
Consciousness and body image: lessons from phantom limbs, Capgras syndrome and pain asymbolia

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Words such as ‘consciousness’ and ‘self’ actually encompass a number of distinct phenomena that are loosely lumped together. The study of neurological syndromes allows us to explore the neural mechanisms that might underlie different aspects of self, such as body image and emotional responses to sensory stimuli, and perhaps even laughter and humour. Mapping the ‘functional logic’ of the many different attributes of human nature on to specific neural circuits in the brain offers the best hope of understanding how the activity of neurons gives rise to conscious experience. We consider three neurological syndromes (phantom limbs, Capgras delusion and pain asymbolia) to illustrate this idea.

Keywords: phantom limb; plasticity; body image; somatosensory cortex

1. INTRODUCTION

You never identify yourself with the shadow cast by your body, or with its reflection, or with the body you see in a dream or in your imagination. Therefore you should not identify yourself with this living body, either.

Shankara (788–820 AD) *Viveka Chudamani*
(Vedic scriptures)

In the first half of the next century, science will confront its greatest challenge in trying to answer a question that has been steeped in mysticism and metaphysics for millennia: what is the nature of the self?

Unfortunately, the word ‘self’ is like the word ‘happiness’; we all know what it is and that it is real, but it is very hard to define it or even to pinpoint its characteristics. Like quicksilver, the more you try to grasp it the more it tends to slip away. When you think of the word ‘self’, what pops into your mind? When I think about ‘myself’, it seems to be something that unites all my diverse sensory impressions and memories together (unity), claims to be ‘in charge’ of my life and makes choices (free will), and seems to endure as a single entity in space and time inhabiting a single body (‘body image’). It also sees itself as embedded in a social context, balancing its checkbook and might even plan its own funeral arrangements. Actually we can make a list of all the characteristics of the ‘self’—just as you can for happiness—and then look for brain structures that are involved in each of these aspects. And this, in turn, will one day enable us to develop a clearer understanding of self and consciousness, although I doubt whether there will be a single, grand, climactic solution in the same way that DNA is the solution to the riddle of heredity.

It has always seemed very puzzling to me that the most interesting aspects of human conscious experience have

received the least attention. Consider questions such as: why do we laugh? Why do we cry? Why do we dance? Or why do we appreciate art and music? And the big question: what is consciousness? Everyone finds these questions fascinating and yet they are largely ignored by mainstream neurology and experimental psychology.

The main concern of this article will be the question of how the self constructs a body image and what we can learn about this process by studying patients with phantom limbs. The second half of the article is much more speculative and deals with two poorly understood syndromes: Capgras syndrome and pain asymbolia. All three syndromes have been known since the end of the nineteenth century but there has, on the whole, been a tendency to regard them as clinical curiosities. In this article I shall show that, far from being mere oddities, these disorders can give us valuable insights into the functional organization of the normal human brain.

2. PHANTOM LIMBS, BODY IMAGE AND NEURAL PLASTICITY

I shall describe a number of experiments that we have conducted on both normal people and amputees. These experiments demonstrate that there is a tremendous amount of latent plasticity even in the adult human brain and that one’s body image is surprisingly malleable; more so than anyone would have imagined.

Phantom limbs have probably been known since antiquity; not surprisingly, there is an elaborate folklore surrounding them. After Lord Nelson lost his right arm during an unsuccessful attack on Santa Cruz de Tenerife, he experienced compelling phantom limb pains, including the sensation of fingers digging into his phantom palm. The emergence of these ghostly sensations led the Sea Lord to proclaim that his phantom was a ‘direct proof of the existence of the soul’ (Riddoch

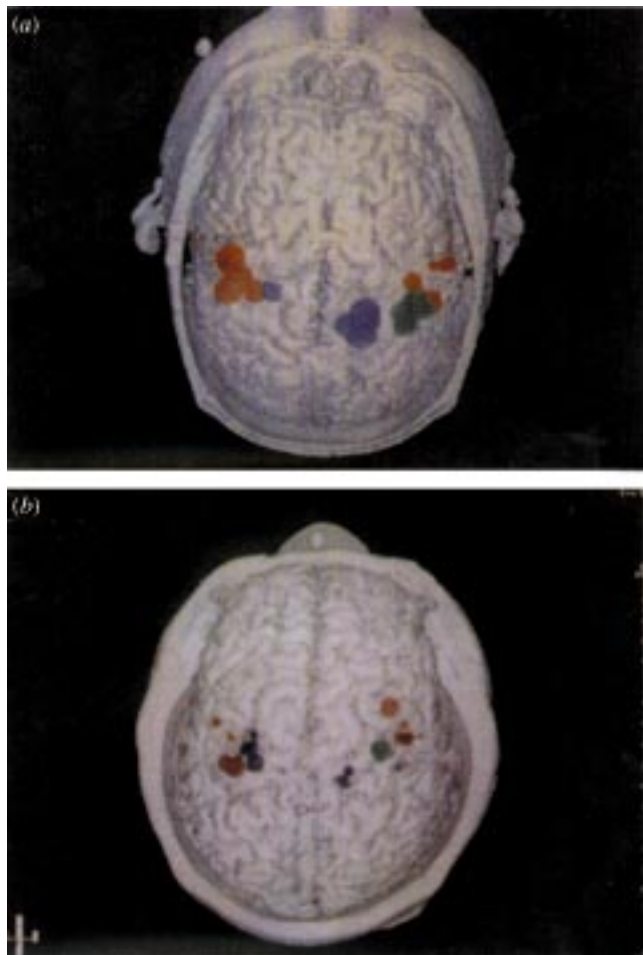


Figure 1. (a) Top view of a combined MEG and 3D surface-rendered magnetic resonance imaging (MRI) scan of an adult whose right arm was amputated below the elbow at the age of 11 years. The right hemisphere is normal and shows the primary somatosensory face area (red) lateral, anterior and inferior to the hand localizations (green), which are in turn lateral, anterior and inferior to the upper arm region (blue) extending into the expected hand territory, reflecting the reorganization of the sensory map as a result of amputation. (b) Combined MEG and 3D surface-rendered MRI of patient F.A. The unaffected right hemisphere shows three spots corresponding to the left face (red), hand (green) and upper arm region (blue). This patient's right arm was amputated below the elbow eight years before these recordings (for details see Ramachandran (1993) and Yang *et al.* (1994a,b)).

