Brain (1999), **122,** 1867–1874

Perception of self-generated movement following left parietal lesion

Angela Sirigu,[1] Elena Daprati,[4] Pascale Pradat-Diehl,[3] Nicolas Franck[1,2] and Marc Jeannerod[1]

[1]*Institut des Sciences Cognitives,* [2]*JE 1943, Université Claude Bernard, Lyon,* [3]*Rééducation de Neurologie, Hôpital de la Salpêtrière, Paris, France and* [4]*Istituto di Fisiologia Umana, Università di Parma, Parma, Italy*

Correspondence to: Angela Sirigu, Institut des Sciences Cognitives, 67, Bd Pinel, 69675 Bron, France
E-mail: sirigu@isc.cnrs.fr

Summary

Three apraxic patients with lesions in the left parietal cortex were required to execute finger movements with either hand, while the visual feedback they received about the movement was manipulated systematically. We used a device which allowed us to present on a video monitor either the patient's hand or the examiner's hand simultaneously performing an identical or a different movement. In each trial, patients were required to decide whether the hand shown on the screen was their own or not. Hand movements produced in response to verbal command included simple (single-finger extension) and complex gestures (multi-finger extension). Ownership judgements were analysed and compared with those produced by six normal controls and two non-apraxic neurological patients. Apraxic patients and controls accurately recognized their own hand on the screen (own movement condition) and correctly identified the viewed hand as the examiner's when it performed a movement different from their own movement (incongruent movement condition). However, when the viewed hand was the examiner's hand executing their own movement (congruent movement condition), apraxic patients were significantly more impaired than controls. When the results were analysed as a function of gesture type, the number of correct responses was significantly lower for apraxic patients with respect to controls only for complex gestures. Interestingly, when patients executed the finger gestures inaccurately, they still failed to recognize the examiner's hand as alien, and claimed that the correct movement presented on the screen was their own. These results confirm that parietal lesions alter the representational aspects of gestures, and suggest a failure in evaluating and comparing internal and external feedback about movement. We conclude that the parietal cortex plays an important role in generating and maintaining a kinaesthetic model of ongoing movements.

Keywords: apraxia; parietal lesion; neuropsychology

Introduction

For over a century, the posterior parietal regions have been known to be involved in arm and hand movements, from reaching–grasping movements to symbolic gestures and the manual use of tools and objects. This role is supported by numerous neuropsychological observations. Damage to the left parietal lobe frequently has been associated with the syndrome of apraxia, as classically described by Liepmann (Liepmann, 1905). In broad terms, apraxia refers to a disturbance, distinct from paralysis, which affects the patient's ability voluntarily to produce skilled motor actions. It applies to both meaningful gestures (symbolic gestures and tool use) and meaningless gestures. Apraxia can affect the spatiotemporal aspects of movement production (Poizner *et al.*, 1990; Hermsdörfer *et al.*, 1996) or the acquisition of complex hand postures (Sirigu *et al.*,1995). The disturbance extends to pantomime of object use (Goodglass and Kaplan, 1963) and to the recognition of gestures executed by another person (De Renzi *et al.*, 1982; Heilman *et al.*, 1982; Rothi and Heilman, 1995; Rothi *et al.*, 1985; De Renzi and Lucchelli, 1988; Ochipa *et al.*, 1989; Sirigu *et al.*, 1995).

A possible interpretative framework on the nature of apraxia can be tracked back to Liepmann (Liepmann, 1920). He suggested that the correct execution of a complex motor act depends on the activation of motor engrams which define and control the spatial and temporal parameters of the single movements comprising the action. Although Liepmann denied the existence of *praxis centres* in the brain, he was positive in claiming that there are systems concerned with the organization of higher order motor behaviour, and whose damage is responsible for apraxia. As later pointed out by Heilman (Heilman *et al*., 1982), action patterns (*visuo-kinaesthetic motor engrams*) are probably stored in the parietal cortex of the dominant hemisphere, and are responsible for gesture production, discrimination and recognition. Damage to this store gives rise to a failure in producing and recognizing action, thus suggesting that the main impairment in apraxia is likely to reside at the level of stored representations of learned movements.

Previous studies on apraxia have focused on execution (De Renzi and Lucchelli, 1988; Ochipa *et al.*, 1989), imitation (De Renzi *et al*., 1982; Goldenberg, 1996; Goldenberg and Hagmann, 1997) or recognition (Heilman *et al.*, 1982; Sirigu *et al.*, 1995) of complex gestures. Experiments involving gesture recognition have provided a major argument for a representational basis of apraxic disorders. In these studies, patients were required to interpret movements performed by another person.

