Movement Intention After Parietal Cortex Stimulation in Humans

Michel Desmurget,[1,2] Karen T. Reilly,[1,2] Nathalie Richard,[1,2] Alexandru Szathmari,[3] Carmine Mottolese,[3] Angela Sirigu[1,2]*

Parietal and premotor cortex regions are serious contenders for bringing motor intentions and motor responses into awareness. We used electrical stimulation in seven patients undergoing awake brain surgery. Stimulating the right inferior parietal regions triggered a strong intention and desire to move the contralateral hand, arm, or foot, whereas stimulating the left inferior parietal region provoked the intention to move the lips and to talk. When stimulation intensity was increased in parietal areas, participants believed they had really performed these movements, although no electromyographic activity was detected. Stimulation of the premotor region triggered overt mouth and contralateral limb movements. Yet, patients firmly denied that they had moved. Conscious intention and motor awareness thus arise from increased parietal activity before movement execution.

A central question in the study of human behavior concerns the origin of willed actions. Where in the brain are intentions formed? How do we become aware of these intentions? According to the dualist philosophy (1), our encephalon is just the recipient of conscious intentions formed elsewhere in a non-physical realm. This implies that conscious intention comes first, as the leading cause of our actions. Although appealing from a spiritual point of view, this hypothesis was progressively challenged by a large set of studies (2–4). Results showing that the decision to move did not precede, but instead lagged, the onset of brain activity signaling motor preparedness were especially convincing (5–7). Thus, researchers suggested that conscious intention of a movement emerged as a consequence of increased neural activity in a premotor-parietal circuit, which elaborates motor plans before action (2). This cortical circuit has also been involved in motor awareness, that is, the awareness that we are actually executing the intended action (7–10).
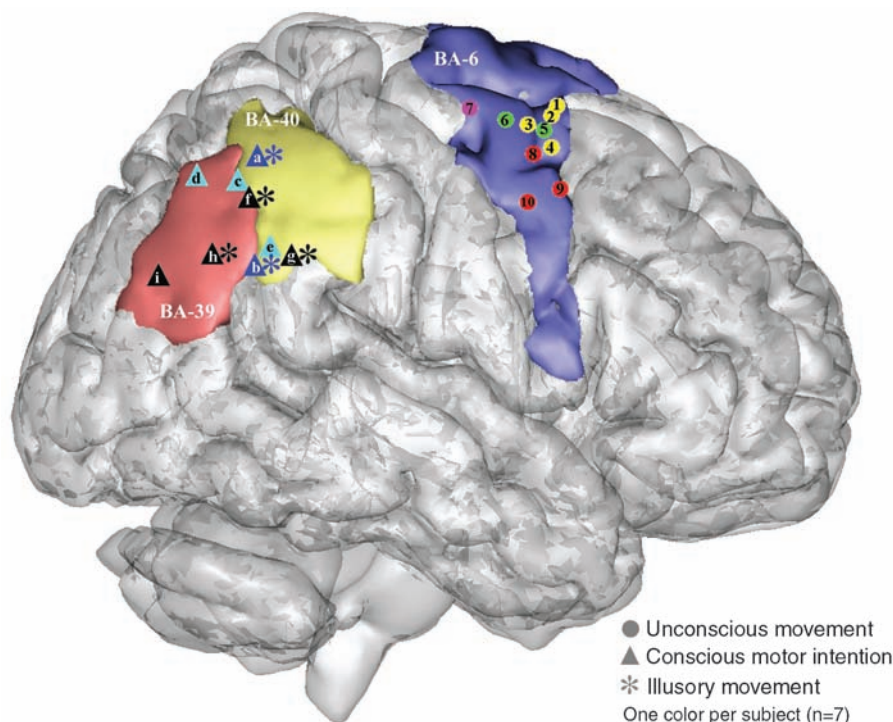
However, the specific contribution of premotor and parietal regions to conscious intention and motor awareness remains unclear. We reasoned that, by directly stimulating parietal and premotor cortex regions, we should be able to evoke motor responses in specific body parts and that, in areas involved in carrying out advance computations related to conscious intention and motor awareness, these movements should be accompanied or preceded by the subjective experience of willed actions. We used direct electrical stimulation (DES) in seven individuals with brain tumors located anteriorly (N = 4, PM1 to PM4) or posteriorly (N = 3, PP1 to PP3) to the central sulcus. Patients were operated under local anesthesia by using DES as a functional mapping technique in order to minimize the risk of postoperative sequelae (11). DES was delivered with a bipolar

