

Grasping the past: delay can improve visuomotor performance

AD Milner^{1,4}, HC Dijkerman², L Pisella³, RD McIntosh¹, C Tilikete³, A Vighetto³, Y

Rossetti³

¹Department of Psychology
Science Laboratories
University of Durham, UK
Durham DH1 3LE
England.

²Psychological Laboratory
University of Utrecht
Heidelberglaan 2
3584 CS Utrecht
The Netherlands.

³Espace et Action
INSERM Unité 534
16 avenue Lépine
69676 Bron
France.

⁴Corresponding author.

Running head: Memory-based grasping in optic ataxia.

Abstract

‘Optic ataxia’ is caused by damage to the human posterior parietal cortex (PPC). It disrupts all components of a visually guided prehension movement – not only the transport of the hand towards an object’s location [1], but also the in-flight finger movements pre-tailored to the metric properties of the object [2-4]. Like previous cases [4, 5], our patient (I.G.) was quite unable to open her handgrip appropriately when directly reaching out to pick up objects of different sizes. When first tested she failed to do this even when she had previewed the target object 5 sec earlier. Yet despite this deficit in ‘real’ grasping, we found, counter-intuitively, that I.G. showed *good* grip scaling when ‘pantomiming’ a grasp for an object seen earlier, but no longer present. We then found that after practice, I.G. became able to scale her handgrip when grasping a *real* target object that she had previewed earlier. By interposing catch trials where a different object was covertly substituted for the original object during the delay between preview and grasp, we found that I.G. was now using memorized visual information to calibrate her real grasping movements. These results provide new evidence that ‘off-line’ visuomotor guidance can be provided by networks independent of the PPC.

Results

Experiment 1

In the first test session, I.G. performed three different tasks in the following order: (a) perceptual matching; (b) delayed real grasping; (c) delayed ‘pantomimed’ grasping. The perceptual task required the patient to make a simple manual size estimate using her forefinger and thumb. The two delayed grasping tasks were based on methods devised by Goodale and colleagues [6]. In the case of pantomimed grasping, the subject was required to delay grasping the object for 5 sec – during which the object was removed – and then to *pretend* to grasp it (see Figure 1). In the delayed ‘real’ grasping task, the object remained present during and after the delay, so that visual information remained available ‘on-line’ to guide the grasping action. This task was used for comparison because it more closely resembles the time-course of the pantomime-grasping task than does a straightforward immediate grasping task.

The results are shown in Figure 2. We found that our optic ataxic patient I.G. reliably varied her finger-thumb grip in proportion to the object size in the perceptual task (Figure 2a), as has been reported before in such patients [5]. As predicted, she also showed reliable grip scaling in the delayed pantomime task (Figure 2c). Thus I.G. could tailor her grip to the size of the object both in an explicitly perceptual task (matching), and in one that relied on a visual memory (pantomimed grasping). Yet much as expected, there was only weak evidence of grip scaling in the delayed *real* grasping task (Figure 2b). I.G.’s grip aperture was variable from trial to trial, in contrast with the high consistency of her perceptual estimates and pantomimed grasps.

These data demonstrate a paradoxical *improvement* of grip scaling when the stimulus was no longer present: the opposite of what would be expected in normal subjects [6]. Nevertheless, there was still a mild trend for grip scaling in the delayed real grasping task, a trend that had not been predicted. We therefore tested I.G. in a second session in which we compared delayed real grasping with standard immediate grasping.

