MAGNETIC MISREACHING

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ABSTRACT

Ms D., a 76 year-old woman with a slowly progressive bilateral parietal lobe degeneration, showed an unusual variant of misreaching as yet unreported. When required to reach to a target in extrafoveal vision, she slavishly reached straight to the foveal fixation point instead ("magnetic misreaching"). Three dimensional recordings of limb movements to foveal and extrafoveal targets revealed that her reach endpoints were determined by the place she was looking, independent of the distance between target and fixation point. The sign was present in both hands. Magnetic misreaching differs from motor misbehaviours which follow frontal lobe damage. The neuropsychological and behavioural profile of Ms D., coupled with neuroimaging evidence suggest that magnetic misreaching is a manifestation of parietal lobe dysfunction.

INTRODUCTION

Poor reaching to non-foveated visual targets has been labelled as visual "mislocalization", and is usually a consequence of parietal lobe dysfunction (e.g. Levine, Kaufman and Mohr, 1978; Ratcliff and Davies-Jones, 1972). This sign is typically considered as a less severe variant of "optic ataxia", where patients are unable to reach to a visual target in spite of being able to "see" the target with full foveal vision. Patients with full-blown optic ataxia may attempt to improve their visuomotor performance by prolonged fixation of the target before movement onset (e.g. Pause, Kunesch, Binkofski et al., 1989; Rondot, De Recondo and Ribadeau Dumas, 1977) or by looking back and forth between the target and the reaching hand (Rizzo, Rotella and Darling, 1992). In most instances where visual mislocalization has been observed, the pattern of endpoint errors has not been reported (Levine et al., 1978; Rondot et al., 1977), the errors were randomly distributed, or, in some patients, were clustered in a direction towards the side of the lesion (Ratcliff and Davies-Jones, 1972). In this paper we report the case of a patient who misreached when the targets were placed in extrafoveal vision. The peculiarity of her behaviour is that she consistently reached towards the point that she was foveating.

CASE REPORT

Ms D., a 76 year-old right-handed woman, presented with a three-year history of increasing clumsiness with her right hand, as well as gait and balance difficulties. During performance of a visual spatial tracking task, Ms D. showed an unusual variant of misreaching. When required to reach to a target in extrafoveal vision, she slavishly reached straight to the foveal fixation point instead ("magnetic misreaching"). Three dimensional recordings of limb movements to foveal and extrafoveal targets revealed that her reach endpoints were determined by the place she was looking, independent of the distance between target and fixation point. The sign was present in both hands. Magnetic misreaching differs from motor misbehaviours which follow frontal lobe damage. The neuropsychological and behavioural profile of Ms D., coupled with neuroimaging evidence suggest that magnetic misreaching is a manifestation of parietal lobe dysfunction.
difficulties. Her initial complaint was that fine movements of her right hand were impaired, causing deterioration of her handwriting. She then noticed difficulties with balance and walking. When our assessment began, she displayed a dramatic limb-kinetic apraxia in both hands (although the right hand [25/72] was more impaired than the left [46/72] as assessed by the Ideomotor Apraxia Test of De Renzi, Motti and Nichelli, 1980). A CT scan showed no abnormalities. However, two SPECT scans (January 1996 and October 1996) revealed progressive bilateral parietal hypoperfusion, worse in the left hemisphere (see Figure 1). A recent (June 1997) magnetic resonance scan

Fig. 1 – SPECT scans of Ms D.’s brain taken on two separate occasions, January 1996 (A, C) and October 1996 (B, D). The first scan shows hypoperfusion in the left parietal cortex. After nine months, the left parietal hypoperfusion worsens and becomes apparent in the right parietal lobe as well. Anatomical left appears on the left side of each image.
revealed cortical atrophy in the parietal cortex, worse in the left hemisphere and largely restricted to the superior parietal lobe (Figure 2).