1941). If an arm can survive physical annihilation, why not the entire person?

The first clinical description of phantom limbs was provided by Silas Weir Mitchell (1872); see Melzack (1992) for a superb review. Although there have been hundreds of case studies since then, systematic experimental work on them began only ten years ago, inspired in part by the demonstration of striking changes in somatotopic maps after deafferentation (Wall 1977; Merzenich *et al.* 1984). Eleven years after dorsal rhizotomy in adult monkeys, the region corresponding to the hand in the cortical somatotopic map, area 3b, can be activated by stimuli delivered to the monkey's ipsilateral face (Pons *et al.* 1991): direct proof that a massive reorganization of topography had occurred in area 3b. That a similar reorganization occurs in the adult human cortex over distances of 2–3 cm was

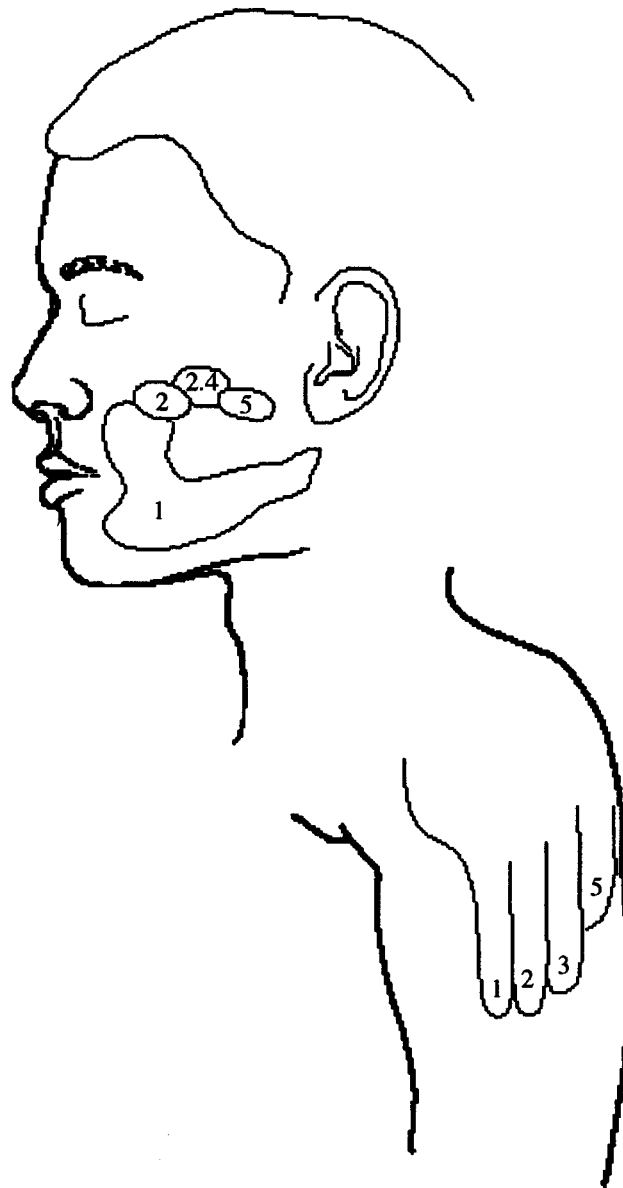


Figure 2. Distribution of reference fields in patient D.S. Notice the prominent representation of the thumb (1)—which we have seen in several patients—and the roughly topographic arrangement of digits 2, 3, 4 and 5 on the face and on the upper arm.

shown by us with the use of magnetoencephalography (MEG) (Ramachandran 1993; Yang *et al.* 1994a,b; Flor *et al.* 1995). After amputation of an arm, sensory input from the face activates the hand area of the Penfield homunculus in S1 (figure 1).

Given this massive reorganization, what would the person feel if his or her face were touched? Because the tactile input on the face now activates the hand area of the cortex, would the person feel that he or she was being touched on the hand as well?

(a) *Referred sensations in phantom limbs*

After testing 18 patients with either arm amputation or brachial avulsion, we found that eight patients systematically referred sensation from the face to the phantom. In many of them, there was a topographically organized map of individual fingers of the hand on the lower face region (figure 2) and the referred sensations were