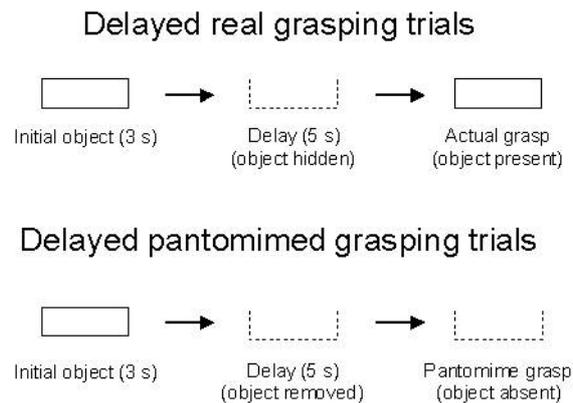


Figure 1

In this second session, I.G. performed the following tasks: (a) immediate grasping, (b) delayed real grasping, and (c) delayed pantomimed grasping. The three tasks were presented according to an 'abccbba' design. As predicted, we found no significant grip scaling during immediate grasping (Figure 3a), as in previously reported patients [4, 5]. In delayed real grasping, however, grip scaling was now very clearly observed, with I.G. opening her hand significantly less wide for the narrowest object in comparison with the other three objects (Figure 3b). Finally, as predicted, clear grip scaling was again found in the delayed pantomime-grasping task (Figure 3c). There was also a notable general reduction in I.G.'s initially exaggerated grip apertures from the first to the second testing blocks.

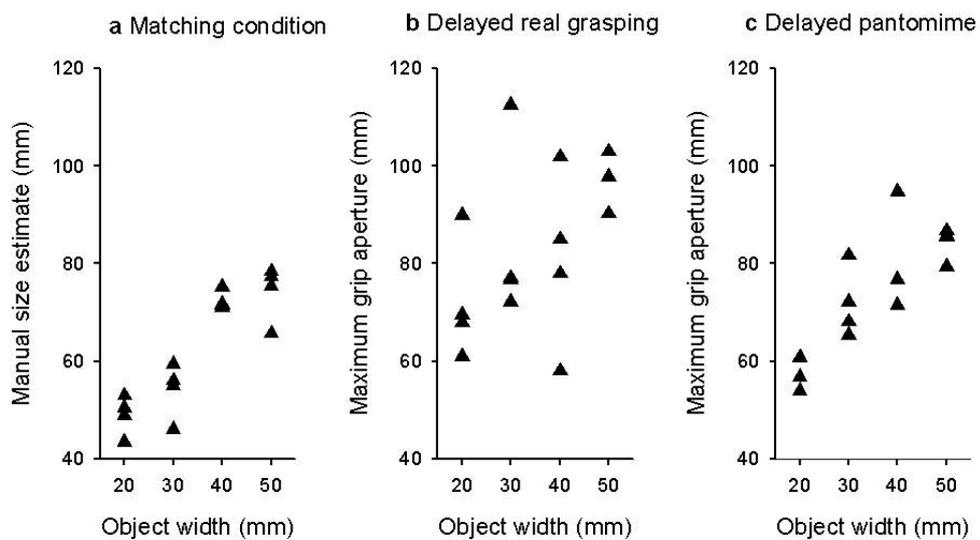


Figure 2

Thus I.G. was unable to scale her grip size when a simple immediate grasp was required: yet having previewed the object 5 sec *before* being offered it to grasp, she now adjusted her grip aperture quite well. Of course, in contrast to the immediate or pantomimed tasks, for which only one source of visual information could be used, both present *and* past visual information is potentially available in the delayed real grasping task. We had initially assumed, however, that the past information would be entirely superseded by the new sensory information available to the visuomotor system, as was shown in a different context for proprioceptive targets [7].

