

## Disorders of Body Scheme P. Haggard<sup>1</sup> & D. M. Wolpert<sup>2</sup>

<sup>1</sup>Institute of Cognitive Neuroscience, University College London,  
17 Queen Square, London WC1N 3AR, UK. Email: p.haggard@ucl.ac.uk

<sup>2</sup>Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology,  
University College London, Queen Square, London WC1N 3BG, UK. Email: wolpert@ion.ucl.ac.uk

### **Introduction**

The brain contains multiple representations of the body. First, afferent inputs from the skin and proprioceptive receptors project to maps of the body surface and body segments respectively in the primary somatosensory cortex (Penfield, 1950). These somatotopic maps reflect the distribution of sensory receptors within the body, and underpin somatic sensation (Romo, Hernandez, Zainos, & Salinas, 1998). For example, area 3b contains a distorted “homunculus”, with enlarged lips and hands. Neuroanatomical, neuropsychological and neurophysiological evidence all suggest that this primary information is further processed to construct higher-order, more cognitive representations of the body. These representations differ from primary maps in providing a supramodal, coherent scheme for body representation and skilled action. These higher-order representations form the focus of this chapter.

At the cognitive level, a fundamental distinction can be made between two different higher-order body representations which have been called *body schema* and *body image* (Paillard, 1999). *Body schema* refers to a representation of the positions of body parts in space, which is updated during body movement. This typically does not enter into awareness, and is primarily used for spatial organization of action. The body schema is therefore a central representation of the body’s spatial properties, that includes the length of limb segments, their hierarchical arrangement, the configuration of the segments in space and the shape of the body surface.

*Body image* refers to a conscious visual representation of the way the body appears *from the outside*, typically in a canonical position. The scientific concept corresponds roughly to the everyday use of the term. This chapter is not primarily concerned with body image, since there is little evidence of any special connection between disorders of body image (e.g., in anorexia) and movement control. We will not discuss pure disorders of body image, but we will discuss the many cases where abnormalities of body schema lead to altered body image.

Sir Henry Head first introduced the term body schema in discussing disordered spatial representation of the body following parietal lobe damage (Head & Holmes, 1912). His original description covers many different aspects of sensorimotor function, but has at its core the representation

in the brain of “*organised models of ourselves*” (Head & Holmes, 1911: p189). Moreover, the term schema has other uses in cognitive science. Therefore, we prefer the more neutral term *body scheme*. By this, we mean a neural representation of the body used for spatial sensorimotor processing. We exclude representations for primary sensory input and motor execution. In this chapter we first define the body scheme by describing several properties based on research with normal human and animal subjects. Then, we show how various neurological and neuropsychological conditions can be explained as pathologies of the body scheme.

### **Properties of the body scheme**

Human and animal studies have consistently shown the following seven fundamental properties of body representation;

#### **Spatially coded.**

The body scheme represents the position and configuration of the body as a volumetric object in space. Crucially, the body scheme integrates tactile information from the body surface with proprioceptive information about the configuration of the limbs in space (Head & Holmes, 1911). This integration means that a stimulus on the body can be localized in external space. For example, combining a tactile sensation on the left hand, with information about the joint angles of my left arm allows me to program a rapid movement of my right arm to swat the fly. Thus, tactile sensations are obligatorily transformed from body surface locations to locations in external space, suggesting that body scheme representations dominate primary representations in normal human behaviour (Yamamoto & Kitazawa, 2001). For example, a visual stimulus on the right side facilitates processing of a subsequent tactile stimulus on whichever hand is adjacent to the visual event. In a normal posture, the right hand shows this facilitation, but if the hands are crossed then the left hand shows a comparable benefit (Spence, Pavani, & Driver, 2000).

#### **Modular.**

Body postures might, in principle, be stored as individual entries in a database, with each entry describing the entire body configuration and body surface stimuli. However, the evidence suggests that the brain represents different

body parts in different neural modules, using the resulting modular network to represent all postures.

