Mapping phantom movement representations in the motor cortex of amputees

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Limb amputation results in plasticity of connections between the brain and muscles, with the cortical motor representation of the missing limb seemingly shrinking, to the presumed benefit of remaining body parts that have cortical representations adjacent to the now-missing limb. Surprisingly, the corresponding perceptual representation does not suffer a similar fate but instead persists as a phantom limb endowed with sensory and motor qualities. How can cortical reorganization after amputation be reconciled with the maintenance of a motor representation of the phantom limb in the brain? In an attempt to answer this question we explored the relationship between the cortical representation of the remaining arm muscles and that of phantom movements. Using transcranial magnetic stimulation (TMS) we systematically mapped phantom movement perceptions while simultaneously recording stump muscle activity in three above-elbow amputees. TMS elicited sensations of movement in the phantom hand when applied over the presumed hand area of the motor cortex. In one subject the amplitude of the perceived movement was positively correlated with the intensity of stimulation. Interestingly, phantom limb movements that the patient could not produce voluntarily were easily triggered by TMS, suggesting that the inability to voluntarily move the phantom is not equivalent to a loss of the corresponding movement representation. We suggest that hand movement representations survive in the reorganized motor area of amputees even when these cannot be directly accessed. The activation of these representations is probably necessary for the experience of phantom movement.

Keywords: amputation; phantom limb; transcranial magnetic stimulation; motor cortex; movement

Abbreviations: MEP = motor-evoked potential; TMS = transcranial magnetic stimulation


Introduction

Reorganization within the motor cortex following amputation has been described in both animals (Donoghue and Sanes, 1988; Schieber and Deuel, 1997; Wu and Kaas, 1999) and humans (Hall et al., 1990; Cohen et al., 1991; Fuhr et al., 1992; Kew et al., 1994; Ridding and Rothwell, 1995; Chen et al., 1998; Roricht et al., 1999; Irlbacher et al., 2002). Together, these studies show that stimulation of the cortical area previously devoted to the missing limb [using either cortical electrical stimulation or transcranial magnetic stimulation (TMS)] produces movements or muscle contractions of parts of the body that are represented in the cortical somatotopic map adjacent to the missing body part. For example, in the case of an above-elbow amputee, stimulating the presumed hand region in the motor cortex will produce muscle contractions in stump muscles such as the biceps and triceps and/or muscle contractions in the face (since the hand representation shares a medial border with the arm and a lateral border with the face). This has been interpreted as an invasion of the arm representation into a region of the cortex that previously controlled the now-amputated hand (Roricht et al., 1999; Irlbacher et al., 2002). There is now increasing evidence, however, that the motor representation of the missing hand can survive in the amputees’ brain.

Functional imaging studies show that upper-limb amputees who imagine moving their phantom hand activate regions of the motor cortex homologous to those activated...
during actual movements of the intact hand. They also show that cortical activity associated with imagined phantom hand movements is distinct from that associated with actual stump movements (Lotze et al., 2001; Roux et al., 2001; Roux et al., 2003). This suggests that this region still retains its original function, that is, the control of hand movements albeit now a phantom hand, despite the absence of the muscles that are normally targeted by its outputs. It has also been demonstrated that motoneurons within severed nerves that previously supplied hand muscles are active when long-term amputees move their phantom hand, suggesting that phantom movements involve both motor cortex activation and activity in descending motor pathways (Dhillon et al., 2004; Dhillon et al., 2005). Furthermore, we have recently shown that the motor output accompanying phantom movements is not restricted to the severed nerves but is also re-targeted toward stump muscles [see accompanying paper (Reilly et al., 2006)]. As a consequence, voluntary movements of a distal part of the phantom limb (i.e. hand opening/closing) become associated with specific patterns of stump muscle activity, which differ from activity patterns recorded in the same muscles during proximal phantom limb movements (i.e. elbow flexion/extension).

Although these results clearly suggest that a representation of the missing limb is maintained in the motor cortex, it is still unclear how this phantom representation coexists with the enlarged motor representation of the stump that results from amputation-induced reorganization. These results also raise the question of what happens to the motor representation when voluntary movement of the phantom becomes impossible but there is still a vivid sensation that the phantom limb is present. This question is important because most amputees report the ability to voluntarily move their phantom limb soon after the amputation, but in many cases this ability diminishes over time, with the phantom limb becoming more and more difficult to move, and in some cases becoming completely paralysed (Ramachandran and Rogers-Ramachandran, 1996). Does the inability to move the phantom imply that the motor representation has disappeared, or can this representation be maintained but become inaccessible?