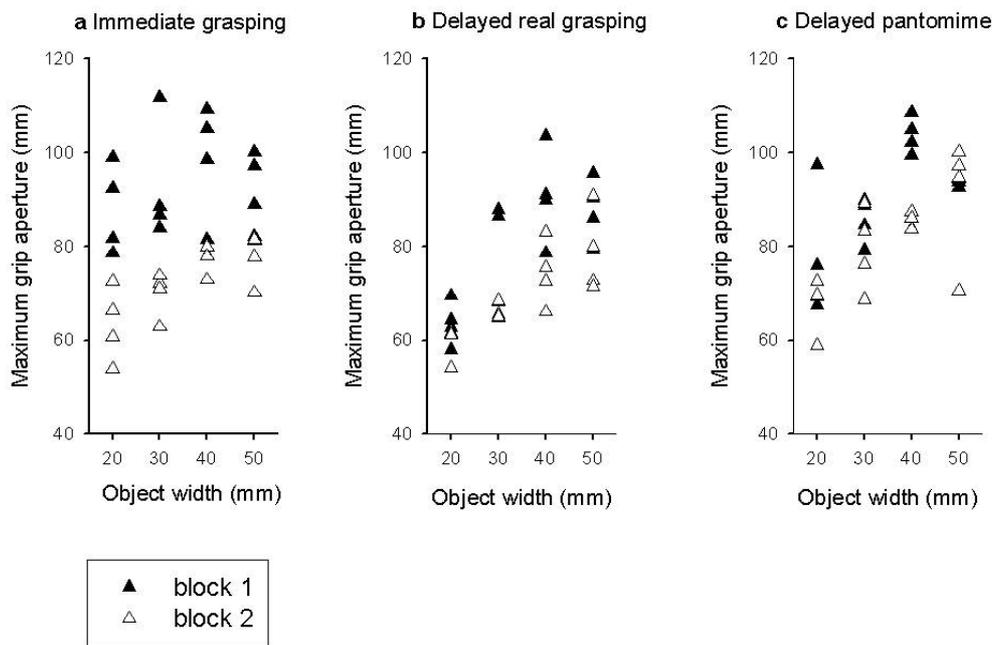


Figure 3

But in I.G.'s case, that past information would *not* be redundant. Indeed her relative success in pantomimed grasping shows that it would now provide her with *better* visual guidance than the current information, processed within her damaged visuomotor system. It is therefore possible that I.G. might have adopted a pantomiming strategy in the delayed real grasping task, rather than relying on the currently visible object to guide her hand. Support for this superficially implausible idea comes from I.G.'s reduced grip aperture during the second block of delayed real grasping trials: previous work has shown that people open the hand less widely when *pretending* to reach out and grasp than when *actually* doing so [6].

In order to determine which of the two sources of visual information was used during delayed real grasping, by I.G. and by healthy subjects, we created a new series of delayed real grasping trials in which occasional special test trials were embedded. The task was given after a series of standard delayed real grasping trials and was presented

as yet more of the same task.

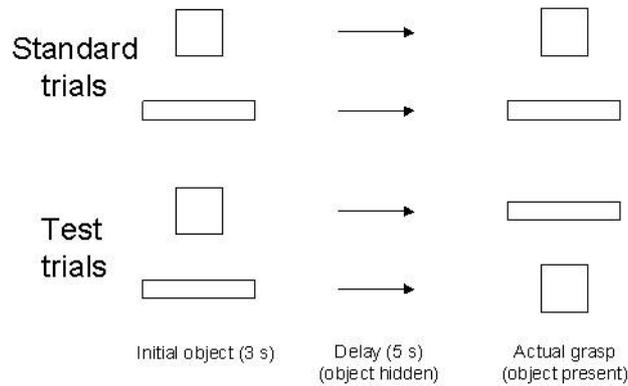


Figure 4

Experiment 2

In this experiment only delayed real grasping was tested. However, although the usual four objects were used throughout, half of the trials with the widest and narrowest objects were made into test trials. On these occasions, the narrowest (2 cm) object was covertly replaced during the delay interval with the widest (5 cm), or the widest replaced with the narrowest (see Figure 4). In total, 48 trials were performed in a pseudo-randomized order (12 for each object, including 6 ‘test’ trials for the widest and 6 for the narrowest object). Six age-matched right-handed healthy control subjects were also tested using the same paradigm.