Neurological examination uncovered some disturbance of horizontal saccades to command and increased muscle tone (worse on the right side). A grasping/groping reflex (De Renzi and Barbieri, 1992) was elicited on one occasion, but such signs were absent at subsequent tests. Her neurological features are similar to those reported in cases of corticobasal degeneration (Gibb, Luthert and Marsden, 1989; Massman, Kreiter, Jankovic et al., 1996; Pillon, Blin, Vidailhet et al., 1995; Rinnie, Lee, Thompson et al., 1994). In many respects Ms D.’s illness is similar to cases of slowly progressive apraxia previously reported in the neuropsychological literature (Azouvi, Bergego, Robel et al., 1993; De Renzi, 1986; Dick, Snowden, Northen et al., 1989). During the period of our assessments, Ms D. was still living alone, she was collaborative and witty, and her social behaviour was always appropriate. In formal neuropsychological testing, she did not show deficits in language (e.g. Token Test = 34/36, De Renzi and Faglioni, 1978) or short or long term memory (e.g. Digit Span = 6, Corsi Block Span = 4; WMS Logical Memory = 8.5). She did not have unilateral neglect in cancellation or copying tasks. Her scores on screening tests for dementia were normal (Mini-mental state examination = 27, Folstein, Folstein and McHugh, 1975). Moreover, she performed within normal limits on tests supposed to assess executive functions (e.g. phonological fluency, F-A-S = 24, Benton and Hamsher, 1976; cognitive estimates = 5 errors, Shallice and Evans,
At the Wisconsin Card Sorting Test (Nelson, 1976), Ms D. achieved 4 categories with 2 perseverative errors. Ms D. had a profound constructional apraxia that affected her drawing, copying, WAIS-R Block Design (scaled score 1) and Object Assembly (scaled score 1) performance. Nevertheless, her performance was better on visuospatial tests which do not call for constructional ability (e.g. Visual Object and Space Perception Battery Dot counting = 8/10, Position Discrimination = 20/20, Number Location = 8/10, Warrington and James, 1991). She also showed a slowly progressive apraxic agraphia, and her reading was troublesome due to her difficulty in scanning the lines. She also had difficulties with multiplication and long division.

A complete ophthalmological examination was unremarkable, including the absence of any visual field defects as assessed by automatized Humphrey perimetry (although the previously described difficulties with horizontal saccades to command were confirmed). However, some disturbances in her visually-guided behaviour were apparent in everyday tasks as well as during formal testing. For example, she missed a hand she was attempting to shake, and placed her pen inappropriately while cancelling lines. Further investigation of her visuospatial and visuomotor performance exposed a hitherto unreported neurological sign, which we have labelled “magnetic misreaching”. This is the focus of the present report.

**Magnetic Misreaching**

*Clinical Observations of Magnetic Misreaching*

Ms D. had absolutely no difficulty pointing to or picking up small visual targets when she was allowed to look at them (i.e. with foveal vision). When she was not allowed to look directly at the targets, she failed. For example she was asked to point to a peripheral twenty-pence coin, while looking at a centrally placed pound coin. Her first attempts were unsuccessful because she could not inhibit eye movements towards the coin she was supposed to touch.

After her early failures on this task, it was apparent that she was making a second kind of error: often her hand would begin moving towards the fixation point (the pound coin) rather than the target (the twenty-pence coin). When we asked her to move very quickly (e.g. ballistically) she invariably reached to the point she was fixating, rather than the target. She was completely aware of these misreaching errors and appalled at the difficulty which she had in suppressing her movements to the non-target, fixation point. Informal testing suggested no obvious hemispatial asymmetries in the magnitude of magnetic misreaching. We observed the sign in several different testing conditions. Magnetic misreaching even occurred when there was no visual target to be reached to: when she was asked to reach into the hemifield opposite to her fixation, her movements were drawn to the place she was looking at. Placing her hand into the opposite hemispace in order to provide additional (proprioceptive) cues did not attenuate her magnetic errors. If Ms D. was allowed to foveate a target, and then close her eyes before she was allowed to point, she was quite accurate.
Three-dimensional Recordings of Magnetic Misreaching

