

PHANTOM LIMBS, NEGLECT SYNDROMES, REPRESSED MEMORIES, AND FREUDIAN PSYCHOLOGY

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I. Introduction

In this essay I would like to consider two of the most fascinating syndromes in clinical medicine: phantom limbs and somatoparaphrenic delusions. In the case of phantom limbs, the patient continues to experience a limb that has long ceased to exist, whereas in somatophrenia he insists that his paralyzed limb does not belong to him. He may even attribute his limb to his spouse or to his physician. These two syndromes have long been regarded as enigmatic clinical curiosities. My goal, how-

ever, will be to bring them into the respectable arena of modern neuroscience and to point out that they illustrate certain important principles concerning the functional organization of the normal human brain.

II. Phantom Limbs as a Perceptual Marker for Somatosensory Plasticity

When I began my career as a medical student nearly 15 years ago, I was taught that no new neural connections can be formed in the adult mammalian brain. Once connections have been laid down in fetal life, or in early infancy, it was assumed that they hardly change later in life. It is this stability of connections in the adult brain, in fact, that is often used to explain why there is usually very little functional recovery after damage to the nervous system and why neurological diseases are so notoriously difficult to treat. In this article I will present some evidence suggesting that we may need to revise these views radically.

It is known that a complete somatotopic map of the entire body surface exists in the somatosensory cortex of primates (Mountcastle, 1957; Merzenich *et al.*, 1983; Kaas *et al.*, 1981; Jones, 1982). In a series of pioneering experiments, Merzenich *et al.* (1984) amputated the middle finger of adult primates and found that within 2 months the area in the cortex corresponding to this digit starts to respond to touch stimuli delivered to the adjacent digits, i.e., this area is "taken over" by sensory input from adjacent digits. If more than one finger was amputated, however, there was no take over beyond about 1 mm of cortex. Merzenich and co-workers concluded from this that the expansion is probably mediated by arborizations of thalamo-cortical axons that typically do not extend beyond 1 mm.

This figure—1 mm—was often cited as the fixed upper limit of reorganization of sensory pathways in adult animals (Calford, 1991). A remarkable experiment performed by Pons *et al.* (1991), however, suggests that this view might be incorrect. They found that after long-term (12 years) deafferentation of one upper limb, the cortical area originally corresponding to the hand gets taken over by sensory input from the face. The cells in the "hand area" now start responding to stimuli applied to the lower face region! Because this patch of cortex is over 1 cm in width, we may conclude that sensory reorganization can occur over at least this distance—an order of magnitude greater than the original 1-mm "limit."

A. PERCEPTUAL CORRELATES OF PLASTICITY IN HUMANS

Despite the wealth of physiological experiments demonstrating striking plasticity in the primary sensory areas of primates, there has been almost no attempt to look directly for the behavioral consequences of this reorganization. The observation by Pons *et al.* (1991), for example, makes the curious prediction that if one were to touch a monkey's face after long-term deafferentation, the monkey should experience the sensations as arising from the hand as well as from the face. To test this prediction, we studied the localization of sensations in several adult human subjects who had undergone amputation of an upper limb. Two of these subjects (VQ and WK) have been described in detail elsewhere (Ramachandran *et al.*, 1992a,b, 1993; Ramachandran, 1993). In this essay, I will briefly summarize our findings for these two patients and will also describe some preliminary results from new patients (FA and DS).

1. Patient VQ

Patient VQ was an intelligent, alert 17-year-old male whose left arm was amputated 6 cm above the elbow about 4 weeks prior to our testing him. He experienced a vivid phantom hand that was "telescoped," i.e., it felt like it was attached just a few centimeters below his stump and was pronated. We studied localization of touch (and light pressure) in this patient using a cotton swab that was brushed at various randomly selected points on his skin surface. His eyes were shut during the entire procedure and he was simply asked to describe any sensations that he felt and to report the perceived location of these sensations. Using this procedure, we found that even stimuli applied to points remote from the amputation line were often systematically mislocalized to the phantom arm (Fig. 1). Furthermore, the distribution of these points was not random (Ramachandran *et al.*, 1992b). There appeared to be two clusters of points, with one cluster being represented on the lower part of the face ipsilateral to the amputation. There was a systematic one-to-one mapping between specific regions on the face and individual digits (e.g., from the cheek to the thumb, from the philtrum to the index finger, and from the chin to the fifth finger). Typically, the patient reported that he simultaneously felt the cotton swab touching his face and a "tingling" sensation in an individual digit. By repeatedly brushing the swab on his face, we were even able to plot "receptive fields" (or "reference fields") for individual digits of the (phantom) left hand on his face surface (Fig. 1). The margins of these fields were remarkably sharp and stable over successive trials. Stimuli applied to other parts of the body such as the



FIG. 1. Regions on the left side of the face of patient VQ, which elicited precisely localized referred sensations in the phantom digits. Reference fields, regions that evoke referred sensations, were plotted by brushing a cotton swab repeatedly on the face. The region labeled T always evoked sensations in the phantom thumb; P denotes the "pinkie" (fifth finger); I, the index finger; and B, the ball of the thumb. This patient was tested 4 weeks after amputation.

tongue, neck, shoulders, trunk, and axilla were never mislocalized to the phantom hand and no referred sensations were ever felt in the other (normal) hand. There was, however, one specific point on the contralateral cheek that always elicited a tingling sensation in the phantom elbow.

The second cluster of points that evoked referred sensations was found about 7 cm above the amputation line. Again there was a systematic one-to-one mapping, with the thumb being represented medially on the anterior surface of the arm and the fifth finger laterally, as if to mimic the pronated position of the phantom hand.

We repeated the whole procedure again after 1 week and found a very similar distribution of points. We conclude, therefore, that these one-to-one correspondences are stable over time—at least over the 1-week period that separated our two testing sessions (but see below).

2. Patient WK

In testing the second patient (WK) we found a very similar pattern of results, although there were some interesting differences as well. This patient had a right forequarter disarticulation, i.e., his entire right arm

and right scapula were removed. We tested him exactly 1 year after amputation.

We had WK close his eyes and we firmly rubbed the skin of his right lower jaw and cheek with one of our fingers or the tip of a ballpoint pen. A representation of the entire phantom arm was found on the ipsilateral face, with the hand being represented on the anterior lower jaw, the elbow on the angle of the jaw, and the shoulder on the temporomandibular joint. Again, as in patient VQ, there appeared to be a precise and stable point-to-point correspondence between points on the lower jaw and individual digits.

A second cluster of reference fields representing the hand was found just below the axilla. Because this region is close to the line of amputation, it may be analogous to the cluster of points we found on the upper arm of VQ. In this region, even a cotton swab was effective in eliciting referred sensations in the thumb, forefinger, fifth finger, or palm. And last, there was also a third cluster of points near the right nipple, and the arrangement of these points also showed some hint of topography. Thus it would appear that there is a tendency toward the spontaneous emergence of multiple somatotopically organized maps even in regions remote from the line of amputation. The exact mechanism by which such maps are formed remains an interesting question for future research.

We have now studied nine patients after upper limb amputation and found that sensations were referred from the face to the phantom arm in only four of them. The cluster(s) of points just proximal to the line of amputation, however, was seen in eight patients. We shall discuss the reason for this variability in a later section.

3. *Patient FA*

FA lost his right arm as a result of an accident on a fishing boat in 1982. His arm had been amputated about 8 cm below the elbow crease. He experienced a very vivid phantom hand that was usually telescoped, i.e., the hand felt like it was directly attached to the stump with no intervening forearm. He could, however, voluntarily extend his hand so that it acquired a subjectively normal length and indeed he could even attempt to grasp objects, fend off blows, or break a fall with his phantom.

FA was one of the subjects we examined who did not initially have a map on his face. As in the other patients, however, he had points near the amputation line that elicited referred sensations. After carefully mapping these points, we established that there were two distinct somatotopic representations that were almost completely identical to each other (Fig. 2). One of these extended from the amputation line to about 3 cm below the elbow, whereas the second one extended from about 6 cm

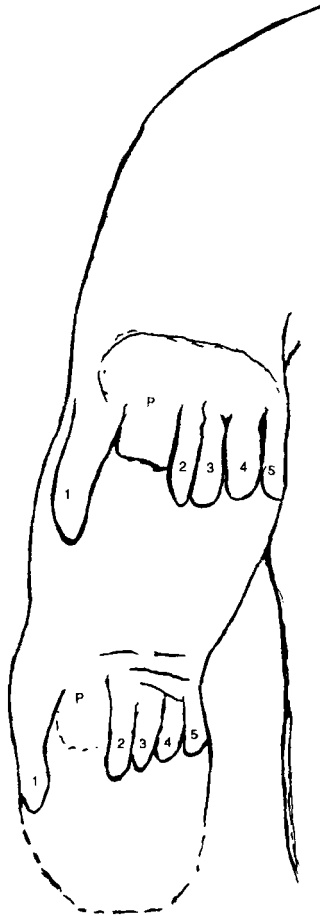


FIG. 2. Somatotopic maps of referred sensations in patient FA. Notice that there are two distinct maps, one close to the line of amputation and a second one 6 cm above the elbow crease. The maps are almost identical except for the absence of fingertips in the upper map. When patient FA imagined he was pronating his phantom, the entire upper map shifted in the same direction by about 1.5 cm (see text). 1, Thumb; 2, index finger; 3, middle finger; 4, ring finger; 5, fifth finger. These reference fields usually elicited sensation in the glabrous portions of these digits. The dorsal surface of the hand was represented on the dorsolateral part of the upper arm lateral to the palm (P) and thumb representations. No referred sensations in the phantom could be elicited by stimulating the skin region in between these two maps.

above the crease to about 14 cm. Stimuli applied to points in between these two maps were completely ineffective in producing referred sensations, even though skin sensitivity was normal in this region. Note that the two maps are very similar except for the absence of fingertips in the second map.

