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Phantom Sensations: What's a Brain to Do? A Critical Review of the Re-mapping Hypothesis

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Biography

I am doctoral candidate in Philosophy at Tulane University, and will be defending my dissertation Spring 2018. I work in the areas of Early Modern Philosophy, Philosophy of Mind, and Neuroscience, and have a particular interest in exploring the connections between the history of philosophy and contemporary neuroscience.

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Phantom Sensations: What's a Brain to Do? A Critical Review of the Re-mapping Hypothesis

Daniel J DeFranco

Abstract

Here, I will review the most widely held account of phantom sensations and the effects of deafferentation in the somatosensory cortex, namely, the "re-mapping hypothesis." According to the re-mapping hypothesis, deafferentation is followed by significant neural reorganization that eliminates the neural structures that give rise to phantom sensations, restoring the alignment between the brain's representation of the body and the actual condition of the body. Implicit in the re-mapping hypothesis is the view that the brain's primary function is the accurate representation of the body. In response to the remapping hypothesis, I propose an alternative theory, which I have dubbed the "preservation hypothesis." The preservation hypothesis argues that the primary function of the brain is to preserve the entirety of the brain's structures and functional capacities. Thus, upon deafferentation, the brain does not eliminate phantom sensations and restore an accurate representation of the body but takes steps to preserve the neural structures underlying phantom sensations, with the effect of maintaining phantom sensations long term. While the effects of deafferentation are certainly an empirical matter, assessing our views on the subject discloses our deeply held assumptions regarding the primary function of the brain: does the brain operate such that it will do all that it can to represent the body accurately? Does the brain have certain limitations in its accurate representation of the body? Or, does the brain care nothing for reality and the accurate representation of the body, operating with the sole purpose of preserving its structure and functional capacities in their entirety? I hope to make some progress in answering these questions.

Keywords

Phantom Sensations, Brain Function, Remapping, Preservation

Whenever I tell someone that I am researching phantom limbs,¹ I often get the same question in response, "why doesn't the brain know that the limb is no longer there?" While the question may be posed by a novice, implicit in the question is a sophisticated understanding of the function of the brain. To ask, "why doesn't the brain know that the limb is no longer there," implies that the brain's production of an experience of the body that does not align with the actual condition of the body constitutes a failing on the part of the brain. Interestingly, it is not just laymen who hold this view; neuroscientists

1. The phantom limb, the lingering feeling that one's amputated limb is still present, is a condition experienced by 98% of amputees, 60-80% of whom experience some degree of pain associated with the phantom (Ramachandran and Hirstein, 1998; Sherman *et al.* 1984).

also inquire, “what happens in the brain upon deafferentation?”² That is, does anything happen in the brain after amputation that might correct the subsequent discordance between the experience and actual condition of the body?

In what follows, I will critically evaluate both current explanations for phantom sensations and the conceptions of the brain to which these explanations are implicitly committed. I will begin with a review of the most widely-held account of phantom sensations, the “re-mapping hypothesis.” Implicit in the re-mapping hypothesis is the belief that the mechanics of the brain work to represent the body accurately, and, upon amputation, significant neural reorganization is initiated to eliminate the causes of phantom sensations and restore the alignment between experience and actual condition of the body. I will assess this hypothesis, raising issues with it, and ultimately proposing an alternative account, which I have dubbed the “preservation hypothesis.” The preservation hypothesis posits that the brain does not attempt to eliminate phantom sensations and accurately represent the body but takes steps to preserve the neural structures underlying phantom sensations, with the effect of the long-term maintenance of phantom sensations.

While the causes of phantom sensations and the effects of deafferentation are certainly empirical matters, assessing our views on the subject discloses our deeply held assumptions regarding the ultimate function of the brain: does the brain operate such that it will do all that it can to represent the body accurately? Does the brain have certain functional limitations in the accurate representation of the body? Or, does the brain care nothing for reality and the accurate representation of the body, operating with the sole purpose of preserving its structures and functional capacities in their entirety? In this essay, I hope to approach answers to these questions.

I. The Initial Appearance of Phantom Sensations

With the aid of modern science, the initial appearance of phantom sensations is well understood. In the parietal lobe, exists an area of the brain called the somatosensory cortex (the primary sensory cortex), which is responsible for mapping sensations of the most peripheral parts of our body (i.e., the sensations of the skin, joints, and muscles). Whenever we touch something, or something touches a part of our body, afferent signals via the peripheral nervous system are sent to the part of the somatosensory cortex that corresponds with that limb, those afferent signals are mapped and we experience a

2. Deafferentation is a disruption of afferent neural connections between the peripheral body and brain. In this piece, I will be considering deafferentation that results from the amputation of a limb.

sensation in the limb. If the nerves connecting a body part to the brain were disconnected or destroyed, or if a body part were amputated (i.e., deafferented), the part of the somatosensory cortex representing that part of the brain would continue to exist, at least for a time, despite not receiving any direct afferent signals from the body. Additionally, the deafferented cortical area continues to receive efferent signals from the motor cortex, signals concerning executive movements of the body. The continued existence of a deafferented cortex's neural structure, in conjunction with its continued stimulation by efferent signals from the motor cortex gives rise to phantom sensations and explains the initial appearance of the phantom limb. The arrival of efferent signals from the motor cortex also explains why so many amputees claim that they can move their phantom.³

Many mysteries continue to surround phantom sensations, and one of these mysteries proves almost as unusual as the case of the phantom limb itself. In 1991, Pons *et al.* made an incredible discovery while recording neuronal activity in the primary somatosensory cortex of four adult macaque monkeys. The macaques "had received deafferentations of an upper limb, three unilateral and one bilateral," by way of dorsal rhizotomy, "more than 12 years before the recording session" (Pons *et al.*, 1857).⁴ Pons noticed that when the faces of his monkey subjects were touched, the effect in the brain was such that the corresponding face area of the somatosensory cortex was activated (which was expected), but so was the area that represented the deafferented limb (Pons *et al.* 1991). Touching the ipsilateral face of a monkey with unilateral arm deafferentation excites the area of the somatosensory cortex that represents the face as well as the area that represents the arm. According to Pons *et al.* "[V]irtually identical findings were obtained in the three other animals," and just "a slight deflection of facial hairs was sufficient to obtain a vigorous neuronal response" in the deafferented zone (Ibid, 1859).