To our knowledge, no study yet has investigated how apraxic patients with parietal lesions perceive and interpret their *own* movements. This may be a relevant issue since a recent study has shown that the ability to simulate hand actions mentally is selectively impaired in patients with parietal lobe lesions (Sirigu *et al.*, 1996). This deficit has been interpreted as a failure to generate and/or monitor an internal model of one's own movement, since in mentally simulated actions no actual motor output is required and no sensory feedback is generated. It may be predicted that such a disturbance will also affect the perception of one's actual movements. Indeed, according to theoretical studies, one of the main stages in motor control is the comparison between an internal model and the expected and actual sensory consequences of the movement (Wolpert *et al.*, 1995). If such an internal model is faulty or unavailable in apraxic subjects, then the capacity to compare or match predicted and actual sensory feedback is likely to be impaired as well. In order to investigate this hypothesis, we tested a sample of patients who had developed apraxic symptoms following left parietal damage. Patients were asked to execute simple and complex hand–finger movements with their unseen hand, and to observe the motor output on a TV screen. A special device (Daprati *et al.*, 1997) enabled us to present in real time on the screen either the patient's hand or that of the examiner. Patients were required to decide whether the hand moving on the screen was their own or not.

Methods

Subjects

Patients

Three apraxics patients (B.A., V.O. and L.B.), all suffering from left parietal lesions (Table 1), were tested in the present paradigm. Patient L.B.'s lesions extended also to the frontal cortex. Patients B.A. and V.O. did not show any elementary motor disorder, while patient L.B. presented a right hemiplegia which had partially regressed at the time of testing. All patients were right-handed according to self-report. Apraxia was clinically tested using the stimuli described in the study of Sirigu *et al.* (Sirigu *et al*., 1995).

All three patients presented impairments when executing symbolic gestures from verbal command, when imitating meaningless gestures and when asked to pantomime the use of an object. Performance improved slightly when patients were allowed to execute the gesture while holding the object in their hand, with the exception of patient L.B., who showed a severe disorder also in this condition. The patients' disturbance also extended to the ability to produce from a visual model complex hand configurations (such as horns, i.e. extending the index and little finger while using the thumb to hold down the middle two fingers, joining together the little finger and the thumb, etc.). In patients B.A. and V.O., gestural disturbance was most severe for the right hand; however, the deficit, even though less prominent, was also present for the left hand. In patient L.B., apraxia affected both hands equally, although the gestural deficit of his right hand was difficult to examine due to the confounding motor disorder he presented.

In order to differentiate the effects of motor disturbance and those linked to general cerebral damage, we tested two additional patients. The first (M.A.) suffered from lesions in the premotor and motor areas, and the second (B.R.) presented bilateral lesions in the inferotemporal cortex. The control patients showed no signs of apraxia. All patients' lesions were of vascular origin. A brief summary of the patients' data is given in Table 1. At the time of testing, all patients were hospitalized at the Neurological Rehabilitation Centre of la Salpêtrière Hospital (Paris, France).

Control subjects

Six normal subjects, matched for age and sex (four females, two males; mean age 51.8 years, SD 6.8 years) and recruited among the hospital staff, volunteered for this study. None of them reported evidence of neurological diseases or psychiatric problems. All were right-handed, and had normal or corrected to normal vision. They were naive as to the purpose of the study.

Both controls and patients gave informed consent to participate in the present study, which was approved by the Ethical Committee of the Centre Leon Bérard, Lyon, France.

Apparatus and procedure

Figure 1 shows a drawing of the apparatus. A detailed description is given elsewhere (Daprati *et al.*, 1997). Each subject entered a cabin and sat in front of a table, on which a rectangular mirror (30×30 cm) was placed at 35 cm from his/her frontal plane. The mirror was oriented with an inclination of 30° on the vertical plane. The subject positioned one hand below the mirror on a support located at ~40 cm from his/her frontal plane. A camera connected to a closed-circuit television system filmed the subject's hand through another mirror. Its image appeared on a TV screen located on a shelf 15 cm above the subject's head, and was reflected by the first mirror. Thus, looking at the mirror, the subject got the impression that he/she watched his/her own hand as