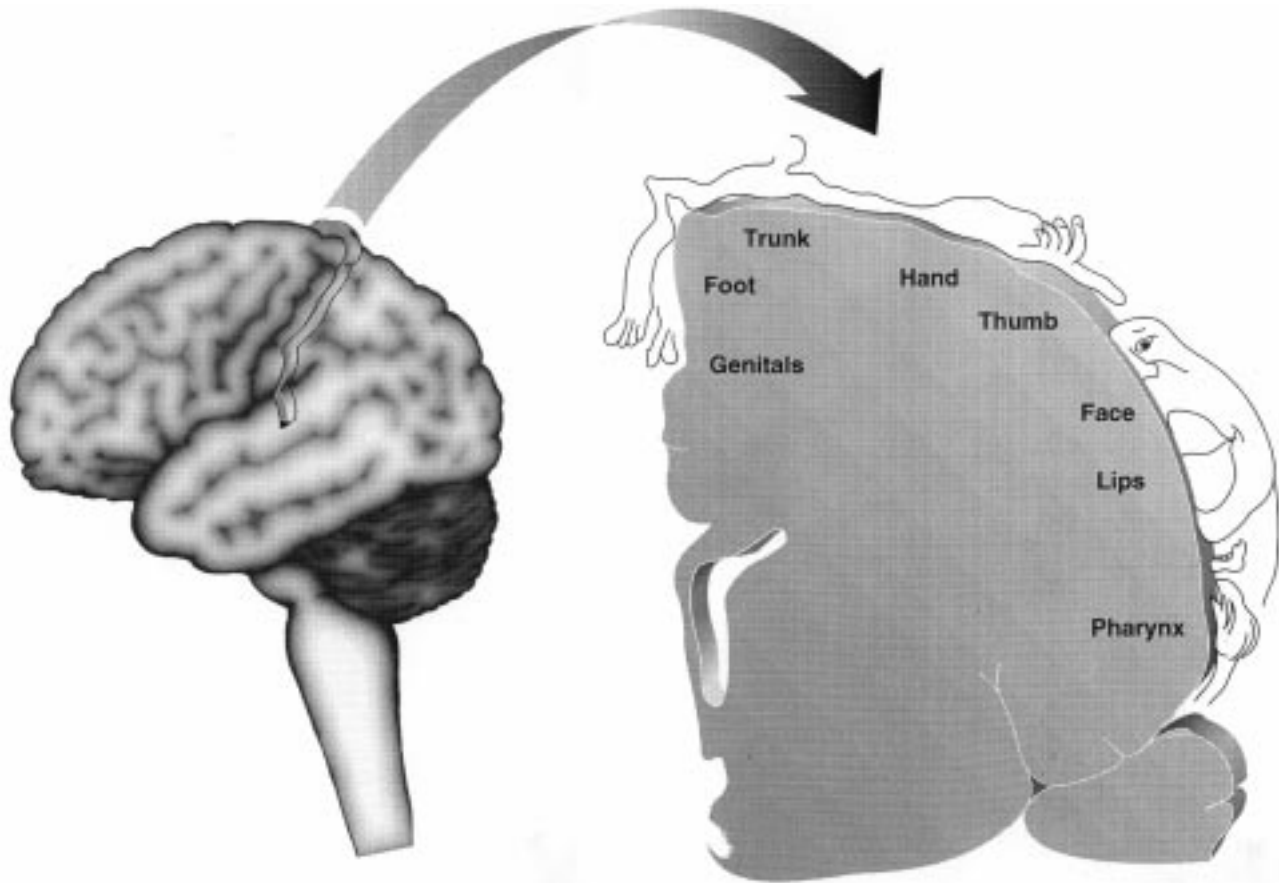


Figure 3. The Penfield 'homunculus'. Notice that the hand area is bordered below by the face, and above by the upper arm and shoulder—the two regions where reference fields are usually found in arm amputees.

modality-specific. For example, hot, cold, rubbing, vibration, metal, or massage on the face were felt as hot, cold, vibration, rubbing, metal and massage at precisely localized points in the phantom. Touching other body parts (e.g. torso, legs, chest) usually did not evoke sensations in the phantom, but there was often a second topographically organized map proximal to the amputation stump. Because the hand area in the Penfield map (figure 3) is flanked on one side by the upper arm and the other side by the face, this is precisely the arrangement of points that one would expect if the afferents from the upper arm skin and face skin were to invade the hand territory from each side.

The fact that stimulating certain 'trigger points' (Cronholm 1951) can elicit referred sensation in the phantom has been noted previously in the older clinical literature, but the occurrence of a topographically organized map on the face and modality-specific referral from face to phantom was not described. Consequently, no attempt was made to relate these findings to somatotopic brain maps, and the referred sensations were often attributed either to stump neuromas or to activation of a 'diffuse neural matrix' (Melzack 1992). Our own results suggest, instead, that referred sensations emerge as a direct consequence of the changes in topography after deafferentation, an idea that we refer to as 'the remapping hypothesis' (Ramachandran 1993).

Based on the remapping hypothesis, we also predicted (Ramachandran 1993) that after trigeminal nerve section, one should observe a map of the face on the

hand, and this has recently been shown in an elegant study by Clarke *et al.* (1996). Also, after amputation of the index finger in one patient a map of the index finger was found neatly draped across the ipsilateral cheek (Aglioti *et al.* 1994a; Aglioti & Berluchi 1998). Finally, our suggestion that these effects are based partly on the unmasking of pre-existing connections, rather than sprouting, receives support from our recent observation that modality-specific referral from the face to the phantom can occur even a few hours after amputation (Borsook *et al.* 1997).

These findings provide strong support for the remapping hypothesis. They might allow us to track the time-course of perceptual changes in humans and relate these in a systematic way to anatomy. The occurrence of topography and modality specificity rules out any possibility of the referral being due to non-specific arousal.

(b) *Reflecting on phantom limbs: synaesthesia*

Some patients claim that they can experience vivid voluntary movements (Melzack 1992) in their phantom limb, presumably because reafference signals from motor commands sent to the phantom are monitored in the cerebellum and parietal lobes. However, with the passage of time the phantom becomes 'frozen' or 'paralysed', perhaps because of a continuous absence of visual and proprioceptive confirmation that the commands have been obeyed. Some patients experience excruciatingly painful involuntary clenching spasms in the phantom; they experience their nails digging into the phantom palm and are unable to open the hand voluntarily to relieve the pain.