Prior to Pons' discovery, all attempts at researching phantom sensations faced the seemingly insurmountable obstacle of determining how to research a phenomenon

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3. Studies by Lotze *et al.* 1999, Raffin, E. *et al.* 2012, and Makin *et al.* 2013 highlight amputees' motor control over their phantom limbs, many of whom can execute fine motor skills even 53 years after amputation. Motor skills executed by amputees include, but are not limited to, (1) elbow flexion/extension (for above-elbow amputees only); (2) wrist flexion/extension; (3) hand closing/ opening; (4) thumb to index opposition; (5) finger abduction/ adduction (Raffin, E. *et al.* 2012, 748).
 4. Pons' study on the "Silver Spring monkeys" was the focus of one of the most public animal abuse cases in the history of the United States, and was the very first animal research case to reach the Supreme Court. Since Pons, phantom limb research has overwhelmingly utilized non-invasive and humane procedures on human patients. This change is in part due to the pioneering work and nuanced approaches to the study of phantom sensations by V.S. Ramachandran.

that had no physical correlate on the peripheral body. Pons *et al.*'s discovery (that the stimulation of intact body parts can excite a deafferented area in the somatosensory cortex) opened the possibility of studying phantom sensations through intact limbs albeit not the limb that corresponds to the phantom sensation. The discovery also raised a multitude of questions that researchers, thanks to Pons *et al.*, now had a means of empirically investigating. For instance, what was being observed in the co-activation of the face and hand cortices when the ipsilateral face of a monkey with unilateral hand deafferentation was stimulated? Additionally, what were the perceptual correlates of this experiment, that is, what were the monkeys feeling when their faces were touched?

Unfortunately, Pons could not ask the monkeys what they felt when their faces were touched, but, the following year, V.S. Ramachandran conducted an experiment with human unilateral hand amputees in which he could ask his patients what they felt when their faces were stimulated. Results showed that the perceptual correlates of this cortical co-activation are the simultaneous experience of sensations on the face and phantom hand (Ramachandran *et al.* 1992). These sensations are: (1) modality specific (i.e., if cold water is dripped on the face, then the patient will feel cold water being dripped on the phantom hand); and, (2) topographically organized (i.e., a point-by-point map of the surface of the hand appears on the face) (Ramachandran *et al.* 1992). Ramachandran's MEG scans of the somatosensory cortex confirm Pons *et al.*'s findings; when the ipsilateral face area of the somatosensory cortex is stimulated, both the face area and large sections of the deafferented hand area are simultaneously stimulated in the brain.

But what could explain the cortical co-activation that both Pons and Ramachandran were observing? As a first step in explaining this phenomenon, Ramachandran notes that the face area neighbors the hand area in the somatosensory cortex. And while the proximity of these two areas does not fully explain observed neural activity nor its perceptual correlates, it offers some insight into why touching the ipsilateral face of a unilateral hand amputee could simultaneously produce sensations in the face and the phantom hand. Unilateral hand amputees can also experience sensations in their phantom limbs when the arm area most proximal to the amputation line is stimulated, which is an area that also neighbors the hand area in the somatosensory cortex (Ramachandran *et al.* 1992). Additionally, stimulation of the contralateral intact hand also elicits sensations in the deafferented hand area; here we are observing "cross-callosal" connections as opposed to intra-hemispheric connections between neighboring areas of the somatosensory cortex (Giummarra *et al.* 2007).

II. The Remapping Hypothesis

As an explanation of the co-activation of intact and deafferented sensory areas, Ramachandran formulates the “Remapping Hypothesis,” which remains to this day the most widely accepted account of cortical co-activation. The remapping hypothesis proposes the following:

1. Upon deafferentation, a cortical area becomes vacant or empty, for no direct sensory input is arriving to this part of the brain from the peripheral body.
2. Neighboring cortical areas sense the vacancy in the deafferented cortex, and actively invade with the effect of reorganizing the deafferented cortical area such that its structure and functional capacities become continuous with the intact cortical area.

As for the mechanism that facilitates this cortical reorganization, Ramachandran offers the following two possibilities:

1. The invasion of a deafferented cortical area occurs when a neighboring cortical area “sprouts thousands of neural tendrils that creep over into” the deafferented area (Ramachandran 2011, 28).
2. Preexisting neural connections exist between neighboring cortical areas, which are “masked” or “inhibited” under normal conditions (i.e., when a cortex is receiving afferent signals from the peripheral body) (Ramachandran *et al.* 1992, 1160; Ramachandran 2011, 28). Upon deafferentation, a cortical area is no longer able to inhibit signals from these pre-existing neural connections, resulting in an invasion of afferent signals from neighboring cortical areas.⁵

5. Ramachandran explains, “Thus even in healthy normal adult brains there might be sensory inputs from the face to the brain’s face map *and* to the hand map area as well. If so, we must assume that this occult or hidden input is ordinarily inhibited by the sensory fibers arriving from the real hand. But when the hand is removed, this silent input originating from the skin on the face is unmasked and allowed to express itself so that touching the face now activates the hand area and leads to sensations in the phantom hand” (Ramachandran 1998, 34).

Considering that phantom sensations can develop quite quickly post-amputation,⁶ Ramachandran suggests that the *initial* work of cortical reorganization is most likely “a result of the unmasking of ‘silent’ synapses, rather than of anatomical changes, such as ‘sprouting’” (Ramachandran *et al.* 1992, 1160). However, the two mechanisms of cortical reorganization are not mutually exclusive. Ramachandran explains that there is presently “no way...of easily distinguishing between these two theories, although my hunch is that both mechanisms are at work.” (Ramachandran 1998, 35). Thus, cortical reorganization could very well be initiated by the unmasking of pre-existing synaptic connections, while the long-term process of reorganization involves the sprouting of new neural connections.

Ramachandran also surmises that this process is evolutionarily “beneficial to the organism” (Ramachandran 2000, 319). For one, the process restores the alignment between the experience of the body and the actual condition of the body by eliminating the neural structures that give rise to phantom sensations. Ramachandran views the restoration of the alignment between experience and the condition of the body as the mechanism’s primary function. In addition to restoring the discordance between experience and actual condition of the body, cortical reorganization prevents cortical degeneration. Without sufficient afferent stimulation, a cortical area would degenerate and ultimately become a rotted-out bit of brain. The neural mechanism at work post-deafferentation prevents this degeneration; an intact cortical area appropriates a deafferented area and includes it in its own representational activities, guaranteeing continued afferent stimulation and thus preventing neural degeneration in the somatosensory cortex (Ramachandran 2000, 319).

