

# Prehension movements in a patient (AC) with posterior parietal cortex damage and posterior callosal section

Victor Frak<sup>a,\*</sup>, Yves Paulignan<sup>b</sup>, Marc Jeannerod<sup>b</sup>, François Michel<sup>b</sup>, Henri Cohen<sup>c</sup>

<sup>a</sup> *Département de Kinanthropologie, Université du Québec à Montréal, Que., Canada*

<sup>b</sup> *Institut des Sciences Cognitives, Bron, France*

<sup>c</sup> *Cognitive Neuroscience Center, Université du Québec à Montréal, Que., Canada*

Accepted 19 September 2005

Available online 3 November 2005

## Abstract

Prehension movements of the right hand were recorded in a right-handed man (AC), with an injury to the left posterior parietal cortex (PPC) and with a section of the left half of the splenium. The kinematic analysis of AC's grasping movements in direct and perturbed conditions was compared to that of five control subjects. A novel effect in prehension was revealed—a hemispace effect—in healthy controls only. Movements to the left hemispace were faster, longer, and with a smaller grasp aperture; perturbation of both object position and distance resulted in the attenuation of the direction effect on movement time and the time to velocity peak, with a reverse pattern in the time to maximum grip aperture. Nevertheless, the correlation between transport velocity amplitude and grasp aperture remained stable in both perturbed and non-perturbed movements, reflecting the coordination between reaching and grasping in control subjects. In contrast, transport and grasp, as well as their coordination in both direct and perturbed conditions, were negatively affected by the PPC and splenium lesion in AC, suggesting that transport and grasp rely on two functionally identifiable subsystems.

© 2005 Elsevier Inc. All rights reserved.

*Keywords:* Posterior parietal cortex; Prehension; Visuomotor channels; Callosal section

## 1. Introduction

One of the most influential theories of prehensile movements in both primates and humans is that proposed by Jeannerod (1981). In this theory, Jeannerod claims that two distinct processing components are involved in prehension movements: one responsible for the transport of the arm to the object—also called the reach channel and experimentally assessed by the wrist movement—and one for the grip of the object—also called the grasp channel and assessed as the distance between the thumb and index fingers. Each channel extracts specific characteristics of the object and transforms them into an adequate prehension act.

The postulated independence of these processing streams has motivated numerous kinematic studies of normal

prehension in humans. One result on which most agree is that transport velocity (of the wrist) and movement amplitude are a direct consequence of the distance between the hand and the object (Gentilucci et al., 1991). However, varying the location of the object yields conflicting results with respect to the grasping module. For example, Jeannerod (1981) found no specific effect, whereas others reported an increase in grip aperture (Chieffi & Gentilucci, 1993).

Several sets of neurophysiological data suggest that the parietal posterior cortex (PPC) possesses a functional modality compatible with the visuomotor transformations. The PPC is activated by the prehensile characteristics of the objects (Kusunoki, Tanaka, Ohtsuka, Ishiyama, & Sakata, 1993), and there exist direct anatomical evidence for largely segregated visuomotor pathways linking PPC with the lateral premotor cortex supporting the notion of parallel visuomotor processing streams (Tanné-Gariépy, Rouiller, & Boussaoud, 2002). Also, individuals with lesions of the PPC have prehension disabilities such as an unskillful grasp and

\* Corresponding author. Fax: +1 514 987 6616.  
E-mail address: [frak.victor@uqam.ca](mailto:frak.victor@uqam.ca) (V. Frak).

a defective or non-existent preshaping (Jeannerod, 1986). Moreover, transcranial magnetic stimulation of the PPC disrupts transport corrections that normally take place when a target position is perturbed (Desmurget et al., 1999).

In this perspective, the purpose of the present study was to investigate direct and perturbed prehension in a patient with a partial PPC lesion without objective evidence of prehension disorders, with a 3-D higher resolution kinematic analyzer. The rapid perturbation of object position adopted here attempted to replicate a unique motor symptom reported by the patient: difficulties with recapturing his partner's hand when dancing rock'n'roll.