4. *Patient DS*

Patient DS had a brachial avulsion following a motorcycle accident, and his arm was amputated a year after the accident. He experienced a vivid phantom that felt "paralyzed"—as if to mimic the paralysis that preceded the amputation. We mapped the distribution of reference fields in this patient extensively on three separate occasions, the first two separated by 24 hours and the third one after 6 months. The arrangement of reference fields is shown in Fig. 3a. Notice the topographic arrangement of digits on the face (e.g., digits 1 to 4 are neatly laid out on the zygoma). The thumb receptive field was especially large, as in some of our other patients. Curiously, there was also a second map of sorts on the mandible, with the digits following approximately the same sequence as on the zygoma. (Stimulating the buccal region in between these two maps elicited diffuse tingling on the palm.) Thus, each digit appeared to be represented twice, but the reference field on the zygoma usually corresponded to the distal interphalangeal joint, whereas the one on the mandible elicited sensations from the base of the digit.

The map remained stable during the first two testing sessions, but when we saw the patient again after 6 months, there had been some small but noticeable changes (Fig. 3b). In particular, the thumb region appeared to have expanded to stretch across the entire mandible—the base of the thumb near the ramus and the tip near the symphysis menti. It was unclear why the map had changed in this manner, but it may have occurred as a result of changing patterns of sensory input (and spontaneous activity) from the face and from the stump. It might be interesting to test this hypothesis by actually stimulating a specific region of the map (e.g., the index finger reference field) for a few days to see if this increases the size of that reference field (see also Section III,E). A second map was found in the region of the deltoid muscle and this, too, was topographically organized. Unlike the face map, however, it remained stable across all three testing sessions (Fig. 3c).

An especially convincing way of demonstrating topography in patient DS was as follows. When the cotton swab was moved continuously from the angle of the mandible to the symphysis menti, the referred sensation also felt like "it was moving from the ball of the thumb to the tip in an arclike motion." This observation was replicated several times. Also, if

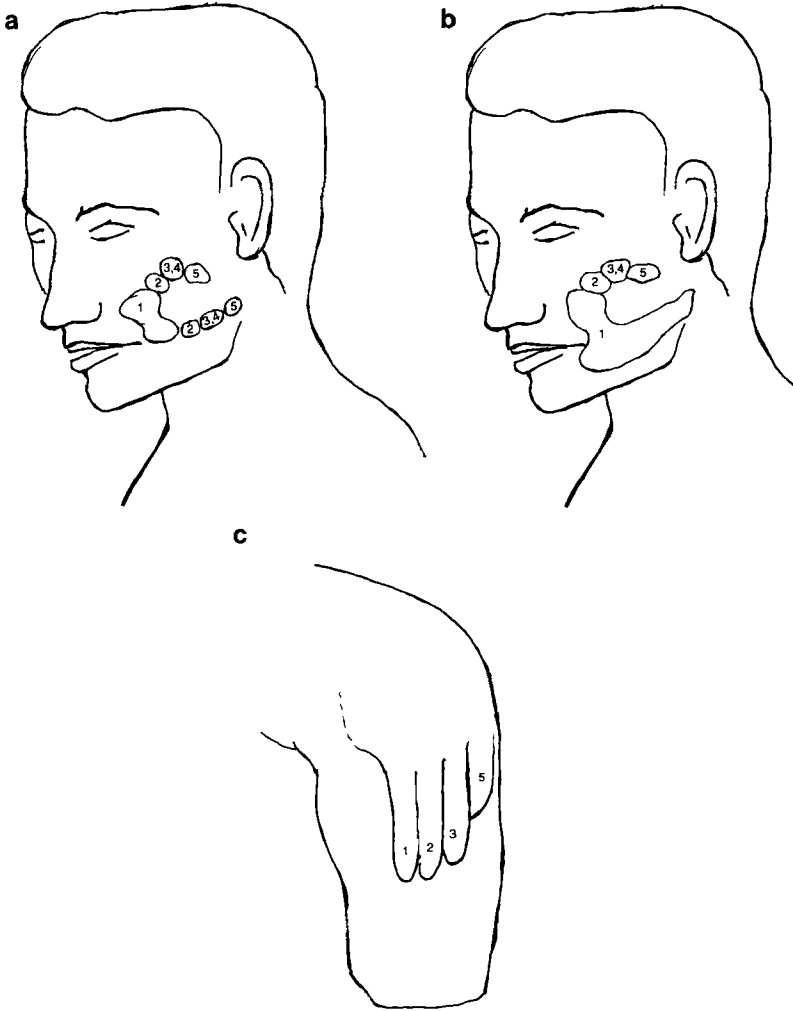


FIG. 3. (a) Distribution of reference fields in patient DS. Notice the prominent representation of the thumb (1), which we have seen in several patients, and the roughly topographic arrangement of digits 2, 3, 4, and 5 on the face. This pattern was nearly identical 24 hours later, but after 6 months the representations of some of the digits had changed noticeably (b). This may occur as a result of sensory input and spontaneous activity from the face (and stump) continuously remodeling neural connections in S1. If this interpretation is correct, then phantom limbs might provide a valuable preparation for studying the manner in which sensory maps emerge and change in the adult nervous system. (c) The second map in the region of the deltoid muscle. Patient DS's arm always felt completely extended and paralyzed; it was never telescoped into the stump.

a short excursion was made on the jaw, the excursion on the hand was correspondingly short. Curiously, however, if the direction of motion of the cotton swab was reversed, DS reported a diffuse tingling in the thumb; there was no reversal of the direction of motion on the thumb. Thus, distance and velocity were accurately referred but direction was not.

A similar effect could also be evoked by moving the cotton swab across the digits' reference fields on the deltoid muscle, and in this case reversing the direction of the cotton swab also reversed the direction of motion on the phantom hand.

Finally, it was our general impression that in patient DS as well as in other patients, the topography was usually much more precise in the map proximal to the stump than on the face. The reason for this difference is not clear.

B. MODALITY-SPECIFIC EFFECTS

The neural pathways that mediate the sensations of pain, warmth, and cold are quite different from those that carry information about touch from the skin surface to the brain (Kenshalo *et al.*, 1971; Landgren, 1960; Kreisman and Zimmerman, 1971). We wondered whether the remapping effect reported by Pons and his collaborators occurs separately in each of these pathways or only in the touch pathways. To find out, we tried placing a drop of warm water on VQ's face. He felt the warm water on his face, of course, but remarkably he reported (without any prompting) that his phantom hand also felt distinctly warm.¹ On one occasion when the water accidentally trickled down his face, he exclaimed, with surprise, that he could actually feel the warm water trickling down the length of his phantom arm! We have now seen this effect in three patients, two patients after upper limb amputation and one after an avulsion of the brachial plexus. The latter patient was better able to use his normal hand to trace out the exact path of the illusory trickle along his paralyzed arm as a drop of cold water flowed down his face. (The distance traversed by this illusory trickle was about five times the

¹ Modality-specific referral from the stump (but not the face) was also noticed by William James (1887) and Wier Mitchell (1871). This finding, however, is open to multiple interpretations, e.g., due to reinnervation of the stump by severed axons (which may also be modality specific) or due to the fact that phantom is often "telescoped" so that its phenomenal location is superimposed on the stump.

distance on the face, as one might expect from the obvious differences in cortical magnification for the face and arm representations.) Finally, in patient DS, a vibrator placed on the jaw evoked a compelling sensation of vibration in the phantom hand.

How does the point-to-point referral of temperature sensations compare with that of touch? To explore this, we tried applying a drop of warm (or cold) water on different parts of the face of patient DS and found that the heat or cold was usually referred to individual fingers so that there was a sort of crude map of referred temperature that was roughly superimposed on the touch map (e.g., touching the thumb reference field on the face with warm water evoked warmth in the thumb alone, whereas touching the fifth finger part of the map evoked a warm sensation confined to that finger). To make sure that these effects were not simply due to simultaneous activation of touch receptors, we also tried touching the thumb reference field on the face with warm water while simultaneously applying tepid water to the fifth finger region of the map. The patient then reported that he could feel the touch in both digits—as expected—but that the warmth was felt only in the thumb. Reversing the stimulation on the face produced corresponding reversal in the phantom. If these preliminary results are confirmed, they would imply that there are independent modality-specific reference fields for touch, heat, and cold on the face and that these reference fields are usually in approximate spatial registration.

C. COMBINING REFERRED SENSATIONS

These observations raise an interesting question. What would happen if dissimilar stimuli were applied to the thumb reference field on the face versus the corresponding reference field on the upper arm? For example, one could put hot water on the thumb region of the face and ice-cold water on the thumb region of the arm. Would the sensation in the phantom thumb then be an average of the two, so that it felt neutral? How would the brain resolve the conflict? The result was clear. On every occasion that we tried the test, the patient reported a clear alternation of the sensations “a flash of heat followed by a flash of cold, followed once again by the heat,” etc. It would be interesting to see what would happen if the two temperature sensations were closer in degree, e.g., tepid and very warm (rather than cold and hot). This might lead to an averaging or blending of the sensations rather than to an alternation.

D. "LEARNED" PARALYSIS OF PHANTOM LIMBS

Another puzzling observation we made also deserves mention. Most patients who have lost a limb experience a very compelling phantom arm or leg that they can "move" voluntarily. (As we shall see later, we have also studied a patient who was congenitally missing both arms, but nevertheless experienced vivid phantom arms.) Yet, we noticed that if the patient had a preexisting paralysis of the arm caused by a peripheral nerve lesion (e.g., a brachial plexus avulsion or infiltration by carcinoma), then the patient usually complained that although he experienced a phantom arm, he could not voluntarily move the phantom—it felt paralyzed and always assumed the same position and length as that of the paralyzed arm before amputation (e.g., neither patient DS nor patient WK could move his phantom voluntarily, whereas FA and VQ had no difficulty). This raises an interesting question. Why does the mere fact that a limb was paralyzed (say) a few months before amputation cause the phantom to be paralyzed, whereas patients who have lost the whole limb, and therefore never moved it for many years, can still imagine voluntary movement? How can a phantom be paralyzed?

The answer is surely that in the former case the patient has learned that the limb is paralyzed, i.e., every time he tried to move his limb there was both visual feedback and proprioceptive feedback from the limb muscles informing him that the limb was not following the command; the brain was receiving evidence that the limb was paralyzed. In the amputees, on the other hand, when the subject tried to move the limb, there was simply no feedback from the limb that either confirmed or contradicted the command signals. Thus, it looks as though the contradictory signals that were sent to the brain during the months preceding the amputation were somehow "wired" into the brain so that there was a permanent memory trace of the paralysis left in place.