Therefore, the body scheme comprises body parts or segments, which bear spatial and categorical relations to each other (e.g., fingers are elements of hands, which form the ends of arms) (Tessari & Rumiati, 2002). For example, Reed and Farah (1995) investigated the effects of moving the legs or arms on visual perceptual judgments. They found facilitation effects for judgements about visual body stimuli, but not for non-body stimuli. These effects were further specific to the body part that the observer was actively controlling, suggesting a division between upper and lower body within the body scheme.

### **Updated with movement.**

Any body representation which is used for action must continuously track the positions of our body parts as we move. Head's definition of the body scheme emphasized automatic updating of the positions of body parts in space during voluntary movement; *"every new posture or movement is recorded on this plastic schema, and the activity of the cortex brings every fresh group of sensations evoked by altered posture into relation with it,"* (Head & Holmes, 1911: p187). The updating process may underlie the finding that the visual receptive fields of many parietal neurons follow the hand when the hand moves (Graziano & Gross, 1993). This mechanism would allow the body scheme to modulate perceptual processing of objects according to their position in peripersonal space. This would be essential for control of grasping or avoidance movement.

### **Adaptable.**

The body scheme must adapt to allow for gradual changes in the spatial properties of the body. For example, the absolute and relative sizes of body parts change over the life span. In addition the body scheme can change on a shorter time scale to incorporate additional objects as new segments of the body representation. In tool use, for example, visual receptive fields of bimodal neurons previously linked to hand position may move towards the tip of the tool, or towards the visual representation of the tool on a video monitor (Iriki, Tanaka, & Iwamura, 1996; Iriki, Tanaka, Obayashi, & Iwamura, 2001). These plastic changes may occur both as gradual extensions to an existing scheme, or as rapid switches between several alternative coexisting schemes (Braun, Heinz, Schweizer, Wiech, Birbaumer, & Topka, 2001).

### **Supramodal.**

The body scheme receives multiple sensory inputs. By definition, the body scheme integrates body surface

information and proprioception to describe the body as a volumetric object in external space. In addition, however, visual information can be in the same representation. Thus, a visual stimulus and a tactile stimulus at the same location on the body surface may form a joint representation within the body scheme (Rorden, Heutink, Greenfield, & Robertson, 1999). This may involve transforming primary representations of vision, proprioception and touch either into a single sensory modality, or into an abstract, amodal code (Lackner, 1988).

### **Coherent.**

The brain maintains a coherent spatial organization of the body scheme across space and time. This ensures a continuity of body experience which may play a major role in individual self-consciousness. A basic principle of body scheme coherence is the resolution of inter-sensory discrepancies. For example, the visual and proprioceptive representations of hand position each have characteristic biases and variabilities, yet we perceive our hand in a single location because the brain optimally combines these sources of information (van Beers, Wolpert, & Haggard, 2002). These discrepancies can be exaggerated by experimental manipulations which put the modalities into stark conflict. For example if the forearm is held at a fixed extension angle and the biceps tendon vibrated an illusory extension of the forearm is experienced. If a blindfolded subject holds his nose during this procedure, the nose is perceived to grow in length as the forearm is felt to extend. In this case, the proprioceptive information from the arm, and the tactile information about the contact between the fingers and nose are preserved and made coherent by adapting the perceived size of another body part (the nose). The overall coherence of the body scheme is thus preserved by altering the representation of a single body segment (Lackner, 1988).

### **Interpersonal.**

A common body scheme is used to represent both one's own body, and the bodies of others. Reed and Farah (1995), for example, showed that participants could better perceive changes in a model's body posture if they simultaneously moved their own corresponding body part. In a related experiment, Tessari and Rumiati (2002) found that memory for observed actions was facilitated when subjects concurrently moved the congruent body part, but not an incongruent body part. These results imply that the observed and self-generated actions were co-represented within a single modular body scheme. An interpersonal function necessarily implies a supramodal body scheme, since information about others' bodies is generally visual, while information from one's own body is generally tactile or proprioceptive.

