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Review of Motor and Phantom Related Imagery

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Abstract

This review presents a summary of recent efforts in understanding the systems of the brain involved in motor imagery. Motor imagery likely involves many cortical regions in its generation, but in action may also involve subcortical structures. The parietal lobe seems to be particularly important, as demonstrated by brain imaging studies and patients with lesions of this region. Brain activity correlated with imagery may be related to an efference copy used to compare with peripheral sensory signals for the correction of movement. Amputees with phantom representations have also provided valuable information in this field, since they demonstrate cortical reorganization which also alters imagery of the missing limb. The following summary explores the recent difficult and challenging studies used to tease out motor imagery in man.

I. Introduction

The internal rehearsal of a motor action, or motor imagery, may very well result in the activation of many of the same brain structures activated by the executive motor system (1). Primary motor cortex is almost certainly involved with motor imagery (2), since patients lesioned in motor cortex suffer from slow imagined movements, similar to the slowness of movements they physically demonstrate contralateral to the lesion. Motor imagery may represent a tap into an “efference copy” stream, or a feed-forward system used by the brain for control and correction of real movements (3). Such a copy of the efferent motor command could be used to compare with peripheral sensory signals to correct for the active and passive mechanical properties of the somatic motor system in close to real-time and to allow for rapid movement (4).

But what structure or structures in the brain would support such an efferent command copy? Parietal cortex seems to be one possibility. Specifically, primate cortical recordings from parietal lobe and patients with parietal lobe lesions demonstrate the importance of this region in maintaining an internal body representation (5). The parietal cortex likely does not act alone. PET studies and EEG localization also point toward the involvement of the anterior cingulate, premotor and opercular motor regions (6). An interesting question has to do with the time and geometric constraints patients experience during imagery tasks (as described by 7). These limits of imagery typically follow normal anatomic constraints, or involve a slowing down of the imagery in the case of lesions in the parietal lobe. Do these

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limits imply the activation of fixed motor programs when imagery is activated? Mental rehearsal of known movements or previously performed movements might be completely different than using the creative imagination to picture the movements (in which nonphysical sorts of movement like wrist rotations of 360° can be imagined). Additionally, it is unknown exactly for what purpose a motor efference copy would be used by the brain. Although there are some hints that the copy and peripheral signal comparison might be used as an error correction signal for movement, and this comparison might occur in parietal cortex (8). There is additionally some evidence that the copy is transmitted caudally to subcortical structures (9, 10).

Patients exhibiting phantom limb sensations after congenital or traumatic amputations have similarly proven useful in teasing out imagery related effects. Functional magnetic resonance imaging (fMRI) of amputees performing imagery tasks with their phantom show sensorimotor activation similar to intact controls (11). Here we review recent studies on motor imagery, drawing from the literature examples of movement imagery in intact patients as well as imagery of phantom limb movements.

II. Imaging Evidence of Motor Imagery Related Activity

Positron emission tomography (PET) imaging of patients has been obtained during motor imagery tasks, including imagery of grasping an object with the hand (12). These studies show activation contralateral to the imagery movement in prefrontal and dorsolateral frontal cortex, the supplementary motor area (SMA) as well as parietal lobe regions (12). Activation can also be observed in bilateral anterior cingulate cortex and the ipsilateral inferior frontal gyrus (Brodmann areas 6 and 44). More recently, (13) studied PET imaging of gait imagery in healthy volunteers, and included scans during imagery of standing, initiating gait, walking, and walking with obstacles. Interestingly, as the complexity of the imagery task increased, activation in the left SMA as well as the right inferior parietal lobe and left parahippocampal gyrus was enhanced implying an increasing cognitive load with task complexity.

In similar studies involving fMRI concurrent with a motor imagery paradigm, (14) found that bilateral premotor areas, the SMA, and the left posterior parietal region were selectively activated in a set of 8 right-handed healthy individuals performing a right-handed task. And when comparing visual imagery of movement versus kinaesthetic imagery, (15) have found that visual imagery selectively activates the occipital visual regions and the superior parietal lobe, while kinaesthetic imagery activates again the inferior parietal lobe. In amputated patients specifically, fMRI can demonstrate activation during phantom movements. Roux et al. studied three individuals (2 upper extremity amputations, and one bilateral lower extremity) using this modality. They showed blood oxygen level dependent (BOLD) signal changes in primary sensorimotor cortex as well as SMA contralateral to the phantom motor imagery, although this study did not demonstrate strong parietal lobe effects (16).

