1.0 μg of UCN, n = 8) had 30-min access to water (days 1 through 6, 8, and 10) or saccharin solution (days 7 and 9) daily for the duration of an 11-day multiple-pairing test conditioning procedure. UCN was administered ICV on days 7 and 9 immediately after access to the saccharin. On day 11, all rats chose between two choices (water or saccharin). A significant taste aversion was observed only at 1.0 μg of UCN. Mean ± SEM milliliters of saccharin intake during the two-bottle test was as follows: vehicle, 16.4 ± 4.5 ml; 0.1 μg of UCN, 12.3 ± 3.5 ml; and 1.0 of μg UCN, 1.1 ± 0.6 ml. Water intake during the same two-bottle test was as follows: vehicle, 8.1 ± 3.1 ml; 0.1 μg of UCN, 9.6 ± 2.3 ml; and 1.0 μg of UCN, 20.3 ± 1.2 ml. Water intake baseline on days 1 through 6, when the animals had access only to water, was 19.1 ± 1.3 (vehicle), 21.2 ± 1.5 ml (0.1 μg of UCN), and 22.2 ± 1.8 ml (1.0 μg of UCN).

15. The nose-poke apparatus consists of an acrylic plastic chamber and wire mesh floor (25 cm by 25 cm by 25 cm) enclosed within a sound- and light-attenuating box. Two holes, one for food and one for water, were made (2 cm above the floor) in two opposite side walls of the chamber. Each nose poke in either the food or water hole activated the delivery of a 45-mg pellet or 100 μl of water, respectively, into a food or water tray situated next to each hole. Nose pokes were recorded by photocell beam interruptions and a microcomputer. Rats (Wistar) were exposed to one session daily for 20 hours and trained during several days to obtain an appropriate baseline level (±20% total food intake from day to day). Six animals were injected ICV with UCN in a within-subjects design; for example, each rat received each dose (0.01, 0.1, and 1.0 μg/2 μl) and vehicle according to a Latin-square design with a minimum of 3 days between injections. Injections were made at 19:30 hours, 90 min after the onset of the dark cycle (12 hours, 6 p.m. to 6 a.m.). Water intake followed food intake on a prandial basis. Results showed that nose pokes for water showed the same decrease as food intake at the same doses of UCN. The data were analyzed at 3, 6, and 12 hours after the injections. Meals or bouts of feeding were defined as continuous sequences of nose-poking for 45-mg food pellets with no inter-poke interval greater than 60 s and a minimum inter-bout interval of 15 min. This analysis is similar to that reported by others, and meals or bouts corresponded to those of visual inspection of the event recorded. J. A. Grinker, A. Drewnowski, M. Enns, H. Kissileff. *Pharmacol. Biochem. Behav.* **12**, 265 (1980).
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18. The locomotor apparatus consisted of 16 wire mesh cages (20 cm by 25 cm by 36 cm) with two horizontal infrared photocell beams located across the long axis of the cage 2 cm above the floor and 16 cm from one another. Beam interruptions and crossovers were recorded (beam 1 broken followed by beam 2 and vice versa) by computer and printed out every 10 min. Activity was recorded over 3 hours, and behavior was observed every 30 min. The day before the experiment, rats were habituated for 1 hour to the room and then for 5 hours to the testing cages. On the testing day, after a 90-min habituation period, rats were injected ICV with UCN (0.1, 1, and 10 μg/2 μl) or vehicle, and the locomotor activity was monitored for the next 3 hours.
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The Mental Representation of Hand Movements After Parietal Cortex Damage

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Recent neuroimagery findings showed that the patterns of cerebral activation during the mental rehearsal of a motor act are similar to those produced by its actual execution. This concurs with the notion that part of the distributed neural activity taking place during movement involves internal simulations, but it is not yet clear what specific contribution the different brain areas involved bring to this process. Here, patients with lesions restricted to the parietal cortex were found to be impaired selectively at predicting, through mental imagery, the time necessary to perform differentiated finger movements and visually guided pointing gestures, in comparison to normal individuals and to a patient with damage to the primary motor area. These results suggest that the parietal cortex is important for the ability to generate mental movement representations.