E. RELEVANCE TO STROKE REHABILITATION: THE VIRTUAL REALITY BOX

If the phantom is paralyzed because the patient's brain has learned that it is paralyzed, then would it be possible to "unlearn" the paralysis so that the limb starts moving once again? Liz Franz and I realized that one way to achieve this might be to encourage the patient simply to perform a mirror-symmetric movement with his other normal hand because this might facilitate the corresponding neural circuits in equiva-

lent locations in the other hemisphere (some phantom limb patients do, in fact, spontaneously report that movement of the phantom can be facilitated by making a corresponding movement with the other hand; e.g., patient FA could not ordinarily flex and extend his phantom hand but could do so if his normal hand performed a similar movement at the same time).

A cleverer method might be to use a “Virtual Reality Box” to trick the patient into thinking that his phantom was moving (Ramachandran, 1994). A mirror is placed vertically on a table in front of the patient. (Imagine a book on the table; if you opened the front cover so that it stood vertically, you would be mimicking the position and orientation of the mirror.) The patient is then asked to view the reflection of his normal hand in the mirror in order to create the illusion that he now has two hands. He would have to move his normal hand until its mirror reflection was superimposed on the felt position of his phantom hand. If he now attempts to execute mirror-symmetric movement with both of his hands, he will see his phantom come to life and move as if it was obeying his commands! Would this revive the lifeless phantom so that it no longer felt paralyzed?

I recently tried this experiment on patient DS and the result was quite astonishing. Just a few seconds after he had tried the “mirror therapy,” he exclaimed, with considerable surprise, that he could vividly feel (and not just see) his phantom arm moving as though it was under his voluntary control again! “Mind-boggling,” he said. “My arm is plugged in again; it’s as if I am back in the past. I can actually *feel* my arm moving, doctor. It no longer feels like it’s lying lifeless in a sling.” I then removed the mirror and verified that, as before, he could no longer feel his phantom moving even if he closed his eyes and tried mirror-symmetric movements. (“It feels frozen again,” he said, “as though in a plaster cast.”) Patient DS also tried moving his index finger and thumb alone while looking in the mirror but, this time, the phantom thumb and index finger remained paralyzed—they were not revived. (This is an important observation, for it rules out the possibility that the previous result was simply a confabulation in response to unusual task demands.) After a dozen such 15-minute sessions of “mirror therapy” (distributed over three weeks), he found, to his amazement, that for the first time in nine years, his hand became telescoped permanently into his stump with his phantom fingers dangling from his shoulder!

The concept of “learned paralysis” may have important implications for rehabilitation from stroke. One ordinarily thinks of the hemiplegia that arises from stroke as being the result of permanent irreversible neural damage. There is undoubtedly some truth to this view, but is it

conceivable that at least some of the paralysis that these patients experience is “learned”? For example, even if there is some degree of recovery—as a result of “remapping”—the patient may not use his arm because his brain has learned during the acute stages that his limb is paralyzed. If so, mirror therapy may eventually prove useful in accelerating recovery of function in at least a subset of stroke victims.

F. DISCUSSION

The occurrence of referred sensations in the phantom limb is not new. It has been noticed by many researchers (Mitchell, 1871; Cronholm, 1951) that stimulating points on the stump often elicits sensations from missing fingers, and the great American psychologist William James (1887) once wrote, “A breeze on the stump is felt as a breeze on the phantom.” Unfortunately, because the results of Pons and his collaborators were not available at that time, such findings were often attributed to direct reinnervation of the stump by the severed axons. Even when points remote from the stump were found to be effective in producing referred sensations, the phenomenon was often attributed to “diffuse” connections in the nervous system (Melzack, 1992). I would argue, instead, that the effects I have observed are a direct consequence of the remapping observed by Pons *et al.* (1991), which in turn is constrained by proximity of maps in the brain. The reason that there are two clusters of points—for example, one on the face and one near the upper arm—is that the hand area in the Penfield homunculus is flanked on one side by the face and on the other side by the upper arm, shoulder, and axilla (Fig. 4). If the sensory input from the face and from around the stump were to “invade” the cortical territory of the hand, one would expect precisely this sort of clustering (Ramachandran *et al.*, 1992b) of points. In the rest of this essay I shall refer to this view as the “remapping hypothesis” of referred sensations (Ramachandran *et al.*, 1992b). I would emphasize, especially, the topographic arrangement of points on the face, the referral of complex sensations such as “trickling” from the face to the phantom, and the extreme rapidity with which the referred sensations emerge. (Our study was carried out after 4 weeks rather than 12 years.)

This rapidity might suggest that the reorganization is based on the unmasking of silent synapses (e.g., through disinhibition) rather than on anatomical sprouting. Whatever the interpretation, however, these findings represent the first clear demonstration that highly organized, modality-specific “rewiring” of the adult mammalian brain can occur in

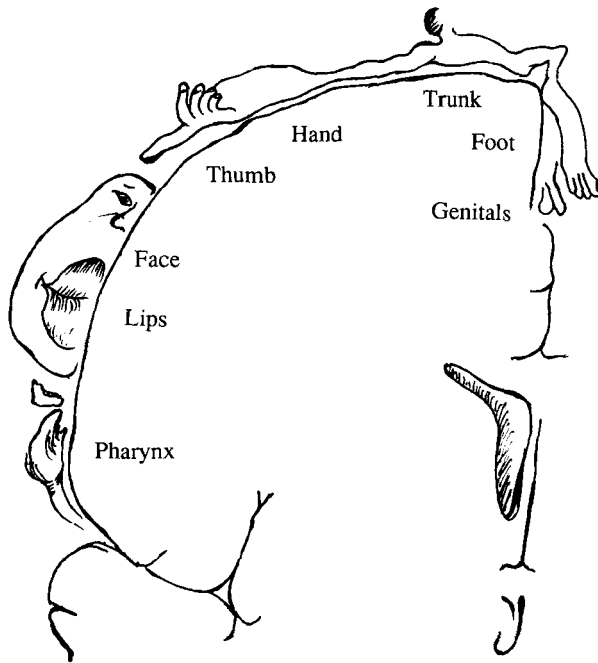


FIG. 4. The Penfield homunculus. Notice that the sensory hand area is flanked below by the face and above by the upper arm and shoulder—the two regions where we usually find reference fields in arm amputees. Also, note that the area representing the genitals is just below the foot representation, a fact that might explain the frequent occurrence of certain foot fetishes even among normal individuals.

as little as 4 weeks and that this rewiring can be functionally effective. It remains to be seen, of course, whether this latent capacity can be exploited for therapeutic purposes.

1. *Sprouting or Unmasking?*

What is the actual neural mechanism underlying the expanded hand representation in the Silver Spring monkeys and in our patients? We need to consider two theories:

1. When a patch of sensory neurons (e.g., in the “hand area”) is deprived of sensory input, it might begin to secrete some neurotrophic factors that provide sprouting of new axon terminals from neurons supplying anatomically adjacent cortical areas. These same trophic factors might subsequently “attract” these terminals to the denervated zone.

2. Perhaps even in normal individuals any given point on the skin projects simultaneously to several locations, e.g., the sensory input from the face projects simultaneously to both the face and the hand neurons in the cortex (or thalamus). The unwanted input to the hand area, however, might be subject to tonic presynaptic inhibition (e.g., via an inhibitory interneuron) by the "correct" axons that arrive there from the hand. If the arm is amputated, on the other hand, this occult input is unmasked through disinhibition and this would lead to mislocalized sensations.

There is at present no strong reason for favoring one hypothesis over the other. The elegant work of Wall (1971) provided the first clear evidence that rapid "unmasking" of synapses can indeed occur in the somatosensory system (see also Calford and Tweedale, 1990; Rasmusson and Turnbull, 1983; Kelahan and Doetsch, 1981). Such short-term changes, however, usually result in an enlargement of receptive field size, and there is no strong evidence that topography can be altered. Furthermore, the unmasking idea is rendered somewhat unlikely by the fact that neither the arborization of a thalamo-cortical axon terminals nor cortico-cortical connections have been found spanning more than a few millimeters of the cortex (Calford, 1991; Pons, 1992).

Even if the sprouting hypothesis turns out to be correct, however, one would still have to account for the emergence of topography and modality specificity, i.e., the sprouting would have to be organized and the new axon terminals would have to find their appropriate targets. The guidance of such new terminals to appropriate targets would, if it occurred at all, have to depend on poorly understood mechanisms such as chemoaffinity.

III. Some Potential Problems

A. INTERSUBJECT VARIABILITY

How general are the findings we have reported here? Of the nine patients we have seen so far, the map on the face was seen in four. The second cluster of points near the line of amputation, on the other hand, was seen in eight patients.

Why do some patients not have a cluster of points on the face? There are at least five possibilities that are not mutually exclusive. First, the brain maps might vary slightly from patient to patient, and this, in turn,

might influence the degree of remapping. Second, some patients may eventually “learn” to ignore the referred sensations from the face by using visual feedback. Third, even without visual feedback the plasticity exhibited by the afferent pathways may be propagated further along the pathways to perception so that the input gets correctly interpreted as originating from the face alone [i.e., the peripheral organ might “specify” the central connections, as suggested by Weiss (1939)]. Fourth, the aberrant connections that are formed may get deleted by the same genetic mechanism that causes their elimination in the embryo. And fifth, if the patient uses the stump constantly, the skin corresponding to it may “regain” the territory that was initially lost to the face.² Obviously, more extensive testing of a large number of patients is needed before we can distinguish between these possibilities.

Halligan and Marshall (1993) have recently studied a patient whose arm had been amputated at the shoulder level. They were able to replicate and significantly extend our basic observation—the occurrence of a map on the ipsilateral lower face. Curiously, they found that although the map was nearly complete and was in many ways quite similar to the one we had observed, it lacked an index finger and thumb. Careful questioning of the patient revealed that she had completely lost sensations in her thumb and index finger for over a year preceding the amputation (she had suffered from carpal tunnel syndrome). It was as though this sensory loss had been carried over into her phantom!