## 2. Method

### 2.1. Subjects

Patient AC was a 25-year-old right-handed man, university student who, after undergoing surgery for a haemorrhage in his left mesial parietal lobe due to a small arteriovenous malformation, was left with a partial section of the posterior half of the corpus callosum. AC participated in the present experiment 2 years after the surgery. An MRI examination revealed a porencephalic cyst in the left parietal lobe and a cut of the posterior half of the corpus callosum, as shown in Fig. 1A. At the time of the study, AC had normal visual fields, no signs of optic ataxia, no paresis, or hypoesthesia no simultagnosia or visual neglect or apraxia. His only motor complaint was of having some difficulties with recapturing his partner's hand when dancing rock'n'roll. He showed left hemialexia (reported previously in Michel, Hénaff, & Intriligator, 1996, and in Cohen et al., 2000) as observed following posterior callosal lesions (Suzuki et al., 1998).

Five healthy controls (three men and two women), all right-handed with ages ranging from 22 to 38 year and with no detected neurological disorders also participated in the experiment. Before the experiment, all subjects were informed about the methods used and the purpose of the study was revealed once the experiment was over. All subjects gave informed consent and the experiment was approved by the localethics committee.

### 2.2. Experimental design

Five identical cylinders (10 cm high, 1.5 cm in diameter, 50 g weight) were used as targets. The position of the cylinders in the workspace relative to the subject's hand and body axis is illustrated in Figs. 1B, C, and D. Subjects were instructed to reach, grasp, and lift an object using a precision grip [Napier, 1956]. The direct and perturbed movements were randomly produced in a session of 300 movements. In 20% of the cases, the object was illuminated in one of three positions ( $-20^\circ$ ,  $0^\circ$ , or  $20^\circ$ ) and, when the transport of the arm began there was illumination of a second object located on the left or the right side of the first illuminated one resulting in a location perturbation. Thirty movements in a left–right perturbation (10 from  $-20^\circ$  to  $0^\circ$ , from  $0^\circ$  to  $20^\circ$ , and from  $20^\circ$  to  $40^\circ$ , respectively) and 30 movements in a right–left direction (from  $20^\circ$  to  $0^\circ$ , from  $0^\circ$  to  $-20^\circ$ , and from  $-20^\circ$  to  $-40^\circ$ ). As well, 80 non-perturbed movements for each  $20^\circ$ ,  $0^\circ$ , and  $-20^\circ$  object position were also recorded.

**Movement recording:** The spatial positions of three active markers placed on the nails of both the right thumb and index finger, and on the right radial styloid process of the wrist were sampled at 200 Hz by means of an

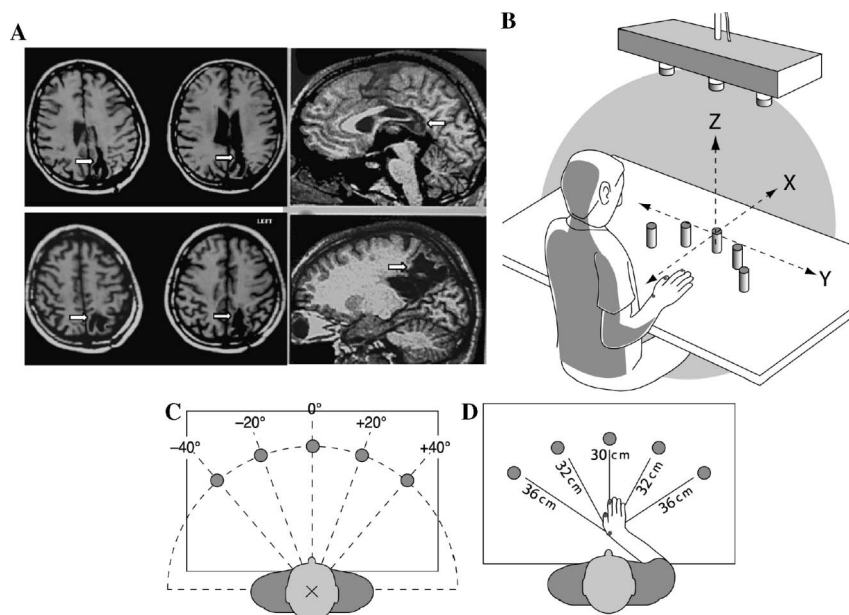


Fig. 1. (A) Sagittal and axial MRI brain sections in AC with posterior callosal and parietal lesions (white arrows). (B) The 3-D kinematic analyzer was fixed at 2.5 m above the workspace with its optical axis aligned with the vertical. Five identical cylinders were used as targets. (C and D) Position of the cylinders in the workspace relative to the subject's hand and body axis.