Prediction is essential to many aspects of motor behavior, from postural compensation to the tracking of moving objects and the planning of a complex trajectory. The capacity of the central nervous system to simulate and anticipate the behavior of the motor apparatus is a central issue not only in experimental and computational studies of motor control (*1*), but also in the study of mental processes. Humans can use this capacity to improve a motor skill or induce sensorimotor plasticity through mental rehearsal (*2*). Decety and his colleagues have shown that motor imagery can be used to predict the time needed to complete a movement, and that the mental reenactment of an effortful exercise causes the same vegetative changes as its actual performance (*3*). Studies of cerebral metabolic activity have demonstrated that most of the regions that are active during overt movement execution such as the parietal and premotor cortices, the basal ganglia, and the cerebellum are active during mental simulation as well (*4*).

These results suggest that motor impairments caused by a cerebral lesion might also affect mentally simulated actions. We reported a case of a patient with motor cortex damage where the simulation of a movement with the affected limb produced a sensation of mental drag and matched that limb's reduced motor efficiency (*5*). Parallel impairments in imagined and executed movements were also observed in patients with basal ganglia dysfunction due to Parkinson's disease (*6*). This observation suggests that the excitatory output produced in the cortico-striatal pathways during motor imagery closely mimics what occurs during movement execution, and that it is accessible to conscious evaluation. Furthermore,

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the motor cortex and basal ganglia do not appear to be instrumental in forming or maintaining a mental image of a limb in action.

In the present study we tested the hypothesis that the parietal cortex might be important for the ability to generate motor images. Parietal lobe lesions produce apraxia, an impairment of skilled movements, in the absence of elementary sensory or motor deficits. Apraxic patients have difficulties in performing symbolic gestures and pantomimes, where movements must be guided by stored representations rather than by contextual cues (*7*). Anticipatory shaping of the hand during grasping gestures can be inaccurate, indicating an impaired recall of finger grip patterns (*8*, *9*). Parietal lesions can affect both motor production and ideation, because some patients with apraxia also have difficulty recognizing the meaning of gestures (*10*) or in judging their accuracy (*9*). These findings suggest that the parietal cortex might be important for storing or accessing motor representations, or both.

We investigated mentally simulated hand movements in four patients with unilateral left or right parietal lobe lesions, and in one patient with a motor impairment associated with a lesion in the right rolandic area. All patients experienced movement difficulties that were restricted to the hand and fingers (*11*). In the first task, participants mentally simulated a continuous thumb-fingers opposition sequence with either the left or right hand to the sound of a metronome. They imagined touching each finger in turn, beginning with the little finger. The speed of the metronome beat, initially set at 40 beats per minute, was augmented every 5 s, until the individual reported that the imagined hand could no longer keep up with the imposed speed (Fig. 1A). The movement sequence was subsequently executed according to the same procedure, and the actual performance break point was recorded.

The results obtained for nine normal individuals showed excellent congruence between maximum imagined and executed movement speeds. In contrast, patients with parietal cortex lesions produced estimates that were systematically inaccurate (too fast or too slow) or that were inconsistent from one trial to the next. Three parietal patients made errors in predicting the break point of the impaired contralesional hand but were accurate in predicting that of the unaffected ipsilesional hand, and a fourth was impaired bilaterally (Fig. 1, B and C). The direction of the error varied among patients, showing either consistent overestimation (R.K.) or underestimation (J.J. and R.L.) of actual motor efficiency. For case J.J., the errors were smaller than for the other patients but showed trial-to-trial variations. These results are in contrast with those previously reported for patient C.P., who has degenerative right motor cortical damage, whose simulated movement speed on the metronome task mirrored exactly the asymmetric motor performance of the contra- and ipsilesional hands (*5*, *12*).

Thus, the ability to estimate manual motor performance through mental imagery is disturbed after parietal lobe damage. However, from the above results, one cannot distinguish whether the patients showed exaggerated positive or negative biases in estimating movement time but otherwise formed accurate mental motor images, or whether the content of the represented movements was altered. To address this issue, we evaluated how closely the imagined movements of patients with parietal lesions reflected the variation in motor performance associated with specific task factors; namely, (i) the complexity of the motor program and (ii) compliance to the perceptual demands of the task. If parietal lesions impair movement representation, a reduced parallelism between the timing of motor performance and imagery can be expected.