## **Disorders of body scheme**

This conceptual framework can be used to classify many of the neurological disorders of body representation. Since each of these disorders has previously been discussed extensively in the literature, we focus on how comparisons across several disorders can clarify the structure of the body scheme in the human brain, rather than on detailed aetiology or presentation. We have classified the disorders according to functional deficits. These disorders typically occur after damage to the parietal lobe, particularly its inferior part. However, the specific neural modules subserving the various properties of the body scheme described above are not clearly understood. In particular, few studies have focussed on *groups* of lesion patients (but see Cutting, 1978). Therefore, it has been difficult to dissociate the neural modules subserving the different aspects of body scheme. Thus, single case studies suggest that both interpersonal body representation (Bottni et al., 2002) and updating of the body scheme with movement (Wolpert et al., 1998) may occur after parietal damage. However, the specific of locus of these functions has not been clearly shown. We speculate that group lesion studies in this area would make an important contribution to future research.

## **Pathologies of sensory input**

### *Deafferentation*

The most straightforward pathology of body representation occurs in conditions where sensory input from the body is reduced or absent. Peripheral deafferentation is the best studied of these. Gallagher and Cole (1995) describe IW, who became completely deafferented below the neck for touch and proprioception following a viral illness. Although he was not paralyzed it took him many months to learn to control his movement and to walk. Over time he has achieved a remarkable degree of control which is heavily reliant on visual feed-back. This control is achieved at the cost of a large attentional demand. Whereas normals can easily perform motor acts while concentrating on other things, IW finds it hard to perform such dual tasks. He requires constant vision of his body to know where his body parts are and how to move them. This demonstrates the primacy of proprioceptive and tactile inputs within the body scheme. In contrast, blind people, can achieve accurate sensorimotor control without such an attentional cost. This contrast suggests the proprioceptive updating of the body scheme is essentially automatic, while the visual input may be less so. Moreover, the automatic proprioceptive updating is a continuous background process: IW's becomes increasingly inaccurate in the absence of vision of his body.

## **Pathologies of bodily spatial organisation**

Studies of patients with central lesions have shown that the brain circuits for localisation of a stimulus on the body surface are separate from those involved in processing tactile form. Thus, patients may be able to perceive a tactile stimulus while not correctly locating it, or may be able locate it without being able to describe it (Paillard, 1999). These dissociations have been taken as evidence for separate pathways for “what” and “where” processing in tactile perception. However, they have additional implications for body representation. In particular, patients with somatosensory lesions may show changes in the implied spatial organisation of the body. Rapp, Hendl, and Medina (2002) asked patients to report where on the hand they have just been touched. The resulting map of localisation errors revealed a systematic distortion of the normal hand shape. Although the patients can detect the tactile stimuli, they have a distorted representation of the body surface, perhaps reflecting changes in neural somatosensory maps induced by the lesion.

### *Macrosomatognosias and Microsomatognosias*

Spatial distortions of body size may also occur as a result of specific psychiatric and neural conditions. These are classified as macrosomatognosias or microsomatognosias, according to whether subjective body size is increased or decreased. They are typically tested by asking the patient to draw themselves, or match their own body to a visual image. Microsomatognosias typically present as a general underestimation of whole body size, and may belong more properly in a discussion of body image than of body scheme (Leker, Karni, & River, 1996). In contrast, macrosomatognosias can be specific to some body parts, and may therefore reflect a distortion of an underlying neural representation of the body. Migraine aura has been associated with an increase in the perceived size of the hands and face (Podoll & Robinson, 2000). This body-part specific effect is consistent with human experimental work in which anaesthetising the thumb induced a perceived enlargement of its primary somatosensory neighbour, the lips (Gandevia & Phegan, 1999). Interestingly, the inverse pattern is seen in some psychotic patients. Their somatic delusions may overestimate the size of midline structures, notably the trunk, genitals and head (Hay, 1970). These conditions suggest that the processing chain between primary body surface representation and a higher level representation of the spatial body configuration can be selective disrupted. However, the neural site within the processing chain at which these disorders arise is not clear.