There have been occasional reports that stimulating the motor cortex contralateral to the side of an upper limb amputation can sometimes evoke sensations of phantom limb movement (Hess et al., 1986; Cohen et al., 1991; Pascual-Leone et al., 1996). None of these studies, however, systematically investigated the relationship between amputation-induced reorganization of stump muscle representations and the sites from which phantom limb movements can be evoked. Furthermore, these reports did not document the types of movements evoked, did not compare evoked movements with the types of movements that the amputees reported being able to produce voluntarily, nor did they examine the relationship between TMS-evoked phantom movement perceptions and motor-evoked potentials (MEPs) in stump muscles.

In the present study, we used image-guided TMS to systematically map the movement perceptions evoked in the phantom limb while recording muscular activity evoked in stump muscles. First, we wanted to determine whether phantom hand movements are elicited by direct stimulation of the hand region in M1. If this were the case, it would support the hypothesis that the original hand representation is maintained in the motor cortex despite cortical motor reorganization induced by the amputation. Secondly, we wanted to determine if the type of phantom movement felt by the amputated subject is related to the pattern of muscle activity evoked in the remaining muscles. Finally, we investigated whether it was possible to artificially evoke phantom movements that patients could not voluntarily produce.

**Material and methods**

**Subjects**

Three traumatic above-elbow amputees (JM, SF, CP) [time since amputation: 2 (SF), 4.5 (JM) and 1 (CP) years; side of amputation: left (SF, JM), right (CP)] participated in this study. Each subject was experiencing phantom limb sensations at the time of testing, associated with moderate to severe phantom pain. They reported being able to move their phantom limb voluntarily, but that these movements were effortful, slow, of small amplitude and limited to movements of the digits. All subjects provided written informed consent to participate in the study, which was approved by the local ethical committee (Centre Léon Berard, Lyon, France).

**Experimental procedure**

TMS mapping was used to obtain muscle representation maps of two muscles on the intact side and one on the amputated side. The muscle mapped on the amputated side was always immediately proximal to the level of the amputation (biceps brachialis or deltoid). Whichever proximal muscle was mapped on the amputated side was always mapped on the intact side, along with the flexor digitorum superficialis. The intact side muscles were always mapped first. The locations of these stimulations were subsequently compared with stimulation sites on the amputated side that evoked phantom hand movements. MEPs were recorded in the mapped muscles, and in other surrounding muscles (e.g. other stump muscles, triceps, deltoid, biceps, and a face muscle, the zygomatic), but stimulation parameters were only optimized for mapped muscles. During stimulation over the cortex contralateral to the amputation, patients were instructed to report after each stimulation whether a movement was perceived in the phantom limb. If this was the case, movements had to be further classified as involving (i) a single finger (in this case he had to name which finger); (ii) the whole hand; (iii) the wrist; (iv) the elbow; (v) the whole arm.

The TMS mapping was performed using a Magstim 200<sup>®</sup> stimulator with a 70-mm figure-of-eight coil. The coil was held tangential to the skull, with the handle pointing backwards. The figure-of-eight junction of the coil was held over the site to be stimulated, and a constant coil orientation was maintained throughout the experiment.

To facilitate positioning of the coil over the sensorimotor cortex during the mapping, subjects wore a tight cap with a grid consisting of...
movement perception was also investigated in a single subject (JM). We chose one stimulation site and delivered pulses at each of nine different stimulation intensities (from 50 to 90% of stimulator output). Each stimulation intensity was delivered 12 times in a randomized manner, making a total of 108 pulses. The location stimulated was the ‘hotspot’ for eliciting phantom finger movements, chosen on the basis of the subject’s verbal report. For each TMS pulse the subject was asked to describe the movement elicited and to rate its perceived amplitude on a numeric scale (0: no movement–10: maximal range of motion with intact limb). For example, a response of 6 corresponded to a perceived movement of the phantom limb with an amplitude of 60% of the possible range of motion of the same joint(s) on the intact limb.