B. MULTIPLE MAPS

The “remapping” hypothesis predicts that after arm amputation two clusters of points should be seen—one near the face and one near the line of amputation. As we have seen, this is generally true (e.g., one hardly ever sees points on the ipsilateral leg, contralateral chest, contralateral leg, and abdomen). However, in two of our patients there was more

² If this argument is correct, then the face map should be seen much more consistently in patients with brachial plexus avulsion than in amputees, and we have some evidence that this is indeed true. The reason for this difference is that in amputees the nerves that supply the arm can reinnervate the stump, after an initial period of retrograde degeneration, whereas in the avulsion cases, the roots degenerate permanently and there is no possibility of reinnervation. It was our impression, also, that patients who referred sensations from the face to the phantom either had a preexisting long-standing pathology in that arm (e.g., a brachial avulsion, infiltration by carcinoma) or were seen soon (e.g., 4 weeks) after amputation. Patients who were seen several years after a “clean” amputation (with no preexisting pathology) tended not to have a map on the face. Clearly, additional experiments are needed to see if this distinction holds up.

than one map near the line of amputation and we have no simple explanation for this. For example, WK (whose arm had been disarticulated at the shoulder) had at least two maps proximal to the amputation line—one below the axilla and a second less distinct one near the ipsilateral nipple. Likewise, patient FA, described above, had two distinct maps—one that was 3 cm below the elbow crease and the second map (identical to the first) 6 cm above the crease, with nothing in-between! There are, of course, multiple representations of the body surface in both the thalamus and in S1, and one wonders whether the multiple maps that we observed reflect remapping occurring separately in each of these brain areas.

In some patients, we have also occasionally seen similar dual representation of the hand on the face (e.g., sensations in the phantom index finger could be evoked by stimuli on either of two separate points on the face—one on the lower jaw and one on the upper cheek). One possible explanation for such dual reference fields comes from the recent physiological work of Lund *et al.* (1994), who found that the “hand area” of the cortex is actually flanked by two separate maps of the face within S1. It is conceivable, therefore, that after upper limb deafferentation, the sensory input from the thumb destined for the hand area of S1 now projects additionally to separate parts of the two flanking face maps, e.g., to the “cheek” region of one map and the “jaw” region of the other. The outcome of such remapping would be two separate thumb reference fields on the patient’s face.

C. CONTRALATERAL POINTS

Points on the normal skin surface that elicited referred sensations in the phantom were usually clustered around regions proximal to the line of amputation and around the ipsilateral face as predicted by the remapping hypothesis, although the details of the maps varied considerably from patient to patient (e.g., in two patients the maps were on the lower jaw but in a third patient they were mainly clustered on and around the temporomandibular joint and pinna).

These observations are, on the whole, consistent with the remapping hypothesis, but mention must be made of the occasional presence of small maps in the contralateral limb at locations that were approximately mirror-symmetrical with the line of amputation. In one patient (RW), for example, in addition to the two clusters that are usually found (i.e., on the face and near the amputation line), a small (2-cm diameter) well-circumscribed region of skin near the contralateral elbow was found that elicited referred sensations in the phantom hand. Moving a pencil in

this region produced a vivid sensation of movement in an equivalent direction in the phantom and scratching it was effective in eliminating itch sensations in the phantom! (Other regions on the normal arm were completely ineffective in producing these effects.) In the second patient (FA) we found a clearly organized map on the normal arm that was an exact mirror image of the map that was above the line of amputation. The curious thing about this map, however, was that the sensation was referred to the finger of the normal limb. Again, no other areas of skin surface on the normal arm were effective in evoking referred sensations.

These contralateral effects are difficult to account for in terms of the remapping hypothesis as it currently stands, but they might be explicable in terms of the transcallosal effects reported by Calford and Tweedale (1990). These authors found that digital nerve block in flying foxes and primates produces the expected immediate expansion of receptive fields in the contralateral hemisphere, but there were also striking changes in the mirror-symmetric locations of the ipsilateral hemisphere corresponding to the normal hand—changes that took less than 20 minutes to emerge. It is not known how long the changes last, but it is conceivable that remapping effects of the kind observed by Merzenich *et al.* (1983, 1984) and Pons *et al.* (1991) may also induce analogous long-term changes in the other hemisphere. If such changes were to occur, they would explain the maps we saw in the normal arms of our two patients.

D. ROLE OF THE CORPUS CALLOSUM

In this section, I will describe a novel perceptual effect that we observed in a 23-year-old patient who fell off a train and lost his left arm 19 days prior to our testing him. The effect provides additional evidence for strengthening or reactivation of preexisting connections. After verifying first that our patient was neurologically intact, we blindfolded him and applied sensory stimuli to various parts of his body and asked him to report where he felt the sensations. Like some of our other patients, he referred sensations from his lower ipsilateral face to his phantom fingers, but there were no clearly defined reference fields—possibly because not enough time had elapsed after the amputation. An especially intriguing effect was observed, however, when we touched the normal hand. For example, when we touched an individual finger of the right hand, he felt he was also being touched simultaneously on the corresponding finger of the phantom hand. Remarkably, even the subjective quality of the sensation was carried over vividly into the phantom, e.g., when his right index finger was scraped with a knee hammer, he experi-

enced his phantom index finger also being "scraped." When we held his fifth finger or index finger and passively dipped either finger into a glass of hot or ice-cold water, he felt the corresponding finger on the phantom also "dipping into water," although, curiously, the warmth or cold was felt only in the right hand—it was not referred to the phantom. This rules out confabulation, for if the patient were confabulating, why should he refer only the "dipping" sensation and not the temperature? We conclude, therefore, that we are dealing with a genuine sensory phenomenon. No referral of sensations occurred from any other part of the body and the effects remained stable across two successive testing sessions separated by 24 hours. (The patient could not be followed up subsequently because he left the country.)

The phenomenon bears a suggestive resemblance to allesthesia, which is seen in neurological patients with right parietal lobe disease. In this condition, the patient detects a stimulus applied to his left arm but attributes it to his normal (right) arm. It has been suggested that the illusion arises because the patient neglects the left side of his body and therefore tends to assimilate sensations from the contralesional (left) side into his "surviving cognitive scheme" or body image associated with the left hemisphere (Brain and Walton, 1969).

What causes allesthesia? We suggest that in parietal lobe syndrome as well as in phantom limbs, the phenomenon arises from activation of preexisting transcallosal connections. There is physiological evidence suggesting that even in normal individuals, touching a finger on (say) the right hand activates not only the "finger" area of the left hemisphere but also the mirror-symmetric location in the right hemisphere (Calford and Tweedale, 1990). One reason normal individuals do not experience allesthesia might be that they ordinarily have countermanding signals from the left hand informing the brain of the absence of touch signals. If the left hand is amputated, however, there is no contradictory information and the brain simply accepts the null hypothesis that both hands are being touched. The fact that temperature is not referred argues against a cognitive explanation and suggests that there are no transcallosal fibers concerned with this modality.

A second possibility is that the callosal input from the right hand to the right hemisphere is ordinarily subliminal, i.e., too weak to activate the neurons in area 3b that get a much stronger input from the left hand. After amputation of the left hand, however, this weak input from the right hand gradually "takes over" these neurons and becomes strong enough to activate them, thereby leading to referred sensations. [This would be analogous to shifts in ocular dominance or eye preference observed in young monkeys after monocular deprivation (Wiesel and

Hubel, 1965).] This interpretation is different from unmasking, of course, but it does imply a strengthening of preexisting connections rather than a sprouting of new axons across the corpus callosum!

1. *Nonspecific Effects*

In addition to the clearly defined reference fields described so far, we also observed that sensations could occasionally be evoked in the phantom using very nonspecific stimuli applied to the normal skin surface. These nonspecific effects could be distinguished from reference fields by using the following criteria: (1) The sensations evoked in the phantom tended to be diffuse and not localized to specific points on the phantom limb. They were also usually less intense. (2) Unlike sensations produced by stimulation of reference fields, the nonspecific referred sensations were usually highly variable in their occurrence and distribution, e.g., stimulating the same point would evoke sensations in very different locations in the phantom on different trials. (3) The distribution of the points that evoked these highly variable sensations was usually quite random, i.e., there was no clearly discernible pattern such as the clustering of reference fields on the lower jaw and around the amputation line. Occasionally, even points on the contralateral torso, neck, shoulder, etc. were effective in producing such nonspecific sensations.

The mechanisms underlying these nonspecific effects are obscure but they may have more in common with diffuse "arousal" than with the remapping that we have considered so far (e.g., the barrage of spontaneous activity from neuromas may be normally gated, but nonspecific arousal might make the cortex more sensitive to such impulses). Some support for the arousal interpretation comes from the recent physiological work of Dykes *et al.* (1994), who found two classes of novel responses in the deafferented hand area of the cortex (S1) of cats. First, there were cells with clearly defined receptive fields of the kind observed by Pons *et al.* (1991) in monkeys. A second class of responses, however, could be obtained by touching almost any part of the animal, i.e., there were no clearly defined receptive fields. Dykes *et al.* suggest that these responses are not "truly sensory in character" and that they may be mediated by brainstem arousal mechanisms. This second category of responses usually appeared very soon after deafferentation and may account for some of the nonspecific referred sensations that we observed in our patients. (The prediction would be that such sensations should also emerge earlier after amputation than specific, organized reference fields.)

2. *Stability of Maps over Time*

In patients WK, VQ, and FA the overall features of the map were remarkably stable with repeated testing across weekly intervals (For 4

weeks). In patient FA, however, we made a very intriguing observation suggesting that the fine details of the map may be dynamically maintained.

Recall that FA lost his arm 10 years ago when the beam of a sailboat landed on his arm and crushed it. His arm was subsequently amputated 8 cm below the elbow crease. On careful questioning, we discovered that FA's phantom hand usually occupied a position halfway between pronation and supination with the fingers slightly flexed as though he was holding an imaginary vertical staff. We were also struck by the fact that the topography of points on the upper arm seemed to approximately mimic the position of the phantom fingers—a tendency that we had also previously noticed in other patients. Out of curiosity, we asked him to pronate his phantom hand all the way and we remapped the points on the upper arm while his hand was still pronated. To our astonishment, we found that the entire map had shifted systematically leftward by about 1 cm as if to follow the pronation partially (Ramachandran, 1993). Because the arm below the elbow was clamped, this shift in the map could not be attributed to accidental upper arm movements. Also, when he returned the phantom to its resting position, the map also shifted rightward and returned to its original location. A particularly convincing way of demonstrating this effect was to place a constant stimulus such as a small drop of water on (say) the fifth finger region of the map. When he was then asked to pronate his phantom, he reported that he very distinctly felt the drop of water moving from the fifth finger to the ring finger.