Following the appropriation of a deafferented cortex by an intact cortex, Ramachandran suggests that we can reasonably anticipate an increase in that intact cortex’s representational and functional powers (Ramachandran 2000, 319). An intact cortex would come to possess a greater area of representation in the somatosensory cortex, and there ought to be measurable and perceptual correlates to this change. Specifically, Ramachandran anticipates “tactile hyperacuity” both in the area most proximal to the amputation line and in any body parts whose area of correspondence in the brain neighbors the deafferented area.

6. Patients can experience vivid phantom sensations immediately following the amputation of a limb, such that “[S]ome patients wake up from anesthesia and are incredulous when told that their arm had to be sacrificed, because they still vividly *feel* its presence” (Ramachandran 1998, 22).

Beyond its ability to explain cortical co-activation, the remapping hypothesis proves fascinating, insofar as it implicitly contains a vision for the primary function, the telos, of the brain. According to Ramachandran, deafferentation initiates a mechanism in the somatosensory cortex by which intact cortical areas invade and reorganize a deafferented cortex with the effect that the deafferented cortex is completely appropriated into intact areas. The perceptual effect of this mechanism is that phantom sensations, over time, decrease in their vividness and eventual vanish completely, restoring the alignment between one's experience of the body and the actual condition of the body. And if this mechanism is indicative of an overall function of the brain, then it seems clear that the brain's utmost concern is producing an accurate representation of the body. Should something challenge the brain's ability to accurately represent the body, such as deafferentation, the brain initiates a mechanism by which it corrects the discrepancy. In the case of phantom sensations, the brain must literally destroy a part of its structure and functional capacities in order to represent the body as it actually is.

The principle question asked by neuroscientists with respect to phantom sensations is, "what are the effects of deafferentation in the brain" (i.e., what happens in the brain after a limb is amputated?). This question proves to be no more than a sophisticated form of the same question posed by non-experts on learning of phantom sensations, which is, "why doesn't the brain know that the limb is no longer there?" The intuitions of experts and laypeople are aligned when it comes to phantom sensations; any condition in which the experience of the body is in obvious discord with the actual condition of the body is an intolerable one. Either the brain must do something to correct this condition or the brain has hit a functional limit in its ability to accurately represent the body. With respect to phantom sensations, the remapping hypothesis takes the position that the brain can come to know that the limb is no longer present, and initiates a mechanism of neural reorganization to eliminate the neural structures that give rise to phantom sensations, restoring the alignment between the experience and condition of the body. Guiding this hypothesis, and the question posed by experts and laypeople alike, is the intuition that the primary function of the brain is to accurately represent the condition of the body. If the brain fails to accurately represent the body, as in the case of phantom sensations, then the brain must do something to correct the situation or the error signals an instance in which the brain has been stumped (i.e., has hit a functional limit in its ability to accurately represent the body). Regardless of what view is taken, both views are equally guided by the assumption that brain's primary function is the accurate representation of the body. Any discordance that arises between the experience and actual condition of the body must be corrected or constitutes a failure of the brain.

III. A Cartesian Interlude

Nearly 400 years ago, another novel scientist took up the challenge of explaining phantom sensations. This scientist determined that sensations must be occurring in the brain, not the body, and that the continued existence of structures in the brain that directly correspond to the peripheral body support the emergence of phantom sensations; “each tiny tube on the inside surface of the brain corresponds to a bodily part” and “each point on the surface of gland H [the pineal gland] corresponds to a direction in which these parts can be turned” (Descartes *The World*, 154). And just as Ramachandran’s explanation of phantom sensations contains within it a vision of the primary function of the brain, this scientist also understood phantom sensations as offering insight into the function of the brain, a window into the teleology of mind. His name: Descartes.

Phantom sensations are often viewed as the exclusive domain of contemporary science, and while some researchers have explored the field’s early modern origins,⁷ most insights remain in the annals of history, failing to transcend into the contemporary scientific discussion. Here, I would like to indulge in an experiment. I will review Descartes’ treatment of phantom sensations, not as a documentation of the past, but as inspiration for assessing the state of phantom limb research as it stands today.

In Meditation 6 of *Meditations on First Philosophy*, Descartes explains the phenomenon of phantom sensations as follows,

...many experiences gradually weakened any faith that I had in the senses...And not just the external senses, but the internal senses as well. For what could be more intimate than pain? But I had heard it said by people whose leg or arm had been amputated that it seemed to them that they still occasionally sensed pain in the very limb they had lost. Thus, even in my own case it did not seem to be entirely certain that some bodily member was causing me pain, even though I did sense pain in it (Descartes *Meditations*, 95).⁸

7. Finger and Hustwit offer an extensive account of the historical development of phantom limb research in their article “Five Early Accounts of Phantom Limb in Context: Pare, Descartes, Lemos, Bell, and Mitchell” (Finger and Hustwit 2003).

8. In addition to Meditation 6 of the *Meditations on First Philosophy*, Descartes discusses phantom sensations on at least two other occasions; in a private letter to Fromondus (1637) and in Part IV Article 196 of *Principles of Philosophy* (1644).

Phantom sensations are treated by Descartes on two different levels of understanding. The first level, the common-sense level of understanding, treats phantom sensations with respect to how they initially strike us as a failing of the body/brain. Phantom sensations lead one to doubt the reliability of the senses, because, in losing a limb, the senses fail to represent the condition of the body accurately. Phantom sensations are to feel what is not there and to know that which is false. This common-sense level of understanding understands phantom sensations themselves to be the error committed by the body/brain, an error in the accurate representation of the body.

The second level, the higher level of understanding, emerges from further reflection on the exact nature of the error to which phantom sensations alert us. As Descartes notes, phantom sensations raise the possibility that “even in my own case it did not seem to be entirely certain that some bodily member was causing me pain, even though I did sense pain there” (Ibid). Phantom sensations led Descartes to reconsider the location in the body/brain at which sensations are produced. Sensations are experienced as occurring in the body, but if sensations continue to be perceived in a limb even after that limb has been destroyed, then clearly the limb itself cannot be the locus of sensation. And if all sensations are experienced as occurring in the body, despite not actually occurring in the body, then the nature of sensations generally would be deceptive.

Descartes ultimately determines that sensations occur not in the body, but in the brain. Descartes explains,

when nerves in the foot are agitated in a violent and unusual manner, this motion of theirs extends through the marrow of the spine to the inner reaches of the brain, where it gives the mind the sign to sense something, namely, the pain as if it is occurring in the foot (Ibid, 102).