Results

All three subjects reported that they could voluntarily produce movements of small amplitude of the phantom fingers and thumb, but that they could not move their phantom wrist or elbow. Figure 1 shows TMS stimulation sites relative to the central and interhemispheric sulci on the cortex contralateral to the amputated side. In all three subjects most stimulation sites were anterior to the central sulcus (primarily over motor cortical areas), while some sites were posterior to the central sulcus (primarily over sensory cortical areas). When stimulation induced a phantom movement perception, all subjects were able to give very precise descriptions of the TMS-induced phantom movements, and these were described as automatic, effortless and of short duration, much like the twitches evoked during TMS over the motor cortex contralateral to the intact side. Stimulation never evoked other kinds of somatosensory sensations, such as touch. Occasionally, patients spontaneously reported increased phantom pain during stimulation (at 2.9% of the 103 stimulation sites tested), but more frequently they reported relief from it (at 10.7% of the stimulation sites) as the painful cramping sensation in their phantom hand was temporarily alleviated by the stimulation-evoked movement of the phantom.

Figure 1 shows that each subject experienced at least three different phantom movement perceptions, and that these were not always at the same phantom joint. For example, at some sites JM experienced movement of only his phantom wrist, while at others he experienced the combined movement of his wrist and digits, and there was at least one site where he reported isolated movement of his elbow with no movement of his digits or wrist. Moreover, the types of movements described at each joint were also diverse. For example, movements reported at the digits and wrist included isolated or combined flexion/extension of the fingers, abduction/adduction of the thumb, flexion/extension of the wrist and radial/ulnar deviation of the wrist. One subject also reported global movements of the whole limb going toward or away from the trunk. When this subject was asked whether the stump was also moving, he answered after a short hesitation: ‘Well, it should be given that the arm is moving!’, which illustrates that movement perceptions...
elicited in the phantom were more salient than the actual contractions of the remaining muscles, or the physical movement of the shoulder. It is important to note that while most of our stimulation sites were anterior to the central sulcus we also stimulated slightly posterior to the sulcus (see Fig. 1), where the main focus of the stimulation was probably over sensory areas. The large number of white dots on or posterior to the central sulcus, however, shows that phantom movement perceptions were rarely evoked from these more posterior sites.

The first column of the Fig. 2 shows a comparison of the map of the phantom digits and that of an intact-side finger muscle. It is important to note that although the cortical sites were stimulated randomly, the topography of evoked phantom digit movements was highly systematic; the sites at which subjects experienced movement of the phantom digits were clustered together and were surrounded by non-responsive sites. The second observation (already mentioned in the description of Fig. 1) is that although stimulation was applied over both the motor cortex and sensory cortices, those sites at which phantom digit movements were evoked were principally concentrated over the motor cortex. Finally, the sites at which phantom digit movements were evoked were comparable with those from which responses were evoked in an intact finger muscle (flexor digitorum superficialis).

The second column of Fig. 2 shows a large overlap between the sites at which stimulation evoked phantom movement perceptions and those at which it evoked responses in stump muscles. Data are only shown for one stump muscle per subject (the muscle for which stimulation parameters were optimized) although MEPs were often simultaneously observed in multiple stump muscles, but never in a face muscle. Indeed, on 86% of the trials on which subjects reported experiencing phantom digit movements this experience was accompanied by muscular responses in the biceps, triceps and/or deltoid. The large overlap between the sites at which stimulation evoked phantom movement perceptions and those at which it evoked responses in stump muscles raised the possibility that the sensation of phantom movement simply arises from a misinterpretation of the afferent feedback generated by stump muscle contractions, and not from activation of the hand representation in the motor cortex. To address this question we investigated whether the type of phantom movement felt by the subject was related to the pattern of muscle activity evoked in the remaining muscles, as a specific pattern of contraction would have generated a specific pattern of afferent feedback. We found
that a particular phantom movement perception was not always accompanied by contraction of a specific stump muscle, or by a specific contraction pattern in several stump muscles. That is, the same phantom movement perception was sometimes associated with a MEP in a particular muscle, but on other trials with a MEP in another muscle, while very similar patterns of MEPs across muscles were sometimes associated with different phantom movement perceptions. Of the 10 different phantom movements or movement combinations reported by the three subjects, 40% were always associated with the same MEP pattern (e.g. biceps only, triceps only, deltoid only, biceps + triceps, biceps + deltoid, triceps + deltoid or biceps + triceps + deltoid), while 60% were associated with different MEP combinations. Conversely, of the 11 different MEP combinations evoked by TMS, only 36% were systematically associated with movement at a particular joint or combination of joints, while 64% were associated with movements of two, three, or four different joints or joint combinations. For example, in JM the combined occurrence of MEPs in a given combination of muscles (biceps + triceps + deltoid, n = 12 stimulation sites) was associated with the perception of either a movement of the fingers only (n = 4), of fingers and wrist (n = 2), of wrist only (n = 5) or of elbow (n = 1), while the occurrence of a given perception of movement in the phantom limb (wrist, n = 9 stimulation sites) was associated with four different MEP combinations: responses in all three muscles (n = 5), responses in the triceps and the deltoid (n = 1), responses in the triceps and the biceps (n = 1) or response in the triceps only (n = 2). To date there are no studies showing that TMS-induced movement perceptions in normal subjects are reliably correlated with MEP response patterns. Thus, it is unclear whether the absence of a strict relationship between movement perception and MEP response pattern that we found is particular to phantom movement perception or reflects a more general effect of TMS. The absence of a strict relationship between phantom movement perceptions and stump muscle contraction patterns is particularly noteworthy, however, as we would not expect this to be the case if phantom movement perception was based solely on afferent feedback from the stump muscles.