To explore this effect further, we also tried placing a drop of hot water on the index finger reference field and, as expected, this produced a referral of both the touch (tingling) and the warmth sensations to the phantom index finger when the hand was in its normal position. When he pronated his hand, the touch sensation was felt to move to his thumb, as one would expect from the previous experiment, but to our surprise (and to the patient's!) the warmth sensation did *not* move; it remained confined to the index finger. This experiment was repeated several times and on two consecutive days, with identical results. The results have at least four interesting implications. First, the referral of warmth does not depend on the simultaneous coactivation of touch receptors. Even though the touch was referred to a new digit, temperature was confined to the original digit. Second, the shift in the reference fields that occurs during pronation of the phantom cannot be confabulatory, for if it were, why should only touch be referred to a new digit and not temperature? Third, there must be separate maps of reference fields for different modalities such as temperature and touch, and these maps must be in approximate registration (although, of course, they may vary in resolu-

tion). Fourth, the dynamic shifts in the sensory map, based, presumably, on the motor commands sent to the hand, occur only for the touch map, but not for the temperature map. One reason for this might be that touch is represented cortically in a topographically organized manner, whereas the response to temperature might be primarily thalamic rather than cortical.

Changes in reference fields were also observed in patient FA when he simply clenched and unclenched his phantom fingers. When he made a fist, for example, touch sensations from the original thumb reference field moved to the palm, but when he unclenched his fingers, the sensation was referred once again to the thumb. (This was true for other digits as well.)

Finally, modulation of referred sensations was also observed, albeit on a smaller scale, on the face map in patient FA. No actual shift was observed, but when he clenched and unclenched his fist, the thumb reference field disappeared and reappeared. (Again, this effect was repeatable across testing sessions.)

These observations are quite remarkable: although their functional significance is not obvious, they suggest that the fine details of the map may be dynamically maintained and that either the map in S1 or in subsequent read-out can be profoundly modified by reafference signals from motor commands sent to the hand. We are currently exploring such effects using magnetoencephalography (MEG) and, obviously, it would also be interesting to study cortical maps in awake, behaving monkeys while they were actually moving their hands.

E. LONG-TERM CHANGES IN MAPS OF REFERRED SENSATIONS: NEURAL DARWINISM?

With regard to the stability of maps, two additional observations were made in patient FA that deserve comment. First, we noticed that during the first three testing sessions there were no reference fields on the face at all. Yet when we repeatedly prodded his face for obtaining MEG recordings (Yang *et al.*, 1993, 1994), he reported, with some surprise, that he had started to notice some sensations in his phantom hand! These referred sensations were more noticeable the following day when he was shaving, than during the actual MEG recording session. It was as though the repeated mechanical stimulation had somehow revived dormant connections that had always been there. (Alternately, the sensations might have emerged spontaneously even without the prodding. This needs to be explored in additional patients.) Interestingly such sensations were

now evoked for both sides of the face, although less reliably from the contralateral side than from the ipsilateral side.

The short-term and long-term changes may be based on very different mechanisms, but whatever the explanation might be, they are sure to have important implications for our understanding of brain function. They provide the first behavioral evidence for the views of Merzenich *et al.* (1983) and Edelman (1989), that even in the adult brain, neural connections are being modified all the time.

F. A THEORY OF PHANTOM LIMBS

The remapping hypothesis not only explains referred sensations but may also provide a novel explanation for the very existence of phantom limbs. The old clinical explanation of phantom limbs is that the illusion arises from irritation of severed axon terminals in the stump by the presence of scar tissue and neuromas. Unfortunately, as emphasized out by Melzack (1992), this explanation is quite inadequate, because injecting local anesthetic into the stump or even removing the neuromas surgically often fails to abolish the phantom or to eliminate phantom limb pain. Indeed, as early as 1836, it was pointed out by Valentin that central factors must be invoked to account for the phenomenology of phantom limbs (Valentin, 1836).

We suggest that the phantom limb experience arises because tactile and proprioceptive inputs from the face and tissues proximal to the stump take over the hand region not only in area 3b as shown by Pons *et al.* (1991), but possibly also in "proprioceptive" maps. Consequently, spontaneous discharges from these tissues would be misinterpreted as arising from the missing limb and might therefore be felt as a phantom (Ramachandran *et al.*, 1992a,b). This hypothesis is different from, although not incompatible with, the view that phantom limbs arise from the persistence of a "neurosignature" in a diffuse neural matrix (Melzack, 1992). We would argue, however, that the effect arises from mechanisms of a more specific nature, such as remapping.

The remapping hypothesis does not, however, explain all aspects of the phantom limb experience. Consider, for example, the observation that phantom limbs are occasionally seen in patients who have congenital absence of limbs. We have recently studied one such patient (DB), a 20-year-old lady whose arms had both been missing from birth. All she had on each side was the upper end of the humerus—there were no hand bones and no radius or ulna. Yet she claimed to experience very vivid phantom limbs that often gesticulated during conversation! It is unlikely

that these experiences are due to confabulation or wishful thinking, for two reasons. First, she claimed that her arms were “shorter” than they should be by about a foot. (She knew this because her hand did not fit into the prosthesis like a hand in a glove “the way it was supposed to”.) Second, her phantom arms did not feel like they were swinging normally as she walked—they felt rigid! These observations suggest that her phantom limbs did not originate simply from her desire to be “normal.” It is also difficult to see how the remapping hypothesis, in its simple form, can explain the vivid gesticulation and other spontaneous movements that both DB and other patients experience. We would suggest, instead, that the sensations arise from refference signals derived from the motor commands sent to the phantom. What is remarkable, however, is that the neural circuitry generating these gesticulatory movements was hard-wired and had actually survived intact for 20 years in the absence of any visual or kinesthetic reinforcement.

Based on these observations, we suggest that the phantom limb experience probably depends on integrating information from three different sources: first, from the spontaneous activity of tissues in the face and tissues proximal to the amputation, i.e., the “remapped” zones; second, from refference signals that accompany motor commands sent to the muscles of the phantom limb; and third, to some extent even from the neuromas—as taught by the old textbooks. Information from these three sources is probably combined in the parietal cortex to create a vivid dynamic image of the limb—an image that persists even when the limb is removed. Indeed, there is at least one case on record of a patient actually *losing* his phantom (Head and Holmes, 1911) as a result of a stroke affecting his right parietal cortex, just as one might expect from our hypothesis.

IV. Conclusions on the Remapping Hypothesis

After 100 years of meticulous neurology, it would have been quite remarkable if no one had noticed referred sensation in phantom limbs, and indeed such effects have been reported several times (see Mitchell, 1871; James, 1887; Cronholm, 1951). In a monograph published in 1951, Cronholm noted clearly that points remote from the stump could sometimes evoke sensations in the phantom. What these researchers did not recognize, however, was that there was something special about the face or that there was a specific pattern of distribution of points yielding referred sensations—one set of points on the ipsilateral lower face and

one on the upper arm. Nor did they suggest that this clustering of points can be explained in terms of Penfield's map, an explanation that we refer to as the "remapping hypothesis." Notice, especially, that this hypothesis dissociates proximity in the brain from proximity on the body surface (because of certain peculiarities of the brain map such as proximity of face to hand and genitals to feet).

With regard to our empirical results, what is new may be summarized as follows:

1. There were well-defined reference fields on the body surface remote from the stump. Also, the reference fields were not distributed randomly; there were two clusters, one on the lower face region and one near the amputation.

2. Reorganization is relatively rapid; the study was carried out after 4 weeks rather than 12 years. This rapidity might suggest that the reorganization is based on the unmasking of silent synapses (e.g., through disinhibition) rather than on anatomical sprouting. This claim receives further support from the recent studies of Aglioti *et al.* (1994a), who reports emergence of referred sensation in 5 days after radical mastectomy.

3. The possible existence of topography³ on the face, e.g., in one patient, when we moved the cotton swab from the temporomandibular joint along the mandible toward the symphysis menti, he experienced an equivalent movement of referred sensation on his phantom arm.

4. The referral of complex sensations from regions remote from the line of amputation, e.g., warm water trickling down the face was felt as a sensation of warm water trickling down the phantom hand.

5. The induction of permanent telescoping using a virtual reality box.

6. The "rivalry" of warmth and cold sensations following simultaneous stimulation of two maps.

7. The shifting map proximal to the stump when the phantom hand is pronated, supinated, or flexed. (This occurred whether the phantom was telescoped or not.)

8. The long-term changes in maps observed in patient FA. It remains to be seen whether this was a consequence of the repeated facial stimulation that occurred during the EMG recording sessions.

In the older neurological literature, there are several examples of vague suggestions to the effect that phantom limbs have a central rather

³ The presence of topography on the stump (but not on the face) was also noted by Cronholm (1951), but he attributed this (incorrectly, in my view) to the fact that the phantom was telescoped onto the stump.

than peripheral origin. Yet surprisingly, no attempt was made to formulate a precise, testable hypothesis. (One reason for this may be simply that the work on primates was not available at that time.) What we call the remapping hypothesis, on the other hand, makes the following simple predictions: (1) Sensations should be referred from the face to this hand after upper limb amputation and a second cluster of points should be seen just proximal to the stump. As we have seen, this is true at least in some patients. (2) There should be topography or both on the face and proximal to the stump. Again, this appears to be true in many patients, although for some reason, the topography is usually clearer near the stump. (3) Sensations should be referred from the genitals to feet after lower limb amputation, and we have seen this in at least two patients (Ramachandran, 1993). There have also been previous reports of defecation and micturition causing referred sensations in the phantom (see Sunderland, 1959, for a review), but no attempt was made to relate this to the Penfield map. Our observations on the link between genitals and feet have now been confirmed and significantly extended by Aglioti *et al.* (1994b). (4) The hypothesis might also explain the widespread prevalence of foot fetishes and the relative scarcity of (say) hand or nose fetishes (e.g., there might be a slight "error" in mapping during fetal development). We prefer this neurological explanation to Freud's psychodynamic interpretation, which postulates a visual resemblance between the foot and the penis. (5) Sensations should be referred from foot to penis after penis amputation (e.g., for carcinoma) and from hand to face after trigeminal ganglion section. The latter prediction would be especially easy to test. (6) To determine whether the remapping is cortical or subcortical, it would be interesting to look for referred sensations in neurological patients who have sustained damage to the sensory thalamo-cortical fibers in the internal capsule. A subset of these patients have complete sensory loss on the contralateral side of the body, but a sparing of the face. Would these patients also refer sensations from the face to hand? If so, one could conclude that the remapping was cortical. (7) Finally, because the "magnification factor" has increased for the face, one might also expect to see an actual improvement in sensory discrimination thresholds on the face.⁴

⁴ Haber (1958) and Teuber *et al.* (1949) noticed an improvement in two-point thresholds on the stump, and suggested, with considerable foresight, that this improvement may occur as a result of "recruitment" of adjacent brain areas. The improvement, however, could also be attributed other nonspecific "practice" effects, because patients tend to use their stumps a great deal. To rule out this possibility it would be especially interesting to look for improvement in touch thresholds, point localization, and two-point discrimination on the face following amputation.

