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Impaired grasping in a patient with optic ataxia: Primary visuomotor deficit or secondary consequence of misreaching?

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ABSTRACT

Optic ataxia is defined as a spatial impairment of visually guided reaching, but it is typically accompanied by other visuomotor difficulties, notably a failure to scale the handgrip appropriately while reaching to grasp an object. This impaired grasping might reflect a primary visuomotor deficit, or it might be a secondary effect arising from the spatial uncertainty associated with poor reaching. To distinguish between these possibilities, we used a new paradigm to tease apart the proximal and distal components of prehension movements. In the "far" condition objects were placed 30 cm from the hand so that subjects had to make a reaching movement to grasp them, whereas in the "close" condition objects were placed adjacent to the hand, thereby removing the need for a reaching movement. Stimulus eccentricity was held constant. We tested a patient with optic ataxia (M.H.), whose misreaching affects only his right hand within the right visual hemifield. M.H. showed a clear impairment in grip scaling, but only when using his right hand to grasp objects in the right visual hemifield. Critically, this grip-scaling impairment was absent in M.H. in the "close" condition. These data suggest that M.H.'s grip scaling is impaired as a secondary consequence of making inaccurate reaching movements, and not because of any intrinsic visuomotor impairment of grasping. We suggest that primary misgrasping is not a core symptom of the optic ataxia syndrome, and that patients will show a primary deficit only when their lesion extends anteriorly within the intraparietal sulcus to include area aIPS.

1. Introduction

In a pioneering series of studies, Jeannerod (1984, 1988) proposed that reach-to-grasp actions, such as picking up a desired object, can be partitioned into distinct and quasi-independent visuomotor parts. He argued that the action of moving the arm to bring the hand to the target object (the "proximal" or "transport" component) is principally influenced by visual information signaling the location of the object, whereas the concurrent anticipatory pre-shaping of the hand and fingers in readiness for the grasp (the so-called "distal" or "grip" component) is guided principally by the geometric properties of the object. Although it is accepted that the two components must be somehow mutually co-ordinated, there is now extensive evidence that the transport component and the grip component are each controlled online by dedicated visuomotor networks within the posterior parietal cortex, in association with linked systems in the premotor cortex (Castiello, 2005; Castiello & Begliomini, 2008; Jeannerod, Arbib, Rizzolatti, & Sakata, 1995; Milner & Goodale, 2006; Tanné-Gariépy, Rouiller, & Boussaoud, 2002).

It has long been known that both components of prehension can be severely disrupted by lesions of the posterior parietal cortex. Damage to this region (particularly around the intraparietal sulcus) in humans is associated with optic ataxia (Karnath & Perenin, 2005; Perenin & Vighetto, 1988), classically defined as a deficit in accurate reaching for visual targets (Bálint, 1909; Harvey, 1995). In the great majority of patients with optic ataxia, grasping turns out to be impaired as well as reaching, and indeed patients will typically fumble for the target with the fingers widely spread, whatever the size of the target (Jeannerod, 1986a, 1986b; Perenin & Vighetto, 1988, Jakobson et al., 1994). This stands in sharp contrast to the normal pattern in which the handgrip opens only so far as to exceed the target size by a safe margin, and then smoothly closes in (Jakobson & Goodale, 1990; Jeannerod, 1984). Such distal impairments have been associated with optic ataxia since the earliest reports of misreaching following parietal damage, in both monkeys and humans (Damasio & Benton, 1979; Faugier-Grimaud, Frenois, & Stein, 1978; Ferrier, 1886, 1890; Jeannerod, 1986a; Lamotte & Acuña, 1978; Perenin & Vighetto, 1988). Indeed this close association between the distal and proximal deficits led Perenin and

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Vighetto (1983, 1988) to follow Bálint (1909) in arguing that optic ataxia reflects a general impairment of visuomotor control rather than a deficit in visuospatial perception (as proposed by Holmes, 1918).

There is of course no necessary contradiction between this neuropsychological association between the distal and proximal deficits, and the quasi-modular visuomotor organization in the brain proposed by Jeannerod and his colleagues. It is entirely reasonable to argue that in most of the patients (and monkeys) studied, the lesions were extensive enough to have compromised both the "grasping" and the "reaching" visuomotor modules. None the less, the question does still arise as to whether an impairment in grip scaling necessarily implies that the patient has damage to such a "grasping" module. Instead, optic ataxia, by virtue of causing inaccurate reaching, might inevitably result in a maximally wide handgrip, simply in order to reduce the margin of error when the patient is trying to grasp an object.

Such an idea would not of course contradict the fact that some patients with parietal lesions misgrasp without misreaching (Binkofski et al., 1998; Jeannerod, Decety, & Michel, 1994). These patients, who would not be considered to have optic ataxia, tend to have lesions that include anterior parts of the intraparietal sulcus, in particular the "grasp" region known as AIP or aIPS (Binkofski et al., 1998). These findings are important, because they refute the argument that misgrasping might *always* be a secondary side-effect of misreaching. The data also mesh nicely with research using functional MRI which has demonstrated distinct activation patterns for grasping (Binkofsky et al., 1999; Cavina-Pratesi, Goodale, & Culham, 2007; Culham et al., 2003; Frey, Vinton, Norlund, & Grafton, 2005), separate from those for reaching (Astafiev et al., 2003; Connolly, Andersen, & Goodale, 2003; Culham, Gallivan, Cavina-Pratesi, & Quinlan, 2008; Prado et al., 2005). Complementary evidence also comes from nonhuman primate studies finding a clear double dissociation between proximal/arm and distal/hand errors following localized microinjections of muscimol (Gallese, Murata, Kaseda, Niki, & Sakata, 1994).