electrode using standard increasing intensities (2, 5, and 8 mA) and durations (1, 2, and 4 s). Up to four replications were performed for each stimulation site. Replications were delivered non-consecutively to avoid provoking seizures. Throughout the experiment, electromyographic (EMG) signals were collected in the contralesional hemibody in 12 muscles covering the face, hand, wrist, elbow, knee, and foot. Stimulation sites were localized with high resolution on individual magnetic resonance (MR) images by using a peri-operative neuronavigation system and reconstructed offline.

Fifty-seven sites were stimulated in the frontal, parietal, and temporal regions (fig. S2A). Posterior

parietal stimulations were performed in Brodmann areas (BAs) 7, 39, and 40. Premotor stimulations were performed in the dorsal sector of BA 6, excluding the convexity and mesial structures involving the supplementary motor area (SMA). Of the stimulated sites, 46% were silent, meaning that DES did not produce any sensations or overt motor responses, and 20% were associated with somatic sensations such as tingling or itching. One participant (PP1) reported a robust visual illusion of background displacement when stimulated in the superior temporal gyrus (BA 22). Of the remaining sites (34%), 16% evoked responses related to motor awareness or movement intention, whereas 18% triggered actual movements. We will focus on these remaining sites, designated as responsive. The distribution of DES effects across brain areas is summarized in fig. S2B.

For the three patients with postcentral tumors, nine responsive sites were found in BAs 39 and 40 (Fig. 1). Stimulation of all these sites produced a pure intention, that is, a felt desire to move without any overt movement being produced or EMG activity recorded in the concerned muscles. In two of the patients (PP1 and PP2), the same sites were stimulated again later but at a higher intensity. Conscious motor intentions were replaced by a sensation that a movement had been accomplished, and yet, just as during the first stimulation trial, no actual movement or EMG activity was observed. Thus, these patients experienced awareness of an illusory movement (Fig. 2). For example, patient PP3 reported after low-intensity stimulation of one site (5 mA, 4 s;

[1]Centre de Neuroscience Cognitive, CNRS, UMR 5229, 69500 Bron, France. [2]Université Lyon 1, 69100 Villeurbanne, France. [3]Neurosurgery Unit 500, Hôpital Pierre Wertheimer, Hospices Civils de Lyon, 69003 Lyon, France.

*To whom correspondence should be addressed. E-mail: sirigu@isc.cnrs.fr

Fig. 1. Premotor and parietal responsive sites shown after registration of the individual MR image to the MNI template. Left stimulations have been reported on the right hemisphere. Colored areas define the anatomical boundaries of BA 40 (yellow), BA 39 (orange), and BA 6 (blue).

site a in Fig. 1), "I felt a desire to lick my lips" and at a higher intensity (8 mA, 4 s), "I moved my mouth, I talked, what did I say?" Similar results were found in patient PP1 for hand (two sites, g and h, in Fig. 1) and foot (one site, f, in Fig. 1) movements. Patient PP2 reported, after stimulation in BA 40 (8 mA, 4 s; site e in Fig. 1), that she felt "like a will to move" her chest (12). The same words were later used for another site with respect to the arm (8 mA, 4 s; site c in Fig. 1). Without prompting by the examiner, all three patients spontaneously used terms such as "will,"

"desire," and "wanting to," which convey the voluntary character of the movement intention and its attribution to an internal source, that is, located within the self (movies S2 and S3).