## Pathologies of segmentation

### *Autotopagnosia*

Autotopagnosia is a disorder of the body scheme typically seen after left parietal lesions. These patients make mislocalisation errors when asked to point to specific body parts. The pattern of errors generally implies confusion between adjacent body parts (e.g., pointing to shoulder when asked to point to the elbow). Thus, the relative positions of body parts appear disorganised. The disorder involves a higher-level, cognitive body scheme rather than a primary sensorimotor representation, because localisation on both the patient's own body and on other bodies may be affected (Sirigu, Grafman, & Bressler 1991). Moreover, naming of body parts pointed to by the examiner may be preserved (Ogden, 1985). Taken together, these findings suggest that knowledge about body part categories is preserved, but that the position of these categorical elements within the overall spatial organisation of the body is lost. The spatial unity of the body is incorrectly segmented into its modular parts.

### *Finger agnosia*

Finger agnosia is a somewhat similar disorder which specifically affects finger segmentation. Like autotopagnosia, it is associated with lesions of the left parietal lobe, and more specifically with the angular gyrus (Kinsbourne & Warrington, 1962). When the examiner touches one of the patient's fingers in the absence of vision, the patient is unable to identify which one is touched. The primary deficit appears to involve *individuating* the fingers: it is as if the patient's fingers become collectively fused and undifferentiated. Because finger agnosia can be found without autotopagnosia for other body parts, the concept of a distinct "finger schema" has been proposed (Benton, 1959). The important evolutionary changes in primate finger dexterity may have driven evolution of a separate abstract representation of the digits. A developmental literature has focussed on Gerstmann's syndrome, in which finger agnosia may coexist with a range of deficits including dyscalculia, left-right confusion and agraphia (Gerstmann, 1942). The association with arithmetic suggests that the modular nature of body representations may be an important precursor of categorical and symbolic representations in general.

## Pathologies of extent

### *Phantom limb*

When all or part of a limb is amputated many patients still feel the presence of their limb (Ramachandran & Hirstein, 1998). Stimulation of the skin on areas such as the face can even cause sensation in a phantom (Ramachandra, Stewart, & Rogers-Ramachandran, 1992; Aglioti, Smania, Atzei, & Berlucchi, 1997; Kew, Halligan,

Marshall, Passingham, Rothwell, Ridding, Marsden, & Brooks, 1997). This is thought to arise from the reorganisation of the deafferented region of cortex after amputation. This marked neural plasticity means that other parts of the body surface project to cortical area that previously represented the phantom limb.

More strikingly, patients may feel they can *move* their phantom immediately after amputation, but lose this ability over time (Ramachandran, 1993). The ability to move the phantom voluntarily may rely on efferent signals that normally update the body scheme (Wolpert, Ghahramani, & Jordan, 1995). Efferent copy of motor commands may be processed normally and used to estimate and update the configuration of the limb. This efferent signal is sufficient to cause the sensation of movement in the phantom. This demonstrates that efferent commands can contribute to the body scheme. However, this percept based on the efferent command is not corroborated by appropriate sensory feedback from the limb. As the body scheme is adaptable, over time the system may learn that the efferent commands are ineffective. Such adaptation could explain why, as the efferent signal no longer predict a change in configuration, patients eventually come to feel that the phantom is "paralysed". However, if the patient is given visual feedback via a mirror box suggesting that the efferent command does move the phantom, this rapidly leads to the perception that they are now able to move the phantom limb again (Ramachandran & Rogers-Ramachandran, 1996).

## Pathology of updating

### *Supernumerary limbs*

Whereas phantom patients must adapt the body scheme to reflect loss of a limb, some patients report experiencing supernumerary limbs (Vuilleumier, Reverdin, & Landis, 1997). Hari, Hänninen, Mäkinen, Jousmäki, Forss, Seppä, and Salonen, (1998) reported a patient with congenital abnormality of the corpus callosum who suffered a subarachnoid hemorrhage leading to an infarction in the right frontal lobe, including damage to the most anterior region of the right SMA. This patient experienced an additional left arm that occupied the position vacated by the real left arm a minute or so previously.

Since the estimated position of a limb is based on integrating information from motor commands and sensory feedback (Wolpert et al., 1995), a failure to integrate these two sources of information could lead to the experience of two limbs rather than one. In the absence of movement, these two sources of information coincide, and indeed the patient does not experience the ghost limb. However, when she moves her arm, the representation of the estimated position is not updated by

the motor commands. Sensory and motor information therefore become discrepant. The normal coherence of the body scheme is lost, and the perceived shape of the body is altered by adding a supernumerary limb to accommodate the discrepancy.