Although misgrasping can clearly arise in the absence of misreaching, it remains uncertain that the converse dissociation holds true in patients with optic ataxia. That is, it remains possible that in many such patients impaired grasping arises purely as a secondary consequence of misreaching, and not as the result of disruption of the "grasp" module.

Flesh can be put on the bones of these doubts. Numerous visuomotor studies show a tendency in healthy subjects to increase the size of their anticipatory grip aperture to compensate for factors that increase transport inaccuracy during reaching toward the object to be grasped. In one such study, maximum grip aperture (MGA) was found to be significantly greater during reaching movements that were performed faster than normal, and also during reaching with the eyes closed (Wing, Turton, & Fraser, 1986). In both cases, this wider hand aperture was accompanied by less accurate transport of the hand toward the target location. It is reasonable to infer that in these circumstances anticipatory grip aperture was enlarged to give a wider margin of error for achieving a successful capture of the object, thereby compensating for the spatial inaccuracy associated with speeded movements. Similar findings of an increased maximum grip aperture during reaching have been reported in cases where the stimulus uncertainty is increased in other ways, for example when visual target size is reduced (Berthier, Clifton, Gullapalli, McCall, & Robin, 1996), when the field of view is restricted (González-Alvarez, Subramanian, & Pardhan, 2007), or when the target eccentricity is increased (Goodale & Murphy, 1997; Schlicht & Schrater, 2007).

In the present paper we aimed to test whether deficits in grip calibration can be separated from deficits in reaching accuracy in optic ataxia, by the use of a task requiring grasping without arm transport. Such a task was developed by Cavina-Pratesi et al. (2006; see also Culham et al., 2008) in order to achieve a clean contrast between 'pure grasping' and 'pure reaching' in a functional MRI study.[1] We adopted this methodology in the present experiment in an attempt to tease apart the transport from the grasp impairment in a patient with optic ataxia (M.H.). Our specific intention was to establish whether his grasping difficulties were primary, or secondary to poor reaching; but at the same time we wished to validate our method so that it might be used for making unambiguous assessments of other such patients in the future.

2. Methods

2.1. Subjects

Patient M.H. had suffered an anoxic episode 8 years prior to the current testing. Structural MRI carried out in 2006 revealed disseminated damage in posterior parietal and frontal regions, concentrated particularly in the vicinity of the intraparietal sulcus of the left hemisphere, with some extension onto the medial aspect and into the inferior parietal lobule. Some atrophy was visible in the left hemisphere both cortically (within the posterior parietal, fronto-temporal and frontal regions) and subcortically (lentiform nucleus and claustrum). The occipital lobes were largely unaffected. The anoxic incident that caused his brain injury resulted in right side muscle weakness and raised sensory thresholds. He was still able to walk and use both hands, though he reported difficulties in everyday living activities, such as dressing, eating with a knife and fork, and writing. Clinical assessment exhibited symptoms of contralateral optic ataxia, most clearly when using his right hand, and when reaching toward targets in right hemispace under conditions of central visual fixation (Rice et al., 2008). M.H. also showed impairments in spatial perception (Riddoch et al., 2004), though clinical assessment showed no evidence of unilateral spatial neglect or agraphia.

Somatosensory performance was assessed using the Rivermead Assessment tests (Winward, Halligan, & Wade, 2000). M.H. scored at ceiling when discriminating surface pressure on both his hands and face (control level); he also detected all bimanual and unimanual stimuli in the Rivermead test of sensory extinction applied both to the face and the hands (tests 2 and 4). His two-point discrimination on each hand was 4 mm (test 5), again within the control range. M.H. had a grating resolution threshold of 2 mm (fair, relative to a group of older controls, in Manning & Tremblay, 2006), for both hands, on a task requiring him to decide whether a grating went along or across his finger (the threshold = minimum width to make 75% discriminations). M.H. was also able to discriminate the 2.83 filament (normal) on his ipsi- and contralesional fingers on the Semmes-Weinstein monofilament test (Bell, 1984). These data indicate that there was no major somatosensory loss in either hand. M.H. was aged 50 at the time of testing.

In addition to patient M.H., 7 age-matched neurologically intact controls were tested (all male; mean age 52.1, range 45–61). Since M.H. has essentially no reaching deficit when using his left hand, or towards targets in the left visual field (confirmed in Section 3 below), M.H. also served as his own control.

The ethics committees of the University of Birmingham School of Psychology and Durham University Department of Psychology approved the experiments described here, and informed consent was obtained prior to the study in accordance with the principles of the Declaration of Helsinki.

2.2. Procedure

Subjects sat comfortably in front of a 50 cm × 50 cm board laid horizontally on a table. As shown in Fig. 1, there were four possible locations where a target object could be placed: two near and two distant from the participant, 30 cm apart on left and right, and forming a square arrangement such that the near locations were 30 cm from the distant locations. A fixation point (a flashing red LED) was located at the centre of the square. At the beginning of each trial, as indicated in Fig. 1, the subject's left or right hand was placed adjacent to one of the potential object locations on a given side of the board (on the right side of the location when using the right hand and on the left side of the location when using the left hand). The starting position of the hand was specified by the use of a small plastic disc (white dot in Fig. 1) fixed to the board, at which the subject placed their pinched forefinger and thumb at the start of each trial. The object was then placed on the same side of the board, either close to the hand or far from the hand. Placing the object at the location adjacent to the hand enabled subjects to grasp the object without making an arm movement (hereafter referred to as the "close" condition). Placing the object at the other location, however, required the subject to move his or her arm towards the object in order to complete the grasp ("far" condition).

[1] Notably, a similar method was devised by Schenk, T., Baur, B., Steude, U., and Bötzel, K. (2003), for use in a different context.