In a subsequent session, two months later, we examined her misreaching in more detail, by recording her limb movements using a MacReflex motion analysis system (Qualisys, Inc.). Videotapes of the session were used as a guide to the success or failure of her fixation efforts and to record her verbal/behavioural responses to her performance on each trial. Four separate target arrays were used. Each array contained 3, 4, or 5 coloured circles. The coloured targets were randomly arranged on separate 42 × 29.6 cm (A3) sheets. Ms D. was required to count (without pointing) the number of targets present in each array and to judge which two of the visible targets were closest to one another and which two were farthest apart. Ms D.’s performance was errorless on these perceptual tasks. After the perceptual judgements were made, Ms D.’s reaching behaviour was examined and recorded.

On foveal reaching trials, Ms D. was asked to point to each of the coloured targets, in turn. She reached to targets with a high degree of accuracy when she was allowed to foveate them (Figure 3, left). After foveal reaching trials were completed, the same array was used to test extrafoveal reaching. Ms D. was required to look at a central black fixation cross while she reached towards a specified target. She was allowed to look back and forth between fixation and the target as many times as she wished before indicating her readiness to reach (reaching movements of patients with optic ataxia improve dramatically when such “pre-reach” eye movements to the target are allowed; Rondot et al., 1977). In these extrafoveal reaching trials, Ms D. reached to the fixation cross rather

**Fig. 3 – Foveal and Extrafoveal Reaching.** Left: Foveal Reaching. The dotted lines are actual x-y coordinates of the first four reaching movements to the first target array, made while Ms D. was free to look at each target. Right: Extrafoveal Reaching. These are the first four recorded attempts at extrafoveal reaching with the first target array. The three black circles and associated numbers were the specified targets for the four reaches, each labelled with appropriate target number. In each case Ms D. was instructed to look at the central fixation cross while reaching for the specified target. In two of the reaches clear magnetic misreaching was observed (reaches 1 and 3a). After the reach attempt at target 2, Ms D. denied moving her eyes to the target. After the apparently successful reach to target 3 (reach 3b) Ms D. confessed to looking towards the target.
than the intended target (magnetic misreaching; see Figure 3, right). These errors were unequivocal on 14 of 23 attempts. She was always aware of her errors and often tried to self-correct before the trial ended. In 8 of the remaining 9 trials, Ms D. admitted to glancing at the target during the movement (e.g. reach 3b in Figure 3). On the remaining trial, when asked if she looked at the target, she responded “No, I don’t think so”. The movement path of this reach is depicted in Figure 3 (reach 2). Her initial heading and subsequent corrective movements suggest a momentary glance towards the intended target. Magnetic misreaching was also found when we made her fixate a target at a location well beyond the testing array (not shown).

**Magnetic Misreaching and Foveation Eccentricity**

We carried out a further experiment in order to ascertain whether or not magnetic misreaching was related to the eccentricity of the fixation point relative to the target to be reached to. The target was located in the same place throughout the experiment, while the position of the fixation point was varied from one block of trials to another. The target to be reached towards was a 1 cm diameter, high contrast (66%) green star (luminance = 69 cd/m²) placed in right hemispace, 22 cm anterior to and 19 cm lateral to a central starting position. In the first six trials Ms D. was required to reach to the green star with her index
finger while she was allowed to foveate it (control trials). For each subsequent block of trials, Ms D. was informed that she was to point to the green star, while looking at a small gray marker which served as the fixation point. Six trials were conducted with three different horizontal fixation locations: 10, 20 and 30 cm to the left of the star. As in the previous experiment, we videotaped the entire session to help confirm the success of Ms D.’s fixation efforts. Additionally, one investigator sat at the end of the table and constantly monitored her eye movements from a position where breaks in fixation were easy to spot. Figure 4 shows Ms D.’s performance using her right and left hand. It is clear from these figures that as the fixation point becomes more eccentric, the magnitude of the patient’s errors increases. The direction of her error is not random; she consistently reached towards the fixation point.