Table 1 *Summary of patients' clinical characteristics*

Patients	Sex	Age	Handedness	Locus of lesion	Time since lesion	Hemiplegia	Sensory deficits	Apraxia	Neglect
Apraxics									
V.O.	F	50	Right	Left/parietal (areas 40, 39)	3 years	Absent	Absent	Present	Absent
B.A.	M	59	Right	Left/parietal (area 40)	5 years	Absent	Absent	Present	Absent
L.B.	M	74	Right	Left frontoparietal (areas 1, 5, 40, 39)	12 months	Motor deficit for the right upper limb	Mild transient	Present	Absent
Control patients									
M.A.	M	50	Right	Left frontal (areas 1, 6, 44, 45)	3 years	Complete for the right hemibody	Absent	Absent	Absent
B.R.	M	55	Right	Bilateral infero temporal (areas 21, 20, 38)	3 years	Absent	Absent	Absent	Absent

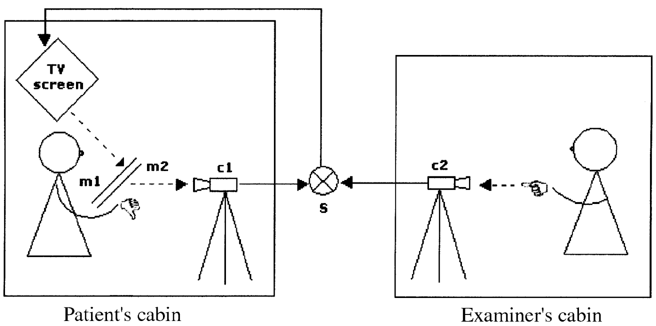

Fig. 1 Schematic representation of the apparatus. The experimental set-up was not visible to the patients.

if through a window. The examiner was hidden in a second cabin, identical to that of the subject, with one hand positioned on a support. Prior to testing, the examiner was provided with a list of the gestures to be executed in each trial. Hand movements were filmed by a second camera, located in front, and connected to the TV screen. A special switch allowed presentation on the screen of the image of either the subject's hand or that of the examiner, according to the trial. The examiner's and the subject's hands were covered with identical gloves, in order to minimize recognition due to morphological differences.

The subject's task was to perform a requested movement with either the right or the left hand, and to monitor its execution by looking at the image in the mirror. At the beginning of each trial, the subject positioned his/her hand on the support with their fist clenched. The screen in front of him/her was dark, no image was presented and an instruction was given as to which movement to execute. Next, the image of a hand appeared on the screen. Simultaneously, a sound produced by a PC was the signal for the subject (and for the examiner) to execute the requested movement. This procedure ensured that the examiner's and the patient's movements were time-locked. Once the movement was performed, the screen returned to darkness, within ~1 s, and the subject was asked the following question: 'You have just seen the image of a moving hand. Was it your own hand? Answer YES if you saw your hand performing the movement you have been executing. Answer NO in any other case, that is if you doubt that it was your own hand and your own movement'.

Both hands were tested and the order was counterbalanced among subjects, with the exception of patient L.B. and the patient with the motor cortex lesion (M.A.) who performed the task only with their left hand because both suffered from right hemiplegia. Several practice trials were run before each block, until subjects became familiar with the apparatus and procedure.

In each trial, one of the following four movements was required: (i) extend thumb; (ii) open hand wide; (iii) cross middle finger on index finger; and (iv) extend index and little finger. For the purpose of data analysis, gestures were later classified as either *simple* (extend thumb, open hand wide) or *complex* (cross middle finger on index finger, extend index and little finger), in accordance with the results of Sirigu and colleagues (Sirigu *et al*., 1996). In each trial, one of the following possible images of the hand appeared on the screen: (i) the subject's hand (own movement condition); (ii) the examiner's hand performing the same movement (congruent movement condition); or (iii) the examiner's hand performing a different movement (incongruent movement condition). The hand presented and movement types were randomized within each block. Sixteen trials were run for each hand condition, i.e. four trials for each movement type. All together, 48 trials were run in each block.

Verbal responses were recorded. The response was considered correct when subjects answered 'YES' when their own hand was presented on the screen, and 'NO' when the examiner's hand was shown. Correct responses obtained by apraxic patients were compared with those of normal controls and of the two control patients. The Mann–Whitney U test was used for between-subjects comparisons.