Optotrak 3020 system. The camera was fixed at 2.5 m above the workspace with its optical axis aligned with the vertical as illustrated in Fig. 1B. Each trial was recorded for 5 s. After the acquisition stage, the position data were filtered with a second-order Butterworth filter with a forward and reverse pass. A lowpass cutoff frequency of 10 Hz was used. Movement onset was determined as the first of seven consecutive measures of increasing amplitude on the fingers' velocity. The movement endpoint was determined as the point where the interfinger distance stopped decreasing while gripping the cylinder. The dependent variables in non-perturbed movements were (a) movement time (MT); (b) transport component parameters, time to first velocity peak (TPV1), amplitude of first velocity peak of the wrist marker (APV1), and (c) grip component parameters, time to first grip aperture (TGA1), amplitude of first grip aperture (AGA1). In the perturbed movements there was (a) movement time; (b) transport component parameters, time to second velocity peak (TPV2), amplitude of second velocity peak of the wrist marker (APV2), and (c) grip component parameters, time to second grip aperture (TGA2), amplitude of second grip aperture (AGA2).

### 2.3. Statistical design

Statistical analyses were conducted to answer three questions: (1) Does object position affect movement parameters similarly for controls and patient? (2) Does the direction of the perturbation of object position affect movement parameters similarly for controls and patient? (3) Do movement parameters in perturbed and unperturbed movements change similarly for controls and patient? To test for the

effect of unperturbed object position or direction of perturbation during movement, a mixed model for repeated measures was specified with movement repetitions nested in subjects and the three positions (question 1), or the two directions of perturbation (question 2) crossed with subjects. The effect of Position and Condition (controls, AC) as well as their interaction were considered. When there was a significant interaction between Condition and Position (question 1), or direction of perturbation (question 2), a mixed model for repeated measures was adopted with repetitions nested in subjects and the three positions (question 1), or directions of perturbation (question 2) crossed with subjects with the Condition factor was removed. To test the effect of perturbation on movement parameters (question 3), a similar mixed model for repeated measures was specified with movement repetitions also nested in subjects and with Position nested in perturbation, whereas this last factor was crossed with subjects. The main effects of perturbation and Condition, as well as their interaction were also considered, controlling for Position. Significance level was set at  $p < .05$ . Post hoc multiple comparisons using Bonferroni adjustment were used. Table 1 gives the descriptive statistics for the dependent variables in perturbed and non-perturbed movements.

## 3. Results

### 3.1. Question 1: Does object position affect movement parameters similarly for controls and patient?

#### 3.1.1. Movement time

A main effect of Position was observed for both controls and AC ( $F_{(2,462)} = 142.532$ ;  $p < .001$ ). Post hoc multiple

Table 1  
Effect of object location on the transport grasp components in controls and AC

	Direct prehension movements mean and standard deviation			Components	Perturbed prehension movements mean and standard deviation	
	Left	Center	Right		Left	Right
<i>Transport grasp components controls</i>						
Movement time (ms)	823 ± 132	742 ± 101	722 ± 96	MT, ms	1012 ± 178	1011 ± 187
Reach				Reach		
Time to velocity peak (ms)	338 ± 50	308 ± 48	294 ± 59	TPV2, ms	601 ± 79	598 ± 117
Velocity peak amplitude (mm/s <sup>2</sup> )	1003 ± 155	928 ± 154	909 ± 168	APV2, mm/s <sup>2</sup>	701 ± 119	574 ± 141
Grasp				Grasp		
Time to maximum grip aperture (ms)	531 ± 94	471 ± 76	462 ± 76	TGA2, ms	691 ± 105	718 ± 154
Maximum grip aperture (mm)	62 ± 6.4	64 ± 6.1	68 ± 6.6	AGA2, mm	69 ± 7.4	79 ± 12.5
<i>Transport grasp components AC</i>						
Movement time (ms)	1010 ± 123	924 ± 86	928 ± 88	MT, ms	1334 ± 121	1337 ± 104
Reach				Reach		
Time to velocity peak (ms)	321 ± 35	284 ± 27	257 ± 39	TPV2, ms	666 ± 131	730 ± 89
Velocity peak amplitude (mm/s <sup>2</sup> )	999 ± 103	901 ± 108	816 ± 68	APV2, mm/s <sup>2</sup>	698 ± 79	679 ± 85
Grasp				Grasp		
Time to maximum grip aperture (ms)	660 ± 70	579 ± 64	564 ± 73	TGA2, ms	921 ± 128	993 ± 95
Maximum grip aperture (mm)	50 ± 5.3	52 ± 6.1	52 ± 6.9	AGA2, mm	52 ± 6.1	49 ± 4.7