#### *Fading limbs*

The inverse situation involves a resting limb fading out of consciousness. Wolpert, Goodbody, and Husain (1998) describe patient PJ, who had a large cyst in the left parietal lobe. She reported that the position and presence of her right limbs faded away over a few seconds if she could not see them. Her experience of a constant tactile stimulus or a weight also faded away, but changes in such sensations could be detected. Slow reaching movements to peripheral targets with the right hand were inaccurate, but reaching movements made at a normal pace were unimpaired. In this case there seemed to be a circumscribed problem with the representation of the current limb position in that it could not be maintained in the absence of changing stimulation.

### **Pathologies of bodily coherence**

#### *Anosognosia.*

Patients with right hemisphere damage leading to paralysis (or weakness) on the left side may develop the false belief that there is nothing wrong with the paralyzed limb. The motor system in these patients may fail to register discrepancies between the actual and predicted states of the system (Frith, Blakemore, Wolpert, 2000). These patients perceive their body scheme to be coherent, despite the impairment.

#### *Somatoparaphrenia.*

In other cases, the patient is clearly aware of the abnormal sensorimotor status of the limb, but the attitude towards the affected limb is clearly abnormal. Such patients often also suffer from neglect, but the relation between the two conditions remain unclear. The patient may have delusions that the affected limb belongs to another person (Bisiach, Rusconi, & Vallar, 1991), or even to an animal (Halligan, Marshall, & Wade, 1995). The attitude to the exiled limb is generally hostile. These patients present with a quite psychotic account of their deficit, but the delusion is highly specific, being confined to their attitude to the affected limb. The patient does not see any impairment in their *own* body, but may attribute the deficit to another individual. By reassigning ownership of the limb, the patient may be preserving a coherence of their own body scheme despite the loss of sensation and movement.

### **Pathologies of interpersonal body representation**

Several lines of evidence show that a common neural body scheme is used interpersonally, to represent both one's

own body and others' bodies. This implies a mapping function linking the codes for specific body parts across people. A patient reported by Bottini et al. (2002) showed a tactile neglect that was sensitive to the interpersonal level of body representation. The patient had a profound hemianaesthesia, and in addition a delusional belief that her left hand belonged to her niece. The patient neglected tactile stimuli when asked to respond to touch on her left hand, but reliably detected identical stimuli when asked to respond to touches on her "niece's hand". This case appears to demonstrate a complex interplay between primary somatosensory maps and a much more cognitive level of body representation, in which body parts are grouped in a coherent way to be assigned to the "self" or to another person. The personal level of body representation may therefore modulate primary tactile processing.

#### *Heterotopagnosia*

In some pathological cases, the interpersonal mapping function can be specifically damaged, leaving other aspects of the body scheme, such as its spatial organisation, unaffected. Heterotopagnosia, which may follow left parietal damage, offers one example of this interpersonal function, and may be a pure pathology of interpersonal body representation (Degos & Bachoud-Levi, 1998). When asked to point to the examiner's own nose, these patients repeatedly point to their own nose. The localisation within the body map is correct, but the body representation is transposed from another person to the self. This disorder therefore seems to involve selective damage to the processing stage at which body parts are assigned to persons.

### **Conclusion**

In this chapter, we have argued from behavioural and neurophysiological data that the human brain contains a cognitive representation of the body. We have shown that this *body scheme* has the essential properties required for multisensory integration and coordinated sensorimotor action. From an understanding of these normal functions, we have shown that several sensory and motor disorders can be explained by reference to damage to one or more of these essential properties. Interestingly, many disorders of body scheme have both neurological and psychiatric aspects, which suggests that a coherent neural representation of the body is a key element of self-consciousness. Finally, a perhaps surprising but fascinating feature of the brain's body scheme is the commonality between the representation of one's own body and the body of other individuals. This suggests that the body scheme could also form a basis for social cognition.

## Acknowledgements

PH was supported by a Leverhulme Trust Research Fellowship. This work was supported by the Wellcome Trust, ESRC, HFSP and McDonnell Foundation. We are grateful to Louise Whiteley for assistance.