Results

Apraxic patients almost always produced the required finger movements. Gross mistakes (executing a gesture different from the one required) were rare (0.04%), as were long latencies (0.04%) or anticipations (0.08%) at the start of movement. Incomplete or clumsy movements, such as poor synchrony between digits, were more frequent (right hand 17.7%, left hand 22.2%), but only for the two most difficult gestures (crossed index and middle finger, and extended index and little finger).

Right hand

Patient L.B. was affected by incomplete right hemiplegia; therefore, data on the right hand were only collected on two apraxic patients, namely V.O. and B.A.

Apraxic patients were able to recognize their own hand when it appeared on the screen (own movement condition), and could easily detect the examiner's hand when it performed a movement different from the one which they were doing (incongruent movement condition), scoring at 100% correct in both conditions (Fig. 2). However, they were impaired in recognizing the examiner's hand when it produced the same gesture as they were executing (congruent movement condition, correct responses 18.7%). Although to a lesser extent, a decline in accuracy in the congruent movement condition was also found in normal controls (correct responses: own movement condition 98.7%, congruent movement condition 79.4%, incongruent movement condition 100%) (Fig. 2). A Mann–Whitney U test performed on the number of correct responses in the three experimental conditions confirms that patients were impaired specifically with respect to controls in the congruent movement condition (mean number of correct responses: patients 3.0, controls 12.7; $U = 0.00001$, $Z = -2.012$, $P < 0.04$, Fig. 2A).

A Mann–Whitney U test comparing the number of correct responses between patients and controls for each group of gestures within the congruent movement condition indicated that apraxics were specifically impaired in the case of complex gestures (Fig. 2B). That is, when patients were required to produce a gesture which involved multiple digit configurations, they failed to discriminate between the movement executed by the examiner and their own significantly more frequently than controls (correct responses: patients 0.5, controls 6.0; $U = 0.00001$, $Z = -2.024$, $P < 0.04$; Fig. 2B). Given this main deficit for complex gestures, we next asked whether success or failure in identifying hand ownership might be related to accuracy of the patients' gestures. Apraxic patients frequently produced inaccurate or clumsy gestures (17.7%), such as failing to cross the middle finger on the index finger or to extend the index and little finger selectively and simultaneously. Therefore, since the patient's hand and the viewed hand were not performing exactly the same gesture, such trials became, in a certain sense, incongruent movement trials. We examined judgements given by the patients when these clumsy movements were produced in the own movement condition and in the congruent movement condition, i.e. when patients could either see their hand or were shown an examiner's hand correctly executing the same movement. In the own movement condition, patients always recognized their own hand, and often regretted that the movement was poorly executed. On the contrary, in the congruent movement condition, patients failed to recognize the examiner's hand in nearly 90% of trials. In other words, patients executed a clumsy movement with their unseen hand, and claimed that the neat movement presented on the screen was their own. For instance, patient V.O. often commented on 'her good' performance, remarking that she had finally managed to execute the required movement properly ['tiens j'ai réussi cette fois!, ('look, this time I succeeded!')].

Left hand

The overall pattern observed for the left hand was similar to that described for the right hand. Apraxic patients were fairly accurate in recognizing movements of their own left hand when it appeared on the screen (own movement condition) and could easily detect the examiner's hand in the incongruent movement condition, scoring at 95.6% and 100% correct, respectively, in the two conditions. However, patients were clearly impaired in recognizing the examiner's hand when it executed the same gesture as their own (correct responses, congruent movement condition 25%). As mentioned before, this is the same condition in which normal controls were also prone to commit errors, although to a much lesser extent (correct responses: own movement condition 95.6%; congruent movement condition 71.9%; incongruent movement condition 100%). A Mann–Whitney U test performed on the number of correct responses in the two groups confirms that patients were less accurate than controls only in the congruent movement condition (mean correct responses: patients 4.0, controls 11.5, $U = 0.00001$, $Z = -2.333$, $P < 0.02$; Fig. 3A).

When performance was analysed as a function of gesture difficulty in the congruent movement condition, a trend towards a significant difference between groups emerged for both simple and complex gestures (simple gestures, correct responses: apraxics 1.3, controls 5.3; $U = 1.5$, $Z = -1.953$, $P < 0.052$; complex gestures, correct responses: apraxics 2.7, controls 6.2; $U = 2.5$, $Z = -1.722$, $P < 0.08$, Fig. 3B).