We placed a vertical sagittal mirror on the table in front of the patient. If the patient's paralysed phantom was, say, on the left side of the mirror, he placed his right hand in an exact mirror-symmetrical location on the right side of the mirror. If he looked into the shiny right side of the mirror, the reflection of his own hand was optically superimposed on the felt location of his phantom, so that he had the distinct visual illusion that the phantom had been resurrected. If he now made mirror-symmetric movements while looking in the mirror, he received visual feedback that the phantom was obeying his command.

Remarkably, six out of ten patients using this procedure claimed that they could now actually feel—not merely see—movements emerging in the phantom limb. This was often a source of considerable surprise and delight to the patient (Ramachandran & Rogers-Ramachandran 1996).

Indeed, four patients were able to use the visual feedback provided to them by the mirror to 'unclench' a painfully clenched phantom hand. This seemed to relieve the clenching spasm as well as associated cramping pain (the burning and lacerating pains in the phantom remained unaffected by the mirror procedure, suggesting that the relief of the clenching was probably not confabulatory in origin). The elimination of the spasm was a robust effect that was confirmed on several patients. The elimination of the associated pain was also pointed out by them but requires confirmation with double-blind controls, given the notorious susceptibility of pain to placebo and suggestion. In one case, repeated use with the mirror for ten minutes a day for three weeks resulted in a permanent and complete disappearance of the phantom arm and elbow (and a 'telescoping' of fingers into the stump) for the first time in ten years. The associated pain in the elbow and wrist also vanished. This might be the first known instance of a successful amputation of a phantom limb.

(c) ***Emergence of 'repressed memories' in phantom limbs***

Another fascinating but poorly understood aspect of phantom limbs concerns not only the continued existence of 'memories' in the phantom—of sensations that existed in the arm just before the amputation—but also the re-emergence of long-lost memories pertaining to that arm. For instance, it is well known that patients sometimes continue to feel a wedding ring or a watch band on the phantom. Also, in the first few weeks after arm amputation many patients report that they experience excruciating clenching spasms in the phantom hand and that these spasms are often accompanied by the unmistakable sensation of nails digging into the palm. It usually takes several minutes—or sometimes even hours—to voluntarily unclench the phantom (unless the subject uses our mirror device!) but when unclenching eventually does take place, the 'nails digging' sensation vanishes as well. The reason for this is obscure, but one possibility is that when motor commands are sent from the premotor and motor cortex to clench the hand, they are normally damped by error feedback from proprioception. If the limb is missing, however, such damping is not possible, so that the motor output is amplified even further, and this overflow or 'sense of effort' itself might be experienced as pain. But why would the 'nails digging'

sensation also be associated with the spasm? This is even more difficult to explain, but one might suppose that the motor commands to unclench the hand and the sensation of the nails digging are linked in the brain, even in normal individuals, by a Hebbian learning mechanism. Furthermore, because the motor output is now amplified, it is conceivable that the associated memory of nails digging is also correspondingly amplified, giving rise to the excruciating pain. The observation that eliminating the spasms (e.g. with intense, prolonged voluntary effort) also abolishes the digging sensation is consistent with this view. What we are dealing with here, then, might be a primitive form of sensory learning that could conceivably provide a new way of experimentally approaching more complex forms of memory and learning in the adult brain.

The reactivation of pre-amputation memories in the phantom has been noted before (Katz & Melzack 1990) but very little systematic work has been done on it, and the significance of the findings for understanding normal memory seems to have gone largely unrecognized. For example, one of our patients reported that before amputation the arthritic joint pains in her fingers would often flare up when the weather was damp and cold. Remarkably, whenever the air became humid the same pains would recur in her phantom fingers. Also, when her hand went into a clenching spasm in the evening, the thumb was usually abducted and hyper-extended ('sticking out') but on those occasions when it was flexed into the palm, the spasm was accompanied by the distinct feeling of her thumbnail digging into the fifth digit's pad. The curious implication of this observation is that even fleeting sensory associations may be permanently recorded in the brain; these memory traces might be ordinarily 'repressed', but might become unmasked by the deafferentation. (Also, surprisingly, the traces might be 'gated' by the felt position of the phantom thumb or even be retrieved on the basis of an unconscious inference: 'if my thumb is flexed it must touch my fifth digit.')

(d) ***Resurrection of long-lost phantoms***

We have also tried the mirror procedure on digit (finger) amputees with very similar results. One patient had his index finger amputated 1 cm distal to the head of the metacarpal about 40 years before we saw him. He had experienced a vivid phantom finger (but no phantom pain) for about a year, but it faded completely after that. Remarkably, when he saw his index finger move in the mirror, he started experiencing proprioceptive sensations in his index finger for the first time in 39 years! He seemed very intrigued and delighted by all this.

(e) ***Phantoms induced in normal individuals***

The question of how the brain constructs a 'body image' has been a topic of considerable interest to neurologists (Head 1918; Brain 1941; Critchley 1953), psychologists (Schilder 1950), and even philosophers (Merleau-Ponty 1967; Dennett 1978; O'Shaughnessy 1980). Even though this image is constructed from evanescent and fragmentary evidence derived from multiple sensory systems such as vision, proprioception and hearing, we have a stable internal mental construct of a unitary corporeal self that endures in space and time, at least until its eventual annihilation in death.

One key difference between tactile sensations and visual sensations is that the former are localized directly on the sensory surface where the receptors are actually located, whereas the latter are 'projected' onto the external world; for example, when light from a tree hits your retinal receptors you localize the tree externally, not inside your eyeball. Indeed, vision probably evolved as a 'remote sensing' device that liberates you from the requirement of direct contact with the object you are trying to localize, whether for dodging or for grabbing (Dawkins 1996).