In one task, four sets of postures were empirically selected on the basis of their degree of difficulty for a group of controls (Fig. 2A). In the imagery condition, the participant simulated one of the movement sequences with the prespecified hand. Movement duration was recorded as the time elapsed between a go signal and the participant's report of having completed five consecutive cycles of the same movement pattern. Cumulating several cycles was necessary because of the short duration of a single movement and the coarse resolution of mental movement time measurements. Participants first completed the imagery task, then executed each movement according to the same procedure.

In normal individuals, imagined and executed movements increased in parallel from the simplest posture to the most complex one (Fig. 2B). The patient with a primary motor impairment (C.P.) predicted the time necessary to execute each of the four postures with equal accuracy with either hand (Fig. 2, C and D). She showed asymmetric motor performance, with her affected contralesional hand being most slowed when executing postures 2 and 4. This had been accurately anticipated during the mental simulation trials, before she was allowed to try any of the movement sequences. In contrast, patients with parietal damage were unable to simulate the behavior of the contralesional hand. With patient J.D., executed movement duration increased steeply with posture complexity, but imagined movement duration did not reflect this accurately (Fig. 2E). In this particular example, imagined movements appear to underestimate the affected hand's slowness, which could suggest that the patient was in fact simulating a movement of

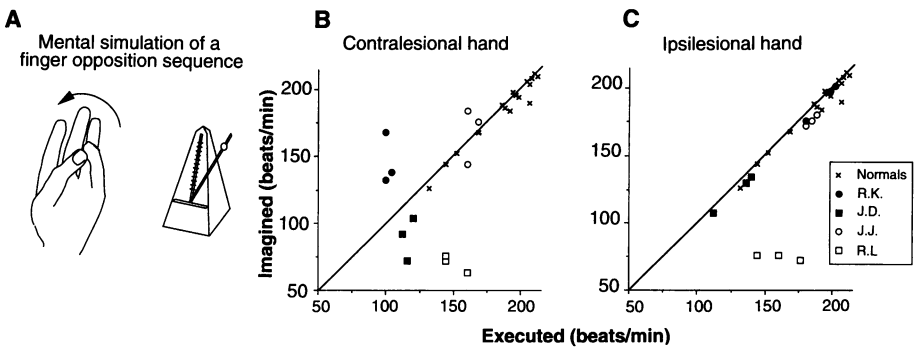

Fig. 1. (**A**) The task consisted of mentally rehearsing a finger opposition sequence to the increasing pace of a metronome. The maximum subjective speed achieved was later compared with the actual break point when the same procedure was physically performed. Imagined movement accuracy was estimated as the normalized difference between maximum speed achieved in the imagined and the executed movement conditions [(imagined − executed)/executed]. Prediction errors in normal individuals ranged from −8% to 0% with a mean of −2.1%, reflecting a small, statistically nonsignificant tendency to underestimate actual movement speed. (**B** and **C**) The data are represented as scatter plots of executed versus imagined movement speed. Points lying on the 45° line represent a perfect match between the two movement conditions. The x symbols represent individual data points for the left- and right-hand performance of nine normal individuals. Other symbols represent the performance of two right (R.K. and J.D.) and two left (J.J. and R.L.) parietal lesion patients. All patients were able to execute the sequence accurately, although movement speed of the contralesional hand was generally less than the normal range. Each patient repeated the imagined-executed movement trials three times in nonconsecutive blocks during a single testing session. Each symbol thus represents the relation between imagery and execution for a single trial. Note the different accuracy and scatter of imagined movement speed for the contralesional and ipsilesional hands in patients J.D., R.K., and J.J.