Electrical stimulation in the frontal cortex contrasted sharply with the above descriptions (Fig. 3). For the four precentral patients, 10 responsive sites were found in the dorsal part of the premotor cortex (BA 6; Fig. 1). These sites triggered movements of various limb segments and the mouth (fig. S2C) (13) devoid of conscious intention and awareness. Patients never

expressed the desire to move and never became aware that they produced a motor response. For example, during stimulation patient PM1 exhibited a large multijoint movement involving flexion of the left wrist, fingers, and elbow, as well as a rotation of the forearm (8 mA, 4 s; site 7 in Fig. 1). He did not spontaneously comment on this, and when asked whether he had felt a movement he responded negatively. The ability of patients to detect electrically evoked movements did not change with the intensity of the stimulation. Higher currents evoked larger movements and
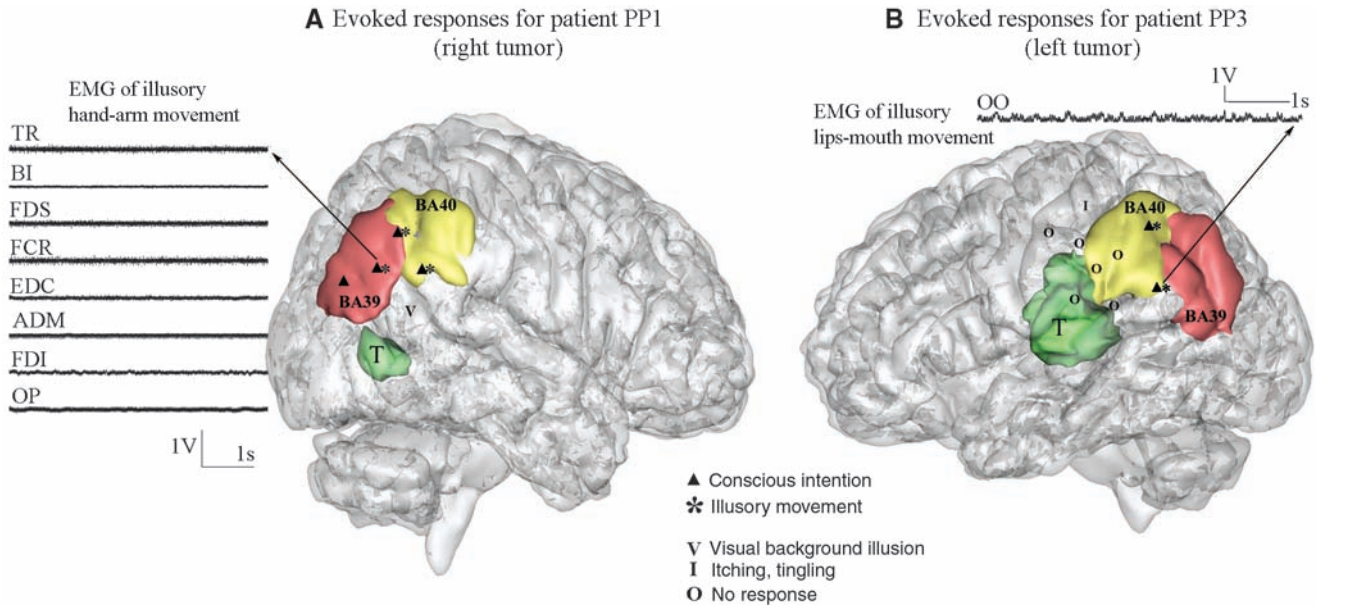