Control patients

As can be seen in Figs 2B and 3B, the two control patients included in the present study showed behaviour different from that of apraxics. In the congruent movement condition, both patients performed in the upper range of normal controls, and neither the inferotemporal patient (correct responses: simple, 8.0; complex, 8.0) (Fig. 2B) nor the motor lesioned patient (correct responses: simple, 7.0; complex, 7.0) (Fig.

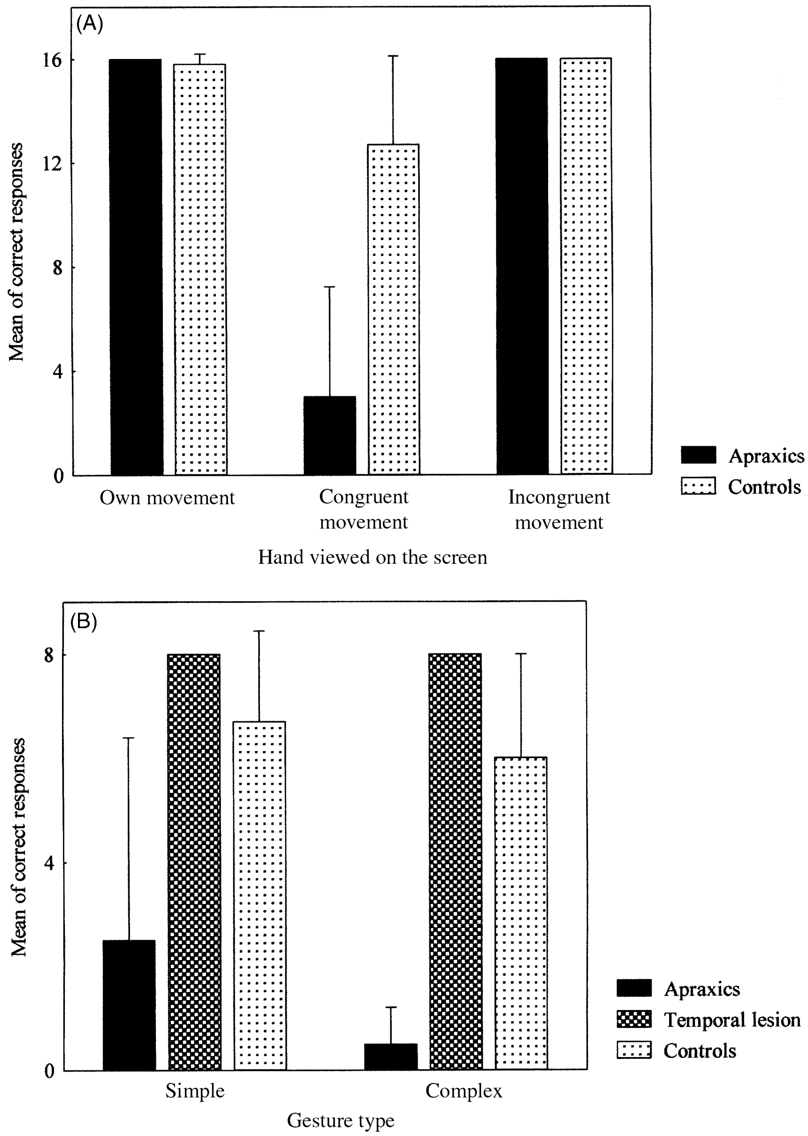

Fig. 2 Mean number of correct responses for the right hand. (**A**) Performance of apraxic patients and six normal controls in the three experimental conditions. The *x*-axis = hand presented on the screen. Own movement = subject's hand; congruent movement = experimenter's hand performing a movement congruent with that produced by the patient; incongruent movement = experimenter's hand performing a movement incongruent with that produced by the patient. Results for combined simple and complex gestures. (**B**) Performance of apraxic patients, one patient with bilateral inferotemporal cortex damage and normal controls according to gesture type (simple versus complex) for the congruent movement condition only. Performance of the inferotemporal patient for the left hand is identical to that shown for the right hand.

3B) showed a trend towards a manifest error increase in the case of complex gestures.