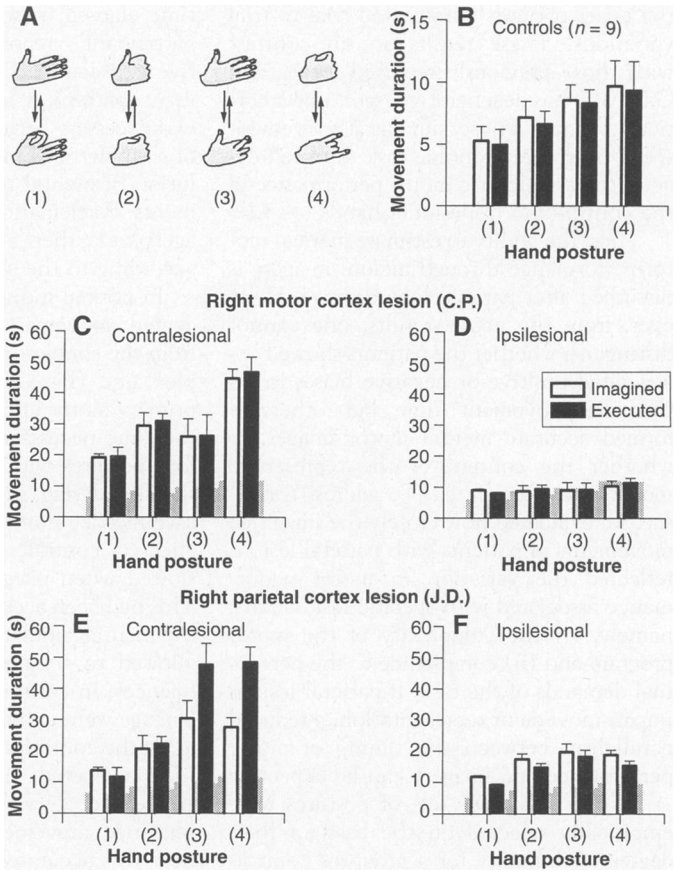

Fig. 2. (**A**) Four pairs of hand postures used in the second motor imagery task, ranked by level of complexity. (**B**) In normal control individuals, mental movement duration accurately predicts actual motor performance: Imagined and executed movement durations increase as a function of posture complexity [$F(3,21) = 34.4$, $P < 0.0001$; all pairwise comparisons significant at $P < 0.05$ or less], although a small but consistent bias was observed in mental movement duration [$F(1,7) = 6.49$, $P < 0.04$]. Error bars represent the standard deviation of the group's mean movement duration. (**C** to **F**) Effects of motor cortex or parietal damage. Each column and error bar represents, respectively, the mean and standard deviation of five nonconsecutive replications of the same trial type. The shaded area in the background illustrates the range of normal performance. Both patients made slower movements with the contralesional than with the ipsilesional hand, but differed in their ability to match this performance in mental simulation trials.