Movements in the peripheral body travel up into the interior of the brain via nerves and the “marrow of the spine,” but it is the movement in the brain that “gives the mind the sign to sense something.” And there seems nothing inherently strange about sensations occurring in an organism’s brain as opposed to its body.

It is unnerving, however, that the sensations the brain produces are experienced as occurring in the body as opposed to being experienced in their true location of occurrence in the brain. As Descartes notes,

...the nature of man could have been so constituted by God that this same motion in the brain might have indicated something else to the mind: for example, either the motion itself as it occurs in the brain, or

in the foot, or in some place in between, or something entirely different (Ibid).

The mechanics of the brain need not have been organized such that the production of sensation be experienced by an agent as occurring in that agent's body. Sensation could have been experienced, Descartes posits, as: (1) directly occurring in the brain; (2) as the actual motions in the body (i.e., violent agitation, as opposed to the feeling of pain); (3) as traveling through the nerves or up the spinal cord; or, (4) something entirely different. All these alternative possibilities posit ways in which sensations could have provided objective insights into the nature of sensation and its actual process of production. However, sensation, as it is, communicates something false about the nature of sensation; sensation is produced in the brain but is deceptively experienced as occurring in the peripheral body.

At a higher level of understanding, we see that phantom sensations do not independently constitute an error of sensation; rather, they alert us to an error that concerns the status of all our sensations. Sensations generally, insofar as they occur in the brain but are experienced in the body, fail to accurately communicate to us the actual happenings of the body. And this illusion, that sensations occur in the body when they in fact occur in the brain, is a direct product of the mechanics of the brain. We are no longer dealing with an error, but a failing, an infirmity of the brain itself. It was assumed that the brain's job is to accurately represent the body and the external world, but we find that the mechanics of the brain produce sensations that are deceptive with respect to sensation's actual nature and its true location of production. Sensation, as a potential mechanism for discerning the objective properties of the external world, is corrupt at its core.

Speaking for myself, I am rather grateful to my brain for failing to represent sensation as occurring at its true location of inception, and I cannot begin to imagine what it would be like to experience all the sensations we associate with our bodies as being experienced in the brain. For those of you who also intuitively grasp the benefit of experiencing sensations as occurring in our extended body, one may begin to see how Descartes has rather cheekily demonstrated that neither sensation nor, consequently, the mechanics of the brain are particularly interested in accurately representing the body and the happenings of the body. While the nature of man could have certainly been constituted differently, "nothing else would have served so well the maintenance of the body" than its present arrangement (Ibid). Thereby, for Descartes, the brain's disinterest in representing the body accurately is to our benefit; it is far better for our preservation

that we experience ourselves as a unified embodied being, and not as a being within a being (i.e., a smaller organism operating out of the head of a larger organism).

In reviewing the nature of sensation, Descartes uncovers two critical insights. Firstly, the nature of sensation as inherently deceptive provides grounds by which Descartes can reject his previously held assumption that the function of the body/brain is the accurate representation of the body. Secondly, Descartes recognizes that, although sensations are deceptive, their nature is such that “nothing else would have served so well the maintenance of the body” (Ibid, 102). Thereby, the true function of the body/brain, that which all the activities of the body/brain are directed towards, must be “the welfare of the body” (Ibid, 103).

There is one last hurdle Descartes must overcome in positing this new function for the body/brain; phantom sensations themselves. It is quite possible that phantom sensations constitute a moment in which the mechanics of the brain have failed to secure what is necessary to preserve the body/brain. It is to our benefit that the mechanics of the brain produce the experience of sensation as occurring in our body, but it is a burden that these same mechanics maintain the experience of a limb even after that limb has been destroyed. According to Descartes, “the nature of man...cannot help being sometimes mistaken,” but “I know that all the senses set forth what is true more frequently than what is false regarding what concerns the welfare of the body” (Ibid, 102–3). While Descartes does not muse much over how phantom sensations could themselves be beneficial to the organism, he does believe that, regardless of whether phantom sensations are a benefit or a burden, the burden is absolutely worth the benefit.⁹ That is: (1) to experience sensations as occurring in the body as opposed to in the brain; and, (2) to have the structural and functional capacities of the body enshrined in the brain such that sensations of the body arise in the brain, is far more beneficial for the welfare of the human than the burden posed by phantom sensations.

The burden, phantom sensations, does offer a conciliation prize, for a reflection upon phantom sensations aid us in further fleshing out the primary function of the brain and mind; the maintenance or preservation of the organism (Ibid 102-3). According to Descartes, “I can think of no better arrangement” than the current mechanics of the

9. Giummarra *et al.* argue that the preservation, and not the reorganization, of a deafferented cortex is necessary for the well-being of the individual. For instance, the successful operation of a prosthesis depends upon that prosthesis having a “neural template” in the somatosensory cortex. Without the preservation of a deafferented cortex, it would be impossible for a patient to operate or even recognize a prosthesis as an extension of his or her body (Giummarra *et al.* 2007, 223).

brain and the kind of sensations that it produces, for the sensation that it does produce, “of all the ones it is able to produce, is most especially and most often conducive to the maintenance of a healthy man” (Ibid, 102). Descartes is unequivocal in “Meditation 6” that the aim of brain mechanics is to produce a condition that best enables the human to maintain its health and preserve itself. And phantom sensations demonstrate a facet of the brain’s function. Specifically, phantom sensations show us that the effort to preserve extends to the brain itself, where the brain preserves its structure and functional capacities even if a part of the body is destroyed. And the fact that the brain maintains its structure and functional capacities in spite of the actual condition of the body, demonstrates to Descartes that the function of preservation is not aligned with the function of accurate representation of the body and external world. The true function, or telos, of the mechanics of the brain is the maintenance and preservation of the entirety of the organism.¹⁰

At a common-sense level of understanding, phantom sensations themselves represent a failing of the brain to produce an experience of the body that accurately represents the body’s present condition. At a higher level of understanding, we see that this failing is no failing at all, but an indication that our initial conception of the function of the brain is false. The function of the brain is the preservation of the organism’s structure and functional capacities, and this function involves an indifference to the accurate representation of the condition of the body and the objective nature of the external world. Sensations are experienced as occurring in the body, despite originating in the mind, because this kind of sensation is most beneficial to the maintenance of the human organism. And while phantom sensations may be a burden to an amputee, the brain’s preservation of its ability to represent the structure and functional capacities of a limb, even after that limb has been destroyed, offers a privileged glimpse into what follows from the brain’s fulfilling its function of maintenance and preservation.