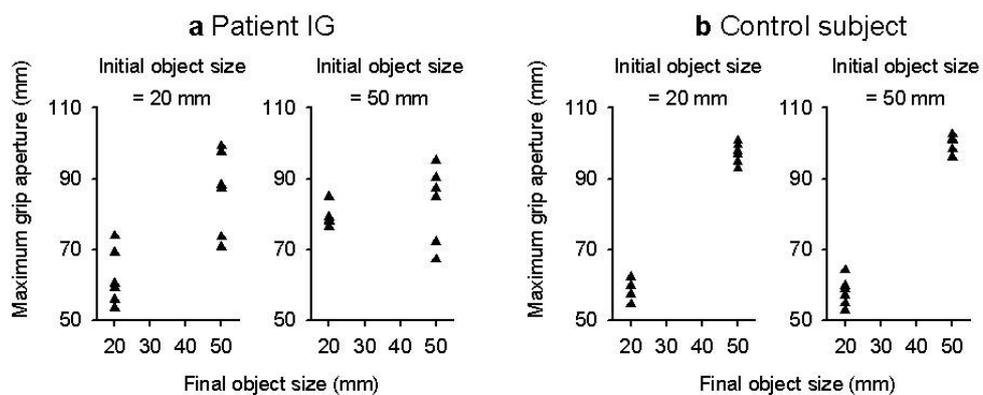


Figure 5

We confirmed that our controls opened their hands during the prehension movement entirely according to the size of the object facing them (Figure 5b). It made no difference whether or not they had been shown a different block 5 sec earlier. In striking contrast, I.G. opened her hand widely when the wide object had been previewed, even when reaching out to grasp the narrow one (Figure 5a, right). Evidently I.G. used a memory-based route to by-pass her visuomotor deficit, while the controls never did this.

On the test trials where the narrow object was replaced by the wide one, I.G.'s maximum grip did reach an appropriately wide aperture (Figure 5a, left). Presumably, the initially programmed small grip aperture had to increase during the course of the reach in order for I.G. to eventually grasp the wide object, and this would be reflected in the maximum grip aperture that we measured. Of course, on the trials when she had to close down her grip from an expected large to an actual small object, that small object was also eventually grasped correctly, but the grip would already have opened widely before that correction occurred.

Discussion and Conclusions

Patient I.G. suffered severe bilateral damage to the homologue of the monkey's 'dorsal stream' of visual processing, causing the visuomotor difficulties typical of optic ataxia. The primate dorsal stream plays a specialized role in the on-line automatic transformation of visual information into action coordinates [8-13], and indeed lesions there result in deficits closely resembling human optic ataxia [14, 15].

This pattern contrasts strikingly with the effects of damage to the primate ventral stream, which cause profound problems in shape discrimination, but spare visually controlled reaching and grasping [16, 17]. In a similar way, the well-tested patient D.F., whose visual-form agnosia renders her unable to distinguish simple shapes or contours, can nevertheless perform a range of actions guided by those same visual features [18, 19]. D.F.'s lesion has bilaterally disconnected her ventral stream from most of its visual inputs [20].

This double dissociation provided the rationale for the present study. The intact visuomotor skills retained both by 'agnosic' monkeys and by the agnosic patient D.F. have been attributed to a (largely) intact dorsal stream [21]. Yet Goodale and colleagues [6] found that D.F. was unable to respond appropriately when asked to perform pantomimed grasps in response to a memorized object no longer present. They proposed that healthy observers perform such delayed acts by means of a conscious perceptual representation of the object, rather than through direct visuomotor control. This indirect route would not be available to D.F., because she cannot achieve the necessary perceptual representation.

This proposal of two separate routes from vision to action [6] is attractive, but it relied entirely on *negative* evidence, in that patient D.F. performed exceptionally badly on the delayed task. We therefore sought complementary *positive* evidence from optic ataxia, making the prediction that patient I.G. should show *improved* performance in delayed pantomime grasping relative to immediate grasping, a converse pattern to that seen in healthy subjects. We assumed that I.G. might be able to circumvent her

damaged visuomotor system by bringing her relatively intact perceptual system into play.

We established in Experiment 1 that I.G.'s visuomotor difficulties included the misgrasping of objects of different widths presented to her in peripheral vision. At the same time we showed that, like previous patients [5], I.G. perceived the object widths quite accurately, and could signal these percepts using her finger and thumb. Most crucially, we confirmed our prediction that she should show an improvement in her grasping movements when performing a pantomime task: I.G. now showed good grip scaling. These findings thus provide the other half of a double dissociation along with the data from D.F. [6]. They are clearly consistent with the idea that posterior parietal visuomotor systems are part of the neural circuitry for mediating normal immediate object grasping [11, 13]: but are not essential for mediating *delayed* responses of an ostensibly similar kind [7, 22].