Thus, although the remapping hypothesis may eventually turn out to be wrong in detail, it leads to several testable predictions and may point to some new directions of research. As noted by Charles Darwin: "False facts are highly injurious to the progress of science, for they often endure long; but false views, if supported by some evidence, do little harm, for everyone takes a salutary pleasure in proving their falseness."

V. Neurology, Freud, and the Inner Ear

The social scientists have a long way to go to catch up, but they may be up to the most important scientific business of all, if and when they finally get to the right questions. Our behavior toward each other is the strangest, most unpredictable, and almost entirely unaccountable of all the phenomena with which we are obliged to live. (Lewis Thomas)

A. NEGLECT, DENIAL OF ILLNESS (ANOSAGNOSIA), AND SOMATOPARAPHRENIC DELUSIONS

We will now consider what might be loosely regarded as the converse of the phantom limb experience, namely, the syndrome of somatoparaphrenic delusions associated with right hemisphere stroke. Whereas in the case of phantom limbs the patient vividly experiences an arm that he knows to be missing, in somatoparaphrenia he asserts that his own paralyzed limb does not belong to him. "This is your arm, doctor," he might say, pointing to his own left arm!

Right hemisphere stroke usually results in a paralysis of the left side of the body (hemiplegia) and somatoparaphrenia is seen only in a small subset of these patients, usually those who have associated right parietal lobe damage. Curiously, left parietal disease does *not* result in denial of the right arm. A much more common manifestation of right parietal disease, however, is not an actual delusion that the arm belongs to someone else, but simply a denial of paralysis. [For insightful discussions on these topics, see Edelman (1991), Prigatano and Schacter (1991), and Weinstein and Kahn (1950).] Such a denial of paralysis was first recognized by Babinski, who coined the term "anosagnosia" to describe the condition. I became interested in this condition for two reasons. First, I believe that this disease provides an experimental approach to certain hitherto mysterious aspects of human nature such as the denial or repression of unpleasant memories or unpleasant facts about oneself. These phenom-

ena are normally relegated to the realm of Freudian psychology, but I believe they can be brought into the realm of experimental neurology. Second, as we shall soon see, denial and neglect have considerable relevance to the problem of rehabilitation of neural function.

Consider, first, the relevance to rehabilitation from stroke. We usually think of neglect and denial as manifestations of injury or lesions in the right parietal cortex. But if this is true then how does one explain the fact that parietal lobe syndrome is one of those few neurological disorders that show a very high rate of spontaneous remissions within a few weeks? One argument might be that, as in the case of phantom limbs, some other part of the cortex (e.g., the other hemisphere) might take over some of these functions, but a remarkable discovery made by Bisiach and co-workers (1991) suggests an alternative, more exciting possibility. Bisiach *et al.* studied a patient who had sustained a right hemisphere stroke and was suffering from the delusion that his left arm belonged to someone else. They found, to their surprise, that when they simply poured cold water in the patient's left ear canal, there was a complete disappearance of symptoms! Unfortunately, a few hours after the caloric stimulation had worn off, the symptoms returned and the patient once again started denying ownership of his arm. Even so, Bisiach's discovery has two important far-reaching implications. First, it may pave the way toward a therapy for neglect and denial and other manifestations of parietal lobe disease. (Would repeated caloric stimulation accelerate recovery? Can a more pleasant substitute be found for vestibular stimulation?) Second, the result implies that many of the so-called syndromes of clinical neurology, which we ordinarily think of as irreversible hardware damage, may in fact represent deranged software (although, admittedly, the distinction between hardware and software is somewhat poorly defined for a biological system such as the brain). If this conclusion is correct, then the relevance of these findings for all kinds of therapy and rehabilitation becomes obvious.

B. HOW DEEP IS THE "DENIAL" OF ANOSAGNOSIA?

Since Babinski's original description of this disease, there have been remarkably few studies on the question of *why* patients with right parietal disease deny their paralysis. With most neurological syndromes, the patient loses some specific mental capacity (e.g., syntax in Broca's aphasia and face recognition in prosopagnosia), but the integrity of the person remains largely intact. What is especially fascinating about anosognosia, on the other hand, is that the very notion of a "person" or self" is called into question (Galín, 1992).

To emphasize this further I will briefly describe a patient I saw recently after she was admitted to our hospital following a right hemisphere stroke. This patient (BM) was a 76-year-old lady who had complete paralysis of the left side of her body as a result of her stroke, but she persistently denied the paralysis even on repeated questioning. The following conversation⁵ was typical:

- VSR: *Mrs. M, when were you admitted to the hospital?*
 BM: *I was admitted on April 7th because my daughter felt there was something wrong with me.*
 VSR: *What day is it today and what time?*
 BM: *It is sometime late in the afternoon on Tuesday. [This was an accurate response.]*
 VSR: *Mrs. M., can you use your arms?*
 BM: *Yes.*
 VSR: *Can you use both hands?*
 BM: *Yes, of course.*
 VSR: *Can you use your right hand?*
 BM: *Yes.*
 VSR: *Can you use your left hand?*
 BM: *Yes*
 VSR: *Are both hands equally strong?*
 BM: *Yes, they are equally strong.*
 VSR: *Mrs. M, point to my student with your right hand. [Patient points.]*
 VSR: *Mrs. M, point to my student with your left hand. [Patient remains silent.]*
 VSR: *Mrs. M, why are you not pointing?*
 BM: *Because I didn't want to.*

The same sequence of questions was repeated the next day with identical answers, except that toward the end of the session the patient looked at me and asked:

- BM: *Doctor, whose hand is this [pointing to her own left hand]?*
 VSR: *Whose hand do you think it is?*
 BM: *Well, it certainly isn't yours!*
 VSR: *Then whose is it?*
 BM: *It isn't mine either.*
 VSR: *Whose hand do you think it is?*
 BM: *It is my son's hand, Doctor.*

⁵ Patient BM was of Hispanic origin and a Spanish-speaking interpreter had to be used both during the testing and for subsequent analysis of the videotaped sessions.

Notice how the patient begins with rather simple denial of paralysis, but when her paralysis becomes increasingly obvious to her with repeated questioning, she is pushed into a corner and the only way she can rationalize the failure of her arm to perform is to progress into the even more full-blown delusion that the arm belongs to her son! (This is analogous to what Freud might call projection.) What we are seeing here on a compressed time scale, is an amplified version of the same kinds of delusions and rationalizations that all of us engage in some time or the other (Ramachandran, 1994).

A second, less psychodynamic interpretation of the same findings is also possible. One could argue, for instance, that she denies her arm because her body image is damaged in such a way that she “loses” half the image. (Even if this were true, however, one would still have to account for the subsequent bizarre rationalization that her arm belongs to her son.) What is puzzling about these cases, however, is the extreme lengths to which the patients will take the process, even though their intelligence, clarity of thought, and mentation are relatively unaffected in every other domain *except* for matters concerning the left hand! The patient I just described, for example, refused a box of candy, saying, “I am diabetic, Doctor—I can’t eat candy. You should know that!” Thus, her anosagnosia included her limb but did not extend to her diabetes.

How does one go about studying anosagnosia in the laboratory? And to what extent does it affect prospects for effective rehabilitation? For if the patient is in a state of denial, what possible motivation could she have for engaging in rehabilitative therapy? These are difficult questions but we have recently begun a series of experiments that might help provide some answers. One interesting question, for example, concerns the depth of the patient’s denial. Is the denial of paralysis mainly at the semantic/verbal level or does it run deeper? If it is mainly semantic, then is it possible that the patient is subconsciously aware that he/she is in fact paralyzed? We devised four novel tests to try and answer these questions (Ramachandran, 1994).

1. Test 1

We used our virtual reality box to trick the patient into thinking that her left hand was moving in response to her command. This was achieved by simply placing an 18” by 18” mirror vertically on the table so that it was perpendicular to the table as well as to the patient’s chest (e.g., imagine opening a book so that the front cover stands upright; the front cover would then be the mirror). The mirror was concealed in a large wooden box and the patient simply inserted her two hands through circular windows cut into the face of the box, so that they lay on either

side of the mirror, palm down on the floor of the box. The top of the box was opaque on the left so that the patient could not directly see her left hand, but the box was clear on the right so that she could see both her right hand and the reflected image of her right hand in the mirror. Because the hands were placed parallel and close to each other on either side of the mirror, the mirror image of the right hand was exactly superimposed on the “felt” position of the left hand. The result was that the patient was tricked into thinking that she was seeing her own two hands. When instructed to move both hands up and down, she obviously saw her right hand obey her command but she also saw her lifeless left hand suddenly coming to life, moving as though it was following her command! Our question was, would the patient register surprise, especially if she knew at some subconscious level that her left arm was paralyzed? Or would she fail to find it surprising because she believed that both her hands were functioning normally? Surprise was measured using two techniques—a galvanic skin response (GSR) and changes in expression, such as raised eyebrows. (The entire session was videotaped for subsequent analysis.)

2. *Test 2*

In the second test, the patient was given a choice between performing two tasks: a simple bimanual task (e.g., tying a shoelace into a bow knot or cutting a circular disk out of a sheet of cardboard) versus a unimanual task (e.g., threading a bolt). She was told, also, that she would be given a large box of candy if she successfully completed the first task and a small box of candy for the second task. (She was, of course, not explicitly told that the tasks were unimanual and bimanual.) Our question was, would the patient show a spontaneous preference for the unimanual task, despite the smaller reward, even though she denied the paralysis of her left arm? In other words, can a dissociation between “procedural” and “declarative” knowledge be observed in anosognosia?