Returning to our contemporary study of phantom sensations, we see that Ramachandran certainly appreciates the common-sense understanding of phantom sensations, but perhaps has not grasped the higher sense understanding. Ramachandran recognizes that the phenomenon of phantom sensations constitutes a disconnect between our experience of the body and the actual condition of the body, but simultaneously maintains that the brain has a mechanism by which it can correct this disconnect and

10. In his book *Cartesian Metaphysics and the Whole Nature of Man*, Richard Hassing reiterates the point that “Descartes makes clear that the soul-body composite has a natural teleology: its natural end is the health and conservation on the body” (Hassing 2015, 57).

realign our experience of the body with the body's physical condition. By way of this mechanism, the brain can fulfill its function of accurately representing the body.

Ramachandran's diagnosis and suggested remedy of the problem, however, fails to recognize that the original sin is not phantom sensations themselves, but that sensations generally are deceptive; sensations occur in the brain but are experienced as occurring in the body. While this phenomenal aspect of sensations might be incredibly beneficial to the organism, it contradicts our account of the brain as working toward providing an accurate representation of our body and the external world. The brain does not only just fail to accurately represent the condition of the body post-amputation but it also fails to do so with respect to all sensations.

The illusion that Ramachandran simultaneously falls prey to and maintains is that there ever was a point in time at which the mechanism of the brain produced sensations that accurately communicated the condition of the body. Sensations, at their core, are deceptive, insofar as they do not accurately communicate the process of their production and the location of their inception, and thus have never satisfied the function of accurately representing the condition of the body. And, if that is the case, then it would be odd that, following deafferentation and the emergence of phantom sensations, the brain would suddenly take up an interest with the accurate representation of the body and initiate a rather dramatic process of neural reorganization such that the experience of the body is realigned with the actual condition of the body.

IV. The Preservation Hypothesis

The intuition that overwhelmingly guides the remapping hypothesis is that the primary function of the brain is the accurate representation of the body. Descartes, however, makes a persuasive case that sensations have never constituted an accurate representation of the body, and, therefore, it would be misguided to interpret neural changes following deafferentation as indicative of a restoration of accurate representation. How can something that never was be restored? This suggests that the remapping hypothesis could be correct in its observations of phantom sensations, but misguided in its assessment of the significance of these observations. Descartes' point warrants a re-examination of the evidence that ostensibly substantiates the remapping hypothesis.

The remapping hypothesis, in large part, depends upon evidence of cortical co-activation (i.e., the stimulation of a deafferented cortex by way of afferent signals overflowing from a neighboring intact cortex). Traditionally, cortical co-activation has been understood to indicate dramatic cortical reorganization, but it is critical to point out

that we do not literally see cortical re-organization in neural scans. Neural scans indicate where in the brain cortical activity is occurring, but do not show structural changes. In amputees, scans show activity in a deafferented cortex when there is activity in a neighboring intact cortex. Ramachandran takes this co-activation to indicate that an intact cortex has initiated an invasion of a deafferented cortex, such that the deafferented cortex is being reorganized and will become continuous with the intact cortex. But certainly, this cannot be the only possible explanation of cortical co-activation. It's possible that the co-activation of a deafferented and intact cortex has no significant effect at all. It is also possible that the co-activation of a deafferented and intact cortex serves to preserve the structure and functional capacities of the deafferented cortex.

Without afferent stimuli a cortex will degenerate, but the observed phenomenon of cortical co-activation indicates a way in which a cortex could continue to receive necessary afferent stimulation and avoid degeneration post-deafferentation. However, it is unclear if the origin of afferent signals affect the effects these signals can have on a deafferented area. That is, must the afferent stimuli arriving at a cortex originate from that cortex's corresponding limb to contribute to its preservation? If the answer is yes, then it seems doubtful that the effect of cortical co-activation is the preservation of a deafferented cortex. But if the answer is no, that afferent signals can stimulate a cortex regardless of their location of origin, then the afferent stimuli of diverse origins could very well serve to preserve the structure and functional capacities of a deafferented cortex.

Fortunately, progress has been made in answering the above question. In an extensive review of the phantom limb literature, Giummarra *et al.* argue that afferent stimuli continue to arrive at a deafferented limb from a variety of sources and that this continued afferent stimuli has the effect of preserving the neural structure of the deafferented area, giving rise to a "normal (non-painful)" phantom limb (Giummarra *et al.* 2007, 228). A deafferented cortex's sources of afferent stimulation include: (1) "the residual limb and stump;" (2) sensations arriving from the intact contralateral limb via "cross-callosal pathways;" (3) "activation of mirror neurons" from watching others move their intact limbs; and, (4) visual feedback from the use of a prosthetic (Ibid, 224; 226; 225; 223). Giummarra *et al.* argue that the arrival of afferent stimuli from these diverse sources counteract cortical reorganization initiated by neighboring intact cortices.¹¹ Thus,

11. This position is also echoed by Lotze *et al.*, who argue that "frequent and extensive use of a myoelectric prosthesis is correlated negatively with cortical reorganization and phantom limb pain and positively with the reduction in phantom limb pain over time. This suggests that the ongoing stimulation, muscular training of the stump and visual feedback from the prosthesis might have a beneficial effect on both

afferent stimuli arriving at a deafferented cortex can aid in preserving the structure and functional capacities of that cortex despite not originating from the peripheral limb to which the cortex corresponds.

Now, if afferent signals coming from other sources have the effect of stabilizing the structure and functional capacities of a deafferented cortex, why must afferent signals originating in neighboring intact cortices destabilize the structure of a deafferented cortex, as suggested by the remapping hypothesis? It is certainly possible that Ramachandran is correct, that afferent signals from an intact cortex have the effect of reorganizing a deafferented cortex, but such a position requires further explanation; we must know what accounts for the different effects had by afferent stimuli in a deafferented cortex.