In one subject we investigated the effect of TMS pulse strength on phantom movement perception. When we stimulated at a single point at a range of stimulus intensities, the phantom movement elicited remained constant (combined flexion of the fourth and fifth fingers) regardless of the intensity. Thus, increasing stimulus intensity did not evoke the perception of movement in additional phantom joints. Figure 3 shows that there was a close association between the perceived amplitude of TMS-evoked phantom finger movements and the intensity of stimulation. We interpret this correspondence as further support for the validity of the subject’s reports of his phantom movement perceptions. The perceived amplitude of phantom finger movement was correlated with MEP amplitude in stump muscles (biceps and triceps). Although an expansion of the representation of face muscles has been shown in upper limb amputees (Pascual-Leone et al., 1996; Lotze et al., 2001), we did not observe twitches in the face while stimulating over the ‘hotspot’ for eliciting phantom finger movements at higher stimulus intensities.
Mapping phantom movements

Fig. 3  Effect of the stimulation intensity on the perceived amplitude of phantom finger movements. When a given cortical site (in the presumed hand region of the motor cortex, indicated by the purple arrow) was repeatedly stimulated at different intensities, the perceived amplitude of movement increased accordingly, but the type of movement was unchanged (here a flexion of the fourth and fifth fingers). The close association between MEP amplitude and perceived amplitude of movement experienced during TMS might suggest that the sensation of phantom movement is solely determined by the afferent feedback from stump muscle. However, it is important to note that at different stimulation intensities the co-contraction level between biceps and triceps varied, but this did not alter the type of movement reported, which might have been expected if movement perception was being solely derived from afferent feedback. The mean (and standard deviation) are in millivolts for biceps and triceps MEPs. The perceived movement amplitude (and standard deviation) is reported on a numeric scale [0 (no movement) to 10 (maximal range of motion with intact limb)].

Discussion

Our results show that TMS applied over the presumed hand area of the motor cortex elicits sensations of movement in the phantom hand and that the amplitude of the movement perceived is positively correlated with the intensity of stimulation. The types of movements evoked were diverse and included movements at several different joints of the phantom limb. Notably, we also found that phantom limb movements that cannot be produced voluntarily by the subject can be elicited by TMS.

These results support the argument that even though there is considerable post-amputation reorganization within the motor cortex the representation of the amputated hand does not disappear. The observation that stimulation over the presumed hand area of the motor cortex simultaneously evoked phantom hand movements and stump muscle contractions suggests that cortical neurons that previously targeted motoneurons controlling the missing hand now target motoneurons in the remaining stump muscles. Evidence from animal studies suggests that this could occur either by deprived motoneurons previously supplying hand muscles re-innervating stump muscles or by cortical neurons that previously targeted motoneurons in the missing muscles re-targeting motoneurons in the remaining stump muscles (Wu and Kaas, 2000). An increased number of neurons targeting the remaining stump muscles might be one reason for the reduced threshold when stimulating these muscles by TMS compared with stimulation of the same muscles on the intact side.