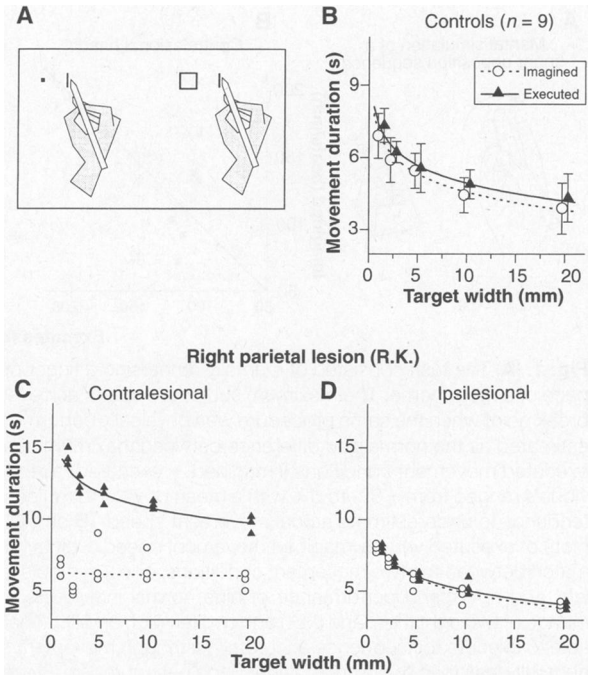

Fig. 3. (**A**) The manual pointing task required a rapid and accurate movement with the tip of a hand-held stylus from a starting position to a square visual target, which was 1.25, 2.5, 5, 10, or 20 mm wide, across a fixed distance of 30 mm. (**B**) In normal individuals, correlation coefficients between movement duration for executed and imagined movements were high for both hands (left, $r = 0.88$; right, $r = 0.93$). The plot shows the mean and standard deviation of executed and imagined movement duration as a function of target width, and a nonlinear logarithmic regression fit to both sets of data, using the equation $f = \alpha + \beta[\ln(2A/W)]$, where A and W represent movement amplitude and target width, respectively. (Coefficients obtained for imagery: $\alpha = 2.6$, $\beta = 1.1$; for execution: $\alpha = 3.0$, $\beta = 1.1$). A better fit was obtained with logarithmic regression (r^2 of 0.61 and 0.65 for imagery and execution trials, respectively) than with linear regression (r^2 of 0.52 for both trial types). (**C** and **D**) Motor imagery and movement execution for parietal lesion patient R.K. Symbols represent three nonconsecutive replications of the same trial type, and the dashed and solid lines correspond to the nonlinear regression fit applied to the patient's individual trial data for imagined and executed movements, respectively.

the normal hand, disregarding the motor impairment. Comparison of the test results for the two hands (Fig. 2, E and F) shows that this is not the case because the mean duration and variability differ for three of the four postures, depending on whether the patient was instructed to simulate a movement of the contralesional or the ipsilesional hand. A similar pattern of results was observed in another patient with a right parietal lesion (R.K.). In the left parietal patient R.L., the motor imagery impairment was bilateral and characterized by longer imagined movement duration compared with executed movement duration (Table 1).

In another task the effects of a perceptual rather than a motor variable were tested. In reaching for a visual target, the hand must decelerate more slowly as it homes in on a small target than on a large one (*13*). This speed-accuracy trade-off is expressed in Fitts' law (*14*), which states that total movement duration is inversely related to the logarithm of target width. To test whether this relation applies to imagined movements, we had participants maintain the tip of a hand-held stylus stationary at the starting location and at a go signal, mentally place it inside a variable-size open square (Fig. 3A). Different combinations of target hand and size were randomly interleaved. As in the previous task, participants performed the same movement five consecutive times, and motor imagery trials were completed before actual execution of the movements. Imagined and executed movement times were highly correlated in normal individuals, and Fitts' law accounted equally well for imagined and executed movements (Fig. 3B). In patients with parietal lesions, actual movement execution was modulated by target size, but motor imagery was not. This is illustrated in Fig. 3C for case R.K., whose imagined movements with the affected hand were too rapid and failed to show any sensitivity to target size. As in the previous task, simulation of ipsilesional hand movements (Fig. 3D) showed a very different pattern, ruling out the possibility that the patient imagined a normal movement when in fact he was instructed to imagine using his affected hand.

For the two patients with right parietal lesions, imagined movements of the intact hand accurately predicted actual motor performance, indicating that their deficit is a selective incapacity to generate a mental representation of the contralesional hand's movements (Table 1). The patients with left parietal lesions were somewhat different in that they showed partial (J.J.) or complete bilateral impairments (R.L.). Although this is consistent with the observation that left parietal lesions can produce bilateral apraxia (*15*), the possibility of

nonspecific mental imagery impairments must first be eliminated through appropriate control experiments. All patients were tested for motor imagery of hip, shoulder, elbow, and wrist joint movements. Both left and right parietal patients showed normal and congruent imagined and executed movement duration with both body sides. In some patients, a small asymmetry of performance could be detected for imagined wrist movements. In case R.L., the selectivity of motor imagery impairments for distal extremities was further confirmed in another pointing task involving a rigid arm with rotation at the shoulder joint and no wrist or finger movements. When pointing movements were thus restricted to the proximal limb joint, executed and imagined movement times were well correlated bilaterally (*16*).

In two previously investigated brain regions, the motor cortex and basal ganglia, impaired motor behavior was accurately reflected in mental movement times (*5*, *6*). To our knowledge, the present results on the effects of parietal lesions constitute the instance of focal cerebral damage associated with an impaired capacity to mentally simulate a movement. The selectivity for hand movements is in accord with previous studies of movement disorders in such patients (*8*, *9*) and with electrophysiological data from nonhuman primates showing that sensorimotor transformations for complex hand movements are performed in the parietal cortex (*17*).