There is, of course, the possibility that afferent stimuli arriving from intact cortices serve to preserve a neighboring deafferented cortex. And, in fact, the findings of Giummarra *et al.* substantiate the view that cortical co-activation constitutes a mechanism by which the structure and functional capacities of a deafferented cortex are preserved long-term. I call this alternative account of cortical co-activation the “preservation hypothesis,” and I summarize it as follows:

1. Upon deafferentation, the brain’s normal mode of operation is maintained, and the effect is the preservation of the structure and functional capacities of the deafferented area.
2. Preservation is accomplished by an overflow of afferent signals from intact cortical areas arriving at and stimulating the deafferented area.
3. The arrival of these afferent signals is made possible by pre-existing neural connections that exist between the deafferented area and many other cortical areas.
4. The flow of afferent signals does not represent a significant change in cortical organization or operation, but reveals the normal functioning and communication between brain regions.¹²

cortical reorganization and phantom limb pain” (Lotze *et al.* 1999, 502).

12. Afferent signals normally overflow into other cortical areas, but usually are inhibited by afferent signals coming from intact peripheral limbs (Ramachandran 1998, 34).

The preservation hypothesis proposes that no significant cortical reorganization occurs in a cortical area following deafferentation. Rather, the brain's structures and modes of operation facilitate the stimulation of a deafferented area with afferent stimuli from intact cortical areas, the effect of which is that the deafferented area receives stimuli aiding it in preserving its overall structure and functional capacities. In this view, a cortical area would have many pre-existing neural connections between itself and a variety of other cortices, receiving continuous afferent stimulation. Under normal operating conditions, these neural signals are inhibited by afferent signals arriving directly from the peripheral body. When the arrival of afferent signals directly from the peripheral body desists following deafferentation, afferent signals coming from other cortices – signals that have been arriving the entire time but until this point have failed to excite the intact cortical area – now successfully excite the deafferented area. The arrival of these afferent signals serve to stimulate the deafferented cortex with the effect of preserving the deafferented cortex's structure and functional capacities. This is not to say that afferent signals coming from other cortical areas are sufficient for maintaining the structure and functional capacities of the deafferented cortex, but that this is the end that the afferent signals serve.

Note that the preservation hypothesis utilizes a great deal of Ramachandran's initial insights regarding phantom sensations and the effects of deafferentation. The point of disagreement concerns the effect of overflow afferent signals on a deafferented cortex. Ramachandran maintains that the effect of overflow afferent signals is the restructuring of a deafferented area, such that its structure becomes continuous with a neighboring intact cortical area. The preservation hypothesis proposes that overflow afferent stimuli contribute to the structural and functional preservation of a deafferented cortex.

In contrast to the remapping hypothesis, the preservation hypothesis maintains that a primary function of the brain is the preservation of the brain's structures and functional capacities, even if this conflicts with accurately representing the condition of the body. Following deafferentation, the brain engages in no activity that would eliminate phantom sensations. On the contrary, activities in the brain would work to preserve the neural structures giving rise to phantom sensations, thus maintaining the structures and functional capacities of the brain regardless of whether the consequence is an inaccurate experience of the body.

V. Remapping vs. Preservation: An Empirical Comparison

The preservation hypothesis proposes that cortical co-activation alerts us to a mechanism by which the brain continues to supply a deafferented cortex with afferent stimulation, contributing to that cortex's long-term structural and functional preservation. This is in direct opposition to the remapping hypothesis, which proposes that the mechanism indicative of cortical co-activation simultaneously serves to deconstruct a deafferented cortex and reorganize it to be continuous with the structure and functional capacities of a neighboring intact cortex. Aside from the fact that afferent stimulation can arrive at a deafferented cortex from numerous other cortical areas with the effect of contributing to the long-term preservation of the deafferented cortex, is there any other empirical evidence that could support the preservation hypothesis? Indeed, there is.

A serious challenge facing the remapping hypothesis is the need to explain certain discrepancies between the entailments of the remapping hypothesis and the phenomenal experiences of amputees with phantom sensations. For instance, the remapping hypothesis posits that the cortical reorganization that follows deafferentation dismantles the neural structure that gives rise to phantom sensations, resulting in a significant reduction in the vividness of phantom sensations and, ultimately, the complete disappearance of phantom sensations. Conversely, we ought to expect an increase in the sensitivity and/or functional capacity of intact cortices that now have a greater area of representation in the brain.

Unfortunately for the remapping hypothesis, this progressive elimination of phantom sensations is just not experienced among amputees with phantom limbs. Amputees typically experience their phantom limbs long-term, without any decrease in the vividness of the phantom. A study by Lotze *et al.* on the effects of myoelectric prosthetics of the somatosensory cortex includes a patient who continues to experience phantom sensations 53 years after amputation (Lotze *et al.* 1999). Another study by Makin *et al.* includes a patient who continues to experience phantom sensations 47 years post-amputation, with the average post-amputation time of all 18 of their subjects being 18 years (Makin *et al.* 2012). And even after extended periods of time following amputation, amputees not only continue to feel their phantoms but can execute fine motor skills, such as the opening and closing of their phantom fists and “thumb to index opposition” (Raffin, E. *et al.* 2012, 748). Additionally, Ramachandran proposes that we ought to see functional enhancements associated with body parts whose area of representation has expanded into a deafferented area (Ramachandran 2000, 319). While more research may have to be conducted on this front, currently, no major research study

has found a noticeable increase in the functional capacity of intact body parts following deafferentation.

Phantom sensations do not disappear over time, do not decrease in vividness, and amputees tend to maintain motor control and the ability to execute fine motor skills even five decades, or more, after amputation. These experiences of phantom sensations do not sound like the perceptual correlates of dramatic cortical restructuring. Rather, these perceptual correlates appear perfectly consistent with continued structural and functional preservation of a deafferented cortex.

For Giummarra *et al.*, the evidence overwhelmingly suggests that a deafferented cortex continues to receive afferent signals from diverse sources in the brain, and that the arrival of these afferent signals support the continued preservation of the deafferented cortex. However, Giummarra does not dispute the arguments that cortical co-activation points to “rapid cortical reorganization contralateral to the deafferented limb” (Giummarra *et al.* 2007, 227). The picture painted by Giummarra *et al.* is one of a brain divided. On the one hand, deafferentation initiates a process of neural restructuring, such that an intact cortex invades a neighboring deafferented area, utilizing pre-existing neural connections to expand its area of representation in the brain. On the other hand, a deafferented cortex continues to receive stimulation from diverse neural sources, with the effect of preserving some of its neural structure and functional capacities. The result is that the somatosensory cortex is subject to two simultaneous, yet contrary, mechanisms; one which erodes the structure of a deafferented cortex and another that preserves it. As Ramachandran, Giummarra *et al.* maintain that a primary function of the brain is the accurate representation of the body, but simultaneously suggest that the very structure and normal functioning of the brain itself stands in the way of the brain fulfilling that function.