Fig. 2. (A and B) Individual brains and stimulation sites reconstructed for two patients harboring postcentral tumors. EMG signals are shown for the stimulation sites identified by arrows. T indicates tumor; TR, triceps; BI, biceps; FDS, flexor digitorium superficialis; FCR, flexor carpi radialis; EDC, extensor digitorium communis; ADM, abductor digiti minimi; FDI, first dorsal interosseous; OP, opponens pollicis; and OO, orbicularis oris. Colored areas define the anatomical boundaries of the tumor (green), BA 40 (yellow), and BA 39 (orange).
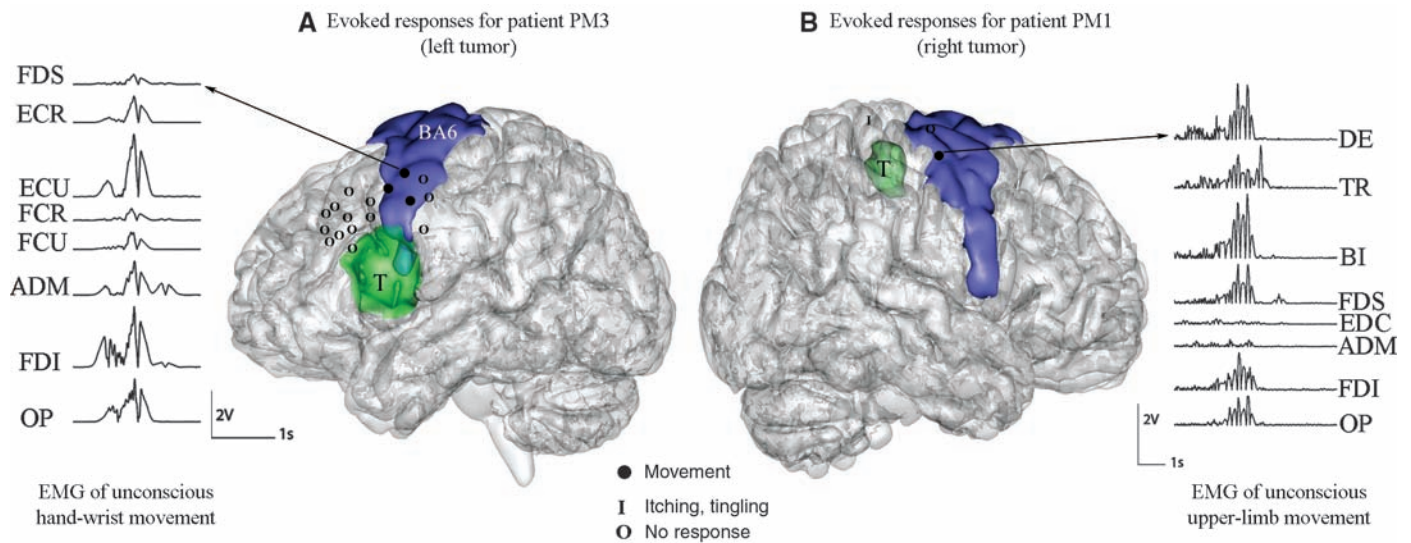
Fig. 3. (A and B) Individual brains and stimulation sites reconstructed for two patients harboring precentral tumors. EMG signals are shown for the stimulation sites identified by arrows. DE, deltoid; ECR, extensor carpi radialis; ECU, extensor carpi ulnaris; and FCU, flexor carpi ulnaris. Colored areas define the anatomical boundaries of the tumor (green) and BA 6 (blue).