Terminal accuracy for each reach was calculated by averaging the X, Y and Z coordinates of the final frames of her movements and comparing the three-dimensional difference of these coordinates to calibration marker coordinates. The calibration coordinates were obtained by placing a MacReflex marker on the green star after each block of six trials was completed. Similar calibration trials were collected for each of the 3 extrafoveal fixation targets. On one trial (in the block of left 10 cm trials of the left hand), Ms D. actually looked at the green star. This trial was omitted from the analysis. Means and standard deviations of her endpoint errors as a function of target-fixation distance and hand used appear in Table I. These data confirm our earlier observations that Ms D.’s magnetic misreaching is present in both left and right hands, and is largely independent of the distance between the target and the fixation point. Since we predicted that Ms D.’s terminal errors would increase as fixation eccentricity increased, for each hand, Page’s L test (Siegal and Castellan, 1988) was used to test these two predictions. In both cases the hypothesis of increasing terminal errors with increasing target eccentricity was supported (left hand: $L_{(3)} = 179$, $p < 0.01$; right hand $L_{(3)} = 180$, $p < 0.01$).

**DISCUSSION**

We observed a patient, Ms D., probably affected by corticobasal degeneration, presenting with slowly progressive apraxia and balance difficulties. Longitudinal neuroimaging and neuropsychological examinations suggested primarily bilateral parietal pathology, more evident in the left hemisphere. The most unusual feature of this case is “magnetic misreaching”. When she
attempted to reach for a target in extrafoveal vision, she consistently reached for the fixation point instead. To the best of our knowledge this sign has not been previously reported.

It seems unlikely that her difficulties were related to parafoveal field defects or difficulties in processing multiple visual targets. First, no field defects were noted in a full ophthalmological examination. Second, Ms D. showed no evidence of simultanagnosia (e.g. her description of the Cookie Theft Picture [Goodglass and Kaplan, 1983] was complete and accurate). Third, she could count the number of target elements in the experimental arrays and describe the spatial relationships between them. Fourth, magnetic misreaching was also observed in the absence of a visual target to be reached towards: when Ms D. was required to fixate on a target far into one hemispace, and to reach towards the opposite side of the table surface, she still reached towards the fixation point (i.e. problems “seeing” a peripheral target to be reached towards cannot account for magnetic misreaching). Fifth, patients with parafoveal field defects can be inaccurate when pointing to targets appearing in their blind visual field. However, their reach endpoints are by no means clustered around the fixation point or any other intact portion of their visual field (Perenin, 1997; Rizzo and Darling, 1996). In other words, if patients have trouble “seeing” a target, they do not point “by default” to places which they can see.

Ms D.’s magnetic misreaching was not due to subtle difficulties with understanding or remembering task instructions. She was quite able to carry out complex multi-step procedures (e.g. in the modified Token Test, De Renzi and Faglioni, 1978). During the reaching tests, Ms D. was frequently reminded of task instructions to the point where she actually exclaimed “I know what I’m supposed to do, I just don’t know how to do it!... My hand just won’t do what I tell it to do!” Furthermore, on some trials, Ms D. did manage to partially overcome the attraction of the fixation point, and landed in between the place where she was looking and the place she was attempting to reach (see Figure 4, Left 20 and Left 30 cm Foveation). This pattern is difficult to interpret in terms of confusion about the desired target in extrafoveal reaching trials.