Not all researchers, however, take cortical reorganization as a given. Tamar Makin, for instance, argues that dramatic cortical reorganization is not the effect of deafferentation, and that the evidence in support of it “is largely based on...crude measurements” (Makin *et al.* 2015, 2140). To remedy the often “crude” and inconsistent measurements of cortical co-activation,

...we assessed remapping of sensorimotor lip representations using an unfolded model of the cortex, allowing us to measure surface-based cortical distances while considering individual cortical folding patterns (Maeda et al., 2014) in 17 unilateral upper limb amputees and 21 intact controls. We found consistent shifts in lip representation

along the homunculus contralateral to the missing hand in amputees (hereafter 'deprived homunculus') towards the hand area. However, this shift didn't reflect full invasion of the lips into the hand territory as previously described, but rather a small local shift in the centre of gravity of the lips (Makin *et al.* 2015, 2141).

Using an "unfolded model of the cortex," Makin *et al.* determine that there is a shift in contralateral intact lip representation in unilateral hand amputees, but that this shift is much smaller than previously documented and does not constitute a "full invasion of the lips into" the deafferented cortex. Measured "shifts in lip representation" were consistent among unilateral hand amputees, but so slight that Makin *et al.* could not establish "any statistical relationship between cortical reorganization and phantom sensations" (Makin *et al.* 2015, 2145). Makin *et al.*'s findings, while they certainly require further corroboration, open the possibility that cortical co-activation may not be as dramatic as previously thought. And, while Makin *et al.* do not posit why shifts in lip representation are observed, it is possible that these consistent, yet slight, shifts of lip representation toward the deafferented cortex are the effect of overflow afferent signals from the lip area successfully reaching and exciting the deafferented hand cortex by way of new or pre-existing neural connections, as suggested by the preservation hypothesis.

In addition to raising concerns over the extent of cortical reorganization/co-activation that is being reported in phantom limb studies, Makin is one of a handful of researchers spearheading a new approach in the way that phantom sensations are studied. As mentioned above, phantom sensation research has historically been challenging because its subject concerns a non-existing limb; the study lacks a peripheral limb at which to focus its inquiry. When Pons discovered that touching an intact body part could excite activity in a deafferented cortex, the field of phantom limb research exploded; finally, researchers could utilize the intact peripheral body as a means of studying sensations corresponding to a non-existent limb. In recent years, a new revolution in the study of phantom limb research has begun through the identification of an alternative medium by which to study phantom sensations. This medium; the movement of a phantom limb.

In their research, Karen T. Reilly and Estelle Raffin have sought to determine whether executive movements of a phantom limb are purely imaginary or resemble the executive movements of intact limbs. Reilly *et al.* and Raffin *et al.* measured EMG activity in the stumps of unilateral hand amputees and two-handed subjects both when they moved their phantom limbs and when they were asked to imagine moving their phantom limbs (Reilly *et al.* 2006; Raffin *et al.* 2012). No significant activity was recorded for either

amputees or two-handed subjects when asked to *imagine* moving their limbs. However, both Reilly *et al.* and Raffin *et al.* observed “significant movement-related bursts of EMG activity in stump muscles” when unilateral hand amputees completed executive motions with their phantom limb (Raffin *et al.* 2012, 753). Additionally, amputees “insist” that imagined movements of their phantom limbs feel like imagined movements of other intact body parts, whereas “motor execution with the phantom evokes sensations close to those experienced when they actually move a body part” (Raffin *et al.*, 754-5). Raffin *et al.* conclude that “amputees moved their phantom limb during our execution condition and imagined moving it during our imagination condition” (Raffin *et al.*, 753).

The possibility of observing the effects of deafferentation in the brain by way of patients “moving” their phantom limbs is being pursued further by Tamar Makin. Using 18 unilateral upper-limb amputees with an average of 18 years since amputation and “22 intact controls (two handers),” Makin *et al.* conducted a series of fMRI scans to determine neural activity that corresponded exclusively to the movement of a phantom limb (Makin *et al.* 2012, 2). Makin *et al.* found that “group activation for phantom movements was similar to that found during two-handers’ non-dominant hand movements in the primary sensorimotor cortex...suggesting preserved functional representations” (Makin *et al.* 2012, 2-3). Prior to Makin *et al.*’s research, we have never observed neural activity isolated to the deafferented cortex; we could only estimate the effects of deafferentation by way of the phenomenon of cortical co-activation. Makin *et al.*’s focus on phantom movements themselves, has finally provided a means to isolate neural activity in the deafferented cortex, and the findings are such that there is a great degree of preservation of the cortex’s original area of representation.

Makin *et al.*’s findings certainly prove problematic for the remapping hypothesis. If cortical co-activation directly corresponded with cortical reorganization, then neural activity in the deafferented cortex ought to be significantly reduced in size compared to the size of the cortical territory prior to deafferentation. However, that’s just not what we see; when we observe neural activity isolated to the deafferented cortex, we observe preservation of cortical structure. Now perhaps something like what Giummarra *et al.* have suggested is occurring, that is, following deafferentation, there are two mechanisms at work simultaneously, one which deconstructs a deafferented cortex and another that preserves the structure of a deafferented cortex. This may very well be the case, and if it were true could partially vindicate the remapping hypothesis. Something like remapping may be occurring, it just would take much longer to obtain because of counteracting forces. But, at the very least, the remapping hypothesis and researchers must concede a critical piece of evidence on which the remapping hypothesis rests; cortical co-activation

does not directly correspond to cortical reorganization. The remapping hypothesis takes cortical co-activation to be identical with cortical reorganization, such that cortical co-activation indicates the new boundaries of an intact cortical area and the extent to which it has invaded a deafferented cortex. However, if neural activity isolated to the deafferented cortex shows a preservation of the original neural structure, then clearly cortical co-activation does not align with cortical reorganization.

Makin *et al.*'s findings could also be indicative of another possibility, that something like what the preservation hypothesis proposes is at work following deafferentation. Note that the preservation hypothesis argues that cortical co-activation is not indicative of cortical reorganization but reveals intercortical transference of afferent stimuli with the effect of preserving the structure and functional capacities of a deafferented area. Thereby, if we were ever able to observe the isolated activity of a deafferented cortex, we ought to see a great degree of structural preservation. And that, in fact, is exactly what we observe, now that Makin *et al.* have pioneered a means to isolate the neural activity of a deafferented cortex.