recruited more muscles as compared with movements triggered by lower currents. Despite increasing stimulation intensity, patients remained completely unaware that a movement occurred (movies S1 and S4) (14).

We report two main contrasting findings: (i) Stimulation of the posterior parietal cortex caused human participants to intend to move and to report having moved, even in the absence of actual motor responses. (ii) Stimulation of the premotor cortex triggered limb and mouth movements that were not consciously detected by the patients.

Clinical observations of high-level movement deficits in patients with apraxia after parietal damage have led to the hypothesis that the posterior parietal cortex contains stored movement representations (15, 16). It can be proposed that direct stimulation of the parietal cortex activates such representations. However, the fact that patients experienced a conscious desire to move indicates that stimulation did not merely evoke a mental image of a movement but also the intention to produce a movement, an internal state that resembles what Searle called "intention in action" (17). This finding is consistent with nonhuman primate results suggesting that the posterior parietal cortex harbors a "map of intentions," with different subregions dedicated to the planning of eye, reaching, and grasping movements (18), and that activity of parietal neurons is highly correlated to processes of motor planning and decision-making (19, 20). It is tempting to propose that electrically induced intentions arise, in our study, from the activation of some nodes within this intentional map. Interestingly, when the stimulation intensity was increased, motor intentions were replaced by a form of illusory movement awareness. In the absence of any muscle contraction, the patients reported that they had actually performed the movement they previously intended to do. Although the nature of this phenomenon cannot be formally elucidated here, it may be hypothesized that motor intention arises from the activation of a limited subregion within the cortical network activated during movement execution. According to this view, higher intensities of stimulation would not simply prime a motor representation to consciousness (giving rise to intention) but also recruit the executive network responsible for movement monitoring through forward modeling. This process of forward modeling has been shown to rely on posterior parietal computations (21–23). It could form the basis of the illusory movement awareness experienced by our patients, assuming that the signal we are aware of when making a movement does not emerge from the movement itself but rather from the predictions we make about the movement in advance of action (3, 4, 7, 24, 25).

It has been reported that stimulation of the SMA triggers an urge to move that resembles an irrepressible desire to move going beyond patients' will (26). This suggests a potential role of SMA in generating motor intentions (2, 27). However, intentions evoked by stimulation of SMA stand

in contrast with what was described by our patients, who reported experiencing an endogenously generated wish to move. The imperative character of the motor intention with SMA stimulation is demonstrated by the fact that higher currents triggered movements (26), whereas none of the stimulated parietal sites ever evoked actual muscle contractions. It is possible that both the parietal cortex and the SMA are linked to motor intentions but that intentions processed in these two regions correspond to different stages of movement planning: Intentions in the parietal lobe may be processed in relation to sensory predictions, whereas in the SMA intentions may be more closely related to motor commands.

Regarding the dorsal premotor cortex, stimulations triggered complex multijoint movements, as already reported in awake monkeys (28). Stimulation intensities were comparable to those performed in the parietal cortex. Yet, patients remained unable to detect the limb and mouth movements evoked by electrical stimulations. This suggests that the proprioceptive volleys associated with the movement were disregarded or not decodable by the brain areas which normally receive these feedback signals. This finding strengthens the conclusion that awareness of initiating and executing a movement is not derived from afferent inputs but rather from the internal computations carried out in the posterior parietal cortex before action (2–4, 7). Our data are compatible with behavioral studies showing that we are largely unaware of sensory feedback about the ongoing state of our motor system, as long as our intentions are achieved (4). Peripheral inputs probably intervene at a further stage for comparing expected and actual movements, that is, when we need to construct a veridical motor awareness (2, 24, 25). Recently, Berti et al. (9) have linked the comparative process leading to veridical awareness to the functioning of the dorsal premotor cortex (BA 6). As shown by the authors, this structure is the most commonly lesioned in hemiplegic patients who obstinately claim that they can move their paralyzed limbs. In our study, premotor stimulations did not evoke any form of conscious intention. As a consequence, the proprioceptive inputs could not be compared to any expected input to estimate movement state to construct a veridical motor awareness.

Our study suggests that motor intention and awareness are emerging consequences of increased parietal activity before movement execution. The subjective (and potentially illusory) feeling that we are executing a movement does not arise from movement itself, but it is generated by prior conscious intention and its predicted consequences.