Abbreviations: MT, movement time; TPV2, Time to 2nd velocity peak; APV2, 2nd velocity peak amplitude; TGA2, time to 2nd maximum grip aperture; AGA2, 2nd maximum grip aperture.

comparisons indicated that movement times to the left took 80 ms more than movements to the center ( $p < .001$ ) and 90 ms more than movements to the right ( $p < .001$ ). Movements to the center and the right did not differ significantly for controls or for patient. Control subjects had a significantly shorter movement time at all positions compared to AC's performance ( $F_{(1,112)} = 104.052$ ;  $p < .001$ ). On average, unperturbed movements were 190 ms faster for controls.

### 3.1.2. Reaching

There were significant interactions between Condition and Position on time to velocity peak ( $F_{(2,613)} = 4.493$ ;  $p = .012$ ) and velocity peak amplitude ( $F_{(2,237)} = 20.815$ ;  $p < .001$ ). Post hoc multiple comparisons showed that time to velocity peak differed between patient and controls ( $p < .001$ ). ANOVA results also showed that time to velocity peak was shorter for AC at all three positions ( $p < .02$ ). The position of the object influenced velocity peak amplitude for both patient and controls. Furthermore, post hoc multiple comparisons indicated that velocity peak amplitude was significantly smaller for AC compared to the controls ( $p < .005$ ). Thus, movements to the left were characterized by longer latencies and larger amplitudes than movements to the center or the right for both controls and patient.

### 3.1.3. Grasping

A main effect of Position on time to maximum grip aperture ( $F_{(2,914)} = 91.212$ ;  $p < .001$ ) was revealed for both controls and patient. Post hoc analyses revealed that time to maximum grip aperture is significantly longer for movements to the left compared to movements to the center by an average of 70 ms ( $p < .001$ ), and significantly longer for movements to the left than to the right by an average of 80 ms ( $p < .001$ ). The difference of 10 ms between the movements to the center and those to the right is not significant ( $p = .112$ ). Also, control subjects showed a significantly shorter time to maximum grip aperture by 110 ms compared to AC ( $F_{(1,239)} = 159.438$ ;  $p < .001$ ). There was an interaction between Condition and Position ( $F_{(2,781)} = 15.394$ ;  $p < .001$ ) showing that maximum grip amplitude is dependent upon the position of the object, for controls only ( $p < .001$ ). There was no change in maximum grip amplitude for AC at all three positions ( $p = .083$ ). Post hoc multiple comparisons showed that all pairwise comparisons, for controls only, are different for maximum grip aperture ( $p < .001$ ). Although movements to the left were characterized by longer time to maximum grip aperture for both patient and controls, AC did not show a difference in grip aperture per se between the three positions whereas the control subjects had significantly smaller apertures for movements to the left and to the center than for movements to the right. Grip apertures were smaller and time to maximum grip aperture was significantly longer at all positions for AC relative to the controls' performance. Thus, the longer movement times observed in AC reflect the longer time needed for maximum grip aperture.

3.2. *Question 2: Does the direction of the perturbation of object position affect movement parameters similarly for controls and patient?*

#### 3.2.1. Movement time

This measure was not affected by the direction of perturbation, for both controls and patient ( $F_{(1,81)} = 1.468$ ;  $p = .229$ ). However, the control subjects' movements were faster in the two positions, relative to the patient's ( $F_{(1,20)} = 24.136$ ;  $p < .001$ ).

#### 3.2.2. Reaching

There was an interaction between Condition and Position on time to velocity peak ( $F_{(1,116)} = 5.990$ ;  $p = .016$ ). Results indicated that the direction of perturbation affects time to velocity peak for AC ( $F_{(1,8)} = 13.332$ ;  $p = .006$ ) but not for controls ( $F_{(1,111)} = 0.059$ ;  $p = .808$ ). For AC, the time to velocity peak was slower with perturbations to the right than to the left (731 ms vs. 667 ms), and was also slower in both directions of perturbation compared to controls ( $p < .02$ ).