Magnetic misreaching has some similarities with the motor misbehaviours reported in frontal lobe dysfunction. Denny-Brown (1956, 1958) described patients with frontal lesions who failed to inhibit their grasp of felt or seen objects. There is no mention of a special role of foveal vision in generating this complex manual activity, that Denny-Brown labelled “magnetic (or frontal) apraxia”. Magnetic apraxia was unilateral in most instances. More recently, Lhermitte (1983) called attention to a bilateral form of magnetic apraxia, that he named “utilization behaviour”. Frontal patients with utilisation behaviour are compelled to use objects even when there is no attempt to draw attention to them and they are hidden from sight (Shallice, Burgess, Schon et al., 1989; Brazzelli, Colombo, Della Sala et al., 1994; De Renzi, Cavalleri and Facchini, 1996). Clearly, utilisation behaviour does not depend upon the patients foveating on the objects they covet. Moreover, patients showing either of these two signs, are typically unaware of the motor misbehaviour of the affected hand(s) (Lhermitte, Pillon and Serdaru, 1986; Brazzelli et al., 1994). A third motor misbehaviour which also resembles magnetic misreaching is anarchic hand
(Della Sala, Marchetti and Spinnler, 1994). This sign is characterised by unwanted, unilateral goal-directed actions with one hand, often performing at odds with the other hand: for example, the left hand doing up a shirt button which the right hand has just undone (Bogen, 1993, p. 360). It is extremely difficult to account for such intermanual conflict in terms of the misbehaving hand being drawn to the place where the patient is looking. In fact, in the very first report of anarchic hand, Goldstein observed that even when his patient H.M. was asleep, she would attempt to grab and squeeze her throat (Goldstein, 1908).

Nevertheless, Ms D.’s verbal reports of her hand having “a will of its’ own” parallel the anecdotes of anarchic hand patients. Similar complaints have been reported in about 50% of patients with corticobasal degeneration (Rinnie et al., 1994; see Black, 1996, for review). The upper limb of these patients often moves without purpose, wandering or levitating spontaneously. These behaviours are usually (e.g. Gibb et al., 1989) labelled as instances of “alien hand” (Brion and Jedynak, 1972), although the patients with corticobasal degeneration never fail to recognize their hand as their own (the defining characteristic of alien hand), nor, are any of their involuntary movements goal-directed (a defining characteristic of anarchic hand).

The motor misbehaviours described above have been interpreted as signs of frontal lobe dysfunction (Lhermitte, 1983; Goldberg, 1985; Shallice et al., 1989; De Renzi and Barbieri, 1992; Della Sala et al., 1994; De Renzi, Cavalleri and Facchini, 1996). Ms D. never showed “frontal” behaviour and performed normally in all tests assessing executive functions. Her neuropsychological profile (apraxia, agraphia, visuo-constructive deficits) can be accounted for by parietal dysfunction, which is demonstrated by the neuroimaging investigation, and is compatible with the diagnosis of corticobasal degeneration (Sawle, Brooks, Marsden et al. 1991). Therefore we argue that magnetic misreaching is due to parietal lobe dysfunction.

Subjects direct their gaze to targets that they are reaching towards. When a target suddenly appears, the movement of the eyes (and usually the head as well) precedes the movement of the hand (reviewed in Jeannerod, 1988). Fisk and Goodale (1985) have demonstrated that the eye movements are “yoked” to the hand movements: for example, if the hand movement onset is delayed, the eye movement which precedes the hand movement is also delayed by an equivalent amount of time. Normal subjects can, if required, inhibit eye movements to suddenly appearing targets (or direct them away rather than towards the target, the so-called “anti-saccade” task). Similarly, they can reach towards a target which they do not look at. Some patients with frontal lesions cannot inhibit eye movements to suddenly appearing visual targets (Guitton, Buchtel and Douglas, 1985; Pierrot-Deseilligny, Rivaud, Gaymard et al., 1991). On the contrary, patients with parietal lesions do not have difficulties inhibiting their eye movements during an anti-saccade task (Pierrot-Deseilligny et al., 1991).