## References

- Aglioti, S., Smania, N., Atzei, A., & Berlucchi, G. (1997). Spatio-temporal properties of the pattern of evoked phantom sensations in a left index amputee patient. *Behavioral Neuroscience*, **111**(5), 867-872.
- Benton, A.L. (1959). *Right-left discrimination and finger localization: Development and pathology*. Hoeber-Harper: New York.
- Bisiach, E., Rusconi, M.L., Vallar, G. (1991). Remission of somatoparaphrenic delusion through vestibular stimulation. *Neuropsychologia*, **29**(10), 1029-1031.
- Bottini, G., Bisiach, E., Sterzi, R., Vallar, G. (2002). Feeling touches in someone else's hand. *Neuroreport*, **13**(2), 249-252.
- Braun, C., Heinz, U., Schweizer, R., Wiech, K., Birbaumer, N., & Topka, H. (2001). Dynamic organization of the somatosensory cortex induced by motor activity. *Brain*, **125**, 2259-2267.
- Cutting J (1978) Study of anosognosia. *J Neurol Neurosurg Psychiatry* **41**:548-555.
- Degos, J.D., & Bachoud-Levi, A.C. (1998). La designation et son objet. Pour une neuropsychologie de l'objectivation. *Revue Neurologique*, **154**(4), 283-290.
- Frith, C.D., Blakemore, S.J., & Wolpert, D.M. (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, **355**(1404), 1771-1788.
- Gallagher, S., & Cole, J. (1995). Body image and body schema in a deafferented subject. *Journal of Mind and Behavior*, **16**(4), 369-389.
- Gandevia, S.C., & Phegan, C.M.L. (1999). Perceptual distortions of the human body image produced by local anaesthesia, pain and cutaneous stimulation, *Journal of Physiology*, **514**(2), 609-616.
- Gerstmann, J. (1942). Problem of imperception of disease and of impaired body territories with organic lesions: relation to body scheme and its disorders. *Archive of Neurology and Psychiatry*, **48**, 890-913.
- Graziano, M.S.A. & Gross, J. (1993). A bimodal map of space – somatosensory receptive fields in the macaque putamen with corresponding visual receptive fields. *Experimental Brain Research*, **97**(1), 96-109.
- Halligan, P.W., Marshall, J.C., & Wade, D.T. (1995). Unilateral somatoparaphrenia after right hemisphere stroke: a case description. *Cortex*, **31**(1), 173-182.
- Hari, R., Hänninen, R., Mäkinen, T., Jousmäki, V., Forss, N., Seppä, M., & Salonen, O. (1998). Three hands: fragmentation of human bodily awareness. *Neuroscience Letters*, **240**, 131-134.
- Hay, G.G. (1970). Dismorphophobia. *British Journal of Psychiatry*, **533**, 399-406.
- Head, H. & Holmes, G. (1911). Sensory disturbances in cerebral lesions. *Brain*, **34**, 102-254.
- Iriki, A., Tanaka, M., & Iwamura, Y. (1996). Coding of modified body schema during tool use by macaque postcentral neurons. *Neuroreport*, **7**, 2325-2330.
- Iriki, A., Tanaka, M., Obayashi, S., & Iwamura, Y. (2001). Self-images in the video monitor coded by monkey intraparietal neurons. *Neuroscience Research*, **40**, 163-173.
- Kew, J.J.M., Halligan, P.W., Marshall, J.C., Passingham, R.E., Rothwell, J.C., Ridding, M.C., Marsden, C.D., & Brooks, D.J. (1997). Abnormal access of axial vibrotactile input to deafferented somatosensory cortex in human upper limb amputees. *Journal of Neurophysiology*, **77**(5), 2753-2764.
- Kinsbourne, M. & Warrington, E.K. (1962). A study of finger agnosia. *Brain*, **85**, 47-66.
- Lackner, J.R. (1988). Some proprioceptive influences on the perceptual representation of body shape and orientation. *Brain*, **111**, 281-297.
- Leker, R.R., Karni, A., & River, Y. (1996). Microsomatognosia: whole body schema illusion as part of an epileptic aura. *Acta Neurologica Scandinavica*, **94**, 383-385.
- Ogden, J.A. (1985). Autotopagnosia: occurrence in a patient without nominal aphasia and with an intact ability to point to parts of animals and objects. *Brain*, **108**, 1009-1022.
- Paillard, J. (1999). Body schema and body image - a double dissociation in deafferented patients. In G.N. Gantchev, S. Mori, and J. Massion (Eds.), *Motor control*:

today and tomorrow. Academic Publishing House: Sofia, Bulgaria.