III. The Importance of the Parietal Lobe

Sirigu et al. have now developed an extensive body of work concerning the effects of parietal lobe injury on motor imagery and motor task performance. They have reported on a series of three patients with lesions involving the left parietal cortex, and their ability to perform in a hand recognition task while performing movements with the hand (5). These patients could identify their own hand when shown the hand on a video monitor, and they could identify a control hand (not their own) when the control hand was performing movements different from their own task. However, when the control hand was performing congruent movements these ataxic patients had difficulty identifying the ownership of the

hand when compared to a control group. This implies a failure in these patients to accurately evaluate and compare sensory feedback with some sort of internal model (5).

In 2009, Desmurget et al. published an interesting study attempting to characterize the origin of human movement intention. Via cortical stimulation in patients undergoing awake craniotomies, these authors demonstrated strong subjective feelings of movement intention by stimulation of the inferior parietal cortex (Brodmann's 39 and 40) (17). At higher stimulation currents, the subjects even reported the completion of these movements when in fact no electromyographic activity was observed. The substrate of movement intention could also lie in premotor cortical regions, however direct stimulation here in this study never resulted in subjective feelings of intention, only overt movements at high stimulation intensities with frequent denial of the movements by the subjects. Desmurget et al. describe the patients undergoing parietal stimulation experiencing strong subjective feelings of the intention of movement with each subject using phrases such as "will", "desire", and "wanting to" when asked to characterize their sensations. The authors further discuss that the hypothesized forward modeling function of the posterior parietal regions might also play a role in the subject's insistence that movement had taken place during higher stimulation currents (17).

IV. Motor Adaptation and Subcortical Activity

Parietal cortex may not be the only structure in the brain supporting an internal model of motion. For instance, patients with lesions in the ventral intermediate (Vim) subnucleus of the thalamus (the cerebellar relay nucleus) demonstrate difficulties in reaching with the contralateral arm under the influence of time varying force fields compared to the normal side (18). Similarly, in patients undergoing deep brain (DBS) surgery for the treatment of essential tremor, stimulation applied to Vim also impairs their ability to adapt the reach function of the contralateral arm under varying force fields (18). These studies imply that adaptive motor control of the extremities, which likely relies on internal modelling of motion and the effects of force fields, requires cerebellar input into the thalamus.

Recently Kühn et al. recorded motor imagery related activity via local field potential (LFP) recordings in the subthalamic nucleus (STN) during surgery for Parkinson's disease (9). This group explored specifically event related desynchronization (ERD) of the oscillatory beta activity in the LFP of the STN during a cued motor image task involving the imagery of wrist extension. The levels of ERD observed in these recordings during motor imagery were similar to those produced during actual movement of the wrist.

Similarly, Anderson et al. have demonstrated similar results at a single-unit level in sensory and motor regions of the thalamus, both in contralateral regions of the thalamus to an arm amputation as well as intact patients (10). This small study included single-unit potential recordings obtained in the ventral intermediate (Vim), ventral caudal (Vc), and ventral oral posterior (Vop) subnuclei of the thalamus, in 4 patients with either essential tremor or chronic pain disorders. One of these patients was an amputee, losing an arm just below the shoulder approximately 20 years before the microelectrode recordings were made. Interestingly, motor imagery produced single unit responses in these thalamic subnuclei which were similar in time structure to phantom-imagery induced activity in the amputee, implying the possibility that similar subcortical circuitry subserves both phenomena (10).

V. Evidence from Amputees

The reorganization of the motor homunculus after limb amputation in primary motor cortex has been demonstrated by direct transcranial magnetic stimulation (TMS) in affected subjects. Cohen et al. performed TMS in 7 patients with a single upper limb amputation, and

found that when compared to real movements on the unaffected side the sensation of phantom movements could be elicited over a larger region of cortex and required lower stimulation amplitudes (19). Representations of muscles just proximal to the amputation stump are also reorganized compared to normal controls (20).

More recently, Raffin et al. have reported on a series of 19 upper-limb amputees who were asked to perform both imagined movements of their phantom representation as well as execution of movement of the phantom representation (21). The subjects reported clear differences in the speeds at which executed voluntary movements of the phantom took place compared to purely imagined movement of the phantom, with imagined movements happening faster. Additionally, stump muscle motor activation always took place with the voluntary movements, but never with the imagined movements.