Discussion

In this study, we examined the ability of apraxic patients with left parietal lesions to decide whether an ongoing hand movement displayed on a monitor placed in front of them corresponded to their own movement or belonged to another person. Two different types of gestures were distinguished, namely simple and complex gestures. Four main results emerged. First, apraxics were selectively impaired with respect to controls in the congruent movement condition, in which they were required to discriminate from their own an external hand which was performing the same movement. The congruent movement condition seems to be particularly difficult since even normal subjects are prone to commit attribution errors when they see an alien hand on the screen acting in the same way as their own hand. Secondly, within this condition, patients had difficulties in correctly attributing the source of the movement, especially when complex gestures were required. Thirdly, errors in attribution

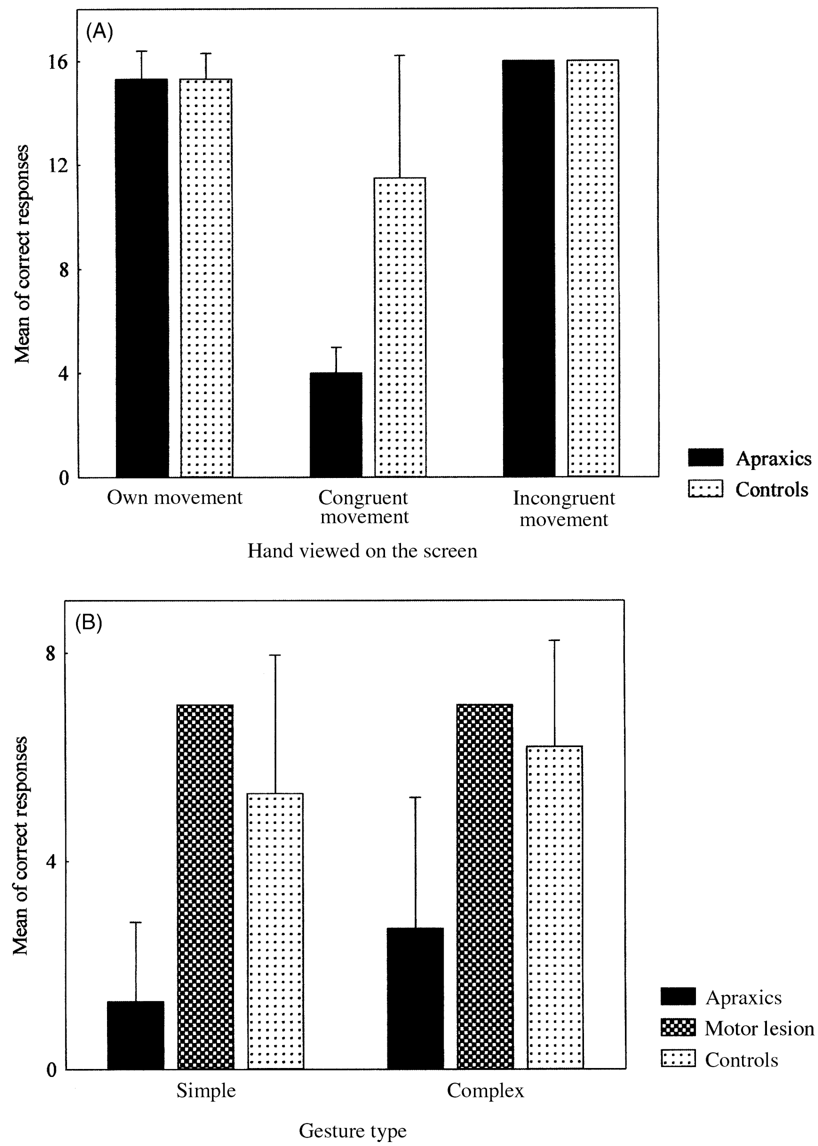

Fig. 3 Mean number of correct responses for the left hand. (**A**) Performance of apraxic patients and normal controls in the three experimental conditions. The *x*-axis = hand presented on the screen. Own movement = subject's hand; congruent movement = experimenter's hand performing a movement congruent with that produced by the patient; incongruent movement = experimenter's hand performing a movement incongruent with that produced by the patient. Results for combined simple and complex gestures. (**B**) Performance of apraxic patients, one patient with motor cortex lesion and normal controls in the congruent movement condition according to gesture type (simple versus complex) for the congruent movement condition only. Data for the motor lesioned patient was collected only for the left hand due to right hemiplegia.

judgements were present for both the right and the left hand, although greater difficulties for complex than for simple gestures were only seen for the right contralesional hand. Fourthly, the complex hand postures used in our paradigm required precise multi-finger patterns and fine synchrony between the moving digits. Not surprisingly, apraxic patients often made those gestures rather clumsily, thus involuntarily creating a mild mismatch between their movement and the viewed examiner's hand in the congruent movement condition. Rather than using this as an additional clue to attribute the movement to its proper source, they modified the perception of their own movement, and accepted the examiner's movement as their own, though improved, performance. At first, such behaviour might suggest that patients were anosognosic about their gestural deficits. However, as revealed by the clinical interview, they often complained about how their hand movement disturbances affected simple life activities.

