Response to Comment on “Movement Intention After Parietal Cortex Stimulation in Humans”

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Karnath et al. argue that the behavioral effects observed in our study after direct parietal and premotor electrical stimulation (DES) could reflect a decrease of local cortical activity. If so, intention and awareness would not reflect the activity of the stimulated area but the recruitment of remote regions. Although tenable, this view does not seem to be the most plausible.

We used direct electrical stimulation (DES) to investigate the origin of motor intentions in the brain (1). Karnath et al. (2) argue that the net effect of electrical stimulation on brain activity is very hard to determine and that this technique can lead to either an increase (activation) or a decrease (inhibition) of neural activity. Also, irrespective of its local effects, it can involve the recruitment of remote areas through current spread along fiber pathways. Consequently, drawing functional conclusions from DES alone might be naively impossible. We fully agree with this claim. However, in our study, DES was not used blindly but with the purpose of testing a functional model elaborated in light of a large set of available evidence. In the literature, there is clear support for the idea that the posterior parietal cortex (PPC) is involved in the generation of motor intention and awareness [for a review, see (3)]. Andersen and colleagues have shown that neural activity in this region correlates with the goal of the movement, that is, intention, rather than particular signals from various muscles information or attention per se (4). Neuroimaging studies in healthy human subjects have linked PPC activity with motor awareness (5). Clinical data have shown that PPC contains stored movement representations, the activation of which could underlie conscious intention (6). Neuropsychological findings have demonstrated that patients with selective lesions in PPC lose the early subjective experience of wanting to move, being aware of their movement only when it is about to be executed (7). A recent study also indicated that circumscribed lesions of the PPC can cause the occurrence of alien movements generated without conscious intention through the recruitment of the precentral executive motor regions (8).

It is true that intentional responses observed in our study after PPC stimulations could have been caused by the inactivation of the inferior parietal lobule (IPL) or the activity or disinhibition of another (“so far unknown”) structure connected to the IPL, as suggested by Karnath et al. (2). However, this contention seems to ignore previous studies on intention and awareness. So far, previous work has identified only two potential candidates besides PPC: the supplementary motor area (SMA) and the premotor cortex (PMC) [for a discussion, see (3)]. The SMA is activated when subjects focus their attention on their intention to move (9). At the same time, this area is commonly injured in patients suffering from anarchic hand syndrome (10), a neurological disorder in which hand movements are performed without conscious will. It also generates irrepresible intentions to move when electrically stimulated (11). However, these intentional responses, characterized by an “urge to move,” are very different from the responses observed after PPC stimulations (3). Consequently, it is unlikely that intentional responses observed in response to PPC stimulations are routed through the SMA.

Regarding the PMC, a distinction must be made between motor awareness (the subjective feeling that we are moving) and veridical motor awareness (the objective knowledge that we are actually moving). In our study (1), PMC stimulations did not elicit conscious intentions or motor awareness. It is therefore unlikely that parietal DES acted through the remote recruitment of PMC. This being said, it has been shown that hemiplegic patients with PMC lesions can be aware of movements that did not occur, as if these patients were in fact aware not of their movements but of what should result from carrying out their initial motor intentions (3). This suggests that PMC could be involved in the emergence of veridical motor awareness by comparing expected and actual sensory inputs (12). It is possible, as claimed by Karnath et al. (2), that this computational process was disrupted in our study by PMC stimulation, thus causing the subjects to remain unaware of movements that were triggered without intention. However, it seems unlikely that PMC was inhibited by DES while overt movements were induced through distant activation of the primary motor cortex (M1). Indeed, it is known that PMC projects heavily to the spinal cord (13), and it is not clear why DES should not spread along these projections. Also, DES triggers different types of movements when applied over M1 or PMC (14). Finally, the premotor neurons that trigger limb movements when electrically stimulated are also responsive during active goaldirected reaches (15).

Besides the observations above, two other arguments lead us to consider that the inhibition hypothesis is less plausible than the activation one. First, we employed stimulation parameters typically used in perioperative functional investigations. If these parameters inhibit PPC and/or activate remote structures, thus leading to the identification of functions located outside the stimulated area, then resection of the PPC regions identified as eloquent during perioperative mapping (for instance for language) should have no major impact on the tested function. Clearly, this is not the case, as shown by the substantial decrease of postoperative deficits when surgery uses functional mapping in awake conditions (16). Second, inhibiting the PPC means that the output of this region will drop to zero. As a consequence, the cortical areas connected with the PPC will stop receiving any signal, and their firing level will drop too (nonlocal cortico-cortical projections are glutamatergic, i.e., excitatory). In this context, one may see how functional disruptions can occur in the examples reported by Karnath et al. (2), for language production and visual object processing. However, in our study, there was no ongoing task, and it is not clear how cortical deactivations could evoke positive behavioral responses such as motor intentions and awareness. Finally, Karnath et al. (2) seem to concur with us when claiming that “the inference that can be drawn with absolute certainty from [our] interesting observations is that the IPL is part of a network used for generating movement intention.” However, in contrast to these authors, we do not believe that the deactivation or remote-activation hypotheses are as plausible as the “increased parietal activity” model in explaining the actual functional contribution of this structure.

References and Notes


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