Subjects with traumatic amputations concurrent with phantom limb pain show pronounced reorganizations of somatosensory cortex, with invasion of the amputated regions by surrounding regions of the sensory homunculus. This has been demonstrated in EEG source localization imaging studies of phantom-limb patients in comparison to congenital amputees and normal controls (22). The extent of cortical reorganization also seems to differentiate between amputated patients having phantom limb pain, and amputated patients with no associated painful symptoms as shown by fMRI studies during real and phantom movements (23). The degree of cortical reorganization (specifically the degree to which remaining surrounding somatopic structures invade the cortical representations of the amputated region) is also indicative of the amount of pain experienced implying that this type of reorganization may be causally related to the pain itself (23). The internal cortical representation of a limb may also manifest or modulate pain experience as well. Acerra et al. exposed 10 subjects with complex regional pain syndrome type 1 (CRPS1) to a reflected mirror image of their normal limb (24). While watching this image, an examiner stroked the normal limb, and the subjects had the perception of the production of their usual neuropathic pain by this maneuver. This type of dysynchiria would imply that it is possible to produce this type of pain sensation simply by tricking the patients internal representation of the body, and not solely reliant on pathologic peripheral input (24).

VI. Anatomic Pathways

To investigate the possibility of a feed forward efference copy system in humans, Wolpert et al. looked at measures of prediction of position by normal subjects of their upper extremity location after movements in the dark, either in the presence or absence of motion altering force fields (25). With a model incorporating a forward copy of the arm dynamics as well as an anticipated sensory model, these authors were able to reproduce position estimation errors associated with various applied force fields (25). Wolpert et al. additionally provided evidence for the neural substrate of this constantly updated representation of the environment supplied by a visuomotor integration system by presenting a patient with a left superior parietal lobe lesion and an altered sense of limb position (26). This patient and other similar cases demonstrated perceptions of movement in isolated stationary limbs when visual confirmation was withheld (26).

One could hypothesize that primary motor cortex is bypassed when performing pure motor imagery tasks, since the descending information to spinal cord and muscle is somehow disabled. However, premotor and motor structures are consistently shown to play a role in motor imagery tasks, although in general there is less activity than with motor execution in these structures (1). Older lesioning studies in primates have demonstrated significant connectivity between the posterior parietal regions and the premotor areas, and there appears to be analogous connectivity in the human (27). It is possible that the imagery related

activity observed in primary and supplementary motor regions is the downstream efference copy representation (3). One future challenge will be in determining exactly which region of the parietal lobe might subserve motor imagery, i.e. the superior parietal lobule (Brodmann area 7) analogous to the area PG in the macaque (27) and/or the inferior parietal lobule which also appears to be activated in metabolic and functional imaging studies.

There are several possibilities for the substrate of any efference copy signal observed in subcortical structures. In general, corticothalamic connections are much more common than ascending inputs to the thalamic nuclei (28). Both output from layer 5 and layer 6 of the cortex form thalamic synapses (28). Vop, whose primary drivers are from the pallidum, projects to and receives afferents from premotor, supplementary motor, and motor cortices (29). Similarly there are reciprocal connections between the sensory nucleus Vc and somatosensory cortex, and the cerebellar relay Vim and primary motor cortex, SMA, and premotor cortex (29). The reciprocal connections between the motor cortex and the thalamic motor relays are one possible source of the subcortical imagery related signals recorded during imagined and phantom movements (9,10). The ventral connections coming from the rostral portion of the superior parietal lobule (SPL) represent an additional possible source for the cellular activity observed in thalamus during phantom and imagined movements (30).

VII. Conclusions

Motor imagery likely involves multiple brain regions in its generation, but seems to be supported primarily by the parietal lobe based on multiple lesioning and imaging studies. Signals correlated with motor imagery can also be detected in subcortical structures during imagery paradigms, and seem to indicate wide spread effects of this system. This activity may be related to an efference copy used to compare with peripheral sensory signals for the correction of movement. Amputees, with phantom representations of the missing limb, have also provided valuable information on the imagery system. Cortical reorganization after the amputation along with concomitant phantom limb pain seems to alter imagery of the limb. The anatomic pathways subserving motor imagery and their physiologic use are slowly becoming clearer from the difficult physiologic and imaging studies reviewed here.

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