There was also an interaction between Condition and Position on velocity peak amplitude ( $F_{(1,129)} = 5.978$ ;  $p = .016$ ). Direction of perturbation influences velocity peak amplitude for controls only ( $F_{(1,113)} = 61.171$ ;  $p < .001$ ), the amplitude being larger for perturbations to the left than to the right (701 vs. 575 mm/s<sup>2</sup> respectively). The difference of 19.5 mm/s<sup>2</sup> between the two directions of perturbation in AC was not significant ( $p = .170$ ). In addition, the ANOVA results revealed that velocity peak amplitude is larger for AC, compared to controls, but only in movements with perturbations to the right ( $p = .007$ ).

Although perturbations to the right were characterized by longer latencies than perturbations to the left in AC, there was no such effect seen in controls. On the other hand, velocity peak amplitude was larger for movements to the left in controls only. Also, relative to controls, time to velocity peak for AC was slower in both directions of perturbation, and amplitude was larger only in perturbations to the right.

#### 3.2.3. Grasping

ANOVA results showed a main effect of perturbation on time to maximum grip aperture for both controls and patient ( $F_{(1,105)} = 13.197$ ;  $p < .001$ ), longer for movements perturbed to the right than to the left, by an average of 35 msec ( $p < .001$ ). Also, the controls were faster than AC by 250 msec ( $F_{(1,27)} = 34.097$ ;  $p < .001$ ). There was an interaction between Condition and Position ( $F_{(1,127)} = 20.267$ ;  $p < .001$ ) on maximum grip aperture. Results indicated that direction of perturbation influences maximum grip amplitude for controls ( $F_{(1,110)} = 63.200$ ;  $p < .001$ ) but failed to reach significance for AC ( $F_{(1,26)} = 3.743$ ;  $p = .064$ ). Maximum grip aperture is larger in controls for perturbations to the right than for those to the left (79 mm vs. 69 mm respectively). Further analysis also showed that AC had a significantly smaller grip aperture for both directions of

perturbations compared to controls ( $p < .001$ ). Thus, perturbations to the right were characterized by longer time to maximum grip aperture than perturbation to the left in both AC and controls; the controls had significantly larger apertures for perturbations to the right whereas AC showed no difference between the two directions of perturbation; grip apertures were smaller and time to maximum grip aperture was longer in both directions in AC. In contrast to direct movements where only grasping contributes to the longer latencies, the longer movement times in perturbed movements observed in AC reflect the time needed for both reaching and grasping.

### 3.3. Question 3: Do movement parameters in perturbed and unperturbed movements change similarly for controls and patient?

#### 3.3.1. Reaching

There was a significant interaction between Condition and perturbation on time to velocity peak ( $F_{(1,988)} = 99.977$ ;  $p < .001$ ) suggesting that time to velocity peak is slower for control subjects with unperturbed movements whereas it is significantly faster with perturbed ones ( $p < .002$ ). Velocity peak amplitude was significantly larger for unperturbed movements compared to perturbed ones in controls ( $F_{(1,276)} = 1041.376$ ;  $p < .001$ ) as well as in AC ( $F_{(1,180)} = 105.490$ ;  $p < .001$ ).

#### 3.3.2. Grasping

ANOVA results showed the usual effect of perturbation on time to maximum grip aperture for controls ( $F_{(1,895)} = 620.197$ ;  $p < .001$ ) and for AC ( $F_{(1,155)} = 568.203$ ;  $p < .001$ ). For all subjects, the time to maximum grip aperture was faster for unperturbed than for perturbed movements. There was a significant interaction between Condition and perturbation on maximum grip aperture ( $F_{(1,1031)} = 38.900$ ;  $p < .001$ ). The perturbation of the objects' position influences maximum grip aperture for controls ( $F_{(1,806)} = 175.509$ ;  $p < .001$ ) but not for AC ( $F_{(1,191)} = 0.413$ ;  $p = .521$ ). For controls, it is significantly larger with the perturbed than with unperturbed movements (75 mm vs. 65 mm respectively).