Penfield, W., & Rasmussen, T.L. (1950). *The cerebral cortex of man; a clinical study of localization of function*. Macmillan.

Podoll, K., & Robinson, D. (2000). Macrosomatognosia and microsomatognosia in migraine art. *Acta Neurologica Scandinavica*, **101(6)**, 413-416.

Ramachandran, V.S. (1993). Filling in gaps in perception: II. Scotomas and phantom limbs. *Current Directions in Psychological Science*, **2(2)**, 56-65.

Ramachandran, V.S., & Hirstein, W. (1998). The perception of phantom limbs: The D. O. Hebb lecture. *Brain*, **121(9)**, 1603-1630.

Ramachandran, V.S., & Rogers-Ramachandran, D. (1996). Synaesthesia in phantom limbs induced with mirrors. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, **263(1369)**, 377-386.

Ramachandran, V.S., Stewart, M., & Rogers-Ramachandran, D.C. (1992). Perceptual correlates of massive cortical reorganization. *Neuroreport*, **3(7)**, 583-586.

Rapp, B., Hendel, S.K., & Medina, J. (2002). Remodeling of somatosensory hand representations following cerebral lesions in humans. *Neuroreport: For Rapid Communication of Neuroscience Research*, **13(2)**, 207-211.

Reed, C.L., & Farah, M.J. (1995). The psychological reality of the body schema: a test with normal participants. *Journal of Experimental Psychology: Human Perception and Performance*, **21(3)**, 334-343.

Romo, R., Hernandez, A., Zainos, A., & Salinas, E. (1998). Somatosensory discrimination based on cortical microstimulation. *Nature*, **392**, 387-90.

Rorden, C., Heutink, J., Greenfield, E., & Robertson, I.H. (1999). When a rubber hand 'feels' what the real hand cannot. *Neuroreport*, **10(1)**, 135-138.

Seppä, M., & Salonen, O. (1998). Three hands: fragmentation of human bodily awareness. *Neuroscience Letters*, **240**, 131-134.

Sirigu, A., Grafman, J., Bressler, K. (1991). Multiple representations contribute to body knowledge processing: evidence from a case of autotopagnosia. *Brain*, **114**, 629-642.

Spence, C., Pavani, F., & Driver, J. (2000). Crossmodal links between vision and touch in covert endogenous spatial attention. *Journal of Experimental Psychology: Human Perception and Performance*, **26(4)**, 1298-1319.

Tessari, A. & Rumiati, R.I. (2002). Motor distal component and pragmatic representation of objects. *Cognitive Brain Research*, **14(2)**, 218-227.

van Beers, R.J., Wolpert, D.M., & Haggard, P. (2002). When feeling is more important than seeing in sensorimotor adaptation. *Current Biology*, **12**, 834-837.

Vuilleumier, P., Reverdin, A., & Landis, T. (1997). Four legs - Illusory reduplication of the lower limbs after bilateral parietal lobe damage. *Archives of Neurology*, **54(12)**, 1543-1547

Wolpert, D.M., Ghahramani, Z., Jordan, M.I. (1995). An internal model for sensorimotor integration. *Science*, **269(5232)**, 1880-1882.

Wolpert, D.M., Goodbody, S.J., & Husain, M. (1998). Maintaining internal representations: the role of the human superior parietal lobe. *Nature Neuroscience*, **1(6)**, 529-533.

Yamamoto, S., & Kitazawa, S. (2001). Reversal of subjective temporal order due to arm crossing. *Nature Neuroscience*, **4(12)**, 1265-1265.