We correctly predicted good *pantomimed* grasping by I.G.. However, we expected that her *real* grasping behaviour would be equally impaired whether or not she had seen the object a few seconds earlier. We wrongly assumed that I.G. would try to use the same visual information in both cases, namely the information present in her visual field at the moment of reaching out for the object. In Experiment 2 we showed that she achieved her surprisingly good delayed real grasping through a strategy quite different from that used by normal observers. We showed this by interspersing 'invalid' probe trials within a delayed real grasping session. Healthy control subjects invariably ignored the previewed information. In sharp contrast, I.G. evidently *depended* on this previewed information, programming her grasp without regard to

the current visual information. Consequently when a wide object was, unbeknown to her, replaced by a narrow one, she opened her handgrip too widely for the object in front of her.

The present data support the idea that vision can guide action through networks other than the dedicated systems of the parietal lobe. These networks have different time constraints and different cognitive loadings attached to them [7, 23, 24]. Use of such alternative networks evidently allowed I.G.'s grasping difficulties to be circumvented, albeit by taking a slow and circuitous route from vision to action.



Figure 6

Materials and methods

Patient

I.G. had suffered a bilateral parieto-occipital infarction 17 months before the present testing (see Figure 6). She initially presented with severe headache, and dysarthria and bilateral blindness lasting for three days. Following this, bilateral optic ataxia and simultanagnosia became apparent [11, 25]. The patient had a diagnosis of ischaemic stroke, related to acute vasospastic angiopathy in the posterior cerebral arteries. MRI revealed hyperintense signal on T2 sequences that was near-symmetrically located in the posterior parietal and upper and lateral occipital cortico-subcortical regions. Reconstruction of the lesion indicated that it involved mainly Brodmann's areas 7, 18, 19, the intraparietal sulcus, and part of area 39. I.G. was aged 31 when we tested her, by which time her simultanagnosia had subsided, at least for presentations of two to three objects [11].

Apparatus

As in several previous studies [6, 19], we used rectangular blocks varying in width, but of constant surface area. Four different blocks were used, with the dimensions: 5 cm x 5 cm, 4 cm x 6.25 cm, 3 cm x 8.3 cm, and 2 cm x 12.5 cm. They were made of dark grey plastic with a thickness of 1 cm, and were presented on a table against a white background. Due to the fact that I.G.'s optic ataxia chiefly affects non-foveal vision, we presented the objects eccentrically, using a central red fixation spot. The left edge of each object was positioned 6 cm (approximately 5°) to the right of this spot. Fixation was checked continually by an experimenter facing the patient.

Grip aperture was measured by means of a magnetic movement recording system (*Minibird*, Ascension Technology Ltd), with markers attached to the tips of the forefinger and thumb. This allowed us to record finger-thumb separation, in 3D space, throughout all of the reaching and grasping movements, or for 1 sec in the case of I.G.'s size judgements in the perceptual matching task. The dependent variable of interest was the maximum grip aperture attained during reaching (MGA), or the mean finger-thumb aperture in the case of perceptual matching. These measures are linearly related to object size in healthy subjects in all of the tasks. In grasping tasks, MGA provides a direct index of the use of visual information in advance of contact with the object [26].

Acknowledgements

The authors are grateful to I.G. for her good-humoured cooperation in this research, and to the Wellcome Trust (grant no. 052443), Leverhulme Trust (grant no. F00128C), and INSERM PROGRES (grant no. 4P012E) for financial support.

References

1. Bálint R: **Seelenlähmung des 'Schauens', optische Ataxie, räumliche Störung der Aufmerksamkeit.** *Monats Psychiat Neurol* 1909, **25**: 51-81.
2. Perenin M-T, Vighetto A: **Optic ataxia: a specific disruption in visuomotor mechanisms. I. Different aspects of the deficit in reaching for objects.** *Brain* 1988, **111**: 643-674.
3. Jeannerod M: **The formation of finger grip during prehension: a cortically mediated visuomotor pattern.** *Behav Brain Res* 1986, **19**: 99-116.
4. Jakobson LS, Archibald YM, Carey DP, Goodale MA: **A kinematic analysis of reaching and grasping movements in a patient recovering from optic ataxia.** *Neuropsychologia* 1991, **29**: 803-809.
5. Jeannerod M, Decety J, Michel F: **Impairment of grasping movements following bilateral posterior parietal lesion.** *Neuropsychologia* 1994, **32**: 369-380.
6. Goodale MA, Jakobson LS, Keillor JM. **Differences in the visual control of pantomimed and natural grasping movements.** *Neuropsychologia* 1994, **32**: 1159-1178.
7. Rossetti Y, Pisella L: **Several 'vision for action' systems: a guide to dissociating and integrating dorsal and ventral functions.** In *Attention and Performance XIX: Common mechanisms in perception and action*. Edited by. Prinz W and Hommel B. Oxford: Oxford University Press; 2001: in press.
8. Jeannerod M, Arbib MA, Rizzolatti G, Sakata H: **Grasping objects: the cortical mechanisms of visuomotor transformation.** *Trends Neurosci* 1995, **18**: 314-320.