This inability to inhibit the “visual grasp” has also been occasionally observed in patients with corticobasal degeneration (Vidailhet, Rivaud, Gouider-Khouja et al., 1994). However this subgroup of corticobasal degeneration
patients also display prominent grasp reflexes and several other signs of frontal lobe dysfunction (Jacobs, Adair and Heilman, 1994), which Ms D. did not. Even though we were unable to use sophisticated eye tracking to record the exact position of her eyes during the tests, the few saccadic intrusions that she made while we recorded magnetic misreaching were easily identified. Furthermore, her reach endpoints provide strong additional evidence of the success of her fixation efforts in extrafoveal reaching trials.

Extrafoveal reaching requires inhibition of eye movements, as does the antisaccade task. In addition, subjects must then move their hand to a target they are not foveating. Ms D. was quite capable of inhibiting her eye movements to targets that she was attempting to reach. However, she invariably failed in her attempts to reach to non-foveated locations, because, much to her dismay, her hand was “magnetized” by the direction of her gaze. Her magnetic misreaching can be interpreted as a pathological manifestation of the eye-hand yoking described above.

Under normal circumstances, Ms D. did not find her hand drawn towards any and all locations she happened to be looking at (although on some occasions she claimed that she found herself grasping an object which she did intend to pick up. Such a symptom could be understood in relation to magnetic misreaching). Magnetic misreaching was observed when we required her to initiate a goal-directed movement to an extrafoveal target. Before she was required to reach, she routinely claimed that she could “see” both the target to be reached to and the fixation point. The pathological “imbalance” of the motor affordances of each potential “target” (the correct extrafoveal target versus the incorrect foveal “target”) was revealed only once the response was initiated. It is possible that target selection mechanisms of parietal-frontal systems were impaired such that the movement was pre-programmed to be directed at the fixation point, in spite of Ms D.’s protests that she always knew what the target to be reached towards was. Another possibility is that target selection was appropriate, but that the oculomotor systems maintaining fixation somehow “hijacked” the manual response, leaving her at the mercy of a “default” eye-hand coordinating mechanism (which yokes gaze to hand movements). Although the exact nature of the neural control of target selection, localization and acquisition have yet to be specified, these sorts of mechanisms are likely to involve cooperation of parietal and frontal lobe systems.

A number of neurophysiological studies have linked posterior parietal lobe mechanisms to the guidance of both limb and eye movements. Cells with firing activity related to reaching are numerous in the posterior parietal cortex and have been well-described for over 20 years (e.g. Mountcastle et al., 1975; see Andersen, Snyder, Bradley et al., 1997; Lynch, 1980, for reviews). More recently, sophisticated behavioural training and testing paradigms have uncovered similar single-cell coding involved in the control and monitoring of eye and head movements to visual targets. These cells related to the direction of gaze are located in regions of the posterior parietal cortex which overlap with reaching-related cell activity (e.g. Brotchie, Andersen, Snyder et al., 1995; Galletti, Battaglini and Fattori, 1995; for review see Johnson, Ferraina, Garasto et al., 1997). For example, Brotchie et al. (1995) report that some neurons in the
posterior parietal cortex code for a particular gaze direction, whether or not that direction is specified by eye position in the head or head position on the torso. Such signals may provide important information for the control of hand movements to a visual target, since gaze movements are usually completed before head movements (see Jeannerod, 1988, 1997, for more complete discussion of these issues).