## 4. Discussion

Few studies (Connolly & Goodale, 1999) have examined the effect of movement direction on prehension. In their study with right-handed healthy subjects, Paulignan, Frak, Toni, and Jeannerod (1997) reported a longer movement time, a smaller grasp aperture, and late and higher wrist velocity peaks with movements to the left than to the right. However, it was not clear whether the object position or the distance between wrist and object (i.e., movement amplitude) was responsible for this effect. In the present study, the distance between the wrist starting position and the object in the direct prehension condition was the same (32 cm) on the right and on the left hemispaces. Thus, in

healthy individuals, movement direction impacts on the grasp and transport components. Similar results have also been reported in monkeys (Roy, Paulignan, Meunier, & Boussaoud, 2002). In direct prehension (without perturbation), right hand movements in the left hemisphere are of longer duration and latencies, faster velocity peaks and with a smaller aperture than for movements in the right hemisphere.

It is known that a perturbation of the spatial position of an object alters an ongoing motor command to engage the same object at the new location (Goodale, Péliesson, & Prablanc, 1986). Also, the trajectory of the hand can be adjusted early in the movement when the spatial location of a target has been modified at movement onset (Prablanc & Martin, 1992). In the current study, this adjustment to perturbation is reflected by a slower transport velocity, longer movement time and latencies as well as a larger grasp aperture. This is in agreement with our previous observations with healthy subjects (e.g., Paulignan, Mackenzie, Marteniuk, & Jeannerod, 1991). In addition, grasp aperture for movements to the left direction is always smaller—whether in direct or perturbed conditions. Object location perturbations are also perturbations in distance between wrist marker and target (e.g.,  $-20^\circ/32$  cm to  $0^\circ/30$  cm;  $0^\circ/30$  cm to  $20^\circ/32$  cm). It is well known that movement amplitude has an effect on movement time, latency, transport velocity and grasp aperture (Gentilucci et al., 1991). The perturbation of both object position and distance results in the attenuation of the direction effect for most measures seen in direct prehension, except for transport velocity amplitude and grasp aperture, suggesting a putative coordination between the two visuomotor channels.

Although there was no objective clinical evidence of grasp disorders—in addition to performing three hundred movements without once letting the cylinders slip or drop—prehension impairments were revealed in the kinematic analyses of AC's movements, thus confirming the role of the damaged brain regions in prehension. In contrast to the performance of control subjects, there was no relationship between transport velocity amplitude and grasp aperture in the two conditions, suggesting an absence of coordination between channels in AC.

Furthermore, each processing channel appears affected by the lesion. The hemisphere effect seen in controls with direct movements does not hold for the grasp channel in AC: maximum grip aperture remained the same in both hemispaces. With perturbed movements, the direction effect seen in controls no longer holds, revealing an additional impairment in the reach channel: there is no influence of direction on velocity peak amplitude in AC. These findings suggest that transport and grasp rely on two functionally identifiable systems. In contrast to what is seen in healthy individuals, these observations reveal that both visuomotor streams as well as the coordination of these two components are disturbed in AC. Thus the injury to both channels has been independently detected according to their functional requirement.



The principal argument against the dual channel theory is that an alteration in the extrinsic properties of the object (such as position) also affects the grasp component (Castiello, Bennet, & Chambers, 1998). In the present study, this is also what we observed: a perturbation of object location impacts upon aperture size in healthy subjects. However, AC's performance reinforces the hypothesis of the presence of two interdependent channels.

It has been shown that ipsilateral muscles to the transport grasp mechanism can be influenced by bilateral hemispheric networks in both humans (Farnè et al., 2003) and monkeys (Brinkman & Kuypers, 1973) and it has been presumed that interhemispheric communication is necessary for the coordination of reaching and grasping (Gazzaniga, 2000). However, it is not clear how the lesion to the splenium of the corpus callosum contributes to the absence of coordination between the two visuomotor channels seen in AC. In direct prehension, AC showed a direction effect similar to that of healthy subjects in movement time and latencies. In addition, AC reaches velocity peak before controls for wrist movements in direct prehension in both hemispaces, while grasp aperture remains constant in both direct and perturbed prehension. In perturbed prehension, movement time does not differ between movements in both hemispaces. These results highlight the important contribution of the PPC to these processes.

### Acknowledgments

The authors are indebted to Julie Lamoureux MSc, Centre de recherche interdisciplinaire en réadaptation du Montréal métropolitain, for her invaluable contribution in the statistical analyses.