9. Andersen RA, Snyder LH, Batista AP, Buneo CA, Cohen YE: **Posterior parietal areas specialized for eye movements (LIP) and reach (PRR) using a common coordinate frame.** In *Sensory Guidance of Movement*. Edited by Bock GR and Goode J. Chichester: Wiley; 1998: 122-128.
10. Milner AD, Dijkerman HC: **Visual processing in the primate parietal lobe.** In *Comparative Neuropsychology*. Edited by Milner AD. Oxford: Oxford University Press; 1998: 70-94.
11. Pisella L, Gréa H, Tilikete C, Vighetto A, Desmurget M, Rode G, Boisson D, Rossetti Y: **An 'automatic pilot' for the hand in human posterior parietal cortex: toward reinterpreting optic ataxia.** *Nature Neurosci* 2000, **3**: 729-736.
12. Desmurget M, Epstein CM, Turner RS, Prablanc C, Alexander GE, Grafton ST: **Role of the posterior parietal cortex in updating reaching movements to a visual target.** *Nature Neurosci* 1999, **2**: 563-567.
13. Gréa H, Pisella L, Rossetti Y, Desmurget M, Tilikete C, Prablanc C, Vighetto A. **A lesion of the posterior parietal cortex disrupts on-line adjustments during aiming movements.** *Neuropsychologia*, in press.
14. Ettliger G. **Parietal cortex in visual orientation.** In *Physiological Aspects of Clinical Neurology*. Edited by Rose FC. Oxford: Blackwell; 1977: 93-100.
15. Faugier-Grimaud S, Frenois C, Stein DG: **Effects of posterior parietal lesions on visually guided behavior in monkeys.** *Neuropsychologia* 1978, **16**: 151-168.
16. Gross CG: **Visual functions of inferotemporal cortex.** In *Handbook of Sensory Physiology, Volume VII/3. Central Processing of Visual Information, Part B: Visual Centers in the Brain*. Edited by Jung R. Berlin: Springer-Verlag; 1973: 451-482.
17. Glickstein M, Buchbinder S, May JL: **Visual control of the arm, the wrist and**

- the fingers: pathways through the brain.** *Neuropsychologia* 1998, **36**: 981-1001.
18. Milner AD, Perrett DI, Johnston RS, Benson PJ, Jordan TR, Heeley DW, Bettucci D, Mortara F, Mutani R, Terazzi E, Davidson DLW: **Perception and action in ‘visual form agnosia’.** *Brain* 1991, **114**: 405-428.
 19. Goodale MA, Milner AD, Jakobson LS, Carey DP: **A neurological dissociation between perceiving objects and grasping them.** *Nature* 1991, **349**: 154-156.
 20. Murphy KJ, Carey DP, Goodale, MA: **The perception of spatial relations in a patient with visual form agnosia.** *Cognit Neuropsychol* 1998, **15**: 705-722.
 21. Milner AD, Goodale MA: *The Visual Brain in Action*. Oxford: Oxford University Press; 1995.
 22. Rossetti Y: **Implicit short-lived motor representations of space in brain damaged and healthy subjects.** *Consc Cognit* 1998, **7**: 520-558.
 23. Rossetti Y, Pisella L, Pélisson D: **Eye blindness and hand sight: temporal aspects of visuo-motor processing.** *Vis Cognit* 2000, **7**: 785-809.
 24. Milner AD, Dijkerman HC: **Direct and indirect visual routes to action.** In *Out of Mind: Varieties of Unconscious Processes*. Edited by De Gelder B, De Haan E and Heywood C.: Oxford: Oxford University Press; 2001: 237-260.
 25. Pisella L, Tiliket C, Rode G, Boisson D, Vighetto A, Rossetti Y: **Automatic corrections prevail in spite of an instructed stopping response.** In *Studies in Perception and Action*. Edited by. Grealy M and Thomson JA. Hillsdale NJ: Erlbaum; 1999: 275-279.
 26. Jeannerod M: **Intersegmental coordination during reaching at natural visual objects.** In *Attention and Performance XI*. Edited by Long J and Baddeley A. Hillsdale NJ: Erlbaum; 1981: 153-168.