With so ancient a phylogenetic rift between the two systems, it would be very surprising if one could 'project' somatic sensations onto the external world, yet anyone who has used a screwdriver or a razor and a mirror will realize that this must be possible, at least to a limited extent. After extended use of the screwdriver one often begins to 'feel' the tip of the screwdriver. Similarly, when using a shaving mirror one experiences a peculiar mental diplopia—the razor is felt simultaneously on one's own face but to a limited extent also on one's *doppelgänger* in the mirror.

Although we ordinarily regard phantoms as pathological, it is relatively easy to generate such illusions, even in otherwise normal individuals. Consider the 'phantom nose' illusion that we recently discovered in our laboratory (Ramachandran & Hirstein 1997). The subject sits in a chair blindfolded, with an accomplice sitting in front of him, facing the same direction. The experimenter then stands near the subject, and with his left hand takes hold of the subject's left index finger and uses it to tap and stroke the nose of the accomplice repeatedly and randomly, while at the same time, using his right hand, he taps and strokes the subject's nose in precisely the same manner, and in perfect synchrony. After a few seconds of this procedure, the subject develops the uncanny illusion that his nose has either been dislocated, or has been stretched out several feet forwards, demonstrating the striking plasticity or malleability of our body image. The more random and unpredictable the tapping sequence the more striking the illusion. We suggest that the subject's brain regards it as highly improbable that the tapping sequence on his finger and the one on his nose are identical simply by chance and therefore 'assumes' that the nose has been displaced—applying a universal Bayesian logic that is common to all sensory systems (Ramachandran & Hirstein 1997). The illusion is a very striking one, and we were able to replicate it in 12 out of 18 naive subjects.

Our 'phantom nose' effect is quite similar to one reported by Lackner (1988) except that the underlying principle is different. In Lackner's experiment, the subject sits blindfolded at a table, with his arm flexed at the elbow, holding the tip of his own nose. If the experimenter now applies a vibrator to the tendon of the biceps, the subject not only feels that his arm is extended—because of spurious signals from muscle stretch receptors—but also that his nose has actually lengthened. Lackner invokes Helmholtzian 'unconscious inference' as an explanation for this effect (I am holding my nose; my arm is extended; therefore my nose must be long). The illusion that we have described, in contrast, does not require a vibrator and seems to depend entirely on a Bayesian principle: the sheer statistical improbability of two tactile sequences' being identical. (Indeed, our illu-

sion cannot be produced if the subject simply holds the accomplice's nose.) Not all subjects experience this effect, but that it happens at all is astonishing: that a lifetime's evidence concerning your nose can be negated by just a few seconds of intermittent tactile input.

Another striking instance of a 'displaced' body part can be demonstrated by using a dummy rubber hand. The dummy hand is placed on in front of a vertical partition on a table. The subject places his hand behind the partition so he cannot see it. The experimenter now uses his left hand to stroke the dummy hand while at the same time using his right hand to stroke the subject's real hand (hidden from view) in perfect synchrony. The subject soon begins to experience the sensations as arising from the dummy hand (Botvinik & Cohen 1998).

We found that it is even possible to 'project' tactile sensations onto inanimate objects such as tables or shoes that do not resemble body parts (Ramachandran & Hirstein 1998). The subject is asked to place his right hand below a table surface (or behind a vertical screen) so that he cannot see it. The experimenter then uses his right hand to randomly stroke and tap the subject's right hand (under the table or behind the screen) and uses his left hand to simultaneously stroke and tap a shoe placed on the table in perfect synchrony. (A tablecloth can be used to make sure that the experimenter's right hand and the subject's own hand are completely invisible to the subject). After 10–30 seconds, the subject starts developing the uncanny illusion that the sensations are now coming from the shoe and that the shoe is now part of his body (Ramachandran *et al.* 1998). We have seen this effect in about half of the subjects tested. Even those who do not initially experience the effect often do so after several minutes of stimulation. On some occasions, when the experimenter had accidentally made a longer excursion on the shoe than on the hidden hand, the subjects exclaimed that felt that their hand had become elongated as well!

But how can we be sure that the subjects are not simply using a figure of speech when they say 'I feel that the sensations are arising from the shoe'? To rule out this possibility, we waited until the subjects started 'projecting' their sensations onto the shoe and then simply hit the shoe with a giant rubber hammer as they watched. Remarkably, the subjects not only winced visibly but also registered a strong increase in skin conductance when we measured their galvanic skin response (GSR) (Ramachandran *et al.* 1998). Such a change was not seen in a 'control' condition in which the shoe and hand were stroked non-synchronously before the shoe was hit. The surprising implication of these observations is that the shoe was now assimilated into the subject's own body image; that he or she was not just being metaphorical when asserting that the shoe feels like the hand. Indeed, we can conclude that the shoe is now 'hooked up', in some sense, to the subject's limbic system so that any threat to the shoe produces emotional arousal (Ramachandran & Hirstein 1998).

(f) *Phantom limbs and sensory codes*

According to the 'labelled lines' theory of sensory coding, every neuron in the sensory pathways, e.g. 3b, or S2 or area 17, has a specific 'hardwired' signature, i.e. it signals a highly specific percept such as 'light touch on my right elbow.' It is obvious, however, that sensory coding

cannot be based exclusively on an endless hierarchy of labelled lines and maps. At some stage, 'pattern coding,' i.e. the total spatio-temporal pattern of activity must take over and determine what the subject actually perceives.