The patients' impairment cannot be reduced to a general consequence of brain damage or to the presence of a general motor disturbance, but it is directly related to the conjunction of the parietal lesion and the presence of apraxia. In fact, our control patients, one with a lesion in the motor and premotor areas and the one with inferotemporal damage, performed at the same level as normal controls for the two types of gestures. It can be argued that the impairment observed here is consistent with the idea that apraxia is a disturbance primarily affecting skilled hand movements (Liepmann, 1920; De Renzi *et al.*, 1982; Heilman *et al.*, 1982), and that gestures are impaired at the representational level in this syndrome.

Previous studies have used gesture recognition tasks (Heilman *et al.*, 1982; Sirigu *et al.*, 1995) in order to demonstrate that apraxia is not solely an execution disorder but also affects the cognitive aspects of gesture processing. In the present study, no recognition of the meaning or of the finality of movements was required. Subjects were simply asked to identify the source of a gesture as being self-produced or as arising from an external agent in a situation where a possible ambiguity between the two sources was deliberately introduced. This task probably involves several decisional stages: first, the subject has to programme a specific gesture, a process which presumably involves setting up internal representations of the expected visual and kinaesthetic feedback that gesture execution subsequently will generate. Next, the expected visual feedback has to be registered with the actual visual feedback. Such a comparison can be used in particular to decide whether the movement displayed on the screen corresponds to the requested movement. In addition, the visual feedback also has to be compared with the kinaesthetic feedback in order to decide whether the movement displayed on the screen agrees with what the subject's hand is actually doing. In the incongruent movement condition, both sources of information contain potential cues, and the mismatch between the actual and predicted feedback probably facilitates forming the decision that the viewed hand is alien. The accurate performance of apraxic subjects in this condition suggests that they use a sufficiently accurate internal visual model of the target movement in order to attribute the viewed hand to its proper source. In the congruent movement condition, visual cues are minimized, hence subjects must use the second source of information, namely the subtle position and timing differences between kinaesthetic and visual feedback, in order to distinguish their own hand from the viewed alien hand. This discrimination is somewhat difficult for normal subjects but it is disproportionately difficult for apraxic patients.

Kinaesthetic feedback may come from the somatosensory (proprioceptive and cutaneous) input generated by the movement and also from an internal feedback of the motor outflow. Studies of normal subjects performing mentally simulated movements have amply demonstrated that the timing of an imagined movement is strongly correlated with and predicts with high accuracy the timing of the same movement when it is actually performed (Decety *et al.*, 1989). This phenomenon argues for the existence of internal models which can be used as an internal feedback during movement execution, independently of the actual sensory feedback generated by the movement. Sirigu and colleagues have demonstrated that patients with lesions of the parietal cortex are very inaccurate in motor imagery of hand gestures (Sirigu *et al.*, 1996), suggesting the existence of a selective impairment in the ability to form and/or monitor internal models for this class of movements. Such a deficit is likely to play an important role in the difficulties experienced by apraxic patients in the present study, in disambiguating the visual stimulus in the congruent movement condition. Our data indicate that the suggested predictive model is defective (Sirigu *et al*., 1996) or, alternatively, that it cannot be updated adequately on-line during movement execution.

None of the patients with parietal lesions whom we tested presented sensory *or* proprioceptive disturbances (with the exception of patient L.B., who had experienced mild sensory deficit soon after the lesion, but had recovered at the time of testing). However, all three patients failed to detect the mismatch between kinaesthetic information and the visual feedback provided by the image on the screen. This failure to detect the alien hand is perhaps even more surprising for the trials in which gestures were poorly executed, and the examiner's hand was displayed on the screen doing the correct movement. Even if, in this case, vision provided an image clearly in conflict with internal feedback, patients erroneously attributed the gesture to themselves. Two patients (V.O. and B.A.) overtly expressed their own surprise at how well they had executed the movement. Therefore, it seems that they behaved as if the kinaesthetic feedback was obliterated, and hence not used to modify the actual internal representation and correct the judgement. In other words, visual feedback given on the screen became dominant and replaced the patients' internal representation, thus constituting the unique information available for conscious decision.