References and Notes

1. R. Descartes, *Méditations Métaphysiques (1641)* (Flammarion, Paris, 1992).
2. P. Haggard, *Trends Cogn. Sci.* **9**, 290 (2005).
3. P. Haggard, *Nat. Rev. Neurosci.* **9**, 934 (2008).
4. C. D. Frith, S. J. Blakemore, D. M. Wolpert, *Philos. Trans. R. Soc. London Ser. B* **355**, 1771 (2000).
5. B. Libet, C. A. Gleason, E. W. Wright, D. K. Pearl, *Brain* **106**, 623 (1983).
6. P. Haggard, M. Eimer, *Exp. Brain Res.* **126**, 128 (1999).
7. A. Sirigu et al., *Nat. Neurosci.* **7**, 80 (2004).
8. A. Sirigu, E. Daprati, P. Pradat-Diehl, N. Franck, M. Jeannerod, *Brain* **122**, 1867 (1999).
9. A. Berti et al., *Science* **309**, 488 (2005).
10. P. Haggard, E. Magno, *Exp. Brain Res.* **127**, 102 (1999).
11. Materials and methods including stimulation protocol, EMG recording procedure, and reconstruction of stimulation sites are available as supporting material on *Science* Online.
12. This result is consistent with monkey studies showing that protrusion movements of the chest are represented in the posterior parietal cortex (29).
13. The movements observed in our premotor patients were similar to the movements reported in monkeys after long train stimulations (28). It is thought that these electrically induced movements are functionally meaningful (30, 31).
14. The possibility can be ruled out that the absence of motor awareness in our patients was due to a low level of vigilance. The anesthetic and stimulation protocols were identical in premotor and in parietal patients who did report illusory movements. During peri-operative functional evaluation, premotor patients appeared well awake (see movies S1 to S4) and otherwise behaved as the parietal patients: They could talk, count, or move in response to verbal commands. Some of them reported sensory feelings of tingling or itching, indicating that they could introspect on stimulation-induced experiences. In one patient (PM4), we examined whether unseen passive movements of the forearm were perceived and reported. Indeed, this was the case.
15. L. J. Rothi, C. Ochipa, K. M. Heilman, in *Apraxia: The Neuropsychology of Action*, L. J. Rothi, K. M. Heilman, Eds. (Psychology Press, Hove, UK, 1997), pp. 29–49.
16. A. Sirigu et al., *Cortex* **31**, 41 (1995).
17. J. R. Searle, *Intentionality: An Essay in the Philosophy of Mind* (Cambridge Univ. Press, Cambridge, 1983).
18. R. A. Andersen, C. A. Buneo, *Annu. Rev. Neurosci.* **25**, 189 (2002).
19. L. H. Snyder, A. P. Batista, R. A. Andersen, *Vision Res.* **40**, 1433 (2000).
20. J. I. Gold, M. N. Shadlen, *Nature* **404**, 390 (2000).
21. J. R. Duhamel, C. L. Colby, M. E. Goldberg, *Science* **255**, 90 (1992).
22. M. Desmurget, S. Grafton, *Trends Cogn. Sci.* **4**, 423 (2000).
23. A. Sirigu et al., *Science* **273**, 1564 (1996).
24. A. Berti, L. Spinazzola, L. Pia, M. Rabuffeti, in *Sensorimotor Foundations of Higher Cognition Series: Attention and Performance XXII*, P. Haggard, Y. Rossetti, M. Kawato, Eds. (Oxford Univ. Press, New York, 2007), pp. 163–182.
25. A. Fotopoulou et al., *Brain* **131**, 3432 (2008).
26. I. Fried et al., *J. Neurosci.* **11**, 3656 (1991).
27. H. C. Lau, R. D. Rogers, P. Haggard, R. E. Passingham, *Science* **303**, 1208 (2004).
28. M. S. Graziano, C. S. Taylor, T. Moore, *Neuron* **34**, 841 (2002).
29. H. Gemba, K. Matsuura-Nakao, R. Matsuzaki, *Neurosci. Lett.* **357**, 68 (2004).
30. M. Graziano, *Annu. Rev. Neurosci.* **29**, 105 (2006).
31. E. Stark, I. Asher, M. Abeles, *J. Neurophysiol.* **97**, 3351 (2007).
32. This study was funded by Centre National de la Recherche Scientifique, Agence Nationale de la Recherche (Neuro-031-02), and by Human Frontiers Science Program (RGP0056/2005-C) to A.S. We thank J. R. Duhamel for helpful discussion. We thank patients for their cooperation and L. Pouga and the clinical staff for help during testing.

Supporting Online Material