Overlap of phantom movement and stump muscle representations

The co-occurrence of evoked phantom digit movements and stump muscle contractions raises the question of whether the perceived phantom movements actually resulted from stimulating the hand area or the proximal arm area of the motor cortex. Although we cannot answer this question directly, several observations suggest that this overlap does not exclude the possibility that phantom digit movements were evoked from the hand area of the motor cortex. The main reason for this is that we expected to see a large overlap between stump muscle representations and phantom digit representations, as it is well known that following amputation the motor representation of stump muscles expands into the hand region (Hall et al., 1990; Cohen et al., 1991; Fuhr et al., 1992; Kew et al., 1994; Ridding and Rothwell, 1995; Chen et al., 1998; Roricht et al., 1999; Irlbacher et al., 2002). Another reason is that we mapped phantom digit representations at an intensity determined by the stump muscle motor threshold, which probably corresponded to an above-threshold intensity for the mapping of digit representations and therefore an enlarged map of the phantom digits. For comparison, during mapping of the intact biceps, if we recorded a MEP in the biceps, a MEP in the intact FDS was always recorded. Thus, at the appropriate intensity for creating a biceps muscle map this map will always overlap the simultaneously acquired finger muscle map.

The observation of an overlap between phantom hand representations and stump muscle representations suggests that activity in these ‘hand’ cortical neurons continues to signal a movement of the missing limb, even though this same activity now produces contractions in stump muscles. A similar dissociation between the ‘anatomical’ and the ‘functional’ reorganization following amputation has been demonstrated in the sensory domain. Davis et al. (1998) recorded and stimulated through microelectrodes within the thalamus of amputees and demonstrated that thalamic neurons deprived of their afferent input acquired new receptive fields, but that when the same neurons were stimulated their activity was perceptually associated with the missing part of the limb that they had originally represented. Our results suggest that a similar phenomenon might occur in the motor domain, that is, that the activation of the hand area of the motor cortex continues to be associated with a hand movement despite the fact that the descending motor...
commands generated by this activation now result in stump muscle contractions.

**Role of efferent versus afferent information in phantom movement perception**

One could argue that the sensation of phantom limb movement is caused by the TMS activation of the hand representation in the adjacent sensory cortex, which might be particularly excitable as a consequence of the peripheral deafferentation. We think that this explanation is unlikely, since the sites of stimulation covered both the sensory and motor cortices, but phantom movement perceptions were exclusively found when stimulating the anterior bank of the central sulcus (see Figs 1 and 2). If the movement perceptions produced when stimulating over M1 resulted from the spread of current from M1 to the hyperexcitable S1 rather than from the direct activation of the motor cortex itself, then we would also expect to see evoked phantom sensations when stimulating directly over S1, but this was not the case; stimulation over S1 did not evoke phantom movement perceptions. The absence of evoked movement perceptions from sensory cortex stimulation is consistent with the results of a study of normal subjects in which stimulation intensities of 20 and 40% above motor threshold applied over parietal sensory structures did not evoke movement perceptions (Andre-Obadia et al., 1999). Thus, we believe that the sensation of phantom movement is related to activity in the motor representation of the missing limb.

Although we believe that activation of the motor representation of the missing limb is essential for the sensation of phantom movement, it is more difficult to assess the relative contributions of the efferent information resulting from this activation versus the afferent feedback that arises from contractions elicited in the stump muscles. On 86% of the trials on which phantom movement perceptions were evoked, these sensations were accompanied by muscular responses at the level of the stump. Thus, it is possible that the sensation of phantom movement arises from either the efferent information resulting from activation of the motor cortex or from a misinterpretation of afferent feedback. It has been reported that the electrical stimulation of severed nerves in long-term amputees can trigger proprioceptive sensations in the phantom limb (Dhillon et al., 2004, 2005), which would suggest that afferent feedback alone is sufficient to perceive movement of a phantom limb. These experiments differ from the present experiment, however, in that Dhillon et al. stimulated afferents in truncated nerves (by intraneural stimulation) that were previously devoted to the hand, while in our TMS experiment the afferent feedback always arose from the contraction of muscles that never moved any joints of the wrist or hand (such as biceps and triceps since all subjects were above-elbow amputees). We cannot completely rule out the possibility that stump muscle contractions might have elicited some activity in the severed nerves, since regenerated peripheral branches of severed sensory nerves might have innervated skin or muscle spindles surrounding the stump. There is some evidence of an increased number of terminal ramifications of the severed nerve in the dissected skin tissue adjacent to the stump following a digit amputation (Manger et al., 1996), but there is no evidence that such a process can take place following a much more proximal amputation, as the re-growth would have to occur much further from the site of the original terminal ramification. Furthermore, our observation that passive stump manipulations or voluntary stump contractions were never associated with the perception of movement in the phantom hand suggests that even if this re-innervation did occur, activity in regenerated peripheral branches of severed sensory nerves does not automatically lead to phantom movement perceptions. Finally, there is no evidence at this time that sensory information coming from proximal muscles can be re-interpreted as coming from the hand on the basis of afferent feedback alone. Indeed, our results show that a contraction of a given amplitude within one muscle is sometimes interpreted as coming from the stump, and other times is attributed to movements of the phantom digits. We also observed that very similar patterns of MEPs were sometimes associated with very different movement perceptions and vice versa. Thus, on the basis of our results it would be difficult to argue that the phantom movement perceptions arise only from afferent feedback generated by the contraction of stump muscles.