A further observation from our study was that the three apraxic patients all suffered from lesions in the left parietal cortex, although in patient L.B. the lesion extended more anteriorly in the frontal cortex. This is consistent with the association between apraxia and left parietal damage frequently described in the literature (Heilman *et al.*, 1982). Moreover, the role of the parietal cortex in coding arm and hand movements has been demonstrated in neuroimaging studies (Stephan *et al.*, 1995; Gerardin *et al.*, 1998). The behaviour of the patients observed in this study suggests that the parietal cortex plays an important role in generating and maintaining a kinaesthetic model of ongoing movements.

These results confirm that parietal lesions alter the representational aspects of gestures, and suggest that a lesion in these regions results in an inability to evaluate and compare internal and external feedback about movement.

Acknowledgements

We wish to thank Dr J. R. Duhamel for helpful discussion of these data. A.S. is supported by CNRS.

References

Daprati E, Franck, N, Georgieff N, Proust J, Pacherie E, Dalery, J, et al. Looking for the agent: an investigation into consciousness of action and self-consciousness in schizophrenic patients. Cognition 1997; 65: 71–86.

De Renzi E, Lucchelli F. Ideational apraxia. Brain 1988; 111: 1173–85.

De Renzi E, Faglioni P, Sorgato P. Modality-specific and supramodal mechanisms of apraxia. Brain 1982; 105: 301–12.

Decety J, Jeannerod M, Prablanc C. The timing of mentally represented actions. Behav Brain Res 1989; 34: 35–42.

Goodglass H, Kaplan E. Disturbance of gesture and pantomime in aphasia. Brain 1963; 86: 703–20.

Gerardin E, Sirigu A, Lehericy S, Poline JB, Leclech G, Thiebot J, et al. The role of the parietal cortex in the representation of hand movements: a fMRI study. Neuroimage 1998; 7 (4 Pt 2): S120.

Goldenberg G. Defective imitation of gestures in patients with damage in the left or right hemisphere. J Neurol Neurosurg Psychiatry 1996; 61: 176–80.

Goldenberg G, Hagmann S. The meaning of meaningless gestures: a study of visuo-imitative apraxia. Neuropsychologia 1997; 35: 333–41.

Heilman KM, Rothi LJ, Valenstein E. Two forms of ideomotor apraxia. Neurology 1982; 32: 342–6.

Hermsdorfer J, Mai N, Spatt J, Marquardt C, Veltkamp R, Goldenberg G. Kinematic analysis of movement imitation in apraxia. Brain 1996; 119: 1575–86.

Liepmann H. Die Linke Hemisphare und das Handeln. Münch Med Wochenschr 1905; 49: 2322–6.

Liepmann H. Apraxie. Ergebn ges Med 1920; 1: 516–43.

Ochipa C, Rothi LJ, Heilman KM. Ideational apraxia: a deficit in tool selection and use. Ann Neurol 1989; 25: 190–3.

Poizner H, Mack L, Verfaellie M, Rothi LJ, Heilman KM. Three-dimensional computergraphic analysis of apraxia: neural representations of learned movement. Brain 1990; 113: 85–101.

Rothi LJ, Heilman KM. Ideomotor apraxia: gestural discrimination, comprehension and memory. In: Roy EA, editor. Neuropsychological studies of apraxia and related disorders. Amsterdam: North-Holland; 1985. p. 65–74.

Rothi LJ, Heilman KM, Watson RT. Pantomime comprehension and ideomotor apraxia. J Neurol Neurosurg Psychiatry 1985; 48: 207–10.

Sirigu A, Cohen, L, Duhamel JR, Pillon B, Dubois B, Agid Y. A selective impairment of hand posture for object utilization in apraxia. Cortex 1995; 31: 41–55.

Sirigu A, Duhamel JR, Cohen L, Pillon B, Dubois B, Agid Y. The mental representation of hand movements after parietal cortex damage. Science 1996; 273: 1564–8.

Stephan KM, Fink GR, Passingham RE, Silberweig D, Ceballos-Baumann AO, Frith CD, et al. Functional anatomy of the mental representation of upper extremity movements in healthy subjects. J Neurophysiol 1995; 73: 373–86.

Wolpert DM, Ghahramani Z, Jordan MI. An internal model for sensorimotor integration. Science 1995; 269: 1880–2.

Received January 20, 1999. Revised March 26, 1999.
Accepted April 28, 1999