The exact contribution of the parietal cortex in predicting manual motor performance remains to be clarified. At least two mechanisms can be considered. One possibility is that kinesthetic representations stored in parietal cortex must first be activated and organized to entrain other brain regions that are active during movement simulation. Another possibility is that the parietal cortex is involved in monitoring the motor outflow, through the efference copy received from downstream motor areas. Such signals do converge on parietal cortex, and it has been shown that parietal neurons can predict sensory changes in anticipation of intended movements (*18*). A broader perspective on the contribution of individual brain areas to mentally simulated actions will be gained from further studies of motor imagery in patients with other lesion locations, for example, in premotor structures, such as the supplementary motor area, and in the cerebellum, which has recently been proposed as a substrate for an internal model of arm dynamics (*19*).

Table 1. Prediction of motor performance through mental simulation of finger postures and visuo-manual pointing movement. All patients except R.L. showed performance asymmetry between the contralesional and ipsilesional hands when executing these movements (actual mvt time). Executed and imagined movement durations of each hand were compared with linear regression analysis. Consistently accurate prediction is expressed by a slope of 1.0, intercept at 0, and perfect correlation (r^2 = 1.0). Normal individuals' performances were close to these values in both tasks. Prediction accuracy in the patient with motor cortex damage was in the normal range. In parietal patients, imagined movement duration correlated poorly or showed markedly distorted relations (or both) with executed movement duration, for the contralesional hand in all cases, and for the ipsilesional hand as well in left-lesioned patients. NT, not tested.

Participant and hand	Posture alternation				Visuo-manual pointing			
	Actual mvt time (s)	Slope	Intercept	r^2	Actual mvt time (s)	Slope	Intercept	r^2
Controls								
Mean	7.3	0.98	1.0	0.92	5.7	0.89	0.9	0.89
Min.	5.3	0.78	−1.0	0.74	5.2	0.63	−1.8	0.65
Max.	8.5	1.20	2.3	0.99	7.7	1.40	2.1	0.99
Right motor cortex lesion								
C.P.								
Contra	30.9	0.94	1.0	0.98	22.0	0.85	1.2	0.79
Ipsi	9.9	0.73	2.4	0.71	8.4	0.76	1.4	0.73
Right parietal lesion								
R.K.								
Contra	13.8	0.05	10.5	0.11	12.0	0.17	4.2	0.10
Ipsi	7.3	0.82	2.2	0.81	6.0	1.10	0.5	0.97
J.D.								
Contra	32.5	0.39	10.0	0.77	13.2	0.22	13.9	0.26
Ipsi	14.4	0.90	4.0	0.83	9.4	1.70	−3.6	0.76
Left parietal lesion								
R.L.								
Contra	16.2	−3.66	72.0	0.21	7.7	0.07	25.9	0.01
Ipsi	16.3	−2.45	62.5	0.09	7.6	−1.19	29.4	0.22
J.J.								
Contra		NT			13.2	−0.01	7.3	0.01
Ipsi		NT			9.1	0.11	5.5	0.04

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11. All participants gave their informed consent before taking part in this study. Nine naïve right-handed normal volunteers between the ages of 30 and 74 (mean = 49 years) participated as controls. Patients were selected on the basis of lesion location and the presence of hand movement difficulties. Lesion sites were reconstructed from magnetic resonance imaging scans and were confined to the parietal cortex. The site of lesions varied among patients and involved the superior and inferior parietal lobule in posterior parietal cortex (areas 7, 39, and 40), and more anterior lesions located in the hand representation region of the postcentral gyrus. Despite this diversity of lesion sites, all patients showed overlapping symptoms with regard to hand movement execution and motor imagery impairments, but none had visuo-spatial or attentional problems, which are frequently observed with right parietal cortex lesions. All patients but case R.L., who had a tumor, suffered a cerebrovascular injury about 2 months before their participation in our study. None of the patients showed motor or sensory deficits, hemispatial neglect, body schema disorders, or asomatognosia at the time of testing. Patient R.K. is a 38-year-old male with a right anterior parietal lesion partially extending into the precentral gyrus. His chief complaint consisted of difficulties with differentiated movements of the left fingers. Patient J.D. is a 62-year-old woman with a right superior parietal lesion, who presented left-sided difficulties with hand movements and visually guided reaching. Patient R.L. is a 28-year-old male with left parietal astrocytoma occupying the angular and supramarginal gyri. There was a mild ideomotor apraxia, and the patient reported needing to look more carefully at his hands when manipulating objects. Simple hand movements were fast and accurate when made under visual control but deteriorated when visual feedback was removed. Patient J.J. is a 60-year-old male with a left lesion of both inferior and superior parietal lobules and presented bilateral ideomotor apraxia and manual grasping difficulties restricted to the right hand. In addition to the patients with parietal lobe lesions, we tested C.P., a 73-year-old female with a degenerative pyramidal syndrome of the left upper limb. A position emission tomography scan showed cortical hypometabolism in the middle rolandic region, corresponding to the hand representation in primary motor cortex. Her case is reported in detail