The basic presumption of the remapping hypothesis of referred sensations is that the labelled lines have been switched so that the same sensory input now activates a novel set of labelled lines (e.g. the face input activates 'hand neurons' in S1). As we have seen, this is consistent with both the MEG changes in sensory maps that we observed as well as with the referred sensations reported by many patients (see also Kew *et al.* 1997). But it is possible that the subsequent changes in pattern-coding somewhere further along in the nervous system eventually lead to the deletion of these anomalous sensations in some patients.

The word 'remapping' carries connotations of actual anatomical change, whereas most of the evidence points to the unmasking or disinhibition of pre-existing pathways (see, for example, Ramachandran *et al.* (1992*a,b*), Ramachandran & Rogers-Ramachandran (1996) and Borsook *et al.* (1997)). A more theory-neutral word, such as 're-routing', might be preferable, to indicate that information from a specific location on the sensory surface (e.g. face or shoulder) is now shunted or re-routed so as either to evoke new patterns of neural activity, or to activate new anatomical sites that have different perceptual signatures and therefore lead to novel sensations. But in either case, the findings imply that there must have been a relatively permanent or stable change in the processing of sensory signals by the adult brain.

3. CAPGRAS SYNDROME

The disorder called Capgras delusion is one of the rarest and most colourful syndromes in neurology (Capgras & Reboul-Lachaux 1923; Young *et al.* 1993). The most striking feature of this disorder is that the patient comes to regard close acquaintances, typically either his parents, children, spouse, or siblings, as 'impostors'; in other words he might claim that the person 'looks like' or is even 'identical' to his mother, but really is not his mother. Although frequently seen in psychotic states, more than a third of the documented cases of Capgras syndrome have occurred in conjunction with traumatic brain lesions, suggesting that the syndrome has an organic basis. The remarkable thing about these patients is that they are relatively intact in other respects: they are mentally lucid, their memory is normal, and other aspects of their visual perception are completely unaffected.

Because this is a very bizarre syndrome, it is hardly surprising that many theories have been put forth to explain it. The most popular view of Capgras syndrome, oddly enough, is a Freudian view. The idea goes something like this: the patient, like all of us, felt a strong sexual attraction for his mother in early childhood (the so-called Oedipus complex) and therefore felt strong jealousy toward his father as a rival for sexual attention. Fortunately, all of this is repressed as the child grows up, so no normal person would ordinarily be attracted to his mother or father. However, as a result of the blow to the head, somehow these latent sexual impulses are unmasked and come flaming to the surface. Suddenly and inexplicably, the patient finds himself sexually attracted

to his mother, and he says 'my God, if this is my mother, how come I'm sexually attracted to her? This must be some other strange person.'

This argument is ingenious, as indeed most Freudian arguments are. However, I recently encountered a patient who experienced the Capgras delusion not only with his parents, but also with his pet dog (i.e. the patient claims that his pet poodle has been replaced with a duplicate). How does the Freudian argument apply to this particular case? I started thinking about this and realized that there was a much simpler explanation for this syndrome, which is as follows. The messages from this area of the brain are usually transmitted to the limbic system, which is composed of clusters of cells concerned mainly with the perception, experience and expression of emotions. The 'gateway' to the limbic system is the amygdala. Thus, the visual centres of the brain in the temporal lobes send their information to the amygdala, which assesses the emotional significance of the incoming visual input and then transmits this to other limbic structures where these emotions are 'experienced'. Is it possible that in this patient there has been a disconnection between the face area of the temporal lobes and the part of the temporal lobes concerned with the experience of emotion? Perhaps the face area and the amygdala are both intact, but the two areas have been disconnected from each other. As a result of this, the patient can recognize people's faces (this is what makes the syndrome different from prosopagnosia). When he looks at his mother, however, even though he realizes that she resembles his mother, he does not experience the appropriate warmth, and therefore says 'well, if this is my mother, why is it I'm not experiencing any emotion? This must be some strange person.' However bizarre this may seem to you and me, it is the only interpretation, perhaps, that makes sense to him with this peculiar disconnection. This interpretation of Capgras syndrome is similar to that proposed by Young *et al.* (1993), except that they postulate a disconnection between ventral and dorsal 'stream' pathways rather than an disconnection between the amygdala and the inferotemporal cortex.

How can a hypothesis of this kind be tested? What Bill Hirstein and I did was to obtain GSRs from one of these patients. When a normal person looks at something emotionally salient like his mother or father, this message is transmitted from the visual centres of the brain to the amygdala, where the emotional significance of this visual event is judged. The message goes to the limbic system and then to the hypothalamus and from there to the autonomic nervous system. This produces sweating, an increase in heart rate, an increase in blood pressure, and other signs of noradrenergic activity. It turns out that the sweating can be measured by simply measuring changes in skin resistance. This is the so-called GSR, which forms the basis of the lie-detection test, and it is a fairly reliable index of emotional arousal. Almost all normal people give a strong GSR when they see their mother, but when we tested our patient, D.S., who had Capgras syndrome, we found that when he looked at a photograph of his mother there was very little change in GSR, supporting the disconnection hypothesis (Ramachandran 1996; Hirstein & Ramachandran 1997). It is important to emphasize that D.S. had no problem in seeing that the photograph looked like his mother and he had no

problem with experiencing emotions in general. During the interview he experienced joy, fear, impatience, boredom, and all other emotions that one would normally expect a human being to experience, because his limbic system was unaffected. What was deranged is the communication between vision and emotion. (Consistent with this, D.S. never experienced this delusion when talking to patients on the telephone.)