3. *Test 3*

The patient was asked to reach out and grab a tray with glasses on it. Would her right hand automatically reach for the handle on the right side or would it go straight for the middle of the tray?

4. *Test 4*

In the fourth task, the subject simply viewed herself in a large full-length mirror while being instructed to perform simple movements with her left or right hand alternately. Ordinarily, the patient will refuse to acknowledge her failure of performance by saying, “I didn’t want to

move it that time," or, "I'm not usually very ambidextrous" (as one patient once told us). Would seeing her failure in a mirror or videotape, however, enable her to adopt a more "abstract" attitude, i.e., enable her to distance herself from her own body image and thereby allow her to acknowledge her paralysis?

We have, so far, tried some of these experiments on two patients (BM and LR)⁶ and the preliminary results were most intriguing (Ramachandran, 1994). The patients (LR) clearly did *not* show "subconscious knowledge" of paralysis on any of the tasks, suggesting that the denial must be very deep indeed; it cannot simply be the inverse of hysterical paralysis (or of malingering). On the unimanual versus bimanual task, for example, patient LR consistently chose the bimanual task (e.g., tying a shoelace). This was true even when the test was prefaced with the question "Mrs. LR, how many hands would you need to tie a bow knot or a shoelace? And how many hands do you need to thread the bolt?" She answered both questions correctly, but went straight for the shoelace task and kept trying to tie a bow knot with one hand! Patient BM, however, chose the nut/bolt unimanual task on one occasion. When asked why she preferred this task even though a larger reward had been offered for tying a shoelace, she replied, "The other task might be too difficult for me" "Why is it more difficult?" I asked, to which she replied, "I don't know why. It just seems like it might be." Computerized tomography scans revealed right parietal lesions in both patients, but one wonders whether there were subtle differences to the extent of lesion that might account for the differences in depth of anosagnosia.

C. REPRESSED MEMORIES

An even more remarkable piece of evidence for subconscious knowledge comes from another experiment we did on patient BM. The reader

⁶ Patient LR was a 79-year-old, right-handed lady who was admitted for left-sided weakness; she was seen by us for 4 weeks after her admission. A CT scan revealed an infarct in the region supplied by the right middle cerebral artery. Neurological examination revealed a residual left hemiplegia affecting the left upper extremity quite severely, a left-sided neglect (line crossing and line bisection tests), and a mild dysarthria. She was quite alert and attentive and her memory, both short and long term, was intact. She had no obvious dementia or aphasia.

Patient BM was a 76-year-old Hispanic woman who was admitted for sudden-onset left-sided hemiplegia and was seen by us 2 weeks later. She had clear neurological signs of left hemiplegia and neglect (e.g., extreme right gaze preference), was accurate but somewhat sluggish in responding to questions or commands, and was able to communicate clearly with the examiner. An MRI revealed a right parieto-occipital lesion along with slight involvement of the right thalamus and head of the caudate nucleus.

will recall that this patient denied her paralysis even on repeated questioning, and she finally asserted that the arm belonged to her son. Having been inspired by the experiments of Bisiach *et al.* (1991), I decided to try a caloric test on this patient. After she had repeated several times that she was not paralyzed and her arm belonged to her son, I administered 10 ml of ice-cold water into her left ear and waited until nystagmus appeared. My main interest was not only in replicating Bisiach's observation, but also in specifically asking her questions about her memory, an issue that had never been studied directly before on a systematic basis.

VSR: *Do you feel okay?*

BM: *My ear is very cold but other than that I am fine.*

VSR: *Can you use your hands?*

BM: *I can use my right arm but not my left arm. I want to move it but it doesn't move.*

VSR: *[holding the arm in front of the patient] Whose arm is this?*

BM: *It is my hand, of course.*

VSR: *Can you use it?*

BM: *No, it is paralyzed.*

VSR: *Mrs. M, how long has your arm been paralyzed? Did it start now or earlier?*

BM: *It has been paralyzed continuously for several days now.*

After the caloric effect had worn off completely, I waited for $\frac{1}{2}$ hour and asked:

VSR: *Mrs. M, can you use your arm?*

BM: *No, my left arm doesn't work.*

Finally, the same set of questions was repeated to the patient 8 hours later by one of our colleagues.

EX: *Mrs. M, can you walk?*

BM: *Yes.*

EX: *Can you use both your arms?*

BM: *Yes.*

EX: *Can you use your left arm?*

BM: *Yes.*

EX: *This morning, two doctors did something to you. Do you remember?*

BM: *Yes. They put water in my ear; it was very cold.*

EX: *Do you remember they asked some questions about your arms, and you gave them an answer? Do you remember what you said?*

BM: *No, what did I say?*

EX: *What do you think you said. Try and remember.*

BM: *I said my arms were okay.*

These observations have several remarkable implications. First, they confirm the observation of Bisiach *et al.* about remission from anosognosia and delusion following caloric stimulation. Second, they also allow us to draw certain important new inferences about denial and memory repression. Specifically, her admission that she had been paralyzed for *several days* suggests that even though she had been continuously denying her paralysis, the information about the paralysis was being continuously laid down⁷ in her brain, i.e., the denial did not prevent memory consolidation (Ramachandran, 1994). Again, we may conclude that at some deeper level she does indeed have knowledge about the paralysis. (Also, the insight gained during the caloric stimulus seemed to last at least for $\frac{1}{2}$ hour after the stimulation had ceased!) Finally, when tested 8 hours later, she not only reverted to denial, but also “repressed” the admission of paralysis that she had made during her stimulation. The remarkable theoretical implication of these observations is that memories can indeed be selectively repressed although, it is unclear whether this repression occurs during consolidation or during the subsequent split second recollection.

Seeing patient BM also convinced me, for the first time, of the reality of the repression phenomena that form the cornerstone of classical psychoanalytical theory. Selective memory repression was also seen in patient LR, whom we tested three times at weekly intervals, and she denied each time that her left arm was paralyzed. Furthermore, she always chose the bimanual task, instead of the unimanual one, and continued to do so with no sign of frustration despite her repeated failures. Finally, after her last try on the third week, I asked her to point to my student with her left hand. She tried to do so and then admitted for the first time in 3 weeks that she could not use her left hand. (And 10 minutes later when asked to point again, she spontaneously admitted her paralysis.) A week later, when my student questioned LR again in my absence, she admitted to remembering me as, “The nice Indian doctor who asked

⁷ Part of this, of course, could have been a confabulatory “gap-filling,” i.e., she sees now that she is paralyzed and she knew that she was admitted for stroke several weeks ago and therefore makes the obvious inference (whether consciously or not) that she must have been paralyzed for that entire period. Her use of the phrases “several days” and “continuously paralyzed” suggests, however, that this was not the case. Mrs. BM’s apparent lack of surprise when she admitted paralysis (during caloric stimulation) is also noteworthy, because it implies that she was also repressing the denial that she had been engaging in just a few minutes earlier!

me to tie shoelaces,” and then went on to add, “He asked me to tie the laces on a small shoe. I was able to do so *using both my hands*.” But when my student queried her specifically about pointing, she admitted at once, “Yes, he asked me to point with my left hand. I was unable to do so.”

Thus, she had selectively repressed her failure to tie laces (perhaps because her failure was never explicitly pointed out to her), but had not repressed her inability to point! Doing so might have required her to stretch the truth to a degree that she was simply not capable of, given, especially, that she was probably already in partial remission 8 weeks after the stroke.

It would be premature to draw any firm conclusions from these very preliminary observations, but clearly the findings are sufficiently provocative that they warrant a systematic large-scale effort to understand these phenomena. Perhaps the reason Mrs. BM repressed her previous admission of paralysis was that there was no way she could honestly deny her present paralysis and still acknowledge the insight she gained during caloric-stimulation—without falling apart. We may conclude, therefore, that remembering something, even from the recent past, entails a complete reordering of one’s conscious experience to accommodate current demands.

D. A DARWINIAN THEORY OF DEFENSE MECHANISMS

In my view, the various defense mechanisms, rationalization, repression, etc., arise because the brain tries to arrive at the most probable and globally consistent interpretation of the evidence derived from multiple sources. By way of analogy, consider a military general trying to make a major strategic decision, e.g., whether to invade a particular city at a particular time. He would ordinarily collect evidence from a large number of scouts, weigh the evidence, and arrive at a firm decision. Now, if one scout were to provide information that was somewhat contradictory to the rest (e.g., he might indicate that the enemy had 10,000 soldiers rather than 5000), the general would be unlikely to change his strategy. He may ask the scout to be silent and to discard the evidence, or, for fear of mutiny, he may actually ask the scout to “march in tune” with the others, and if necessary, even lie to the other officers in the army. (The former would be analogous to denial and the latter to rationalization or confabulation.) A perpetually indecisive general, on the other hand, would be quite incapable of winning a war.

Oddly enough, something like this also seems to occur when the visual system tries to combine multiple sources of information about relative

depth (e.g., perspective, stereo, occlusion, motion parallax, shading) to yield a vivid coherent impression of depth (Ramachandran, 1988). The rules that the visual system uses to combine multiple cues are poorly understood, but may involve two processes: (1) taking a weighted average (the weighing is important because, statistically speaking, some cues might be inherently more reliable than others, and this “wisdom” might be wired into the visual system during ontogeny and phylogeny) and (2) looking for consistency (e.g., if six cues yield random values and two yield identical values, then the visual system may “choose” the latter instead of averaging the values). This is because accidental inconsistencies are common in nature (due to noise) but accidental consistencies are rare.

Interestingly, once a global interpretation of depth has been reached, the system simply ignores or suppresses the conflicting information. The purpose of this might be to avoid going into a perpetually indecisive state, i.e., the rule might be that any firm decision is better than none at all. Now the remarkable thing is that the visual system may, on occasion, even hallucinate some of the required evidence in order to preserve consistency, i.e., there appears to be a tendency to actually impose coherence. It was as though the general had decided not only to ignore the advice of one of his scouts, but also insisted that the scout actually fabricate the evidence required in order to fit the “big picture.”

