Newport, Husain, Harvey, & Hindle, 2000). However, a closer inspection of the experimental design of those studies reveals that these motor abnormalities in neglect patients resulted from direct comparisons between brain-damaged patients with spatial neglect and healthy controls without brain damage only (Goodale et al., 1990; Harvey et al., 1994; Heilman et al., 1985; Husain et al., 2000; Jackson et al., 2000; Mattingley et al., 1992, 1998). Based on these studies, we thus can reason about the presence of certain deficits in patients with a circumscribed brain lesion. However, this does not necessarily lead to the conclusion that these deficits represent a specific impairment caused by the presence of spatial neglect. To investigate the specific influence of spatial neglect on motor functions, a comparison of neglect patients with patients suffering from brain damage but not spatial neglect is required (i.e. not only a comparison between neglect patients and healthy controls). Interestingly, studies incorporating such a group of brain-damaged control patients did not reveal any specific deficit in neglect patients neither in the spatial (Harvey et al., 2001; Himmelbach & Karnath, 2003; Karnath, Dick, & Konczak, 1997) nor in the temporal (Harvey et al., 2001; Konczak & Karnath, 1998) domain of goal-directed motor performance. Himmelbach and Karnath (2003) investigated the performance of stroke patients with acute neglect, patients who recovered from neglect, stroke patients with right brain damage but no neglect, and healthy subjects in a pointing task. Unfortunately, Coulthard et al. (2006) summarised these results stating that “... neglect patients did have greater absolute curvature ...”. Presumably, they referred to the only significant difference in action control that was found, namely a difference between brain-damaged patients with neglect and healthy controls. As reviewed above, this has been observed in several previous studies. However, the decisive comparisons between brain-damaged patients with and without neglect did not reveal any significant differences (Himmelbach & Karnath, 2003). This result was congruent with another comprehensive group study of reaching movements in brain-damaged patients with and without neglect (Harvey et al., 2001). In contrast to the interpretation given by Coulthard et al. (2006), one would thus have to summarise the
imperfections vanish or decrease considerably. As soon as they fixate the target their motor impairments if their movements are directed to targets in the peripheral visual field, i.e. exactly for the situation in which brain-damaged patients with optic ataxia show a specific disturbance of this function, we suggest that there are two dissociable underlying cognitive functions which can be separately disrupted by distinct brain lesions. The lateral and medial aspects of the POJ appear to be involved in the fast control of visually guided reaching, on the basis of short-lived and implicit eye- or arm-centred space representations, as part of the dorsal action system. In contrast, more ventral regions involving the temporo-parietal junction (TPJ), the superior temporal cortex and insula – damaged in patients with spatial neglect (Heilman, Watson, Valenstein, & Damasio, 1983; Karnath, Fruhmann Berger, Küker, & Rorden, 2004; Mort et al., 2003) – seem to play an essential role in adjusting body position relative to external space (Karnath & Dieterich, 2006). These areas appear to subserve longer lasting, explicit and multimodal spatial representations.

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References