One objection to our interpretation might be that patients with bilateral lesions of the amygdala do not suddenly develop Capgras syndrome. The reason might be that with the entire amygdala damaged, the patient's brain has no baseline for comparison: no stimulus evokes a GSR. To develop the Capgras delusion you may need a loss of GSR to certain categories of sensory images but not to others. Consistent with this, our patient D.S. showed a normal GSR to threat gestures and loud noises, suggesting that his amygdala was probably intact.

Patient D.S.'s tendency to duplicate his parents is surprising enough, but even more remarkably he would sometimes duplicate himself! On one occasion when shown an old photograph of himself he said it was 'a different person . . . see, he has a moustache and I don't'. Or sometimes, during conversation, he would refer to 'the other David' (he once accused his employer of sending the cheque to the other David). Philosophers often remind us that if there is any one aspect of existence that is completely beyond question, it is the fact that 'I exist' as a single conscious individual. But it seems that even this axiomatic foundation of one's existence is called into question by David.

I mention Capgras syndrome because it is a striking example of how a completely bizarre, seemingly incomprehensible psychiatric syndrome can be at least partly understood in terms of the known neuroanatomy of the temporal lobes. This idea can then be tested using a relatively simple technique, GSR, to show that something like this might in fact be going on in the brain.

4. NEUROLOGY OF LAUGHTER AND HUMOUR

Laughter and humour are surely just as important a part of our conscious experience as any other trait. A Martian ethologist visiting our planet would be very puzzled by the fact that every now and then groups of humans produce an explosive fit of rhythmic sounds, in certain social situations. Why do people do this? Can studying neurological syndromes help to answer this question?

A 'disconnection' syndrome that is just as fascinating as Capgras syndrome is a disorder called 'pain asymbolia', in which a patient with damage to the insular cortex will say he can feel the pain of a pinprick but that 'it no longer hurts'. I was amazed to notice recently that a patient whom I recently saw in India with this disorder not only failed to experience the aversive quality of the pain but also started laughing in response to a pin-prick! So here again is a mystery: why would an intelligent, sane human being begin to giggle when poked with a needle? Is this not the ultimate irony: laughter in response to pain? Can this syndrome help us to investigate the evolutionary origins of laughter and humour?

Cultural factors undoubtedly influence humour and what people find funny—the English are said to have a

sophisticated 'sense of humour', whereas the Germans and the Swiss rarely find anything amusing. But even if this is true, might there still be some sort of 'deep structure' underlying all humour? The details of the phenomenon vary from culture to culture and are influenced by the way you were raised, but this does not mean that there is no genetically specified mechanism for laughter, a common denominator underlying all types of humour. Indeed, many people have suggested that such a mechanism does exist and theories on the biological origins of humour and laughter have a long history, going all the way to Schopenhauer and Kant, two singularly humourless German philosophers.

Why are jokes funny? Despite all their surface diversity, most jokes and funny incidents have the following logical structure. Typically you lead the listener along a garden path of expectation, slowly building up tension. At the very end, you introduce an unexpected twist that entails a complete reinterpretation of all the preceding results; moreover, it is critical that the new interpretation, though wholly unexpected, makes as much 'sense' of the entire set of facts as did the originally 'expected' interpretation. In this regard, jokes have much in common with scientific creativity, with what Thomas Kuhn calls a 'paradigm shift' in response to a single 'anomaly'. (It is probably no coincidence that most creative scientists have a great sense of humour.) Of course, the anomaly in the joke is the traditional punch line and the joke is 'funny' only if the listener 'gets' the punch line by seeing in a flash of insight how a completely new interpretation of the same set of facts can incorporate the anomalous ending. The longer and more tortuous the garden path of expectation, the 'funnier' the punch line when finally delivered. Good comedians make use of this principle by taking their time to build up the tension of the story line, for nothing kills humour more surely than a premature punch line.

However, although the introduction of a sudden twist at the end is necessary for the genesis of humour, it is certainly not sufficient. My plane is about to land in San Diego and I fasten my seat belt and get ready for touch-down. The pilot suddenly announces that the 'bumps' that he (and I) had earlier dismissed as air turbulence are really due to engine failure and that we need to empty fuel before landing. A paradigm shift has occurred in my mind, but this certainly does not make me laugh. Rather it makes me orientate and prepare for action to cope with the anomaly. Or consider the time I was staying at some friends' house in Iowa City. They were away and I was alone in unfamiliar surroundings. It was late at night and just as I was about to doze off, I heard a 'thump' from downstairs. 'Probably the wind,' I thought. 'There are no burglars in this neighbourhood.' After a few minutes there was another thud, louder than the one before. Again I 'rationalized' it away and went back to sleep. Twenty minutes later I heard an extremely loud, resounding 'bang' and leapt out of bed. What was happening? A burglar perhaps? Naturally, with my limbic system activated, I 'orientated', grabbed a flashlight and ran down the stairs. Nothing funny so far. Then, suddenly I noticed a large flower vase in pieces on the floor and a large tabby cat right next to it—the obvious culprit! This time I started laughing because I realized that the 'anomaly' I

had detected and the subsequent paradigm shift were of trivial consequence. All of the facts could now be explained in terms of the cat theory rather than the ominous burglar theory.

On the basis of this example, we can sharpen the definition of humour and laughter. When a person strolls along a garden path of expectation and there is a sudden twist at the end that entails a complete reinterpretation of the same facts and the new interpretation has trivial rather than terrifying implications, laughter ensues.

But why laughter? Why this explosive, repetitive sound? To an ethologist, on the other hand, any stereotyped vocalization almost always implies that the organism is trying to communicate something to others in the social group. Now what might this be in the case of laughter? I suggest that the main purpose of laughter might be for the individual to alert others in the social group (usually kin) that the detected anomaly is trivial, nothing to worry about. The laughing person in effect announces her discovery that there has been a false alarm, that the rest of you chaps need not waste your precious energy and resources responding to a spurious threat (or, perhaps, also to playfully censure minor violations of social taboos and norms).