elsewhere (*5*). The movements selected for the experimental tasks were within the range of the motor capabilities of all patients, despite the presence of a performance asymmetry between the affected contralesional and intact ipsilesional sides. It should be noted that increased movement duration, which is the only variable reported here, can be the consequence of different underlying impairments and does not imply necessarily hypokinetic behavior.

12. One potential concern is that opportunity for relearning of mental imagery could explain the dissociation of mental simulation accuracy between motor cortex and parietal damage. Most patients were followed up over an extended period of time. C.P., the patient with motor cortex damage, was first seen 3 months after onset of her motor impairments. She was tested repeatedly for over 2 years, and her performances in motor imagery tasks neither improved nor deteriorated. Patient J.J. was seen only once, shortly after his stroke; hence, we have no follow-up data on him. Patient R.L., whose tumor had been diagnosed 4 years before we began testing him, performed various mental imagery tasks on numerous occasions over a 6-month period. The impairment was quite severe and did not change over time. Quite to the contrary, the patient reported that because these experiments made him acutely aware of a deficit he did not know he had, he deliberately tried to train himself at forming mental images of hand movements but was always unsuccessful. The remaining patients had nonevolving, vascular lesions (R.K. and J.D.). Follow-up testing over a 6- to 9-month period showed no change. Therefore, it appears that the inability to form mental representations of upper limb movements appears to be a stable and permanent consequence of parietal lobe lesions.
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16. The study does not address the contribution of visual imagery to mentally simulated movements, though it has been suggested that a representation of visual context is activated in conjunction with an imagined action [S. M. Kosslyn, *Image and Brain* (MIT Press, Cambridge, MA, 1994)]. Because they are specific to a particular body part, it is unlikely that impairments of the type described here could result from a visual imagery deficit. Another question is whether parietal patients can generate a static representation of their hands. Mental movement simulation would be preempted without this capacity, and the apparent motor imagery impairment would in fact reduce to a selective body schema disturbance. Subjective descriptions obtained from patients ranged from an incapacity to "imagine my hand as other than normal" (R.K.) to a subjective sense of a fading image of the hand during movement (R.L.). In view of the latter comment, and given the marked motor imagery impairment exhibited by R.L., we used a modified mental rotation task [L. M. Parsons, *J. Exp. Psychol. Hum. Percept. Perform.* **20**, 709 (1994)] to assess the capacity to form mental pictures of one's hands. Participants listened, with eyes closed, to verbal instructions asking them to imagine one of their hands in a particular position and report the side on which the little finger (or the thumb, on alternate trials) appeared from their own perspective. A typical instruction was: "Imagine your own left hand, fingers pointing down, back of the hand facing you. Is your thumb on your left or right side?" This task can be performed correctly only if the individual imagined the designated hand (left or right) in the appropriate orientation referred to his or her own point of view. R.L. scored 29/32 (91%) correct on this task, with a mean response latency of 6.2 s, within the range of the control individuals (81 to 100% correct, 3.8 to 7.8 s mean response latency). Hence, the behavior observed after parietal lesion appears to concern specifically the ability to generate a representation of hand movements, not hand shapes or orientations.
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