Figure Legends

Figure 1

The delayed grasping tasks used in the present study.

In both delayed tasks (real and pantomimed), the object was first viewed for 3 sec, and then shielded from view for 5 sec. In delayed real grasping, the subject then had to reach out and grasp the object. In pantomimed grasping, however, the subject had to *pretend* to reach out and grasp the object after this delay, as it had been covertly removed during the delay period.

Figure 2

Experiment 1: I.G.'s maximum grip aperture (MGA) during a perceptual matching task and two delayed prehension tasks. Within each block of trials, each object was presented four times in a pseudo-randomized order (no object was presented twice in succession).

Linear regression analysis showed that I.G. scaled her grip size in relation to object width at high levels of significance in both (a) the matching task [$r^2 = 0.80$; $F(1,14) = 56.08$] and (c) the delayed pantomime task [$r^2 = 0.66$; $F(1,14) = 26.75$, $p < 0.001$].

There was much less indication of grip scaling in the delayed real grasping task (b), though it did just reach significance [$r^2 = 0.26$; $F(1,14) = 4.97$, $p < 0.05$]. Grip size varied from trial to trial considerably more during delayed real grasping (average SD per object width = 13.65) than during pantomimed grasping (average SD = 6.48) or perceptual matching (average SD = 4.38).

Figure 3

Experiment 1: I.G.'s maximum grip aperture during three different prehension tasks.

(a) In the immediate grasping task, the subject simply had to reach out to pick up the target object, front to back using forefinger and thumb, as soon as it became visible.

Linear regression showed no significant relation between object width and MGA

during this task [$r^2 = 0.08$; $F(1, 30) = 2.53$]. (b) During delayed real grasping,

however, clearly significant grip scaling was observed [$r^2 = 0.43$, $F(1,30) = 22.91$,

$p < 0.001$]. (c) As expected, highly significant grip scaling was also found in the

delayed pantomime grasping task [$r^2 = 0.40$, $F(1,30) = 19.83$, $p < 0.001$]. In all three

tasks, MGA was significantly smaller during the second block of trials [immediate

grasping: $F(1,24) = 46.37$, $p < 0.001$; delayed real grasping: $F(1,24) = 20.82$, $p < 0.001$;

delayed pantomimed grasping $F(1,24) = 11.74$, $p < 0.005$].

Figure 4

Schematic depiction of Experiment 2.

In a quarter of all trials, the widest object (50 mm) was covertly replaced by the narrowest (20 mm), or vice-versa, during the delay period (bottom). In another quarter of the trials the narrowest and widest objects remained unchanged (top). In the remaining half of the trials, objects of intermediate widths were used (30 and 40 mm, not depicted here), and remained unchanged throughout each trial.

Figure 5

Experiment 2: Maximum grip aperture a function of final object size for patient I.G. and one representative control subject.

The MGA data were analysed for each subject individually using ANOVAs with initial and final object size as independent variables. For all of the subjects there was a significant main effect of final object size ($p < 0.005$), but none of initial object size. However, there was a significant interaction between initial and final object size for patient I.G. only [$F(1, 19) = 7.01, p < 0.02$]. Inspection of the data reveals that when the initial object was 5 cm wide and covertly replaced by the 2 cm wide object, I.G. programmed her grip size on the basis of the initial large object width. All of the six control subjects always used the final object size for programming their MGA, irrespective of whether it had changed during the trial.

Figure 6

A coronal section through I.G.'s brain, visualized with structural MRI. Extensive damage is present bilaterally in the posterior parietal lobes.