This 'false-alarm theory' of humour might also explain slapstick. You watch a man—preferably one who is portly and self-important—walk down the street when suddenly he slips on a banana peel and falls down. If his head were to hit the pavement and split open his skull, you would not laugh as you saw blood spill out: you would rush to his aid or to the nearest telephone to call an ambulance. But if he got up casually, wiped the remains of the fruit from his face and continued walking, you would probably burst out laughing, thereby letting others standing nearby know that they need not rush to his aid.

The smile, too, may have similar evolutionary origins, as a 'weaker' form of laughter. When one of your ancestral primates encountered another individual coming towards him from a distance, he might have initially bared his canines in a threatening grimace on the fair assumption that most strangers are potential enemies. Upon recognizing the individual as 'friend' or 'kin', however, he might abort the grimace half way, thereby producing a smile, which in turn might have evolved into a ritualized human greeting: 'I know you pose no threat and I reciprocate.' Thus, in my scheme, a smile is an aborted orientating response, in the same way that laughter is.

Let us return now to pain asymbolia, which in my view provides strong support for the false-alarm theory. The insular cortex ordinarily receives sensory input including pain from the skin and viscera and sends its output to parts of the limbic system (such as the cingulate gyrus) so that one begins to experience the strong aversive reaction—the agony—of pain. Now imagine what would happen if the damage were to disconnect the insula from the cingulate. One part of the person's brain (the insula) tells him, 'here is something painful, a potential threat' while another part (the cingulate gyrus of the limbic system) says a fraction of a second later, 'oh, don't worry, this is no threat after all.' Thus the two key ingredients—threat followed by deflation—are present and the only way for the patient to resolve the paradox is to laugh, just as my theory would predict.

The same line of reasoning might help explain why people laugh when tickled. You approach a child, hand stretched out menacingly. The child wonders, 'will he hurt me or shake me or poke me?' But no, your fingers make light, intermittent contact with her belly. Again, the recipe is present and the child laughs, as if to inform other children, 'he doesn't mean harm, he's only playing!' This, by the way, might help children to practice the kind of mental play required for adult humour. In other words, what we call 'sophisticated cognitive' humour has the same logical form and therefore piggybacks on the same neural circuits: the 'threatening but harmless' detector that involves the insula, cingulate and hypothalamus. Such co-opting of mechanisms is the rule rather than the exception in the evolution of mental and physical traits. (However, in this case the co-opting occurs for a related, higher level function rather than for a completely different function.)

5. SUMMARY AND CONCLUSIONS

Neurological syndromes can give us novel insights into human conscious experience. The Capgras delusion, for example, provides clues to understanding the link between visual perception and emotions, as long championed by Young *et al.* (1993), and the pain-asymbolia syndrome provides strong evidence for our evolutionary false-alarm theory of laughter and humour. Thus, two seemingly incomprehensible syndromes become comprehensible in terms of the known neural circuitry of the brain, especially when viewed in an evolutionary context.

The experiments on referred sensations in phantom limbs are important for two reasons. First, they suggest that, contrary to the static picture of brain maps provided by neuroanatomists, topography is extremely labile. Even in the adult brain, massive reorganization can occur over extremely short periods, and referred sensations can therefore be used as a 'marker' for plasticity in the adult human brain. Second, the findings allow us to relate perceptual qualia (subjective sensations) to the activity of brain maps and to test some of the most widely accepted assumptions of sensory psychology and neurophysiology, such as Muller's Law of specific nerve energies, 'pattern coding' as opposed to 'place coding' (i.e. the notion that perception depends exclusively on which particular neuron fires rather than on the overall pattern of activity), and, more generally, to understand how neural activity leads to conscious experience. For instance, after arm amputation, patients usually have dual sensations: sensations are experienced in both the face and the hand, presumably because two separate points are activated on the cortical map. However, after fifth nerve section, the patient feels the sensation only on the face when the hand is touched (Clarke *et al.* 1996). Perhaps there is an initial 'overshoot' during remapping so that the anomalous input from the hand to the face territory actually comes to dominate perception and masks or suppresses the 'real' sensation from the hand.

The experiments with mirrors have three implications. First, they might be clinically useful in alleviating abnormal postures and spasms in phantoms. Indeed, it is not inconceivable that even other neurological syndromes

such as focal dystonias, dyspraxis and hemiparesis might be caused, at least in part, by a temporary 'inhibition' of sorts and might therefore benefit from visual feedback provided by the mirror. Second, it suggests that the modular, hierarchical, 'bucket brigade' model of the brain popularized by computer engineers needs to be replaced by a more dynamic view of the brain in which there is a tremendous amount of back-and-forth interaction between different levels in the hierarchy and across different modules. The fact that the mere visual appearance of the moving phantom feeds all the way back from the visual to the somatosensory areas of the brain to relieve a spasm in a non-existent hand shows how extensive these interactions can be. Third, the resurrection of long-lost phantoms in some patients (Ramachandran & Hirstein 1998), the 'phantom nose' effect, and the projection of sensations onto chairs and shoes, suggest that your body image, despite all its appearance of durability and permanence, is in fact a purely transitory internal construct, a mere shell that your brain creates temporarily for passing on your genes to the next generation.

For in and out, above, about, below,
'Tis nothing but a Magic Shadow-show
Play'd in a Box whose Candle is the Sun,
Round which we Phantom Figures come and go'
The Rubaiyat of Omar Khayyam

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