Although neural activity related to the control of arm movements and gaze has been found in the frontal lobes, and some of this activity may be related to “nonstandard sensorimotor mapping” (see Boussaoud, Di Pellegrino and Wise, 1995; Wise, Di Pellegrino and Boussaoud, 1996, for reviews), several recent reports implicate the posterior parietal cortex in the control of both foveal and extrafoveal reaching. Snyder, Batista and Andersen (1997) found that in the lateral intraparietal area (LIP) and in a medial, posterior region, signals related to forthcoming arm and eye movements are largely segregated. Such signals may be integrated in parietal visuomotor areas downstream from these regions, such as areas 7m and V6 (Ferraina, Johnson, Garasto et al., 1997; Johnson et al., 1997). Some cells in these latter areas responded to an eye movement to a visual target, and some to a hand movement, but a majority had both hand- and eye-movement related properties. Previous studies have implicated these regions in the control of reaching movements. For example, the oculomotor properties of cells in this region are related to both saccadic activity and eye position before as well as after saccades (Galetti et al., 1995; Johnson et al., 1997). These areas also contain cells with arm-movement related properties, and many are influenced by somatosensory information regarding limb position (Galletti, Fattori, Kutz et al., 1997). Of course, eye position information at the end of a saccade is likely to play a crucial role in the on-line modification of a hand movement towards a visual target (e.g. Fisk and Goodale, 1985). Taken together, these findings strongly suggest that the posterior parietal region of the primate brain plays a role in coordinating eye-hand movements to suddenly appearing targets, as well as inhibiting either the eye movement component (Snyder, Andersen and Brotchie, 1997) or the hand movement component (Ferraina et al., 1997).

Less is known about the precise anatomical organization of parietal lobe “extrafoveal” reaching centres in humans. Kawashima, Naitoh, Matsumura et al. (1996) conducted a positron emission tomography study of reaching tasks, under conditions where both eyes and hand were moved to the target or either eyes alone or hands alone were directed to the target. These authors conclude that two fields in the intraparietal sulcus are discernable; an anterior zone with mostly reach-related activation and a posterior zone with mostly saccade-related activity.

In conclusion, the available evidence suggests that magnetic misreaching is a sign of parietal lobe dysfunction. This sign was uncovered while our patient was receiving a series of detailed neuropsychological investigations. In this case, the absence of aphasia, dementia, visual disorientation, gross sensorimotor disturbance and the like made the discovery of magnetic misreaching possible. The slow and specific nature of degeneration in Ms. D.’s case caused the selective disruption of parietal networks, resulting in magnetic misreaching. In
other, less selective instances of parietal lobe damage (such as infarct of the middle cerebral artery) several other related parietal networks will also be compromised, resulting in complete visuomotor/attentional disturbances such as Holmes-Balint syndrome. It is conceivable that if looked for carefully, magnetic misreaching will be detected in other patients with parietal lobe damage.

Acknowledgements. The authors would like to thank Dr. O. Turnbull for administration and scoring of the frontal lobe tests, and Dr. C. Gray for his statistical advice. Prof. E. De Renzi saw the patient with us and provided helpful advice. Mr. H. Atta conducted the ophthalmological examination. Dr. K. Scott-Brown measured the luminance of target and background used in the final experiment. Dr. F. Smith performed the MR scan and provided us with valuable neuroradiological advice. Dr. S. Jackson provided helpful comments on a draft of the manuscript. Finally, we are extremely grateful to Ms D. for her extraordinary patience and endurance.

Some of these data were reported at the British Neuropsychological Society meeting in Birmingham, England (October 1996), and the 15th Annual European Workshop on Cognitive Psychology in Bressanone, Italy (January 1997).

This research was supported by an equipment grant from the Research Committee, University of Aberdeen, to D.P. Carey and O.H. Turnbull.

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(Received 7 July 1997; accepted 14 July 1997)

Note

L.J. Buxbaum and H.B. Coslett (Subtypes of optic ataxia: reframing the disconnection account. Neurocase, 3: 159-166, 1997) observed a patient (D.P.) who reached towards the examiner’s nose while trying to point to an object in his visual periphery. Although detailed description of the conditions in which this behaviour was (and was not) observed were not provided, D.P. may have displayed magnetic misreaching. The potential presence of magnetic misreaching in another patient supports our contention that the sign may be found in other cases of parietal lobe dysfunction.