If afferent feedback alone is not sufficient to generate phantom movement perceptions, then can these sensations result from efferent activity? Ellaway et al. (2004) investigated whether movement perceptions evoked by TMS in normal subjects are generated centrally or arise from sensory feedback. They used TMS to evoke movements on one side of the body and direct electrical stimulation of muscles to evoke movements on the other side. They reported that the TMS-evoked movement was felt on average 20 ms after the electrically evoked movement, which does not support the idea that the TMS-elicited movement perceptions arose solely from centrally generated activity (Ellaway et al., 2004). Another argument against a purely central effect is the observation that deafferentation of the stump region induced by ischaemia leads to the perception that the phantom limb is still present but is frozen, thus supporting a role of afferent feedback in the perception of phantom limb movement (Reilly et al., 2006). On the basis of these observations we acknowledge that it is unlikely that the TMS-induced sensation of phantom limb movement arises exclusively from efferent information generated by motor cortex activation. Instead, we propose that when amputees voluntarily move their phantom limb, the interpretation of the afferent information arising from the movement-related contraction of stump muscles is modulated by the efferent motor command. That is, if the motor command arises
from the original biceps area, for example, the afferent feedback resulting from biceps contraction will be interpreted as a stump movement, while if the motor command arises from the original hand area, the same biceps contraction will now be interpreted as a phantom hand movement. This view, in which efferent information from the motor cortex is essential for the perception of voluntary control over phantom movements, is consistent with the emerging view that M1 plays a role in movement perception (Naito, 2004).

Awakening of voluntarily inaccessible phantom movement representations

The hypothesis that efferent information plays a role in the interpretation of afferent information can account for the fact that some amputees are unable to produce some phantom movements voluntarily even though they can produce various contraction patterns in their stump muscles. On the basis of our hypothesis, when these subjects attempt to make a certain phantom movement they contract their stump muscles but they do not do so by recruiting cortical neurons within the hand area of the motor cortex, and as such, the motor command that is generated is not interpreted as a ‘hand’ movement command. In contrast, TMS stimulation over the presumed hand area also results in stump muscle contractions, but does so artificially via recruitment of ‘hand’ cortical neurons that now have descending projections to stump muscles. This can explain why some movement perceptions were evoked by TMS even when the amputee could no longer perform the same movement voluntarily.

The demonstration that even in the case of phantom joints that cannot be moved voluntarily a latent representation seems to be preserved and can be re-awakened through artificial stimulation suggests that the loss of control over phantom movements is not due to the disappearance of the cortical representation of those movements but rather due to a problem with voluntary access to this representation. This could explain why vision of a virtual hand moving in place of the missing one can often restore (sometimes almost instantaneously) the sensation of being able to produce previously impossible phantom movements (Ramachandran and Rogers-Ramachandran, 1996; Brodie et al., 2003; Giraux and Sirigu, 2003; MacLachlan et al., 2004), and why repeated exposure to such feedback leads to an increased activation in the motor cortex during phantom movements (Giraux and Sirigu, 2003).

Conclusion

The general belief about cortical reorganization in the primary motor cortex following amputation has been that the elbow invades the hand representation and takes control of its cortical territory. Our data do not support this hypothesis, and rather suggest that the hand representation remains even though neurons in this area select other muscular groups to express phantom hand movements. In conclusion, we propose that hand movement representations are preserved and remain at their original location in the motor cortex of amputees, and that the major plastic changes that occur following amputation might take place downstream, at the level of spinal motoneurons.

References