### References

- Brinkman, J., & Kuypers, H. G. (1973). Cerebral control of contralateral and ipsilateral arm, hand and finger movements in the split-brain rhesus monkey. *Brain*, *96*, 653–674.
- Castiello, U., Bennet, H., & Chambers, H. (1998). Reach to grasp: The response to a simultaneous perturbation of object position and size. *Exp. Brain Res.*, *120*, 31–40.
- Chieffi, S., & Gentilucci, M. (1993). Coordination between the transport and the grasp component during prehension movements. *Exp. Brain Res.*, *94*, 471–477.
- Cohen, L., Dehaene, S., Naccache, L., Lehéricy, S., Dehaene-Lambertz, G., Hénaff, M. A., & Michel, F. (2000). The visual word form area. Spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. *Brain*, *123*, 291–307.
- Connolly, J. D., & Goodale, M. A. (1999). The role of visual feedback of hand position in the control of manual prehension. *Exp. Brain Res.*, *125*, 281–286.
- Desmurget, M., Epstein, C. M., Turner, R. S., Prablanc, C., Alexander, G. E., & Grafton, S. T. (1999). Role of the posterior parietal cortex in updating reaching movements to a visual target. *Nat. Neurosci.*, *6*, 563–567.
- Farnè, A., Roy, A. C., Paulignan, Y., Rode, G., Rossetti, Y., Boisson, D., & Jeannerod, M. (2003). Visuo-motor control of the ipsilateral hand: evidence from right brain-damaged patients. *Neuropsychologia*, *41*, 739–757.
- Gazzaniga, M. S. (2000). Cerebral specialization and interhemispheric communication: Does the corpus callosum enable the human condition? *Brain*, *123*, 1293–1326.
- Gentilucci, M., Castiello, U., Corradini, M. L., Scarpa, M., Umiltà, C., & Rizzolatti, G. (1991). Influence of different types of grasping on the transport component of prehension movements. *Neuropsychologia*, *29*, 361–378.
- Goodale, M. A., Pélisson, D., & Prablanc, C. (1986). Large adjustments in visually guided reaching do not depend on vision of the hand and perception of target displacement. *Nature*, *320*, 748–750.
- Jeannerod, M. (1981). Intersegmental coordination during reaching at natural visual objects. In J. Long & A. Baddeley (Eds.), *Attention and performance* (pp. 153–168). Hillsdale, NJ: Lawrence Erlbaum Associates Publishers.
- Jeannerod, M. (1986). The formation of finger grip during prehension. A cortically mediated visuo-motor pattern. *Behav. Brain Res.*, *19*, 99–116.
- Kusunoki, M., Tanaka, Y., Ohtsuka, H., Ishiyama, K., & Sakata, H. (1993). Selectivity of the parietal visual neurons in the axis orientation of objects in space. Society for Neuroscience Abstracts. *J. Neurosci.*, *19*, 770.
- Michel, F., Hénaff, M. A., & Inrtiligator, J. (1996). Two readers in the same brain, after a posterior callosal lesion. *Neuroreport*, *7*, 786–788.
- Napier, J. R. (1956). The prehensile movements of the human hand. *Journal of Bone and Joint Surgery*, *38 B*, 902–913.
- Paulignan, Y., Frak, V. G., Toni, I., & Jeannerod, M. (1997). Influence of object position and size on human prehension movements. *Exp. Brain Res.*, *114*, 226–234.
- Paulignan, Y., Mackenzie, R., Marteniuk, R., & Jeannerod, M. (1991). Selective perturbation of visual input during prehension movements. I. The effects of changing object position. *Exp. Brain Res.*, *83*, 502–512.
- Prablanc, C., & Martin, O. (1992). Automatic control during hand reaching at undetected two-dimensional target displacements. *J. Neurophysiol.*, *67*, 455–469.
- Roy, A. C., Paulignan, Y., Meunier, M., & Boussaoud, D. (2002). Prehension movements in the macaque monkey: Effects of object size and location. *J. Neurophysiol.*, *88*, 1491–1499.
- Suzuki, K., Yamadori, A., Endo, K., Fujii, T., Ezura, M., & Takahashi, A. (1998). Dissociation of letter and picture naming resulting from callosal disconnection. *Neurology*, *51*, 1390–1394.
- Tanné-Gariépy, J., Rouiller, E. M., & Boussaoud, D. (2002). Parietal inputs to dorsal versus ventral premotor areas in the macaque monkey: Evidence for largely segregated visuomotor pathways. *Exp. Brain Res.*, *145*(1), 91–103.