A striking example of this can be observed with the illusory square (Fig. 5), which is created by simply aligning four disks from which pie-shaped sectors have been removed (Kanizsa, 1979). What people usually see when viewing this display is an opaque white square partially occlud-

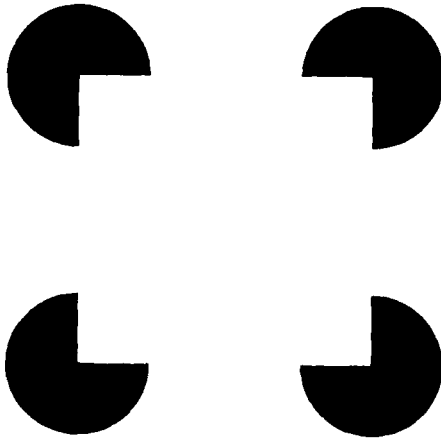


FIG. 5. An illusory square (after Kanizsa, 1979).

ing four back disks, rather than four disks that have been deceitfully aligned by the experimenter.

Now it should be obvious that when seeing a square in this display, the visual system has to discard the contrary evidence from the homogeneous paper surface about the absence of an edge (for such an edge would usually be associated with a change of luminance). But instead of discarding the perception of a square, the visual system seems to opt for actually hallucinating an illusory edge, which even has an illusory brightness change associated with it (an example of "Vector Completion"). So here is a clear example of the visual system "jury rigging" the evidence in order to impose a coherent interpretation.

Now in my view, the same sort of thing happens in the cognitive/emotional domain. We have a tremendous need to impose a sense of order and coherence in our lives—we need a "story." Of course, when most of the evidence favors one particular interpretation of the available data, we have no difficulty in simply accepting that interpretation. For example, even when a patient's arm is paralyzed, her motor cortex sends messages to her limb and there is a comparator in her brain that ordinarily monitors these feed-forward signals and informs her "self" that, "I am moving my limb." Therefore her conscious self tentatively accepts this story.⁸ When the evidence is conflicting, however (e.g., if the patient's vision tells her that her arm is not obeying her commands), then instead of wasting time in conflict or oscillating between alternate decisions, her cognitive system simply picks one story and adheres to it. Again, in order to do this, it either ignores the conflicting evidence (denial) or actually fabricates new evidence (rationalization). The evolutionary purpose of such defense mechanisms might be that when limited time is available, *any* decision, however uncertain, is better than an indecisive vacillation—so long as it is the best interpretation of the current data. But to deal with a conflicting source of information that keeps nagging away at the central processor, the latter may actually insert the relevant evidence so that it can go about the rest of its business. What you end up with, therefore, is a rationalization or a denial.

And that brings me to my last point. The purpose of a rationalization, we have seen, is to eliminate discrepancy by creating fictitious evidence (or false beliefs). But clearly there must be limits to this process, for

⁸ This is similar to the function of the "interpreter" in the left hemisphere that Gazzaniga (1992) has postulated, to account for the rationalization that one observes in split brain patients. What I have tried to do in this chapter, however, is to consider the biological role of such a mechanism. I also postulate a second mechanism in the right hemisphere that is required for monitoring the magnitude of discrepancy and for generating a paradigm shift.

otherwise defense mechanisms would soon become maladaptive and threaten the individual's survival. It may be a good thing to repress an extremely traumatic memory in order to avoid being paralyzed with fear. This *would* be adaptive. It would be maladaptive, however, to repress every memory that was unpleasant because that would defeat the very purpose of having aversive memories in the first place. I suggest, therefore, that there is a special-purpose mechanism in the right parietal lobe whose sole purpose is to periodically "challenge" the left hemisphere's story, detect discrepancies, and make sure that the discrepancy is not too large. (You can think of this, if you like, as a center for intellectual honesty or integrity.) Hence, I might be willing to engage in some minor rationalization, i.e., make some small false assumptions to get on with my life, but when the false beliefs become too far removed from reality, my discrepancy detector kicks in and makes me reevaluate the situation (e.g., if I was a general about to wage war, it would be quite appropriate, usually, to ignore contrary evidence from a single scout, but if he told me that the enemy was waving a white flag or had nuclear arms, I would be foolish to adhere to my original decision). I suggest, further, that the mechanism for imposing consistency (i.e., the small rationalizations and repressions) is located in the left hemisphere, whereas the global anomaly detector that monitors the level of discrepancy and reacts with the appropriate emotion is in the right hemisphere⁹. This would explain why right hemisphere patients are willing to engage in much more elaborate and fanciful rationalizations than normal individuals or individuals with left hemisphere damage. Conversely, left hemisphere stroke patients may not be able to manage even a minimal amount of denial, rationalization, or confabulatory gap-filling and consequently become profoundly depressed. This would be a new interpretation of the common clinical observation that depression is most often associated with left hemisphere stroke. (The more common interpretation is that the right hemisphere is the emotional hemisphere, so that the affect is inappropriate when it is damaged.)

This theory may also help explain another major psychological puzzle—the biological significance of laughter and humor. When the "discrepancy detector" in the right hemisphere questions and corrects the

⁹ Another way to characterize the difference between the hemispheres would be to note that their manner of dealing with anomalies is different. When small local errors are detected, the left hemisphere tries to impose consistency by ignoring or suppressing the evidence, whereas when there are large, global anomalies, an interaction with the right hemisphere forces a change in world view—a paradigm shift. Until we have a clearer idea of the underlying neural mechanisms, such a metaphorical explanation will have to serve as a temporary substitute.

rationalization or tentative hypothesis of the left hemisphere, the net result can be a strong emotional response. The defenses crumble and force a revision of world view; composure must be given up in order to correct behavior (e.g., by fight or flight). But if the correction is only of relatively trivial consequence (e.g., the burglar turns out to be a neighbor's cat), then the tension that is built up is discharged by a convulsive laughter. (This hypothesis would predict that right hemisphere patients should lack a sense of humor but should, paradoxically, sound funny to the physician.)

E. FREUD AND THE INNER EAR: A NEUROLOGICAL APPROACH TO PSYCHOTHERAPY

Finally, let us consider the extent to which these defense mechanisms (such as denial and projection) are domain specific, i.e., do they apply only to the patient's paralyzed limb or to all of the patient's maladies? (The reader will recall that patient BM denied her paralysis but was concerned about her diabetes!) We propose to test this experimentally using our mirror test (Test 2 described earlier), except that the patient will be asked to view the left side of the mirror so that she sees the reflection of her paralyzed left hand superimposed on the felt position of her (hidden) right hand. If she is now asked to move her right hand, she will, of course, feel her movement, but the hand will appear not to move. With this sensory conflict, the question, is, will she now rationalize the disability by saying, "Oh, I did see the hand move" or "I wasn't *really* trying to move my hand," or will she express surprise just as you and I would in that situation? In other words, does her tendency to confabulate affect only her left arm or does it also affect her other arm?

Is it conceivable that there are mechanisms in the right hemisphere that are concerned with detecting not only a discrepancy involving the corporeal self but with detecting other types of discrepancies as well? According to classical psychoanalytical theory, many forms of neuroses arise because the patient's defense mechanisms have become so extreme that they have actually become maladaptive instead of being simply used as a coping strategy. Providing insight into these maladaptive defense mechanisms is, therefore, one of the goals of Freudian psychoanalysis, and this is usually achieved by several months or years of intensive psychotherapy. But if we are correct in arguing that that insight can be enhanced using caloric left ear stimulation in patients with parietal lesions, is it conceivable that such insight could also be induced in neurotic patients who are neurologically intact, in order to eliminate the maladapt-

tive defenses that are said to produce the psychoneuroses? (Notice that the efficacy of this would not necessarily depend on whether the denial is encapsulated; all we have to assume is that caloric stimulation can also eliminate other types of anosagnosia and amnesia by activation of the right hemisphere—a proposition that can be tested experimentally.) After all, in patient BM, caloric stimulation not only eliminated denial but also allowed the repressed memory of the paralysis to be overcome. What I am suggesting, then, is that left ear caloric stimulation¹⁰ may eventually be used actually to replace psychoanalysis as a device for reviving repressed memories and for producing insight. If so, a few dozen sessions of cold water stimulation of the left ear might prove just as effective as several months of costly and intensive psychotherapy. Obviously, these are highly speculative and tentative ideas, but at least they are testable and they may eventually suggest new ways to rehabilitate and reeducate the malfunctioning brain.

VI. Conclusions: Is There Reason for Optimism?

The main conclusion to emerge from our experiments [together with the important recent work of Aglioti *et al.* (1994a,b) and Halligan and Marshall (1993)] is that we must give up the notion that the human brain is composed of a fixed set of anatomical connections specified largely by the genome. Our results suggest, instead, that even the adult brain is a dynamic biological system in which new connections are being created all the time, partly in response to novel sensory inputs and partly as a result of self-organizing principles. Everyone recognizes, of course, that *some* changes must be possible even in the adult brain, for otherwise, it would not be possible to account for phenomena such as learning and memory. What our results imply, however, is that even the basic circuitry or hardware in the brain—such as the sensory maps—can be altered with surprising rapidity. No one would have suspected, for example, that the famous Penfield homunculus, which every medical student learns about, can be reorganized over a distance of 2 cm or more in less than 4 weeks. And remarkably, the remapping, at least in some cases,

¹⁰ Why does caloric stimulation produce these apparently miraculous effects? One possibility is that vestibular stimulation generates “orientating” through cortical activation, as shown in animals (Fredrickson, 1964). A second more intriguing possibility is that the eye movements generated by the caloric stimulation directly derepress memories in the right hemisphere in a manner analogous to the derepression of memories that occurs during dreaming in rapid eye movement (REM) sleep.

is precise and orderly, for if it was not, one could not account for the emergence of a topographically organized, modality-specific reference field on the face or for the referral of complex sensations such as "trickle" from the face to the phantom. It is unclear, at present, how one could harness this latent ability of the brain to accelerate recovery from brain injury, but if our view of the brain is correct, then there is at least reason for optimism.

The second set of experiments, on patients with denial and neglect, also has obvious relevance to stroke rehabilitation. But more importantly, perhaps, these experiments may eventually turn out to have broader implications for understanding the biological origin of defense mechanisms. The results of our studies may, in fact, suggest an experimental approach to these enigmatic aspects of human nature and eventually may lead to new types of treatment for the many intractable psychological problems to which our species is notoriously prone.

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