VI. Concluding Remarks

My intent in writing this piece is not to indisputably prove the preservation hypothesis or debunk the remapping hypothesis. What I want to show is that there is sufficient evidence to suggest that something like the preservation hypothesis could explain research findings on the effects of deafferentation in the somatosensory cortex. And, if this is the case, then additional research ought to be pursued that explores the possibility of neural preservation following deafferentation.

Whether remapping or the preservation of a cortex follows deafferentation is of material consequence to the determination of how best to treat phantom limb pain. Researchers and clinicians who take remapping seriously, tend to approach the medical treatment of phantom pain in terms of (1) expediting cortical reorganization, and/or (2) the pharmaceutical management of pain.¹³ The preservation hypothesis conceives of the health of the brain in terms of its ability to preserve all its constituent structures and functional capacities, suggesting that treatments for phantom pain ought to be directed towards accomplishing that end.

13. Medications currently used to treat phantom pain include "opioids, NMDA receptor antagonists, anticonvulsants, antidepressants, calcitonins, and anaesthetics," with researchers finding these pharmaceutical interventions "unsatisfactory" in managing phantom pain long-term (Alviar *et al.* 2011, 2).

Additionally, phantom sensation research provides critical insights for conceiving a primary function of the brain: does the functioning of the brain produce an accurate representation of the body or preserve the structures and functional capacities of the brain/body? The phenomenon of phantom sensation pits these two accounts of brain function against one another. Eliminating phantom sensations and restoring the alignment between the experience and actual condition of the body requires the destruction of the neural structures that underpin the appearance of a phantom limb. Thereby, the process of ensuring the accurate representation of the body would come at the cost of the preservation of the brain's structures and functional capacities. In contrast, the brain's operating to preserve its neural structures and functional capacities, the very neural structures that give rise to phantom sensations, would result in the continued discordance between the experience and actual condition of the body. Given that the requirements for accurate representation and preservation each entail a condition that would prevent the other from obtaining, it would be impossible for the brain to simultaneously pursue both these ends. If the brain's function is to preserve itself, one's experience of the body will be in permanent discord with the actual condition of the body. And if the brain's function is to accurately represent the body, it must engage in self-destruction, eliminating the neural structures of the phantom limb to restore the alignment between our experience of the body and the body's actual condition.

It is important to note that these philosophical musings over the function of the brain are very much of practical significance. As Socrates points out in the *Phaedrus*, if we want to make the body "healthy and strong" on "the basis of an art," then it is necessary "to determine the nature of...the body" (Plato *Phaedrus*, 270b3-7). In the case of neuroscience, we must extend Socrates' insights to include the brain as well, such that, in pursuing an understanding of the nature of the brain, we simultaneously pursue an understanding of what truly constitutes a healthy and strong brain. Thus, I do hope that my work here encourages future research projects on phantom sensations. But even more so, I hope that this piece inspires researchers, in all areas of neuroscience, to reflect upon their conceptual commitments concerning the ultimate function of the brain, and to consider how these commitments affect their research and approach to the treatment of neurological conditions.

References

- Alviar, MJM., Hale, T., and M. Duncan. 2011. "Pharmacological interventions for treating phantom limb pain." *Cochrane Database of Systematic Reviews* 12 (CD006380): 1-54.
- Descartes. 1998. *Discourse on Method and Meditations on First Philosophy*. Translated by Donald A Cress. Indianapolis: Hackett Publishing Company.
- Descartes. 1998. *The World and Other Writings*. Translated and Edited by Stephen Gaukroger. Cambridge: Cambridge University Press.
- Finger, S. and Hustwit, M. 2003. "Five Early Accounts of Phantom Limb in Context: Pare, Descartes, Lemos, Bell, and Mitchell." *Neurosurgery* 52 (3): 675-686.
- Giummarra, Melita J., Gibson, Stephen J., Georgiou-Karistianis, Nellie, and John L. Bradshaw. 2007. *Brain Research Reviews* 54: 219-232.
- Hassing, Richard. 2015. *Cartesian Psychophysics and the Whole Nature of Man*. Lanham: Lexington Books.
- Lotze, M., Grodd, W., Birbaumer, N., Erb, M., Huse, E., and H. Flor. 1999. "Does use of a myoelectric prosthesis prevent cortical reorganization and phantom limb pain?" *Nature Neuroscience* 2 (6): 501-502.
- Makin, Tamar R., Scholz, Jan, Filippini, Nicola, Henderson Slater, David, Tracey, Irene, and Heidi Johansen-Berg. 2013. "Phantom pain is associated with preserved structure and function in the former hand area." *Nature Communications* 4 (1570): 1-8.
- Plato. 2006. *Plato on Love*. Edited by C. D. C. Reeve. Indianapolis: Hacking Publishing Company, Inc.
- Pons, TP., Garraghty, PE., Ommaya, AK., Kaas, JH., Taub, E., and M. Mishkin. 1991. "Massive cortical reorganization after sensory deafferentation in adult macaques." *Science* 25 (5014): 1857-60.
- Raffin, Estelle, Giroux, Pascal, and Karen T. Reilly. 2012. "The moving phantom: Motor execution or motor imagery?" *Cortex* 48: 746-757.
- Reilly, KT., Mercier, C., Schieber, MH., and A. Sirigu. 2006. "Persistent hand motor commands in amputees' brain." *Brain* 129 (8): 2211-23.
- Ramachandran, V.S. 1998. *Phantoms in the Brain*. New York: Quill.
- Ramachandran, V.S. 2011. *The Tell-Tale Brain*. New York: W. W. Norton & Company.
- Ramachandran, V.S. and W. Hirstein. 1998. "The perception of phantom limbs: the D.O. Hebb lecture." *Brain* 121: 1603-1630.

- Ramachandran V.S., Rogers-Ramachandran, D, and M. Stewart. 1992. "Perceptual correlates of massive cortical reorganization." *Science* 258 (5085): 1159–60.
- Ramachandran, V.S., and D Rogers-Ramachandran. 2000. "Phantom Limbs and Neural Plasticity." *Arch Neurol.* 57 (3): 317-320.
- Sherman, RA., Sherman, CJ., and L. Parker. 1984. "Chronic phantom and stump pain among American veterans: Results of a survey." *Pain* 18 (1): 83–95.